COMPARATIVE STUDY BETWEEN INTENSE PULSED LIGHT "IPL" AND PULSED DYE LASER IN THE TREATMENT OF STRIAE DISTENSAE

Thesis
Submitted for the Fulfillment of (Ph.D) Degree in Medical Applications of Laser
By

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National Institute of Laser Enhanced Sciences
Cairo University
2013
بسم الله الرحمن الرحيم

قالوا سبحانك لا علٌم لنا إلاّ ما علمتنا أنتَ أنتَ العليم الحكيم

صدق الله العظيم
سورة البقرة الآية (32)
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Abstract

Title: Comparative study between Intense Pulsed Light and Pulsed Dye Laser in the treatment of Striae Distensae.

Pulsed dye laser (PDL) and Intense Pulsed Light (IPL) have been used to treat Striae Distensae (SD). Thirty patients with age ranging from 14 - 42 years were included in this study. Twenty patients were treated on one side of their bodies with PDL and on the other side with IPL while seven patients were treated on both sides by IPL and three patients were treated on both sides by PDL for five sessions with four weeks interval between sessions. Skin biopsies were stained with H & E, Masson Trichrome, Orcein, Alcian blue and anti-collagen I α1. After both PDL and IPL treatments striae width was decreased and the texture was improved in a highly significant manners where P value was 0.001. Collagen expression was increased in a highly significant manner and P values were <0.001 and 0.004 after PDL and IPL treatments respectively. However, PDL induced expression of collagen I in a highly significant manner compared to the treatment with IPL where P values were <0.001 & 0.193 respectively. Striae rubra gave a superior response with either PDL or IPL compared to striae alba which was evaluated clinically by the width, color and texture, although the histological changes could not verify this consequence. Both PDL and IPL can enhance the clinical picture of striae through collagen stimulation.

Key words: Striae rubra, Striae alba, Striae width, Collagen, Pulsed dye laser, Intense pulsed light.
# List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHA</td>
<td>Alpha Hydroxy Acid</td>
</tr>
<tr>
<td>ALA</td>
<td>Amino Levulonic Acid</td>
</tr>
<tr>
<td>BMZ</td>
<td>Basement Membrane Zone</td>
</tr>
<tr>
<td>Cm²</td>
<td>Square Centimeter</td>
</tr>
<tr>
<td>Cm³</td>
<td>Cubic Centimeter</td>
</tr>
<tr>
<td>CO²</td>
<td>Carbon Dioxide</td>
</tr>
<tr>
<td>COL</td>
<td>Collagen</td>
</tr>
<tr>
<td>CREST</td>
<td>Calcification, Raynaud's phenomenon, Oesophageal Stenosis, Scleroderma and Telangiectasia</td>
</tr>
<tr>
<td>DAB</td>
<td>Diaminobenzidine tetra hydrochloride</td>
</tr>
<tr>
<td>DDSA</td>
<td>Dodecenyl Succinic Anhydride</td>
</tr>
<tr>
<td>DEJ</td>
<td>Dermo Epidermal Junction</td>
</tr>
<tr>
<td>DLE</td>
<td>Discoid lupus erythematoses</td>
</tr>
<tr>
<td>Er: YAG</td>
<td>Erbium: Yttrium Aluminum Garnet</td>
</tr>
<tr>
<td>FDA</td>
<td>Food and Drug Administration</td>
</tr>
<tr>
<td>FPDL</td>
<td>Flash lamp- Pumped Pulsed Dye Laser</td>
</tr>
<tr>
<td>FPT</td>
<td>Fractional Photothermolysis</td>
</tr>
<tr>
<td>HIER</td>
<td>Heat Induced Epitope Retrieval</td>
</tr>
<tr>
<td>HX &amp; E</td>
<td>Haematoxylin &amp; Eosin</td>
</tr>
<tr>
<td>Hz</td>
<td>Hertz</td>
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<tr>
<td>ILVEN</td>
<td>Inflammatory Linear Verrucous Epidermal Nevus</td>
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<tr>
<td>IPL</td>
<td>Intense pulsed light</td>
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<tr>
<td>J</td>
<td>Joule</td>
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<tr>
<td>Kg</td>
<td>Kilogram</td>
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<tr>
<td>KTP</td>
<td>Potassium Titanyl Phosphate</td>
</tr>
<tr>
<td>LASER</td>
<td>Light Amplification by Stimulated Emission of Radiation</td>
</tr>
<tr>
<td>LIS</td>
<td>Large Scale Laser Isotope Separation</td>
</tr>
<tr>
<td>MMPs</td>
<td>Matrix Metalloprotienses</td>
</tr>
<tr>
<td>MNA</td>
<td>Methyl Nadic Anhydride</td>
</tr>
<tr>
<td>msec</td>
<td>milli second</td>
</tr>
<tr>
<td>MTZs</td>
<td>Micro Thermal Zones</td>
</tr>
<tr>
<td>Nd: YAG</td>
<td>Neodymium: Yttrium Aluminum Garnet</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>nm</td>
<td>nanometer</td>
</tr>
<tr>
<td>PBS</td>
<td>Phosphate Buffer Saline</td>
</tr>
<tr>
<td>PDL</td>
<td>Pulsed Dye Laser</td>
</tr>
<tr>
<td>PWS</td>
<td>Port Wine Stain</td>
</tr>
<tr>
<td>RF</td>
<td>Radiofrequency</td>
</tr>
<tr>
<td>SD</td>
<td>Striae Distensae</td>
</tr>
<tr>
<td>SG</td>
<td>Striae Gravidarum</td>
</tr>
<tr>
<td>SP</td>
<td>Selective Photothermolysis</td>
</tr>
<tr>
<td>SPSS</td>
<td>Statistical Package for the Social Science</td>
</tr>
<tr>
<td>STD</td>
<td>Sexually Transmitted Disease</td>
</tr>
<tr>
<td>TCA</td>
<td>TriChloroAcetic acid</td>
</tr>
<tr>
<td>TC</td>
<td>ThermaCool</td>
</tr>
<tr>
<td>TRT</td>
<td>Thermal Relaxation Time</td>
</tr>
<tr>
<td>ttt</td>
<td>Treatment</td>
</tr>
<tr>
<td>USA</td>
<td>United State of America</td>
</tr>
<tr>
<td>UV</td>
<td>Ultraviolet</td>
</tr>
<tr>
<td>VPL</td>
<td>Variable Pulsed Light</td>
</tr>
<tr>
<td>XeCl</td>
<td>Xenon Chloride</td>
</tr>
</tbody>
</table>
# List of Tables

<table>
<thead>
<tr>
<th></th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Genetically distinct collagen types and their tissue distribution</td>
<td>35</td>
</tr>
<tr>
<td>2</td>
<td>Frequency and percentage of striae rubra and alba</td>
<td>53</td>
</tr>
<tr>
<td>3</td>
<td>Frequency and percentage of patient's age and their mean</td>
<td>53</td>
</tr>
<tr>
<td>4</td>
<td>Frequency and percentage of different sites of striae distensae</td>
<td>54</td>
</tr>
<tr>
<td>5</td>
<td>Frequency and percentage of possible causes of striae distensae</td>
<td>54</td>
</tr>
<tr>
<td>6</td>
<td>Frequency and percentage of previous treatment of striae and types of this treatment</td>
<td>55</td>
</tr>
<tr>
<td>7</td>
<td>Frequency and percentage of treatment used in this study</td>
<td>55</td>
</tr>
<tr>
<td>8</td>
<td>Grading Scale and Scoring</td>
<td>57</td>
</tr>
<tr>
<td>9</td>
<td>Comparison between width of the striae before and after treatment with PDL and IPL</td>
<td>69</td>
</tr>
<tr>
<td>10</td>
<td>Comparison between the color of striae before and after PDL</td>
<td>70</td>
</tr>
<tr>
<td>11</td>
<td>Comparison between the color of striae before and after IPL</td>
<td>71</td>
</tr>
<tr>
<td>12</td>
<td>Comparison between color changes of striae after ttt by PDL and IPL</td>
<td>71</td>
</tr>
<tr>
<td>13</td>
<td>Comparison between improvement of skin textures after PDL and IPL</td>
<td>72</td>
</tr>
<tr>
<td>14</td>
<td>Comparison between side effects with PDL and IPL</td>
<td>76</td>
</tr>
<tr>
<td>15</td>
<td>Special Stains before and after PDL and IPL</td>
<td>84</td>
</tr>
<tr>
<td>16</td>
<td>Special stains after PDL and IPL in striae rubra and alba</td>
<td>87</td>
</tr>
<tr>
<td>17</td>
<td>Correlation between duration of striae and Collagen and Alcian stains after PDL and IPL</td>
<td>88</td>
</tr>
</tbody>
</table>
## List of figures

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Electron micrograph of a collagen fiber</td>
<td>30</td>
</tr>
<tr>
<td>2</td>
<td>Electron microscopic picture of collagen fiber</td>
<td>32</td>
</tr>
<tr>
<td>3</td>
<td>Frequency of possible causes of striae distensae</td>
<td>54</td>
</tr>
<tr>
<td>4</td>
<td>PDL (cynosure)</td>
<td>55</td>
</tr>
<tr>
<td>5</td>
<td>IPL (Plasmalite)</td>
<td>55</td>
</tr>
<tr>
<td>6</td>
<td>Comparison between width of the striae before and after treatment with PDL and IPL</td>
<td>70</td>
</tr>
<tr>
<td>7</td>
<td>Striae rubra on the abdomen before and after treatment</td>
<td>72</td>
</tr>
<tr>
<td>8</td>
<td>Striae rubra on the abdomen before and after treatment</td>
<td>72</td>
</tr>
<tr>
<td>9</td>
<td>Striae rubra before and after PDL (Left side) and IPL (Right side) of the abdomen (same patient)</td>
<td>73</td>
</tr>
<tr>
<td>10</td>
<td>Striae rubra on the right thigh before and after IPL treatment</td>
<td>73</td>
</tr>
<tr>
<td>11</td>
<td>Striae rubra on the right thigh before and after IPL treatment</td>
<td>74</td>
</tr>
<tr>
<td>12</td>
<td>Striae rubra on the breast before and after IPL treatment</td>
<td>74</td>
</tr>
<tr>
<td>13</td>
<td>Striae rubra on the right thigh and leg before and after IPL treatment</td>
<td>74</td>
</tr>
<tr>
<td>14</td>
<td>Striae rubra on the right thigh and leg before and after IPL treatment</td>
<td>75</td>
</tr>
<tr>
<td>15</td>
<td>Striae rubra on the abdomen before and after IPL treatment</td>
<td>75</td>
</tr>
<tr>
<td>16</td>
<td>Percentage of PDL side effects</td>
<td>76</td>
</tr>
<tr>
<td>17</td>
<td>Temporary erythema after PDL treatment</td>
<td>76</td>
</tr>
<tr>
<td>18</td>
<td>Temporary hyperpigmentation after PDL treatment</td>
<td>77</td>
</tr>
<tr>
<td>19</td>
<td>Percentage of IPL side effects</td>
<td>77</td>
</tr>
<tr>
<td>20</td>
<td>Striae Distensae Rubra (H &amp; E X 200)</td>
<td>78</td>
</tr>
<tr>
<td>20A</td>
<td>A photomicrograph of a section from striae distensae rubra before treatment</td>
<td>78</td>
</tr>
<tr>
<td>20B</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with PDL</td>
<td>78</td>
</tr>
<tr>
<td>20C</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with IPL</td>
<td>78</td>
</tr>
<tr>
<td>21</td>
<td>Striae Distensae Alba (H &amp; E X 200)</td>
<td>79</td>
</tr>
<tr>
<td>21A</td>
<td>A photomicrograph of a section from striae distensae alba before treatment</td>
<td>79</td>
</tr>
<tr>
<td>21B</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with PDL</td>
<td>79</td>
</tr>
<tr>
<td>21C</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with IPL</td>
<td>79</td>
</tr>
<tr>
<td>22</td>
<td>Striae Distensae Rubra (Masson Trichrome X 100)</td>
<td>79</td>
</tr>
<tr>
<td>22A</td>
<td>A photomicrograph of a section from striae distensae rubra before treatment</td>
<td>79</td>
</tr>
<tr>
<td></td>
<td>Description</td>
<td>Page</td>
</tr>
<tr>
<td>---</td>
<td>----------------------------------------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>22B</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with PDL</td>
<td>79</td>
</tr>
<tr>
<td>22C</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with IPL</td>
<td>79</td>
</tr>
<tr>
<td>23</td>
<td>Striae Distensae Alba (Masson Trichrome X 100)</td>
<td>80</td>
</tr>
<tr>
<td>23A</td>
<td>A photomicrograph of a section from striae distensae alba before treatment</td>
<td>80</td>
</tr>
<tr>
<td>23B</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with PDL</td>
<td>80</td>
</tr>
<tr>
<td>23C</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with IPL</td>
<td>80</td>
</tr>
<tr>
<td>24</td>
<td>Striae Distensae Rubra (collagen immunostaining with Novocastra hematoxylin counterstain X 200)</td>
<td>81</td>
</tr>
<tr>
<td>24A</td>
<td>A photomicrograph of a section from striae distensae rubra before treatment</td>
<td>81</td>
</tr>
<tr>
<td>24B</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with PDL</td>
<td>81</td>
</tr>
<tr>
<td>24C</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with IPL</td>
<td>81</td>
</tr>
<tr>
<td>25</td>
<td>Striae Distensae Alba (Collagen immunostaining with Novocastra hematoxylin counterstain X 400)</td>
<td>81</td>
</tr>
<tr>
<td>25A</td>
<td>A photomicrograph of a section from striae distensae alba before treatment</td>
<td>81</td>
</tr>
<tr>
<td>25B</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with PDL</td>
<td>81</td>
</tr>
<tr>
<td>25C</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with IPL</td>
<td>81</td>
</tr>
<tr>
<td>26</td>
<td>Striae Distensae Rubra (Orcein X100)</td>
<td>82</td>
</tr>
<tr>
<td>26A</td>
<td>A photomicrograph of a section from SD rubra before treatment</td>
<td>82</td>
</tr>
<tr>
<td>26B</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with PDL</td>
<td>82</td>
</tr>
<tr>
<td>26C</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with IPL</td>
<td>82</td>
</tr>
<tr>
<td>27</td>
<td>Striae Distensae Alba (Orcein X100)</td>
<td>82</td>
</tr>
<tr>
<td>27A</td>
<td>A photomicrograph of a section from striae distensae alba before treatment</td>
<td>82</td>
</tr>
<tr>
<td>27B</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with PDL</td>
<td>82</td>
</tr>
<tr>
<td>27C</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with IPL</td>
<td>82</td>
</tr>
<tr>
<td>28</td>
<td>Striae Distensae Rubra (Alcian blue X 200)</td>
<td>83</td>
</tr>
<tr>
<td>28A</td>
<td>A photomicrograph of a section from striae distensae rubra before treatment</td>
<td>83</td>
</tr>
<tr>
<td>28B</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with PDL</td>
<td>83</td>
</tr>
<tr>
<td>28C</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with IPL</td>
<td>83</td>
</tr>
<tr>
<td>29</td>
<td>Striae Distensae Alba (Alcian blue X200)</td>
<td>83</td>
</tr>
<tr>
<td>29A</td>
<td>A photomicrograph of a section from striae distensae Alba before treatment with PDL</td>
<td>83</td>
</tr>
<tr>
<td>29B</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with PDL</td>
<td>83</td>
</tr>
<tr>
<td>29C</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with IPL</td>
<td>83</td>
</tr>
<tr>
<td>30</td>
<td>Shows Collagen changes by Masson Trichrome Stain</td>
<td>85</td>
</tr>
<tr>
<td>31</td>
<td>Collagen type I changes after PDL and IPL</td>
<td>85</td>
</tr>
<tr>
<td>32</td>
<td>Shows ground substance changes by Alcian blue Stain</td>
<td>86</td>
</tr>
<tr>
<td>33</td>
<td>Shows elastic changes by Orcein Stain</td>
<td>86</td>
</tr>
<tr>
<td>34</td>
<td>A photomicrograph of a section from striae distensae rubra before (TEM X 4000)</td>
<td>89</td>
</tr>
<tr>
<td>35</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with PDL (TEM X 4000)</td>
<td>89</td>
</tr>
<tr>
<td>36</td>
<td>A photomicrograph of a section from striae distensae rubra after treatment with IPL (TEM X 4000)</td>
<td>89</td>
</tr>
<tr>
<td>37</td>
<td>A photomicrograph of a section from striae distensae alba before treatment (TEM X 4000)</td>
<td>90</td>
</tr>
<tr>
<td>38</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with PDL (TEM X 4000)</td>
<td>90</td>
</tr>
<tr>
<td>39</td>
<td>A photomicrograph of a section from striae distensae alba after treatment with IPL (TEM X 4000)</td>
<td>90</td>
</tr>
</tbody>
</table>
## List Of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction and Aim of work</td>
<td>1</td>
</tr>
<tr>
<td>Review of literature</td>
<td></td>
</tr>
<tr>
<td>Chapter (1)</td>
<td>3</td>
</tr>
<tr>
<td>• Striae Distensae</td>
<td></td>
</tr>
<tr>
<td>Chapter (2)</td>
<td>29</td>
</tr>
<tr>
<td>• Collagen</td>
<td></td>
</tr>
<tr>
<td>Chapter (3):</td>
<td>37</td>
</tr>
<tr>
<td>• Pulsed Dye Lasers</td>
<td></td>
</tr>
<tr>
<td>Chapter (4):</td>
<td>45</td>
</tr>
<tr>
<td>• Intense Pulsed Light</td>
<td></td>
</tr>
<tr>
<td>Patients and Methods</td>
<td>52</td>
</tr>
<tr>
<td>Result</td>
<td>69</td>
</tr>
<tr>
<td>Discussion</td>
<td>91</td>
</tr>
<tr>
<td>Conclusion</td>
<td>98</td>
</tr>
<tr>
<td>Summary</td>
<td>99</td>
</tr>
<tr>
<td>References</td>
<td>103</td>
</tr>
<tr>
<td>Arabic summary</td>
<td></td>
</tr>
</tbody>
</table>
Introduction

And

Aim of the Work
INTRODUCTION

Striae distensae (SD), a common skin condition, which does not cause any significant medical problems, however, striae can cause significant distress to those affected. Striae distensae represent linear dermal scars that are accompanied by epidermal atrophy. The literature reports a prevalence rate of SD of 40-90% depending on the population studied. Approximately 90% of pregnant women, 70% of adolescent females and 40% of adolescent males (many of whom participate in sports) have stretch marks (Arnold et al., 2000 & Sing and Kumar, 2009).

Striae distensae or stretch marks arise from progressive or rapid stretching of the dermis (McDaniel DH, 2002) and generally following rapid weight changes during adolescent growth spurts, corticosteroid use, or pregnancy and (less typically) due to Cushing syndrome (Zheng et al., 1985 & Nieman and Ilias, 2005 & Cho et al., 2006). The classic anatomical sites affected include the abdomen and breast for pregnancy-related striae, the outer thighs or lumbosacral regions for adolescent boys, and the buttocks, thighs, upper arms, and breast for adolescent girls (Chang et al., 2004 & Elsaie et al., 2009).

Striae progress through three different stages of maturation: the acute stage is characterized by red and slightly raised striae rubra, the subacute stage is characterized by purpuric stage, and the chronic stage is characterized by hypopigmented and atrophic striae alba (Kim et al., 2008).

Dermoscopy and histological studies have demonstrated that the varying colors of striae are influenced by melanocyte mechanobiology and that striae formation parallels the wound healing process of regular scar formation (Elsaie et al., 2009 & Hermanns and Pierard, 2006 & Atwal et al., 2006).

Initial inflammatory changes are followed by a flattening and thinning of the epidermis due to underlying changes in the numbers and organization of collagen, fibrillin, and elastin fibers (Elsaie et al., 2009). Histologic studies of mature striae reveal stretched collagen fibers aligned parallel to the skin surface, followed by subsequent loss of collagen and increased flattening of rete ridges (Elsaie et al., 2009). Contributing to the atrophied appearance of striae are the reduced amounts of
fibrillin surrounding the dermal-epidermal junction, reduced elastin in the papillary dermis and reorganization of elastin and fibrillin fibers in deep dermis (Watson et al., 1998).

Several treatments have been proposed, yet no consistent modality is superior to others. It has always been suggested that effective treatment of SD be instituted during the active stage, well before the scarring process is complete (Garcia HL, 2002).

The use of topical tretinoin has yielded variable results (Elson ML, 1990 & Pribanich et al., 1994). In the study of Kang et al. (1996), topical tretinoin improved the clinical appearance of stretch marks during the active stage. However, this treatment works poorly if at all on mature striae (striae alba) (Elson ML, 1990).

Microdermabrasion has been suggested for their treatment (Hopping S, 1999 & Rubin and Greenbaum, 2000 & Hernandez-Perez and Valencia-Ibiett, 2003).

Laser and light therapy have been advocated as treatment for stretch marks as well as different forms of scars (Normeth AJ, 1993 & Alster TS, 1994 & Alster and Williams, 1995 & McDaniel et al., 1996 & Narukar and Haas, 1997).

**Aim of the work**

1. To compare between Pulsed Dye Laser and Intense Pulsed Light in the treatment of striae distensae in relation to the degree of striae distensae clinically and histopathologically.

2. Histopathological study with different special staining for collagen, elastin and ground substance for assessment of healing response of striae distensae.
Review of Literature
Chapter (1)

Striae Distensae

Striae Distensae

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Striae Distensae
STRIAE DISTENSAE

Striae distensae (SD) "stretch marks" are a common disfiguring cutaneous condition, characterized by linear smooth bands of atrophic appearing skin caused by dermal damage to stretching (Osman et al., 2007).

A- AETIOLOGY

Striae are skin defects that look like bands or lines that are better known as stretch marks. They are a common disfiguring skin disorder of significant cosmetic concern (Uitto et al., 1989). The factors which govern the development of striae are poorly understood (Groover and Alster, 2000). Many authors have suggested that striae develop as a result of stress rupture of the connective tissue framework although others disagree (Elson, 1990). It has been suggested that they develop more easily in skin which has a critical proportion of rigid cross-linked collagen, as occur in early adult life (Hashimoto, 1998).

On the other hand, the absence of cross-linkage leads to "elasticity" and excessive stretching with eventual rupture of the skin if the stretch goes beyond elastic limit. Striae appear to occur therefore, only in skin in which the rigid cross linkage and "elastic" unlinked collagen, permit a limited intradermal rupture, i.e. striae. However several factors are involved in the development of striae (Pierard et al., 1999).

I- Genetic factors:

Skin affected by striae shows reduction in its components of elastic fibers network and reorganization with fibrillin, thus, the procollagen and fibronectin gene expression are markedly decreased in striae distensae. Such reorganization implies an important role for these two proteins in the pathogenesis of striae. It is possible that individuals susceptible or predisposed to the development of striae have an inherent qualitative and or quantitative deficiency in cutaneous fibrillin (Elson, 1990).

A genetic predisposition is presumed as striae distenase have been reported in monozygotic twins (Di-Lernia et al., 2001).
Fibrillin microfibrils are found in both elastic and non elastic tissue and appear to be ubiquitous although their exact function is still unknown (Fleischmajer et al., 1991).

There are apparent and coordinate decrease in α (I) and α (III) procollagen mRNA levels in striae distensae tissues compared with normal tissues (Lee et al., 1991). Accordingly there is decreased, expression of collagen, elastin, fibronectin mRNA and loss of extracellular matrix in the dermis. One possible explanation is that striae are fibroblast depleted hence the low level of extracellular matrix RNA and fibronectin mRNA (Lee et al., 1994).

Mutation in genes encoding fibrillins (fibrillin 1 on chromosome 15 and fibrillin 2 on chromosome 5) are causative factor in Marfan syndrome, an autosomal dominant connective tissue disorder (Tsipouras et al., 1991) that can be presented with a number of clinical features including striae distensae (Aoyama et al., 1995). In Marfan syndrome there are reduced fibrillin deposition caused by a dominant negative effect of abnormal fibrillin molecules. It is of prognostic and possibly diagnostic significance in these patients (Aoyama et al., 1995). On this basis, alteration in the expression of fibrillin molecules may therefore play a part in the development of striae distensae (Watson et al., 1998).

The importance of genetic factors in determining susceptibility of connective tissue to striae emphasized by the absence of striae in pregnancy in the Ehler-Danlos syndrome. In this syndrome the collagen fibers are smaller and more irregular and more spaced apart than in normal dermis. Conversely, elastic microfibrils and amorphous components are considerably increased. Collagen fibrils show poorly defined outlines and a loose texture, besides being markedly reduced in size (Watson et al., 1998).

II- Stretch factors:

Striae are largely parallel to the resting tension lines which show the orientation of minimal skin extensibility (Daly and Weston, 1986). They result from a failure of the skin to elongate under low intensity forces, with ultimate brittleness of the dermis. Once established, their resistance to stretch deformation is still solicited to sustain the permanent natural tension (Henry et al., 1997 & Elsaie et al., 2009).
Many observers have interpreted the abnormalities in elastin in striae to be due to stretching and rearrangement of old fibers or to splitting of thicker fibers *(Tsuji and Sawabe, 1988).*

Patients with chronic liver disease and ascites may also have striae *(Johnston and Graham-Brown, 2007).* Also obesity and rapid change in weight are associated with the development of striae *(Stevanovic, 1972 & Scheinfeld, 2004).*

### III- Glucocorticoids:

Both systemic and local glucocorticoid therapy can produce cutaneous atrophy by a dose related pharmacological effect. The effect is more severe with more potent steroids but both fluorinated and non fluorinated topical steroids can cause atrophy *(Wach et al., 1998).*

The mode of action of corticosteroids is through inhibition of collagen gene expression at transcriptional, translational and post-translational level *(Lee et al., 1994).* Topical steroids also inhibit the activity of enzymes involved in collagen biosynthesis, and they have been shown to depress collagen synthesis both in vivo and in fibroblast cultures *(Oikarinen et al., 1985).*

They can also suppress collagenase production and collagen breakdown, hence the rate of collagen turnover decreased *(Lee et al., 1994).* Even a weak steroid such as hydrocortisone can suppress the stimulatory effect of cyclic nucleotides on collagenase production *(Lehmann et al., 1983).*

Capillaroscopic studies have shown that steroid induced vasoconstriction involves the superficial capillary network, and prolonged superficial ischemia could also play a role in producing striae distensae *(Zheng et al., 1985).* A single large intravenous dose of corticosteroid produces a rapid and pronounced decrease in the resistance of skin to mechanical stretching also *(Ala-Kokko et al., 1987).* In addition, *Pinnell and Martin (1968)* reported that steroids known to inhibit the formation of glycosaminoglycans.

In 2010, *Cordeiro et al.* reported that a significant increase in the expression of hormonal receptors (estrogen, androgen and glucocorticoid receptors) principally estrogen receptors in recent striae distensae.
IV- Oral contraceptive pills:

Combined pills which contain a low dose of estrogen (ethinyl estradiol about 20 µg) and progesterone (desogestrel 150 µg) shown to be able to induce striae. It has been documented that free cortisol is increased with contraceptive pills intake, whereas, the ability of the liver to metabolize cortisol decreases, thus, the level of cortisol may be slightly elevated (Fotherby, 1992).

Robinson (1997) reported that in nulliparous patient that use contraceptive pills, the formation of striae distensae may be a cumulative effect of the pills or idiosyncratic response to these hormones. Striae improvement is achieved by immediate cessation of oral contraceptive pills (Har-Shai et al., 1999).

V- Pregnancy:

Continuous and progressive stretch cause alteration and damage in the extracellular matrix, which may remodel the elastic fiber network particularly fibrillin component in susceptible individuals and manifest itself clinically as striae distensae (Watson et al., 1998).

In pregnancy, the combination of endocrine changes (increase steroid levels in the blood) and mechanical stretching of the skin is responsible for most striae distensae (Henry et al., 1997).

In addition, some investigators have demonstrated a disturbance in glucose regulation during pregnancy and that fibroblasts no longer synthesize elastin (Chu et al., 1982).

Accordingly, hormonal changes disrupt the collagen bundles and thin out the skin. When an expectant mother puts on weight too quickly, the sudden stretching causes the skin to tear (White and Schnur, 1995).

A recent study done by Kelekci et al. (2011) reported that SD was more common in woman who had been born prematurely than in woman who had been born at term. In a total of 1336 women of reproductive age, group 1 consisted of 1231 women who had been born at term and group 2 included 105 women who had been born prematurely, ranging in age from 18-45 years. The overall prevalence of SD was 34.6% (462/1336). SD was significantly more common in woman who had been born
prematurely (49.5%) than in woman who had been born at term (31.8%). So the premature may be considered a risk factor for the development of SD.

VI- Systemic diseases:

In early 1900s striae were observed to be associated with many debilitating conditions such as tuberculosis, typhoid fever, rheumatic fever and other chronic infections. The aetiology is therefore linked with malnutrition, protein loss, general debility, and toxic states (Arem and Kischer, 1980).

They are found to be a feature of Cushing's disease, Marfan syndrome and diabetes mellitus (Watson et al., 1998). Striae are also reported in anorexia-nervosa in both bulimic and restrictive types. In nutritionally deprived states, such as starvation and in acute or chronic illness the risk of deficiency of ascorbic acid essential for normal collagen synthesis as a required co-factor for the hydroxylation of proline and lysine may be the aetiology of striae development (Strumia et al., 2001).

VII- Breast augmentation:

It was suggested also that patients who are candidates for breast augmentation surgery should be informed of the possible risk of developing striae distensae especially if they are taking or planning to take the contraceptive pills (White and Schnur, 1995).

VIII- Organ transplantation:

Skin diseases are frequent in organ transplant recipients; however, it was found that, striae distensae occurs as a result of steroid treatment after transplantation (Euvarard et al., 2001).

IX- Skin sutures:

Striae distensae can develop along sutures scars after skin surgery. Accordingly, the production of striae should be taken into consideration before surgery of the skin, especially in young women (Ono et al., 1991).

X- Edematous striae distensae:

Edematous striae distensae are uncommon but can develop from the combined effects of glucocorticoid and generalized edema, it can develop in systemic lupus
erythematosis and lupus nephritis. It occurs secondary to dermal edema with separation of collagen fibers and small fragmented elastic fibers (Lee et al., 1999).

XI- Idiopathic striae distensae:

In idiopathic striae distensae, the striae appear on the legs, back and abdomen (Elson, 1990).

B- CLINICAL FEATURES

Striae are very common and occur in most adult women, they readily develop at puberty or during pregnancy. Adolescent striae may first develop soon after the appearance of pubic hair (Watson et al., 1998).

Clinically, they are thin and pink, and may be pruritic initially, but with further development, they usually enlarge both in length and width and acquire a vivid reddish-purple appearance "striae rubra" (McDaniel et al., 1996). Finally as time goes by, striae assume a white sunken appearance "striae alba" and become wrinkled. The natural evolution of stretch marks is similar to that of scar formation or a healing wound (Requena and Sanchez, 1997 & Jiménez et al., 2003).

The commonest sites to be affected are the outer aspect of the thighs, the lumbosacral region in boys and the thighs, buttocks and breasts in girls, but there are considerable variations and other sites including the outer aspects of the upper arm are sometimes affected (Lee et al., 1994).

In pregnancy, the striae appear first and more conspicuous on the abdominal wall and later on the breasts but may involve most or all of the pubertal sites, and the majority of stretch marks appear in the later stages of pregnancy from about seven months onward, and be concentrated on the lower abdomen, and this tends to be concentrated around the areas that store most of the fat and do most stretching (Zheng et al., 1985 & White and Schnur, 1995). However, absence of stretch marks during one pregnancy does not necessarily mean that stretch marks will not occur in the other pregnancies (Viatour et al., 1995).

Those induced by topical steroid therapy occur particularly in the flexures but may appear in other sites if occlusive plastic films increased absorption. They may disappear or become less conspicuous when treatment is stopped (Chernosky and Knox, 1964).
The striae in Cushing's syndrome are characterized by their inordinate breadth, depth and intense color. In linear focal elastosis (elastotic striae), asymptomatic yellow linear bands arrange themselves horizontally over the lower back or legs. These lesions may resemble striae distensae, but they are palpable rather than depressed and yellow rather than purplish or white (Basak et al., 1989 & Arnold et al., 2000).

In idiopathic striae distensae, the striae appear on the legs, back and abdomen. They are commonly irregular linear, symmetrical and mainly vertical on the direction of skin tension when the skin is stretched. They are several centimeters long and 1-10 mm wide. After some years they fade and become inconspicuous (Elson, 1990).

It was reported by Sharquie et al. (2002) that striae distensae may be presented in children by patchy hair loss with great resemblance to alopecia areata.

Bordier et al. (1984) & Bowman and Hogan (2000) described patients with psoriases who were treated with systemic corticosteroids and etretinate (Tigason) who presented ulceration of pre-existing striae.

In 2008 Cordeiro and Moraes reported a case of ulcerated striae associated with corticosteroid therapy (60 mg/day prednisone) and cyclophosphamide pulse therapy (500 mg dose) in a case of systemic lupus erythematosus. Also, Pruitt (2005) & Daly and Schiff (2007) reported cases of striae distensae as a complication seen in primary brain tumor patients requiring chronic corticosteroids therapy.

C- HISTOPATHOLOGICAL FINDINGS

The histopathology of striae distensae varies depending on the age of the lesions (Booth et al., 1982). Under light microscopy, early lesions show superficial and deep perivascular infiltrate of lymphocytes and sometimes of eosinophils, as well as, widely dilated venules and edema in the upper part of the dermis. Fully developed lesions show scant infiltrate of lymphocytes around venules (Ebert, 1933 & Hernandez et al., 2002).

By Verhoeff's stain, the elastic fibers in early lesions appear in the periphery thick and tortuous, but the older lesions show normal appearance of elastic fibers. It is suggested that the elastic fibers in striae are newly synthesized and gradually increase and become thick with age (Requena and Sanchez, 1997).
After flattening and thinning of the striae alba surface, almost complete disappearance of dermal papilla was observed in thin paraffin sections. The papillary dermis was found to be almost completely replaced by straight bundles of collagen fibers running parallel to the skin surface (Hernandez et al., 2002). The hair follicles and other appendages also disappear in this stage (Elson, 1990).

Accordingly, Elson (1990) reported that formation of the striae in this way indicates that these are mini-scars in the skin.

Scanning electron microscopy shows extensive tangles of fine curled elastic fibers with a random arrangement. This histological appearance probably results from mast cell degrauulation with resultant destruction of both collagen and elastin fibers (McDaniel, 2002). This arrangement is different from normal skin which has thick elastic fibers with a regular distribution when viewed by transmission electron microscopy.

**D- PATHOGENESIS OF STRIAE DISTENSEAE**

The pathogenesis of striae is unknown but most probably related to changes in those structures that concerned with elasticity. Such structures are components of the extracellular matrix, including fibrillin, elastin and collagen (Watson et al., 1998).

It is possible that their early countenance is due to an inflammatory response associated with vasodilatation, which over time reduces as the lesion takes on a scar-like appearance (Zheng et al., 1985).

Ultrastructural analysis revealed marked alterations in the appearance of skin affected by striae compared with that of normal unaffected skin (Lee et al., 1994). Little difference was observed in the morphology of the dermoepidermal junction (DEJ) region of striae, suggesting that there is little or no role for the associated molecules (laminin, collagen IV, collagen VI) in the pathogenesis of the lesion (Watson et al., 1998). There is also a marked alteration of fibroblast metabolism in striae distensae (Lee et al., 1994).

The epidermal thinning probably results from a reduction of the mitotic activity in the germinal layer (Hagari et al., 1997). However, in striae compared with normal skin, the dermal matrix appeared looser and more follicular, containing
material of a "ground glass" appearance. The overall effect was of a reduction in the amount of collagen and elastic fibers (Watson et al., 1998).

However, Sheu et al. (1991) reported in their study that the specific changes seen in very early stages of striae distensae, are due to elastolysis accompanied by mast cell degranulation followed by an influx of activated macrophages that envelope fragmented elastic fibers. The relationships among elastic fibers, mast cells, and macrophages seen explained their critical roles in the process of development of striae distensae.

Many observers suggest that striae might be a form of dermal scarring, in which the dermal collagen ruptures perhaps under the influence of steroids. Ruptured collagen separates and the intervening gap becomes occupied with newly synthesized collagen, which then becomes aligned in response to local stress forces (Arnold et al., 2000).

As a result of hormonal influences, fibroblasts which are basic cells in the skin that manufacture elastin and collagen, undergo an imbalance resulting in quantitative and qualitative decrease in elastin and collagen fibers. Consequently the skin becomes thinner and stretch marks appear (Lee et al., 1994).

All the previous changes occur in individual with the genetic susceptibility to striae as a result of decreased procollagen and fibronectin gene expression (Arnold et al., 2000).

**E- MANAGEMENT**

I- Diagnosis:

The diagnosis of striae distensae is usually simple depending mainly on the clinical picture. The possibility of Cushing's syndrome and other endocrine disorders must be considered (Watson et al., 1998).

II- Prevention:

Many of the reasons that make stretch marks are beyond control, but there are some preventive measures to help reduction of the appearance of stretch marks. The only real prevention is to limit weight gain, get lots of exercise and to keep the body
hydrated by drinking plenty of water and using moisturizers on the affected areas (Henry et al., 1997).

Other experts advised massaging the skin with a massage brush to help increasing the circulation and maintain a diet high in vitamin C, E, as well as zinc and silica (Wierrani et al., 1992).

III- Prognosis:

Adolescents with striae can expect their striae to be less visible with time. Treatment with tretinoin, flashlamp pulsed dye laser and chemical peels significantly improves the clinical appearance of early, active stretch marks (Alaiti and Zein, 2003).

IV- Treatment

It is not possible to completely remove stretch marks because they are essentially small scars of torn skin. It is possible, however, for the ridges to smooth out, and the associated redness or whiteness to fade to the point that they become hardly visible. The complete evaluation of a patient who presents for treatment of striae distensae should include consideration of the Fitzpatrick skin type, duration of striae, and any contraindications to the treatment of choice (McDaniel, 2002).

Patients must have realistic expectations and understand that therapies produce gradual, incremental improvement. Persons with Fitzpatrick skin types I, II and III are suitable candidates for pulsed dye laser therapy (Nouri et al., 1999). Also, patients with a history of vitiligo, pigmentation disorders, dark tans or dark pigmentation may require an initial skin test for evaluation prior to treatment (Raulin et al., 1997).

Tanned patients with type III skin must be treated with more caution. Generally persons with skin types IV to VI may be treated with non ablative lasers with proper skin cooling, most topical agents, microdermabrasion, and ultrasonic (sonophoretic) therapy (McDaniel, 2002).


**Treatment Includes:**

**1-Topical agents:**

**A- Tretinoin:**

Retinoids are derivatives of vitamin A, which has been used in the treatment of acne. Its use was increased exponentially since the article by Weiss and Ellis in 1988, who reported benefits from tretinoin in the treatment of photoaging. There are many effects of this chemical upon the skin when applied topically (Elson, 1994).

Oikarinen et al. demonstrated in (1985) that cultured dermal fibroblasts contain a retinoic acid binding protein. In addition, tretinoin increases the deposition of collagen and increases the presence of fibroblast in the area of its application (Goldfarb et al., 1989).

It is metabolized rapidly and mostly excreted in bile when administered topically; a minute amount passes through the dermis but has not detected systemically (Kligman, 1987). However, in epithelial cells, tretinoin affects differentiation, neoplastic transformation, tumor promotion, collagen synthesis, wound healing, stimulation and modulation of immune response, inflammation, cell membrane and many other processes (Kang et al., 1996).

Topical tretinoin in a 0.05% and 1% concentrations has been shown to be effective for early stretch marks (striae rubra). However, it produces no significant improvement in mature stretch marks (striae alba) (Glassberg et al., 1988 & Elson, 1994). Other concentrations of tretinoin have also been explored as potential treatment for stretch marks, with varying degrees of success (Pribanich et al., 1994).

After topical treatment, stretch marks may appear more red after first few days of use, this initial redness reaction signals an active circulation in the damaged area and the start of regeneration process (Normeth, 1993). Authors suggest that the application of 20% glycolic acid with either 0.05% tretinoin, emollient cream or 10% L-ascorbic acid improves the appearance of striae alba (Ash et al., 1998). However, most patients experienced only minimal skin irritation (McDaniel, 2002).

Histological analysis of patients treated with 0.05% tretinoin emollient cream revealed a 22% increase in dermal elastic content compared with untreated striae. But it did not increase in sites treated with 10% L-ascorbic acid compared with untreated striae. The combined epidermal and papillary dermal thickness approached that of
untreated normal skin after treatment with the 20% glycolic acid cream plus 0.05% tretinoin emollient cream or the 20% glycolic acid cream plus 10% L-ascorbic acid (Ash et al., 1998 & McDaniel, 2002).

If reaction suggesting sensitivity or irritation occurred, patients should stop the treatment. Patients on tretinoin therapy should be advised to minimize exposure to sunlight and sunlamps, to wear protective clothes and to apply sunscreen. On the other hand, these drugs are not recommended during pregnancy, lactation or in patients with sun burns (Kang et al., 1996).

**B- Vitamin C**

Ascorbic acid (vitamin C) is a cofactor required for the function of several hydroxylases and monooxygenases. It is not synthesized in humans and has to be provided by diet or pharmacologic means. In fibroblast cultures, vitamin C stimulates collagen production by increasing the steady state level of mRNA of collagen type I and III through enhancement of transcription and prolongation of the half life of the transcripts (Nusgens et al., 2001).

Vitamin C also has the potential to enhance the density of dermal papillae, perhaps through the mechanism of angiogenesis. However, a three month daily regimen of topical ascorbic acid may slightly improve the appearance of mature striae (Ash et al., 1998), it may lead to partial corrections of the regressive structural changes associated with striae distensae (Sauermann et al., 2004).

The Er: YAG laser showed the greatest enhancement of skin permeation of vitamin C, the laser fluence and spot size were found to play important parts in controlling drug absorption. The CO$_2$ laser at a lower fluence promoted vitamin C permeation with no ablation of the stratum corneum or epidermal layers. Further enhancement was observed with the CO$_2$ laser at higher fluences which was accompanied by a prominent ablation effect (Ash et al., 1998).

Microdermabrasion ablated the stratum corneum layers with minimal disruption of the skin barrier properties according to transepidermal water loss levels. The flux and skin deposition of vitamin C across microdermabrasion-treated skin was approximately 20 fold higher than that across intact skin (Lee et al., 2003).
C- Hydrant creams:

Despite the general understanding that proper hydration is necessary to maintain the integrity and barrier of skin, little in the literature is available on the use of such creams in stretch mark prevention. Three studies involving 130 women in total were found (Young and Jewell, 2008). The active creams in the studies described are not widely available, and it was not clear whether any particular ingredient was helpful. The lack of clarity in the studies and the scientific data available makes it difficult to conclude such creams are effective, and larger studies are needed to determine the efficacy and safety of such products in combating stretch marks (Elsaie et al., 2009).

Trofolastin

One study involved 80 women and investigated the effect of massage with a cream containing Centella asiatica extract, vitamin E, and collagen-elastin hydrolysates (Trofolastin, Novartis Barcelona, Spain) and its preventive effect on the development of stretch marks in pregnant women (Mallol et al., 1991).

Forty-one subjects used the cream, and 39 used a placebo. Results showed that 56% of the placebo group and 34% of the treated group developed striae distensae in pregnancy. This study demonstrated that the active component "Centella asiatica" induced significant prevention of stretch mark development. The exact mechanism of action was identified as the stimulation of fibroblastic activity (Velasco and Romero, 1976), and antagonistic effect against glucocorticoids was also reported (Brinkhaus et al., 2000).

Verum

In a study of 50 women, although lacking a placebo control, examined a cream containing vitamin E, panthenol, hyaluronic acid, elastin and menthol (Verum). It was associated with fewer stretch marks during pregnancy than no treatment (Wierrani et al., 1992).

One-third of women in the treated group and two-thirds of those who did not receive any treatment developed striae distensae during pregnancy. The results suggest that the product could be helpful, although the trial had no placebo and the benefit may be of massage alone (Wierrani et al., 1992).
**Alphastria**

It is a cream that is composed of hyaluronic acid, allantoin, vitamin A, vitamin E, and dexpanthenol. The name is composed of the Greek word "alpha" prefix meaning "without" and the Latin word "stria" which means "lines". Hyaluronic acid is an organic substance found in human skin and is the main constituent of the cream. The hyaluronic content stimulates fibroblast activity and collagen production to restore any inhibition and collagen loss induced by hormonal fluctuations or mechanical stretch *(Shah et al., 2008)*.

A study was conducted to demonstrate the efficacy and safety of the cream. Thirty pregnant women were recruited to receive the cream and 30 others received a placebo as a control group. Three subjects in the exposed group and 21 in the control group developed striae distensae. The study concluded that the product markedly lowered the incidence of stretch marks development after pregnancy *(De-Bauman et al., 1987 & Zambon, 2007)*.

**D- Topical oil massage and herbal topical remedies:**

Some unconventional therapies and anecdotal reports recommend applying unproven oils and natural remedies to stretch marks. The underlying principle for this use would probably be keeping the skin well hydrated. Sweet almond oil, wheat germ oil, olive oil, avocado oil, and castor oil and applying seaweed wrap *(Ody P, 1999)*.

Other remedies such as comfrey, hypericum, maritime pine, equisetum, slippery elm, and wheat grass and eucalyptus tree oil are all used in creams or oils, but no efficacy studies have been performed to support these practices *(Goldberg et al., 2005)*.

Olive oil was found to reduce the incidence of severe Striae gravidarum (SG) and increases the incidence of mild SG, but it does not significantly reduce the incidence and the severity of SG and it could not be recommended for SG prevention *(Soltanipoor et al., 2012)*.

**2-Peeling agents:**

**Chemical peeling in treatment of striae distensae:**

Chemical peeling is defined as the application of a chemical cauterant to induce a controlled partial thickness cutaneous insult. It is designed to allow
regeneration of the skin from adenxal structures and dermal fibroblasts (Matarasso et al., 1990).

There are three types of peeling according to the depth penetrated by the wounding agent. Superficial peels are produced by agents that provoke a mild desquamation and refresh the skin. Very low concentration of the exfoliants slough the outer two to three layers of the stratum corneum (Zohair et al., 1996).

Brody (1992) elicited many chemical formulas that used for light peels which include, Trichloroacetic acid (TCA), Jessner's solution, Salicylic acid or alpha hydroxy acids.

Medium-depth peeling is the application of wounding agents that replace the entire epidermis and also create upper dermal destruction, inflammation and fibroplasias, thereby tightening the skin. The chemical formulas that are used include; Solid carbon dioxide with TCA 35%, Jessner's solution combined with TCA 35%, TCA 50% and Pyruvic acid which is α-ketoacid (Rubin, 1992).

Lastly, deep peeling is defined as the chemical peel that penetrates deep into the reticular dermis to correct most severe damage, utilizing a formula containing phenol. The most commonly used peel is the Baker/Gordan formula. It provides the most dramatic results but holds the most potential for systemic toxicity. It is formed of phenol 88% 3 ml added to tape or distilled water 2 ml with septisol liquid soap 8 drops and croton oil 3 drops (Zohair et al., 1996).

**Types of chemical peel in striae distensae:**

Superficial peels generally only have an impact on the epidermis and are helpful in treatment of early striae (rubra). There is little improvement in mature striae (alba) when superficial peeling is performed regularly and on repeated basis (Stagnone, 1989).

Medium depth peel is indicated in old mature striae which form a scar like lesion (Zohair et al., 1996). They are not closely associated with systemic toxicity (Monheit, 1992). They are useful for patients who in fact are appropriate for deep peels but are poor candidates due to medical contraindication (Resnik, 1984).
Accordingly, deep peels are not indicated in treatment of striae as patients should be free of cardiac, renal and hepatic disease as phenol is systemically absorbed and can be toxic to these systems (McCollough and Langston, 1988).

**Commonly used peeling agents:**

**Alpha hydroxy acid (AHA):**

Glycolic acid 50%-70% helps in treatment of early stretch marks (striae rubra). The majority of patients recognize only a mild improvement of early striae at four weeks after the peel is done, after eight weeks the improvement is more recognized. Old mature striae are minimally improved (McDaniel, 2002).

**Jessner's solution:**

It is the combination of resorcinol 14 gm, salicylic acid 14 gm, lactic acid 14 cc and ethanol 100 cc. Jessner's solution can be used alone to produce superficial peeling (Stagnone, 1989).

It can be used and immediately followed by TCA 35% to produce medium depth peel (Monheit, 1989). If the aim is superficial peeling, Jessner's solution can be applied on weekly or on intermittent basis (Matarasso et al., 1990).

**Trichloroacetic acid (TCA):**

A solution of 15-25% concentration of TCA will produce superficial coagulation of epidermal proteins, clinically appearing as a mild epithelial slough. A higher concentration up to 45% will produce epidermal necrosis, partial dermal denaturation and dermal inflammatory cell response. The application technique can affect the depth of damage. Vigorous rubbing of the solution into the skin will increase penetration by removing superficial debris already peeled by the action of the acid (Matarasso et al., 1990).

**3-Microdermabrasion:**

There are scattered anecdotal reports of improved appearance of striae with the use of various peels, but most of them are associated with vacuum-based aluminum oxide microderm-abrasion. Such reports generally describe mild improvement after a series of six to ten or more treatments (McDaniel, 2002).
Microdermabrasion polishes the skin surface, has some stimulatory effects on production of new dermal collagen and also temporarily disrupts the outer epidermal barrier, allowing better penetration of topical agents (Alster, 1999).

Microdermabrasion when combined with topical agent containing glycolic acid, retinal and magnesium ascorbyl phosphate may produce better clinical results than microdermabrasion alone (Ash et al., 1998). Also, combined with ultrasound to increase the transport of topically applied drugs into/or across the skin to achieve better response (McDaniel, 2002).

The microdermabrasion contains aluminum oxide or sodium chloride crystals that strike the skin and produce superficial trauma. It is theorized that the repetitive intraepidermal injury causes gradual improvement in damaged skin by stimulating fibroblast proliferation and collagen production, leading to new collagen deposition in the dermis (Shpall et al., 2004).

Mahuzier (1999) stated that 10 to 20 sessions of microdermabrasion at an interval of not less than 1 month and each session resulted in bleeding points, provide satisfactory improvement in SD.

In a study done by Abdel-Latif and Elbendary in 2008 on the clinical and molecular evaluation of treating SD with microdermabrasion demonstrated a promising effect of dermabrasion on stretch marks. The study was done on 20 patients with SD receiving five microdermabrasion treatments at weekly intervals on half of the body; SD on the other half of the body served as a control. Biopsies from patients were analyzed using real-time reverse transcriptase polymerase chain reaction for assay of type I procollagen I-mRNA levels. The results showed an overall good to excellent response in more than half of the subjects, with improvement more marked in striae rubra, and up regulation of type I procollagen mRNA was found in all treated SD samples.

4-Laser therapy:

Ablative lasers:

CO₂ and erbium: Yttrium-aluminum garnet (Er: YAG) lasers have been used for surfacing many types of dermal scars as acne and wrinkles. Generally, these ablative laser therapies should not be preferred to treat striae, because prolonged
healing, infection, hyper and hypopigmentation are of real concern (Nouri et al., 1999).

**a- Erbium: YAG laser:**

The Erbium: YAG laser is effective in improving striae. It demonstrates greater safety and control than CO$_2$ laser systems as the CO$_2$ laser produces excessive peripheral thermal damage. This damage may impede wound healing, skin tissue regeneration, cell viability, and functionality (Cotton et al., 1996).

The Erbium: YAG laser system produces laser energy in the infrared spectrum with a wavelength of 2940nm, with a maximum water absorption peak. The emission is deposited in the superficial dermal layer with minimal thermal scatter. It delivers laser energy in time duration short enough to prevent heat conduction damage, and enough energy is delivered to the tissue to obtain the desired cosmetic results (Teikemeiere and Goldberg, 1997). Since Erbium: YAG laser produces little thermal damage, complications as pigmentary changes and scarring which are significant following skin resurfacing with chemical peels, dermabrasion, and CO$_2$ lasers are rare. The little thermal damage may explain the short healing time seen in all patients (Teikemeiere and Goldberg, 1997).

**b- Short pulsed carbon dioxide laser:**

Short pulsed carbon dioxide laser provides another system that theoretically could be useful in the treatment of stretch marks. This laser system targets water with an infrared beam at 10,600 nm, causing a controlled abrasion of the skin (Nouri et al., 1999).

Since stretch marks are thought to be dermal scars, the short pulsed CO$_2$ laser may be helpful, indeed, cosmetic improvement of stretch marks following short pulsed CO$_2$ laser treatment, not as much as that seen with the 585 nm pulsed dye laser (Wheeland, 1995).

Persistent erythema with lack of improvement was seen in type IV skin, while hyperpigmentation was seen in skin type VI. Accordingly, it should be avoided in skin types IV, V, and VI or used with great caution and should be limited to use with skin type I, II, and III (Nouri et al., 1999).
Non ablative laser therapy:

Non ablative laser means absence of burning of the skin as seen in routine ablative laser. Both increase and decrease in collagen production have been reported following laser irradiation, depending on the wave lengths and energy used for the treatment (Abergle et al., 1987 & Alster, 1994).

Treatment with lower laser fluencies stimulates certain cells such as fibroblasts and endothelial cells, whereas higher energy densities generally inhibit production and function of these cells (Van Breugel and Bar, 1992 & Normeth, 1993).

a- Pulsed dye laser (PDL; 585 nm):

It was originally designed to destroy blood vessels by targeting red oxyhemoglobin (Lask et al., 1995). The pulsed dye laser induces selective vascular thermal injury, leading to thrombosis, vasculitis and gradual local repair with neovascularization (Anderson and Parrish, 1983 & Garden et al., 1986).

The dilated blood vessels marked at the early stage of the stretch mark formation render the striae rubra a good candidate for PDL (Karsai et al., 2007).

It has been the most commonly reported laser used for the treatment of striae (Jiménez et al., 2003).

Irradiated striae with PDL have also been shown to exhibit a large number of regional mast cells, which may elaborate a number of cytokines that could potentially stimulate the process of collagen remodeling (McCraw et al., 1999 & McDaniel, 2002).

It is also possible that collagen synthesis can be stimulated by dermal heat conduction from the laser irradiated blood vessels (Alster and Handrick, 2000).

The rationale for the use of this laser is that the architecture of the elastic fibers network subjacent to the dermoepidermal junction is markedly affected in skin exhibiting striae (Watson et al., 1998). Equally reduced and reorganized collagen has been observed (McDaniel et al., 1996). Laser therapy of striae may produce continued clinical improvement for six to twelve months after laser irradiation. Although a fluence of 3 J/cm² seemed to be optimal all fluences between 2 and 4 J/cm² were found to improve striae appearance (Yu et al., 1995 & McDaniel, 2002).
In 1995, Lask et al. found that PDL Pulsed dye laser 585 nm when used at a low fluence of 3 J/cm² with a 10 mm spot size produces significant improvement in both striae rubra and striae alba and Goldfarb et al. in 1989 reported that pulsed dye laser therapy improve all types of striae and early striae improve more dramatically while white mature striae are less responsive.

In 1996, Fitzpatrick et al. reported that in their study that there were band of well-organized elastin and collagen fibers, with increase cellularity and mucin deposition consisting with dermal collagen remodeling after PDL treatment. The increase in elastin was deemed responsible for the improvement seen clinically (McDaniel, 2002).

Striae receiving multiple treatments with 585 nm pulsed dye laser at six weeks intervals displayed incremental improvements with up to three treatments. In addition, elastin content of the papillary and reticular dermis increases five months after initiation of treatment when compared with adjacent normal appearing untreated skin (Teikemeiere and Goldberg, 1997).

Jiménez et al. in 2003 noted that using 585 nm pulsed dye laser in the treatment of SD was safe and did not produce pigmentary alteration in type IV skin, but did produce hyperpigmentation in type VI. However, it did not produce noticeable improvement in striae in either skin types because melanin competes as a chromophore with hemoglobin for the light energy (Nouri et al., 1999). Accordingly, pulsed dye laser may not be a suitable treatment option for patients with darker skin because of the potential for pigmentary alteration (Jiménez et al., 2003).

b- 308 nm Excimer laser:

Excimer laser is a monochromatic radiation at a wave length of 308nm, which can be delivered through a fused silica fiber to a handpiece. When compared with standard phototherapy, the 308 nm xenon chloride (XeCl) laser has the advantage of having increased precision and the ability to deliver high energy fluences to the target tissue in less time. It was found that it produces noticeable improvement in the loss of pigmentation with no improvement of the atrophy in striae distensae (Goldberg et al., 2003).
c- Copper-bromide laser:

The copper-bromide laser is a 577 nm laser that used for stretch marks. The study done by Longo et al. in 2003 that treated 15 patients with different stretch marks on different areas of the body, exposing them to laser settings of 4 J/cm² for SD on the breast of women or 8 J/cm² for SD on other parts of the body. The study concluded that the copper-bromide laser was effective in decreasing the size of the SD, although further studies are needed to determine the ideal parameters and the number of sessions needed for an optimum response.

d- 1,450 nm Diode laser:

The diode laser is a mid-infrared non ablative laser with an integrated dynamic cooling device. This type of laser has demonstrated efficacy in the diminution of rhytides, treatment of active acne, and improvement of atrophic scars (Tanzi and Alster, 2004).

A study examined the efficacy and safety of diode laser in the treatment of 11 patients with SD, Fitzpatrick skin types IV to VI. Patients were assigned randomly to receive 4, 8, or 12 J/cm² fluences and treatment sessions were offered every 6 weeks for a total of three sessions. The incidence of post-inflammatory hyper-pigmentation was 64% and there was no improvement in the SD. It was concluded that, for skin types IV to VI, treatment of SD is not useful and the incidence of post-inflammatory hyper-pigmentation is significant (Tay et al., 2006).

e- 1,064 nm Nd: YAG laser:

The 1,064 nm long pulse Nd: YAG laser has also led to an increase in dermal collagen when used in the non-ablative treatment of facial wrinkles (Trelles et al., 2005). In addition, this laser has a strong attraction to vascular targets (Sadick NS, 2003) that associated with its action on dermal collagen, can lead to the beneficial effects observed in the treatment of immature SD. The histopathologic characteristics present in immature SD are similar to those found in recent scars (Groover and Alster, 2000).

This would explain why scars also show a significant improvement after treatment with the 1,064 nm long pulse Nd: YAG laser. A study used the 1,064 nm Nd: YAG on immature SD in 20 patients and observers and patients identified results as satisfactory (Goldman et al., 2008). Owing to its physical characteristics,
represented mainly by the 1,064 nm wavelength, the laser used is safe. Complication rarely results when the device and parameters are appropriately used in epilation or vascular alterations, even in patients with dark skin. In addition, the cooling of striae distensae before and immediately after the use of the laser represents another factor in epidermal protection (Goldman et al., 2008).

**f- Fractional photothermolysis:**

Fractional photothermolysis (FPT) is a newer, non-ablative resurfacing laser technique, which creates microzones or microthermal zones (MTZs) of "injury" onto the skin. Within these areas, localized epidermal necrosis occurs alongside collagen denaturation. Ultimately, the necrotic debris is expelled and neocollagenesis occurs. Additionally, because this laser treatment is non-ablative, the islands of normal skin serve to speed the healing process. Fractional photothermolysis has been FDA approved for dermatological procedures requiring the coagulation of soft tissue; treatment of peri-orbital wrinkles; treatment of acne scars and surgical scars; photocoagulation of pigmented lesions such as lentigos (age spots), solar lentigos (sun spots), melasma, and dyschromia; and skin resurfacing procedures (Wanner et al., 2007).

There are several studies confirming the efficacy of fractional photothermolysis for treatment of facial scarring. Glaich and colleagues reported on seven patients who were treated with fractional photothermolysis for hypo-pigmented scars (secondary to inflammatory acne or gas fire burn). Patients received two to four treatments at 4 week intervals. No adverse events were noted. Independent physician clinical assessment revealed improvements of 51% to 75% in hypo-pigmentation in six of seven patients 4 weeks after final treatment (Glaich et al., 2007).

**Alster and colleagues** reported on 53 patients who were treated with fractional laser photothermolysis for atrophic scars. No complications or adverse events were noted. Ninety-one percent of patients had at least 25% to 50% improvement after a single treatment; 87% of patients receiving three treatments had at least 51% to 75% improvement in the appearance of scars after 1 month, with stable improvement after 6 months (Alster et al., 2007).
**Hasegawa and colleagues** treated 10 patients with acne scars using fractional photothermolysis. There was no hyper-pigmentation reported and results as seen by patients were successful (*Hasegawa et al.*, 2006).

Some authors using hematoxylin and eosin or orcein stain reported evidence of new collagen formation and demonstrated an overall increase in the density of collagen after fractional photothermolysis for improveing the photoaging (*Geronemus RG*, 2006).

This mechanism is the anticipated mode of reversing the signs and atrophy associated with stretch marks using fractional photothermolysis, the technology receiving the most attention in this regard. There are a few published studies on SD and fractional resurfacing. In 2007 Brazillin clinical study showed that (1,550 nm Fraxel SL Laser) improved texture and appearance of mature white SD in skin types I to IV in fifteen female patients. Treatments included four to five sessions at weekly intervals, pulse energy of 8 to 10 mJ/MTZ, and a final density of 2,000 MTZs/cm². The treatment response was assessed by comparing pre- and 2 week post-treatment clinical photography evaluated by two physicians and patient questionnaires. The study demonstrated an early new indication for stretch mark treatment with Fraxel at that time (*Macedo et al.*, 2007).

A Korean study used fractional photothermolysis on four patients of skin type IV with striae gravidarum. All patients received just one treatment and were assessed visually and histologically using skin biopsy. The histology showed an increase in the number of elastic fibers and no side effects were demonstrated (*Petro I*, 2007).

In a study done by *Kim et al. in 2008* on treated six patients with fractional photothermolysis, they all showed clinical improvement in melanin and erythema indices and in elasticity. The authors demonstrated an increase in collagen and elastin deposition in the dermis.

The optimal settings and parameters to use FPT have not been decided upon, but investigators have shown promising results with three to five treatment sessions with their therapeutic approaches (*Sadick N*, 2008).
5-Intense pulsed light:

Intense pulsed light (IPL) is a non coherent filtered flash lamp with a very broad band spectrum (515-1200 nm), whose source emits a visible polychromatic pulsed light of high intensity (Hernandez et al., 2002).

Requena and Sanchez (1997) reported that IPL seems to be a good option for the improvement of striae distensae. Regardless of the fact that all the treated striae in their research were white in color, that is to say in a late stage.

Intense pulsed light is known to act on the blood vessels, the authors found that the epidermal thickness increased from 0.17 to 0.49 mm and the dermal thickness also increased from 2.03 mm to 3.31 mm at the end of the treatment (Spicer and Goldberg, 1996). The increased dermal thickness is primarily as a result of an increase in the collagen fibers, which seem to acquire a more fibrillar aspect and take up more pink stain (Castor et al., 1983).

However, elastic fibers did not show any changes after treatment with intense pulsed light in the experiment performed by (Hopping, 1999). All the previous changes probably account for the clinical improvement especially in late stage striae distensae, and that's why IPL seems to be a promising alternative for the treatment of the striae distensae as it has less side effects and being of benefit (Hernandez et al., 2002).

The generation of IPL systems that deliver the selected wavelenth in micro-pulses, can be adjusted regarding their duration and the delay that separate them one from the other (in milliseconds ms); hence these systems deliver Variable Pulsed Light (VPL). A study done by Hassan and Soleiman in 2006 in the efficacy of VPL in the treatment of SD, the study included twenty patients (4 male - 16 female) with striae (9 rubra - 11 alba), ten sessions of double pass VPL were performed in each patients once weekly, 590 nm, 30 J/cm², 50x10 mm, each shot of VPL comprises a sequence of four rapid micro-pulses, these micro-pulses were of variable durations (2, 2.5, 3 and 4 ms) and were separated by a delay of 20 ms. Patients showed clinical and microscopical improvement.
6-Radiofrequency devices:

The use of radiofrequency (RF) devices has been reported to be an effective and safe noninvasive technique to tighten the face and neck skin. Unlike lasers, which convert light to heat and target a specific chromophore through the selective photothermolysis, RF devices transfer higher energy fluences to the skin through a coupling method. The electrical energy transmitted is converted to heat upon reacting with the skin’s resistance (Hsu and Kaminer, 2003).

It was reported that collagen fibril contraction occurs immediately after RF treatments, which induces new collagen formation (Zelickson et al., 2004).

A study evaluating the effectiveness of a RF device in combination with PDL subjected 37 Asian patients with darker skin tone with striae distensae to a baseline treatment with a RF device and PDL. This was followed by an additional two sessions of PDL performed at weeks 4 and 8. Histological evaluation was done on nine patients who were selected randomly; 89% of the patients showed good to very good overall improvement and 59% were graded as good and very good in elasticity. All histological evaluations demonstrated an increase in the amount of collagen fibers and six of the nine specimens showed an increase in the number of elastic fibers. Hyper-pigmentation developed in one study subject only and improved in 3 months (Suh et al., 2007).

The effects of using RF devices in combination with lasers are yet to be decided, but preliminary studies show a synergistic, effective, safe modality that could be a good alternative for stretch mark therapy (Elsaie et al., 2009).

7-Ultrasound (Sonophoresis):

The stratum corneum acts as a barrier that limits the penetration of substances through the skin. Application of ultrasound to the skin increases its permeability to various topical agents and various drugs, a technique referred to as sonophoresis (Boucaud et al., 2001 & Merino et al., 2003). Low- intensity ultrasound has been used also to enhance the transport of topical agents into the dermis after the skin's barrier layer has been disrupted by microdermabrasion (Lavon and Kost, 2004).
The low-intensity superficially focused ultrasound causes this topical agent to be propelled or massaged into the dermis, ultimately increasing their therapeutic effects (Merino et al., 2003).

In 2012 Bleve et al. demonstrated that the high frequency ultrasound can diagnose stretch marks; can detect and measure striae distensae type and maturation. Furthermore, the high-frequency ultrasound and the 3D image device, can be successfully employed in order to evaluate the efficacy of a topical treatment.

8-Cosmetic surgery:

Usually used as a last resort, for the most severe scarring form of stretch marks. A tiny incision is made along the length of the stretch marks and the affected area is removed and then stitched together (Alaiti and Zein, 2003).
Collagen
COLLAGEN

The term collagen is of Greek origin and is synonymous with glue and also gelatins (the denatured form of collagen obtained from animal tissue by boiling) (Eady et al., 1998).

Collagen fibers are soft and flexible, they are also strong and inelastic enough to the extent that the physiological role of collagen fibers in the skin is to provide the tensile properties that allow the skin to serve as a protective organ against external trauma (Prockop and Kivirikko, 1995). The fact that, a single collagen bundle 1.0 mm in diameter can sustain a load from 10 to 40 Kg without breaking, can demonstrate the remarkable tensile properties of collagen (Sams and Lynch, 1996).

Collagen fibers, as the most abundant constituent of the connective tissue of the dermis, are present either as a finely woven network or as thick bundles (Arndt et al., 1996).

Collagen as a finely woven meshwork of fibers is found in the papillary layer of the dermis, which includes; not only the sub-epidermal papillae situated between the rete ridges, but also the sub-papillary layer forming a narrow ribbon between the rete ridges and the sub-papillary blood vessels; this is referred as the papillary dermis. In addition, the pilosebaceous units and the eccrine and apocrine glands, the periadnexal dermis, are encircled by a thin meshwork of collagen fibers similar to that present in the papillary dermis. Therefore, the papillary and the periadnexal dermis are regarded as a single anatomical unit, the adventitial dermis which is a thin layer of fine collagen fibers also surrounds the blood vessels of the dermis (Murphy, 1997). While the rest of the dermis, which constitute by far the largest portion of the dermis and referred to as the reticular dermis, show the collagen fibers united into thick bundles (Arndt et al., 1996).

These collagen bundles extend in various directions horizontally, and thus some are cut lengthwise and other across, so in histological section, those bundles which are cut lengthwise appear slightly wavy (Fawcett and Jensh, 1997). Biochemically, the papillary dermis is composed primarily of type III collagen, while reticular dermal collagen is primarily of type I collagen (Murphy, 1997).
Collagen fibers are formed by both lateral and longitudinal association of collagen molecules. The collagen molecule aligned in a quarter-stagger arrangement, this arrangement demonstrating the characteristic banding pattern of the collagen fibers "cross-striation" (Uitto et al., 1999) Fig. (1).

![Fig. (1): Electron micrograph of a collagen fiber showing the characteristic cross striation (Stryer, 1995)](image)

The collagen fibrils vary in diameter as a result of varying degree of polymerization of collagen molecules. In the normal dermis, the thickness of collagen fibrils ranging from 70-140 nm but most of the fibrils are approximately 100 nm thick. On the other hand, the diameter of collagen fibrils in transverse section increases progressively from the superficial dermis through the mid and lower dermis, increasing approximately from 20-70 nm in this interval (Smith et al., 1982).

**Structure of collagen:**

Collagen fibers are composed of thinner micro-fibrils that, in turn, are formed by collagen molecules. Each collagen molecule formed by what's called tropo-collagen. The tropo-collagen molecule is the basic unit of the collagen fiber (Mathews et al., 2000).

**Microscopy of collagen**

Under the ordinary light microscope, dermal collagen fibers appear as colorless branching wavy bands with a quite variable diameter. Using dark field microscope, it becomes evident that, each fiber is a bundle of parallel un-branching fibrils (Chapman et al., 1990).
The reticulum fibers represent a special type of thin collagen fibers with smaller diameter; in addition, reticulum and collagen fibers differ in the amount of ground substance present within and around each fiber. These reticulum fibers are not recognizable with routine stains but, being argyrophilic, they can be impregnated with silver nitrate that stains them black (*Fawcett and Jensh, 1997*). Rather, all newly formed collagen consists of large fibers. However, there are a few areas in which normally small collagen fibers are present as reticulum fibers without transforming into large, non-argyrophilic collagen fibers. This occurs above all in the basement membrane zone, the region of the adventitial dermis that lays closet to the epidermis, in addition, the reticulum fibers present normally around blood vessels and as a basket-like capsule around each fat cell (*Murphy, 1997*).

By transmission electron microscope, normal type I collagen fibers; which is the original model for all other collagen variants; possess a characteristic banding patterns, known as cross striation. The most prominent cross striation appear as repeating bands spaced approximately 68 nm apart, this characteristic banding appearance of the collagen fibers originated from the specific way of aggregation of the individual collagen molecules when packing together to form collagen fibers (*Mathews et al., 2000*), which can be explained as follow; The collagen molecules are stacked in parallel way, from nose to tail (N- to C- termini), where each molecule overlaps the adjacent one in the next row by approximately one-quarter of its length (*Young and Heath, 2000*). Although, the neighboring collagen molecules overlap each other, gaps of 40 nm long separate individual molecule on the same row, which is in register across the fibril at 68 nm interval (*Fawcett and Jensh, 1997*). These gaps are referred to as "hole region" that play an important role in which small molecules; such as calcium or proteoglycan can fit into these zones according to the requirement of the tissue (*Eady et al., 1998*). And as collagen triple helices are 300 nm long arranged into rows along the long axis of the fibrils, the banding pattern repeated every five rows (5 X 68 nm = 340 nm).

In which the dark zone is referred to the gaps; as the contrast medium penetrates into the gaps, while the light zone corresponds to the overlap region; as it prevents penetration of the stain (*Garrett and Grisham, 1999*). In spite of the difference between collagen and reticulum fibrils, the later show the same 68 nm periodicity of their cross-striation (*Murphy, 1997*) Fig. (2).
Fig. (2): Electron microscopic picture of collagen fiber showing its characteristic banding pattern formed by specific packing of the collagen molecules (Garrett and Grisham, 1999)

**Genetic Heterogeneity:**

Collagens are a family of closely related, yet genetically distinct macromolecules designated by Roman numerals (I, II, III…) (Kielty et al., 1993). Historically, collagen proteins have been classified according to the order of discovery. Thus collagen type I, which is the first discovered one, was regarded as the only collagen for a long time; until a specific cartilage collagen with same properties but different sequence was discovered (collagen type II); shortly afterward, a third variant (collagen type III) was purified from skin and show to be very important for the normal mechanical integrity of blood vessels (Vuorio and De chrombrugghe, 1990).

There are at least 19 different types of collagen (from type I through XIX). Furthermore, several additional collagenous proteins have been identified through the recombinant DNA technology, but the lack of precise information about their structure and their $\alpha$-chain composition, precludes assignment of a Roman numeral at this time. In addition, there are short triple-helical collagenous segments present in other proteins such as; acetylcholinesterase, Clq component of the complement system and others are not included in the collagen family because of their collagenous domains are not a prominent part of their molecules (Mayne and Brewtown, 1993 & Myllyharju and Kivirikko, 2001).

Some collagens are homotrimers (all their three alpha chains are identical) whereas; some collagens are heterotrimeres (consisting of two or even three different
types of polypeptides, each being a distinct gene product. According to, in the human genome there are as many as 33 different genes encoding α-chains with variant amino acid sequences e.g. collagen type I is a heterogeneous molecule containing separate α1 (I) and α2 (I) chains, in which their nomenclatures for genes are (COL1A1) for pro-α1 (I) and (COL1A2) for pro-α2 (I), where COL for collagen; 1 for the type of collagen; A for α-chain; while the next number for the type of chain. This example can be used as a model for all subsequent collagen genes (Prockop and Kivirikko, 1995).

On the basis of fiber architecture in tissue, the genetically distinct collagens can be divided into different classes, Table (1):

Collagen types I, II, III, V, and XI align into relatively large fibrils, therefore they are designated as fibril-forming collagens or so called interstitial collagens. These types are characterised by having N- and C-globular extension with un-interrupted (-Gly-X-Y-) triple helices. On the other hand, type IV collagen shows numerous triple helical discontinuities. Whereas, types VI and VII are distinct as micro fibril-forming collagens, types VIII, X, and XIII distinct as short-chain collagens (Kuivaniemi et al., 1991).

A described group, includes types IX, XII, XIV, and XVI are so called fibril associated collagens with interrupted triple helices (FACIT), show to be associated with larger collagen fibers and may serve as molecular bridges that are important for the organization and stability of the extra-cellular matrices (Shaw and Olsen, 1991).

**Type I:**

Type I collagen is the most abundant structural protein in the body, found in fibrous supporting tissues such as, the dermis of the skin, tendons, ligaments, and bone (Uitto, 1990).

In skin it is considered the major collagen in the dermis, amounting to approximately 80% of the total skin collagen, where it plays a major role in forming the three-dimensional fibrillar array that provides the framework for extra-cellular matrix. On the other hand, it makes up to over 90% of the organic compound in the bone, while the remainder being ground substance proteoglycan and groups of non-collagenous molecules that appear to be involved in regulation of bone mineralization (Young and Heath, 2000).
Collagen type I, which is considered as the original model for all other collagen variants, is a heteropolymer of two distinct alpha chains that spontaneously self-assemble in a 2:1 ratio to form $[\alpha 1 \text{ (I)}]_2 \alpha 2 \text{ (I)}$ triple helices. On the other hand, there are another molecules that consist of three identical alpha I chains $[\alpha 1 \text{ (I)}]_3$ have been also detected in skin, but these so-called $\alpha 1 \text{ (I)}$ trimer molecules appear to represent a minor fraction of collagen in connective tissues. In contrast, $\alpha 2 \text{ (I)}$ trimer cannot be assembled and are thermo-dynamically unstable (Tolstoshev and Crystal, 1882).

**Type III:**

The major components of this type of collagen fibers are the fibrillar collagen of types I and III (Hatamochi et al., 1996). Type III collagen predominates particularly in extensible tissue such as, the skin, gastrointestinal tract, and the arterial blood vessels; thus it appears to contribute to the extensibility and flexibility of connective tissue. In general, tissue distribution of type III collagen parallels that of type I collagen particularly where very high compliances are necessary and so, organic matrix of bone is an exception; where type I collagen predominates (Arndt et al., 1996).

In fetal skin, type III collagen accounts for about 50% of the total collagen, while it represents approximately 10-20% of the total collagen in adult human dermis. So many studies have suggested that; type III collagen represents a fetal form of collagen since it predominates in human skin during embryonic life, but synthesis of type I collagen accelerates during the early post-natal period until the ratio of type I:III collagen reach about 6:1 in adult skin (Uitto et al., 1999). Immunocytochemical labeling studies using the electron microscope suggest that, formation of type I/III co-fibrils may be relatively common occurrence (Fleischmajer et al., 1990).

Type III collagen is composed of three identical alpha chains $[\alpha 1 \text{ (III)}]_3$ that are unique among fibrillar collagens in relatively high content of hydroxyproline, and the presence of a cysteine residue in the helix that may provide additional stabilization to the fiber through inter-molecular bonding and giving the fiber its affinity for silver stain (Cheung et al., 1983).
Table (1): Genetically distinct collagen types and their tissue distribution (Uitto et al., 1999)

<table>
<thead>
<tr>
<th>Type</th>
<th>Chain composition</th>
<th>Tissue or cell source</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>α1 (I) α2 (I)</td>
<td>Skin, bone, tendon</td>
</tr>
<tr>
<td>I-trimer</td>
<td>α1 (I)3</td>
<td>Tumors, skin</td>
</tr>
<tr>
<td></td>
<td>α1 (II)3</td>
<td>Cartilage</td>
</tr>
<tr>
<td></td>
<td>α1 (III)3</td>
<td>Fetal skin, blood vessels, gastrointestinal tract</td>
</tr>
<tr>
<td>II</td>
<td>α1 (II) α2 (IV)</td>
<td>Basement membrane</td>
</tr>
<tr>
<td>III</td>
<td>α1 (V) α2 (V);</td>
<td>Ubiquitous</td>
</tr>
<tr>
<td></td>
<td>α1 (VI) α2 (VI) α3 (VI)</td>
<td>Ubiquitous</td>
</tr>
<tr>
<td>IV</td>
<td>α1 (VII)3</td>
<td>Anchoring fibrils</td>
</tr>
<tr>
<td>V</td>
<td>α1 (VIII)3</td>
<td>Endothelial cells</td>
</tr>
<tr>
<td>VI</td>
<td>α1 (IX) α2 (IX) α3 (IX)3</td>
<td>Cartilage</td>
</tr>
<tr>
<td>VII</td>
<td>α1 (X)1</td>
<td>Hypertrophic cartilage</td>
</tr>
<tr>
<td>VIII</td>
<td>α1 (XI) α2 (XI) α3 (XI)</td>
<td>Cartilage</td>
</tr>
<tr>
<td>IX</td>
<td>α1 (XII)3</td>
<td>Tendons, ligaments, peri-chondrium, periosteum, cornea.</td>
</tr>
<tr>
<td>X</td>
<td>α1 (XIII)3</td>
<td>Ubiquitous including epidermis</td>
</tr>
<tr>
<td>XI</td>
<td>α1 (XIV)3</td>
<td>Skin, tendons cornea</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>Basement membrane</td>
</tr>
<tr>
<td>XV</td>
<td>α1 (XV)3</td>
<td>Skin, internal organs, cartilage</td>
</tr>
<tr>
<td>XVI</td>
<td>α1 (XVII)3</td>
<td>Hemidesmosomes</td>
</tr>
<tr>
<td>XVII</td>
<td>α1 (XVIII)3</td>
<td>Basement membrane</td>
</tr>
<tr>
<td>XVIII</td>
<td>Unknown</td>
<td>Basement membrane</td>
</tr>
<tr>
<td>XIX</td>
<td>Unknown</td>
<td></td>
</tr>
</tbody>
</table>

Degradation of collagen:

Control of collagen degradation in relation to synthesis, is necessary; not only during normal growth and development but also in disease states involving tissue repair. One of the important steps in this control occurs by different but related enzymes known as matrix metalloproteinase (MMPs) family, which is known to be involved in connective tissue remodeling (Sang and Douglas, 1996).

The matrix metalloproteinases (MMPs) show great deal of overlapping substrate specificities. The identities of the particular enzymes, involved in various instances of remodeling and tissue destruction, are dependent to a large degree on tissue specificity (Birkedal-Hansen, 1995). The fibroblasts and other related cells, secret this enzymes in a complex respond to multiple factors and oncogenes such as; platelet-derived growth factor and lymphokines particularly interleukin-1 respectively (Murphy and Reynolds, 1993). On the other hand, they are repressed by many factors such as, glucocorticoids, transforming growth factor B and retinoic acid (Mauviel, 1993).
At physiological temperature, interstitial collagenase initiates the proteolytic events resulting in complete degradation of collagen. So, it stands as a key point in connective tissue metabolism; it cleaves all the three chain of the collagen molecule simultaneously (at a site of three-quarters distant from the amino-termini, near the carboxy-terminal end) producing two fragments. This cleavage occur at specific bonds present in all interstitial collagens "type I, II and III “in an area of low helix stability and due to its preponderance on the hydrophobic amino acids in this region (Murphy, 1995). In addition to cleaving interstitial collagens, interstitial collagenase also cleaves type VII and X collagens but does not degrade collagen type IV and V. The products of collagen cleavage become soluble, thermally unstable and denature spontaneously at physiological temperature forming gelatins. These denatured gelatins are then susceptible to be attack by other proteinases (Seltzer et al., 1989).

There is another collagenase enzymes so-called neutrophil collagenase which attacks interstitial collagens at the same site, as does interstitial collagenase producing the characteristic three-quarter one-quarter collagen fragments, but with different rate of activity (Hasty et al., 1990). The newest addition to collagenase subfamily is collagenase-3, which unlike the previous enzymes in its activity against their same substrates (Knauper et al., 1996).

There are other matrix metalloproteinases that have been found to be included in the degradation of both collagen and connective tissue matrix such as; stromelysins, which dose not degrade type I collagen, but it can degrade type III, IX collagens and type IV "principally in its non-helical region". It can also cleave intact type II and XI collagens at sites between the cross-linking hydrolysine residue and the start of the triple helix (Parks and Sires, 1996).

Gelatinases are other matrix metalloproteinases (MMPs) enzymes, which unable to cleave helical interstitial collagens, but they show a marked specifity for denatured collagens (gelatins). Thus these enzymes most likely complete the degradation of the gelatinous cleavage products of interstitial collagenase (Tournier et al., 1994). The smallest matrix metalloproteinases (MMPs) has been formally designated "matrilysin" because of its broad substrate specificity (Wilson and Matrision, 1996).
Chapter (3)

Pulsed Dye Lasers
PULSED DYE LASERS (PDL)

The principle of selective photothermolysis (SP) proposed by Anderson and Parrish in the early 1980s, had led to a new generation of highly selective pulsed lasers in dermatology. A 450 µsec yellow dye laser was initially developed for treating PWS lesions and was the first laser cavity design intrinsically motivated by a medical need (Grevelink et al., 1999). There are many different types of dye lasers with widely differing characteristics. Unfortunately, all of the dye lasers must contend with photo-induced dye disintegration necessitating frequent changes of the dye after a certain number of pulses. They differ from each other primarily in the method of optical pumping and wavelength selection (Nelson, 1992). The central concept of pulsed dye laser is the preserving of the epidermis by allowing hemoglobin to be more precisely targeted within lesions (Richards and Garden, 2000).

Pulsed dye lasers are capable of producing yellow light at 577 or 585 nm. This represent optimal wavelength for selective absorption of hemoglobin over melanin, in that it corresponds to the beta-absorption peak of hemoglobin. PDL has the ability to penetrate up to depth from 0.5 mm to 1.2 mm below the dermo-epidermal junction and maintain the same degree of selectivity, thus allowing vessels that lie deeper in the dermis to be impacted with expected clinical effects (Garden and Geronemus, 1990).

The flashlamp-pumped pulsed-dye laser (FPDL) uses a high-power flashlamp to produce a true pulsed beam. The lasing medium is a fluorescent organic dye dissolved in a liquid solvent and housed in a transparent cell; the dye's chemical structure and the solvent and additives used dictate the operating lifetimes, which are limited due to decomposition when exposed to heat and intense light, particularly in the ultraviolet part of the spectrum. Rhodamine 6G dye is efficient, with a long lifetime, and is employed often. The cell containing the dye is surrounded by a flashlamp capable of producing difference pulse durations (Lanigan, 1995). Precise wavelength tuning is controlled by the individual dye's fluorescence spectrum and is accomplished through the use of prisms, diffraction gratings, or birefringent filters placed within the cavity. By choosing different dyes, laser light of virtually any wavelength in the visible spectrum can be produced. These lasers produce high peak power, short single pulses that are ideally suited for selective target damage to
cutaneous structures containing specific chromophores (Nelson, 1992). The 585 nm PDL is established as the standard treatment for small caliber telangiectasias. Long PDLs with wavelengths of 585, 590, 595 and 600 nm have been used to treat spider leg veins of diameters ranging from 0.2 to 1.0 mm with pulse duration of 1500 µsec and fluences up to 15 to 20 J/cm² (Kauver et al., 1996).

For heating of the targeted blood, high temperatures must be achieved within the vessels, followed by slow diffusion of cooler temperatures to the perivascular area (Richards and Garden, 2000). The emitted 450 µsec pulse duration of this laser is shorter than the thermal relaxation time (TRT) of the vessels that constitute most cutaneous vascular lesions (Goldberg, 2000).

Prolonging the pulse width by 1 to 10 ms may improve results with the PDL, but a requisite increase in energy density is necessary (Garden and Bakus, 1996).

Energy densities of PDL are in the range from 5-20 J/cm². Lower fluences usually are used for treatment of macular disorders in young children, whereas higher fluences are used in more mature adult port-wine stains, vascular lesions, hypertrophic scars, and warts (Kauver et al., 1996 & Goldberg, 2000).

The PDL light is delivered through a fiberoptic handpiece. Individual pulses are placed next to each other with a small degree (10%) overlap. It is probable that optical effects, secondary to dermal scattering, affect clinical outcome with changing spot size. Spot sizes (the area of laser impact on tissue during each pulse) typically used are 2, 3, 5, 7 or 10 mm and a 2 x 7 mm elliptical spot size. Although smaller spot sizes are highly useful for small telangiectasias, such small spot sizes can lead to a reticulated post-treatment appearance. Because complete lightening after only one treatment dose not necessarily occur for larger vascular lesions, the reticulated pattern can be treated with a subsequent laser treatment. This effect can be lessened with larger spot sizes which penetrate the dermis more deeply, possibly resulting in an improved response, specially in lesions composed of larger caliber vessels (Ross et al., 1997 & Goldberg, 2000 & Richards and Garden, 2000 & Koster et al., 2001).

**Clinical Applications of PDL:**

The spectrum of clinical applications of the PDL has continued to advance rapidly over years. This progress has been, especially apparent in the treatment of benign cutaneous blood vessel disease processes and is now being expanded to
include many benign nonvascular conditions as well. The development of the dye laser, which can be used in pulsed mode, has allowed the therapist the opportunity to treat these lesions in all age groups and anatomic sites (Richards and Garden, 2000). A great variety of vascular skin lesions have been treated with the PDL.

Port-wine stains are vascular malformations, affecting approximately 3 in 1,000 children. They are composed of networks of ectatic vessels and primarily involve the papillary dermis. Unlike many other birthmarks, port-wine stains do not resolve spontaneously. In contrast, they typically begin as pink macules and become redder and thicker over time due to decreased sympathetic innervation. The depth of the skin lesions ranges from about 1 to 5 mm. Port wine stains are generally located on the face and neck but can occur in other locations such as the trunk or limbs, the PDL has proven itself to be the preferred treatment for port-wine stains in both children and adults (Dougherty and Ryan, 2002). In 2007, Liu et al. reported that the 595 nm PDL appears to be safe, effective and well tolerated in the treatment of PWS in Chinese patients.

Hemangioma is the most common described vascular tumor, it usually present on head and neck either at birth, or shortly thereafter. FPDL, is effective when used to treat superficial hemangiomas, even if ulcerated. The selective vascular injury of the FPDL allows multiple treatment sessions with little risk of complication (Spicer and Goldberg, 1996).

Telangiectases, when small and superficial, are best treated with the FPDL, as well as spider angiomas in one or two treatment, with minimal incidence of adverse effects. Other disorders associated with telangiectasia that respond nicely to the PDL treatment include poikiloderma of Civatte, telangiectasia associated with the CREST syndrome, generalized essential telangiectasia, linear facial telangiectasia, flushing, and rosacea-associated erythema (Spicer and Goldberg, 1996 & Goldberg, 2000 & Clark et al., 2002). PDL has effectively treated areas of permanent erythema and/or diffuse telangiectasia in all cutaneous processes, including post-rhinoplasty red-nose, post-radiotherapy telangiectasias, keratosis pilaris, post-trauma, and scars (Ahmed et al., 1999 & Richards and Garden, 2000).

Pulsed dye laser can also be used for the treatment of many acquired vascular lesions including pyogenic granulomas, venous lacks and cherry angiomas. In
addition, PDL has been used successfully to treat angiofibromas, lymphangiomas, striae distensae, fine leg telangiectasias, cutaneous lesions of Kaposi’s sarcoma and other benign ectasias of the skin (Ross et al., 1997 & Lupton and Alster, 2002).

The 585 nm PDL has been used effectively in improving the quality and cosmetic appearance of surgical scars in skin type's I-IV starting on the day of suture removal; it has been approved to increase the amount of collagen in the extracellular matrix (Nouri et al., 2003). Dilated blood vessels at the early stage of striae are a good candidate for PDL (Karsai et al., 2007).

Moreover, PDL can be used to improve the appearance of erythematous keloidal and hypertrophic scars. Treatment can obliterate the vascular telangiectasia that produces the erythema associated with these scars. The laser also can flatten the scars by decreasing endothelial directed collagen synthesis (Goldberg, 2000 & Manuskiatti and Fitzpatrick, 2002 & Alster, 2003).

Non-primary cutaneous vascular disorders such as plaque-type psoriasis and warts constitute other cutaneous lesions that have been treated by the PDL and evaluated for efficacy. Psoriatic plaques with vertically oriented vessels and few horizontal vessels appear to respond better to treatment than those with numerous tortuous vessels (Zelickson et al., 1996). Several investigators have demonstrated the efficacy of the 585 nm PDL in treating warts. Flat warts respond very well to PDL therapy. However, very keratotic lesions and/or those over the planter or periungual areas have a much greater failure and recurrence rate (Richards and Garden, 2000 & Hruza, 2002).

Other uses for the PDL includeing facial acne scars, xanthelasma palpebrarum, sebaceous gland hyperplasia, molluscum contagiosum, sun-damaged skin, linear porokeratosis and granuloma faciale (Richards and Garden, 2000 & Chatrath and Rohrer, 2002). In addition, PDL has been used with encouraging results in the treatment of; cutaneous lesions of lupus erythematosus concomitantly with the usual systemic therapy (Raulin et al., 1999), inflammatory linear verrucous epidermal nevus (ILVEN) and lichen sclerosus et atrophicus (Spicer and Goldberg, 1996). Moreover, it has been also used in treatment of atrophoderma vermiculata, multiple eccrine hidrocystoma, lupus pernio, morphea, necrobiosis lipoidica diabeticorum and elastosis perforans serpiginosa (Tanzi et al., 2003).
As a general rule, PDL is safest in skin types I-IV. For patients with darker complexions, melanin competes with hemoglobin for laser light absorption, leading to decrease laser penetration and requiring the use of higher fluences in order to produce a clinical effect in these patients (Tan et al., 1984 & Goldberg, 2000).

Pre- and Postoperative Measures of PDL:

Although treatment with PDL may be anxiety provoking for young children, it is well tolerated by adults. Discomfort is described like a rubber band snapped against the skin. Each pulse of laser is described as feeling like a pin or warm needle being touched to the skin. This is generally well tolerated by adult patients except in anatomic sites of increased sensitivity such as the upper lip, the central part of the face, temple, periorbital, anogenital, digital, and plantar regions, or when using larger diameter spot size and higher energies. The addition of forced cold air or a cryogen spray, cold water chambers placed on the skin may be very helpful (Chang et al., 2002; Loo and Lanigan, 2002 & Tunnell et al., 2003). Local, topical or injected anesthesia may be very helpful in reducing the pain. If large areas are being treated, patient may request anesthesia (Richards and Garden, 2000 & Bryan and Alster, 2002).

The use of laser is safe when common sense and preventive measures are practiced. The laser-instrument room should be well ventilated and smoking should be forbidden. If the dye vehicle includes flammable materials, a fire extinguisher should be mounted in the room for added safety. Appropriate safely goggles that filter specific wavelengths of laser light must be worn at all times while the laser is in operation. Eye damage constitutes the greatest risk to the patient and to the treating staff. Both the pigmented and vascular portions of the retina absorb 585 nm light. Therefore, appropriate protective eyewear is to be worn. Because of the risk of flash fires, ethyl chloride cryogen spray, supplemental oxygen, and green vinyl tubing should be avoided (Spicer and Goldberg, 1996 & Richards and Garden, 2000).

Immediately after treatment of a vascular lesion as PWS, purpuric macules develop at the site of laser exposure. It appears as an immediate slate blue-gray discoloration of the skin, which on the ensuing 5 to 10 minutes becomes increasingly purple black. This purpura will usually last 5 to 14 days after which there is a diffuse erythema for up to 2 weeks. Post-treatment purpura can be minimized by the use of
larger spot size (e.g. 10 mm). Epidermal damage is rarely seen following the use of this laser, for that postoperative wound care generally is not required. Immediate applications of ice packs, topical antibiotics, or both are usually sufficient for PDL treated area. Optimal treatment results are observed in 3-4 Weeks (*Spicer and Goldberg, 1996* & *Goldberg, 2000*). After care is minimal, requiring only the application of antibiotic ointment or aloe vera gel and daily application of a sun screen for exposed areas for 3 to 4 months. Make-up can be worn 3 to 4 days post-treatment as long as it can be removed with minimal trauma to the skin (*Applebaum and Nelson, 1992*).

**Complications of Pulsed Dye Laser:**

Complications of PDL are often transitory and those that persist are rare. There have been reports of pigmentary changes, as well as atrophic blanch-type, hypertrophic, and keloidal scarring. Often, these side effects were found to occur after higher fluences than indicated were used, or during concomitant use of isotretinoin, or excessively overlapping pulses (*Richards and Garden, 2000* & *Koster et al., 2001*). On the basis of SP, the FPDL is able to target cutaneous blood vessels with minimal risk of collateral thermal injury and subsequent scarring. Histologic examination of FPDL irradiated skin shows that except for spongiosis, the epidermis is unaffected while the endothelial cells of superficial blood vessels are damaged (*Alster and Williams, 1995*).

Although hypertrophic scarring has been reported, it is a rare event. In a study of 500 patients treated with FPDL for a variety of vascular conditions, there were no reported cases of hypertrophic scarring and the incidence of atrophic scarring was less than 0.1%. Rare transient cutaneous depressions lasting 6 to 8 months have been reported with the use of high fluences. Dermal and epidermal atrophy, which may not be reversible, have also been reported (*Renfro and Geronemus, 1993*).

Hypertrophic scarring has been reported after PDL treatment of anterior chest and shoulder areas. Areas prone to scarring such as the anterior chest or neck, or areas where tissue is delicate, such as the periorbital regions, require a 10% to 20% reduction in fluence. Pulses are placed adjacent to one another without overlapping or with a maximum overlap of 10%. Greater spot overlap may increase the risk of side effects because of nonspecific tissue damage (*Ross et al., 1997* & *Tanzi et al., 2003*).
In a large study on 701 patients treated for PWSs by PDL, Seukeran et al. (1997) found that atrophic scarring occurred more in younger patients and they attributed this side effect to be due to the usage of higher fluences than indicated, and in other studies due to trauma secondary to scratching soon after treatment.

Hyperpigmentation is the most common side effect that has been reported to occur in as many as 10% to 15% of cases. It is more likely to occur in darker-skinned or suntanned patients, with the use of higher fluences, and after post-treatment sun exposure. Hypopigmentation, which is less commonly seen, has been reported in 2.6% to 5% of patients and is usually transient. Persistent hypopigmentation, when observed, occurs most frequently on the neck, legs, and chest (Ross et al., 1997). Thermal damage of epidermis due to higher irradiances however, increases the risk of hypo- and hyperpigmentation and of scarring. Therefore, it is compulsory for PDL treatment to cool the area to be treated prior to the laser pulse to reduce the side effects caused by the heating of the epidermis. A second important effect of selective epidermal cooling is reduced pain during laser treatment (Scherer et al., 2001). Other mild, transient side effects include crusting, scaling, and peeling that last up to 5 days (Ross et al., 1997).

Seukeran et al. (1997) reported that blistering and crusting were seen in 5.0% and 0.83% (respectively) of their group of patients, but were transient events which usually healed without permanent residual sequelae. However, five patients in their study developed hypertrophic scarring; two of them experienced severe blistering that healed by scarring. Seukeran et al. (1997) mentioned that secondary infection of treatment sites is a frequent complication occurring in 25% of PDL treatments of PWS.

The role of cooling:

The concept of cooling the skin in an effort to protect the epidermis during laser treatment of dermal targets was first studied by Gilchrest et al. (1982) with the use of ice before argon laser treatment of port-wine stains.

Epidermal melanin is often an undesired target during the laser treatment. Epidermal damage can be minimized through the use of skin cooling. This is especially important in the treatment of darkly pigmented skin types, in which side effects are common (Hirsch and Anderson, 2003). Skin cooling not only to protect
the epidermis and to prevent other collateral dermal damage, but also to reduce the discomfort associated with treatment (*Dover and Arndt, 2000*).

To enhance patient safety, Laser and IPL actively cool the skin surface through cryogen spray, forced refrigerated air, or contact cooling which may be integrated in to the distal end of the hand piece (*Ross, 2006*).
Chapter (4)

Intense Pulsed Light
INTENSE PULSED LIGHT

Intense pulsed light (IPL) is non-coherent non-collimated pulsed broad spectrum light; this is not a laser but has similar indications to many lasers (Lanigan, 2000). They are effective in the treatment of vascular and pigmented lesions of the skin and hair removal (Bitter, 2000).

IPL uses flash lamps, computer controlled power supplies, and hand pass filters to generate light pulses of prescribed duration, intensity, and spectral distribution. Flash lamps are gas discharge lamps of high intensity filled with xenon gas that produce bright light when an electric current passes through the gas. The lamp output is directed towards the distal end of the handpiece and is usually coupled in to the skin surface via a sapphire or quartz block (Ross, 2006). Cutoff filters are used to remove unwanted shorter wavelength light. For example, using a 550 nm cut-off filter, only light from 550 to 1200 will be transmitted. The light source allows great flexibility in pulse duration and fluence (Lanigan, 2000).

Engineers have improved IPL power supplies, optical components, and accessories which resulted in enhanced reliability, increased predictability of the skin response, and wider range of clinical applications (Raulin et al., 2003). One generational improvement is the replacement of quartz with sapphire. Although quartz is almost as durable and much cheaper, sapphire has a much higher thermal conductivity and thermal diffusivity than quartz. The end result is improved epidermal protection (Ross, 2006).

**IPL advantages:**

IPL devices deliver a broad spectrum of wavelengths (515 to 1200 nm), which can be altered by cut off filters to adjust to the patient's skin type and lesion depth (Butterwick et al., 2006). This is advantageous because the longer the wavelength the deeper the penetration and telangiectasia, which occur at different depths of the dermis, can be treated accordingly (Taub, 2003).

The variable pulse duration of IPL devices (0.5 to 100 msec) allows for targeting of different sized vessels at different depths (Butterwick et al., 2006), and allows greater penetration depths to be reached without damaging surrounding tissue and thus enhance the versatility of this system (Angermeier, 1999).
The larger spot size of most IPL devices facilitates treatment (Butterwick et al., 2006), and offers a more time efficient treatment and less patient discomfort. The large spot size also enables much deeper light penetration into the skin (Schroeter et al., 2005).

The beam divergence of IPLs makes them more intrinsically eye-safe than laser (The collimated nature of laser light makes it less eye safe than IPL). However, patient eye injuries have been reported after IPL treatment, and eye protection is recommended for both operator and patient (Ross, 2006).

Since treatment is relatively painless, it is carried out without anesthesia (Schroeter et al., 2005).

Complications are rare and there is no downtime (Butterwick et al., 2006). The pronounced purpura that has been reported with the pulsed dye laser was not observed during treatment with IPL (Angermeier, 1999).

Theoretically, IPL that produces a non-coherent light, as a continuous spectrum longer than 550 nm should have several advantages over a single wavelength laser system, as both oxygenated and deoxygenated hemoglobin will absorb at these wavelengths (Goldman and Fitzpatrick, 1999).

**IPL disadvantages:**

There are some drawbacks with IPL. Because such devices do not enjoy the monochromaticity of an optically pumped laser rod, even with the same filter configuration, the spectrum may not be consistent from pulse to pulse (or during the pulse). IPLs are vulnerable to the instantaneous "pumping" voltage of the capacitors. It follows that during the course of a pulse, the spectrum changes as the power ramps up and down. Most modern systems use a sophisticated computer control system that minimizes the so called spectral jitter (Ross, 2006).

Larger IPL spot sizes, while ideal covering large areas, also pose the risk of large side effects. The large spots also make it difficult to work in tight concave areas e.g. nasal ala crease (Sperber et al., 2005).

Because of the broad - spectrum nature of most IPLs, there is a risk of hair reduction in male patients (Ross, 2006).
The multiplicity of treatment parameters available to the clinician has resulted in some delay in establishing optimal parameters for each condition treated and contradictory published results (Lanigan, 2000).

Although some operators contend that IPL are safer than lasers, complications are not uncommon (Moreno - Arias et al., 2002). Some wavelength ranges are not possible with available IPLs (Ross, 2006).

**Indications and uses of IPL:**

IPL can be used in different types of indications including vascular lesions, pigmented lesions, hair removal and skin photo-rejuvenation (Christian et al., 2003).

**A) Vascular lesions**

1- **Benign venous malformations**

Deep benign venous vascular anomalies can generally be treated by means of surgery, sclerotherapy treatment, high doses of corticosteroids and with Nd: YAG laser (percutaneously or interstitially). These methods are often restricted by the adverse effects of treatment and by the extent of the malformation, the size or depth of the vessel and localization of lesions. IPL systems are preferred in these conditions (Christian et al., 2003).

2- **Essential telangiectasia**

Telangiectasis on the nose and cheeks present a significant cosmetic problem for many patients. Older forms of treatment such as electric cauterization with a diathermy needle or sclerotherapy treatment not only entails severe adverse effects but also much less widely used due to the much more effective therapeutic options of laser and IPL technology (Raulin et al., 2001).

IPL systems are preferred in treating essential telangiectasia as it shows high success rates and low occurrence of adverse effects (Bjerring et al., 2001).

3- **Haemangiomas**

Haemangiomas frequently demonstrate a great tendency to grow in the first months of life. Although a significant number of such lesions regress, they may
continue to expand significantly; in 20% of patients they remain unchanged. This is the reason why treatment should begin as soon as possible (Christian et al., 2003).

Therapeutic tools include contact cryotherapy, pulsed dye lasers, percutaneous or interstitial Nd: YAG laser, the long pulsed KTP-Nd: YAG laser and various IPL systems. IPL in particular offers many options in selecting parameters (Adamic et al., 2007).

4- Poikiloderma of Civatte

Poikiloderma is a morphologic descriptive term defined as a combination of atrophy, telangiectasia – induced erythema and irregular hyperpigmentation. It may be caused by congenital diseases, autoimmune processes and malignancy. Poikiloderma of Civatte is a specific variant that is thought to be caused by cumulative sunlight exposure exacerbated by a photoallergic reaction to fragrances applied to the neck (Weis and Goldman, 2000).

Patients with poikiloderma of civatte seek treatment for cosmetic reasons to improve the erythematous, pigmented and finally wrinkled appearance that occur in this condition in visible areas on the neck (Goldman and Fitzpatrick, 1999).

The IPL source can be considered a safe and effective therapeutic option for poikiloderma of Civatte, allowing a marked improvement of vascular and pigmented lesions with minimal side effects (Rusciani et al., 2007).

5- Port-wine stains

The pulsed dye laser is the preferred treatment for port-wine stains but IPL systems is more effective in both therapy resistant cases as well as in purple and hypertrophic port-wine stains (Christian et al., 2003).

6- Leg telangiectasias

The incidence of unsightly venulectasis and/or telangiectsis on the leg occurs in up to 41% of woman and 15% of men (Kauver, 2000).

The utilization of lasers and IPL sources for the treatment of lower extremity veins has gained increased popularity over the past years (Kauver, 2000).

The choice of wavelengths, degree of energy fluence and pulse duration of light exposure are all related to the type and size of target vessel treated and scattering
phenomenon. Deeper vessels require a longer wavelength to allow penetration to their depth (Dover et al., 1999).

B) Pigmented lesions

The spectrum of pigmented lesions treated by IPL technology extends from epidermal lentigines and café au lait macules to acquired Ota"s / Ito"s nevus, Becker's nevus and congenital nevi all the way to post-inflammatory hyperpigmentation, melasma and decorative and traumatic tattoos (Kawada et al., 2002).

Both Q-switched lasers and IPL are currently used for treatment of pigmented lesions. These different devices vary considerably regarding wavelengths, energy levels and pulse duration (Lin and Chan, 2006).

The biological effect of IPL occurs through the thermal damage of the pigment containing cells. The final cosmetic outcome of treatment with IPL is highly dependent on light source, the number of treatments and the intervals between these, as well as biological variables, for example: type and depth of lesion and anatomical location (Shimbashi et al., 1997).

C) Hair removal

Unwanted pigmented hair is a common problem for many patients. Traditional methods for hair removal have been limited by their pain, inconvenience and poor long term efficacy. A number of lasers and intense pulsed light source have been developed specifically to target hair follicles. These devices offer the potential for rapid treatment of large areas and long lasting hair removal (Christine and Dierickx, 2000).

IPL produces selective damage to hair follicles based on the principles of selective photothermolysis, they operate in the red or near-infrared wavelength region, where they under go selective absorption by melanin combined with deep penetration into the dermis (Anderson, 1999).

Deep, selective heating of the hair shaft, the hair follicle epithelium and the heavily pigmented matrix is therefore possible in the 600 to 1200 nm region. However melanin in the epidermis represent a competing site for absorption (Bjerring et al., 2000).
Pulse width also plays an important role, as suggested by thermal transfer theory. Thermal diffusion from the melanin rich shaft and matrix will heat surrounding follicular structures (Anderson, 1999).

Spatial confinement of thermal damage is obtained if the pulse duration is shorter than or equal to the thermal relaxation time of that hair follicle that is estimated to be about 10 to 100 ms depending on the size of hair follicle. IPL devices used for hair removal have pulse duration in the millisecond domin region (Bjerring et al., 2000).

D) Photo rejuvenation

IPL photo rejuvenation repairs photo-aging in the skin without disruption of the cutaneous integrity, minimal downtime and low risk profile (Bjerring et al., 2004).

Photo rejuvenation could be type I, which involves epidermal and superficial dermal structures that manifest as vascular, pigmentary and pilosebacious changes or type II, which involves changes in fine rhytides as well as lipoatrophy (Goldberg and Cutler, 2000).

The non ablative technologies may be divided into short wavelength vascular targeting technologies (585-600 nm) and longer wavelength technologies, where water is felt to be the primary target causing heating of collagen and subsequent dermal remodeling (Nell et al., 2002).

Theories of improvement in photoaging using non-ablative technologies involve 3 major mechanisms:

1. Dermal heat wounding which lead to a repair mechanism of fibroblast activation and subsequent collagen remodeling (Bjerring et al., 2004).

2. IPL induced displacement of chronically actinically damaged dermis (Goldberg, 2000).

3. Shorter wavelength technologies have been hypothesized to induce a heat mediated cytokine activation leading to secondary collagen remodeling via heat shock protein vascular endothelial and B-fibroblastic growth factor modulation (Prieto et al., 2002).
Feng et al. have stated that IPL is effective in improving wrinkles and skin texture. The mechanism of action may be through the hyperplasia and increasing activity of the fibroblasts and rearrangement of both collagen and elastin within the stroma (Feng et al., 2008).

The adjunctive use of 5-ALA in the treatment of facial photoaging with IPL provides significantly greater improvement in global photodamage, mottled pigmentation and fine lines than treatment with IPL alone, without a significant increase in adverse effects. This combination treatment enhances the results of photo rejuvenation and improves patient's satisfaction (Dover et al., 2005).

E) Treatment of some inflammatory dermatosis

Rosacea is a common disease which occurs in men and women most frequently between 30-50 years of age. Signs and symptoms include facial erythema, flushing episodes and acneform eruption (Wilkin et al., 2002).

Although pustules, papules and nodules improve with topical and oral antibiotics, control of flushing and erythema is suboptimal, lasers have been shown to be effective in eradicating superficial vascular lesion e.g. Pulsed dye lasers. However IPL systems are preferred for treatment as pulse duration can be varied and they have deep penetration. Also the larger spot size of IPL makes it simpler to treat an entire face making this a "no-down-time" procedure (Angermeier, 1999).

F) Treatment of hypertrophic scars, keloids and striae distensae

Keloids and hypertrophic scars are extremely disturbing to patients, both physically and psychologically. IPL is effective not only in improving the appearance of hypertrophic scars and keloids regardless of their origin, but also in reducing the height, redness and hardness of scars (Erol et al., 2008).

Striae distensae are dermal atrophic scars with epidermal thinning and decreased collagen and elastic fibers (Shin et al., 2011). IPL is effective in improvement of striae distensae clinically and microscopically (Hernandez et al., 2002).
Patients and Methods
PATIENTS AND METHODS

This study was carried out on thirty patients; who were recruited from the outpatient clinic of Cairo Hospital for Dermatology and Sexually Transmitted Diseases (STDs), Cairo-Egypt. Diagnosis was based on clinical examination and confirmed by histopathological evaluation. An informed consent before enrollment approved by our dermatology research ethical committee. Full detailed history and complete clinical examination were done for all patients. Patients with; skin disorders such as: photosensitivity, premalignant or malignant skin diseases, collagen or elastic disorders or keloids, history of oral retinoid intake within a year and pregnant females were excluded. Detailed consent forms were completed by all patients before starting the study.

Patients were randomly selected; twenty three with striae rubra (76.7%) and seven with striae alba (23.3%) as shown in table (2). Patients were 29 females (96.7%) and one male (3.3%), their ages ranged between 14 - 42 years with mean of age 22.77 ± 5.54 as in table (3).

According to Fitzpatrick skin type classification, 16 patients were skin type III (53.3%) and 14 patients were skin type IV (46.7%). These striae were found within the thighs in 30% (9 patients), axillae, forearms and arms in 26.66% (8 patients), abdomen in 16.7% (5 patients), buttocks in 10% (3 patients), breast and sub mammary in 10% (3 patients), and legs in 6.7% (2 patients) as in table (4).

Twenty one patients had no associated diseases (70%), while nine patients (30%) had discoid lupus erythematoses (DLE), vitiligo, liver disease, chronic idiopathic urticaria or anemia.

There were various causes that led to the appearance of striae distensae in this group of patients including weight gain in 19 patients (63.33%) which were due to increase food intake (20%), steroid intake (30%), pregnancy (10%) and early adolescence with weight gain (3.33%). Weight loss was in 5 patients (16.66%), pregnancy with proportionate increase of weight was in 3 patients (10%) and steroid intake without increase of weight was in 3 patients (10%) as in table (5) and Fig. (3).
Twenty one patients (70%) did not have any previous treatment, six patients (20%) had Dermapure cream [Hydroxyprolisilane, Tamanu extract, Darutoside], two patients (6.7%) had Retin A cream and one patient (3.3%) had Acne-free [Trazodone Hydrochloride 0.05%] as in table (6). All patients stopped their treatments 6 months before starting this study.

Twenty patients were treated with PDL on one side and IPL on the other side (66.7%), while seven patients were treated on both sides by IPL (23.3%) and three patients were treated on both sides by PDL (10%) as in table (7).

Table (2): Frequency and percentage of striae rubra and alba

<table>
<thead>
<tr>
<th>Type of Striae Distensae</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Striae Rubra</td>
<td>23</td>
<td>76.7</td>
</tr>
<tr>
<td>Striae Alba</td>
<td>7</td>
<td>23.3</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Table (3): Frequency and percentage of patient's age and their mean

<table>
<thead>
<tr>
<th>Age</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>15</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>16</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>19</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>20</td>
<td>7</td>
<td>23.3</td>
</tr>
<tr>
<td>21</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>23</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>24</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>25</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>26</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>27</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>30</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>33</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>42</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Table (4): Frequency and percentage of different sites of striae distensae

<table>
<thead>
<tr>
<th>Site of Striae Distensae</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thighs</td>
<td>9</td>
<td>30.0</td>
</tr>
<tr>
<td>Abdomen</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>Axillae</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>Buttocks</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>Legs</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>Arms</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>Breast</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>Sub mammary</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>Forearms</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

Table (5): Frequency and percentage of possible causes of striae distensae

<table>
<thead>
<tr>
<th>Possible Cause</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight Gain</td>
<td>6</td>
<td>20.0</td>
</tr>
<tr>
<td>Weight Gain &amp; Steroids</td>
<td>9</td>
<td>30.0</td>
</tr>
<tr>
<td>Weight Gain &amp; Pregnancy</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>Weight Gain &amp; Early Adolescence</td>
<td>1</td>
<td>3.33</td>
</tr>
<tr>
<td>Weight Loss</td>
<td>4</td>
<td>13.33</td>
</tr>
<tr>
<td>Weight Loss &amp; Excess Exercise</td>
<td>1</td>
<td>3.33</td>
</tr>
<tr>
<td>Steroid intake</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

Fig. (3): Frequency of possible causes of striae distensae
Patients and Methods

Table (6): Frequency and percentage of previous treatment of striae and types of this treatment

<table>
<thead>
<tr>
<th>Previous treatment</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>21</td>
<td>70.0</td>
</tr>
<tr>
<td>Dermapure cream</td>
<td>6</td>
<td>20.0</td>
</tr>
<tr>
<td>Retin A cream or gel</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>Acne free cream</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

Table (7): Frequency and percentage of treatment used in this study

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both (IPL&amp;PDL)</td>
<td>20</td>
<td>66.7</td>
</tr>
<tr>
<td>IPL only</td>
<td>7</td>
<td>23.3</td>
</tr>
<tr>
<td>PDL only</td>
<td>3</td>
<td>10.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>30</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

Treatment Protocol:

Twenty patients were subjected to Pulsed dye Laser (PDL) on one side of their bodies and IPL on the other side; while ten patients were treated by either PDL or IPL on both sides for five sessions with four weeks interval between sessions, 4-mm punch biopsies were taken before starting treatment and one month after the end of treatment.

Patients were treated with PDL (Cynosure – Chelmsford, MA-USA) with wavelength 595 nm, spot size 10 mm, pulse duration 0.5 ms, energy density (Fluence) 2.5 J/cm², pulse rate 1 Hz, with cold air and no anesthetic preparations were used, Fig. (4).

Patients were treated with fluorescence IPL source (Medical Bio Care - Sweden) with wavelength 565 nm, spot size 10 x 20 mm, pulse duration 50 -70 ms, energy density (Fluence) 17.5 J/cm². Each treatment was given by applying a thin layer of cold optical contact transparent gel (Sonogel-Germany) over the area to be treated, then energy was delivered through a pre-cooled sapphire crystal, Fig. (5).
Patients and Methods

Evaluation:

Patients were evaluated clinically before and one month after the last treatment by two fixed dermatologists. The width of the widest striae of each lesion was measured at baseline and at 20\textsuperscript{th} week. The difference in width was converted to the percentage of reduction from the baseline. Skin texture was evaluated using various parameters including: skin elasticity, horizontal striations, the level of elevation of striae rubra, the depth of striae alba and over all skin improvement. The degree of progress of improvement was graded from 0 to 5 as in table (8) according to De Angelis and his colleagues (2011). Digital photos were taken by a digital camera (Panasonic X9-Japan) for patients before and one month after the treatments. Formalin-fixed, paraffin-embedded skin biopsies were cut into 5 \textmu m thick sections.
and placed on glass slides from patients before and after treatments. They were stained with Hematoxylin and Eosin (HX&E), Masson Trichrome for collagen, Orcein for elastic and Alcian blue for ground substances. Sections were also labeled with primary monoclonal antibody (anti-collagen I α1) supplied by (Santa Cruz Biotechnology, Inc.- CA- USA) for immuno-histochemical evaluation. Electron microscopic study was done in selected cases to evaluate the ultra-structural changes. Clinical and histological pictures were assessed using computerized Image analysis; the data were obtained using Leica Qwin 500 image analyzer computer system (England). The image analyzer consisted of a colored video camera, colored monitor, hard disc of IBM personal computer and controlled by Leica Qwin 500 software. The image analyzer was first calibrated automatically to convert the measurement units (pixels) produced by the image analyzer program into actual micrometer units. The results of treatment were analyzed subjectively and digitally at baseline and after 20 weeks. Statistical calculations were performed by Student t-test, Pearson correlations and Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 15 for Microsoft Windows (SPSS).

<table>
<thead>
<tr>
<th>% of improvement</th>
<th>Treatment effect</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>0%</td>
<td>No change or worsened scar</td>
<td>0</td>
</tr>
<tr>
<td>1%-25%</td>
<td>Slight improvement</td>
<td>1</td>
</tr>
<tr>
<td>26%-50%</td>
<td>Mild improvement</td>
<td>2</td>
</tr>
<tr>
<td>51%-75%</td>
<td>Moderate improvement</td>
<td>3</td>
</tr>
<tr>
<td>76%-99%</td>
<td>Significant improvement</td>
<td>4</td>
</tr>
<tr>
<td>100%</td>
<td>Complete clearance</td>
<td>5</td>
</tr>
</tbody>
</table>

(De Angelis et al., 2011)

**Light Microscopic Study:**

The obtained specimens were fixed in 10% saline then washed and dehydrated in ascending grades of alcohol (70%, 90%, 100%). This was followed by clearing overnight in xylene. Embedding was done and paraffin blocks were obtained. 5-6 µm
Patients and Methods

thick serial sections were cut and mounted on charged and polylysine coated slides to prevent section loss.

**They were stained with:**

1. Hematoxylin and Eosin (Hx and E) *(Cook and Fimlt, 1974a).*
2. Masson Trichrome *(Modified from Masson, 1929 after Culling, 1974).*
3. Orcein *(Cook and Fimlt, 1974b & Bancroft and Gamble, 2008).*
4. Alcian blue *(Lendrum et al., 1969).*
5. Immunohistochemical stains for detection of collagen I α1. *(Watson et al., 1998).*

**Masson trichrome:**

Masson trichrome technique *(Modified from Masson, 1929 after Culling, 1974):*

**A-Materials:**

1) **Cytoplasmic (plasma) stain:**
   - 1 % ponceau de xylidine (ponceau 2R in 1% acetic acid) (2 parts).
   - 1% acid fuchsin in 1% acetic acid (1 part).

2) **Differentiation and mordant:**
   - 1% phosphomolybdic acid in distilled water.

3) **Fiber stain:**
   - Ether, 2% light green (or fast green) in 1% acetic acid or 2% aniline blue in 2% acetic acid.

**B- Method (Technique):**

1. Sections were taken to water.
2. Nuclei were stained either with Weigert's iron hematoxylin or with celestine blue-haemabum.
3. They were washed well in water.
4. Nuclear stain was differentiated with 0.5% HCL in 70% alcohol.
5. They were then washed well in tap water, and then rinsed in distilled water.

6. They were then stained in the red cytoplasmic stain 5-10 minutes.

7. This was followed by rinsing in distilled water.

8. Differentiated in 1% phosphomolybdic acid until collagen was discolourized, muscle, red blood cells and fibrin remaining red.

9. Rinsing in distilled water was done.

10. Counterstained in aniline blue or light green for 2-5 minutes.

11. Washing well in 1% acetic acid for at least 1 minute was then done.


\textbf{C- Results:}

- Nuclei dark red.
- Muscle, red blood cells, fibrin and some cytoplasmic granules → red.
- Collagen, some reticulin, amyloid and mucin → green or blue according to counter stain (Aniline blue counter-stain was the one used in this study so obtained color was blue.

\textbf{Orcein:}

For detection of elastic tissue fibers. Orcein is a naturally occurring vegetable dye, which has now been synthesized. Variations between belches of dye may produce erratic results with insufficient depth of stain on occasions. The main advantage of this stain is the simplicity of preparation. (Bancroft and Gamble, 2008).

\textbf{Orcein technique (Cook and Fimlt, 1974b):}

\textbf{A- Comments:}

This method shares with the Weigert type techniques a remarkable selectivity for elastic tissue. Occasionally it may fail to give good results and this can nearly always be traced to a faulty batch of the dye. The fact that it stains elastin a brown
colour makes it less suitable than the Weigert methods for use in conjunction with a van Gieson counterstain.

**B- Solution:**

Dissolve orcein (preferably synthetic) 1 g in 100 cm$^3$ of 70% alcohol with the aid of gentle heat. Cool, filter and add 1 cm$^3$ of concentrated hydrochloric acid.

**C- Technique:**

1. Take sections to 70% alcohol.
2. Stain with the orcein solution for 1-2 hours at 37°C.
3. Rinse in 70% alcohol, differentiate in 1% acid-alcohol if necessary then wash well in water.
4. Counterstain as required. Suitable counterstains are haematoxylin, 0.1% aqueous azure A or methylene-blue for 1-2 minutes.
5. Finally, dehydrate, clear and mount as desired.

**D- Results:**

- Elastin → dark brown.
- Background according to the counterstain used.

**Alcian Blue:**

**Alcian Blue technique (Lendrum et al., 1969):**

**A- Comments:**

It will be recalled that amyloid contains a mucin fraction and various workers have used alcian blue as a mean of identification. For example, *Pennock et al. (1968)* used both pH 1.0 alcian blue and pH 5.7 alcian blue with added magnesium chloride concentration of between 0.3 M and 0.7 M. Also, *Mowry and Scott (1967)* employed a pH 5.7 alcian blue solution plus 0.4 M magnesium chloride to a similar effect.

One major snag, however, is that alcian blue will also stain various acid mucins depending on the pH or molarity salt used which tends to lessen the value of the method. Even so, the following technique, which uses an alcian blue solution containing sodium sulphate to suppress background staining, is a reliable one and may be of use in equivocal cases. The post-staining alkali treatment renders the alcian blue
'fast' by decomposing the "solubilizing" groups present in the dye; this results in the pigment's being deposited in the tissue (Lendrum et al., 1972).

**B- Solutions:**

- Acetic-alcohol (prepare fresh):
  - 95% ethanol 45 cm$^3$
  - Distilled water 45 cm$^3$
  - Acetic acid 10 cm$^3$

- Alcian blue solution (prepare fresh):
  - 1% alcian blue in 95% ethanol 45 cm$^3$
  - 1% aqueous hydrated sodium sulphate 45 cm$^3$
  - acetic acid 10 cm$^3$
  - Allow to stand for 30 minutes before use.

- Saturated borax in 80% alcohol.

**C- Technique (slightly modified):**

1. Take sections to water. Wash. Rinse in the acetic-alcohol solution.

2. Stain with the alcian blue solution for 2 hours. Rinse in the acetic-alcohol solution, then in water.

3. Alkalinize in the borax solution for at least 30 minutes. Wash.

4. Carry out van Gieson's technique.

5. Rinse in distilled water or alcohol. Dehydrate, clear and mount as desired.

**D- Results:**

- Recent amyloid, some colloids → dark blue
- Old amyloid → paler blue or non-reactive
- Collagen → red
- Muscle → yellow
Immunohistochemical Staining:

A- Materials:

**Primary antibody:**

For detection of collagen α-type I of mouse, rat and human origin by western blotting (starting dilution (1:200) dilution range (1:100-1:1000), immunoprecipitation [(0-2 µg per 100-500 µg) of total protein (1 ml of cell lysate)], immunofluorescence and immunohistochemistry (including paraffin embedded sections) (starting dilution 1: 50 dilution range 1: 50-1: 500). Code number Sc-25974 Santa Cruz Biotechnology, Inc.- CA - USA Collagen type I (D-13).

- Storage: store at 4°C, do not freeze. Stable for one year from the date of shipment, non hazardous.

- Source: Collagen type I (D-13) is an affinity purified goat polyclonal antibody raised against a peptide a mapping near the C-terminus of the mature chain of collagen α-1 type I of human origin.

- Product: Each vial contains 200 µg IgG in 0.1 ml of PBS with 0.1% sodium azide and 0.1% gelatin.

**Epitope Retrieval:**

Formaldehyde fixation impairs or totally destroys immunoreactivity of many antigens and epitopes.

**Heat induced epitope retrieval (HIER) in citrate buffer**

Non enzymatic epitope unmasking techniques had been recently introduced to improve the immunoreactivity of many antigens in formaldehyde fixed tissues. Heat induced epitope retrieval in citrate buffer had been reported to improve the reactivity of many antibodies in formalin fixed tissues. Citrate buffer pH 6, [Catalogue number AP-9003-050] (10X), lab. Vision Corporation, Thermo Fisher Scientific, CA- USA was a 10X stock solution that was diluted 10 folds with distilled water before use.
Patients and Methods:

Detection system:

1) Goat Immuncruz staining system: code number Sc-2053, Santa Cruz Biotechnology, Inc.-CA-USA.

The goat immunocruz staining system includes 15ml each negative control (normal goat IgG), peroxidase block, serum block (5% normal donkey serum, two 15 ml vials provided), biotinylated secondary antibody and HRP-streptavidin reagent. Each of these reagents is pre-diluted and ready to use for immunohistochemical staining formalin-fixed, paraffin-embedded tissue sections.

Also included 50X peroxidase substrate, 50X DAB chromogen and 10X substrate buffer. It is sufficient for 150 slides while has to be stored at 4°C & not to be freezed and it is stable for one year from the date of production.

2) DAB substrate system: DAB chromogen (3` diaminobenzidine tetrahydrochloride), 50X concentrate and store at 2-8°C.

B- Steps of Staining (Method):

1) Paraffin sections were deparaffinized in xylene for 30 minutes and then rehydrated in descending graded series of ethanol (100%, 95%, 70%) two changes for 5 minutes each, then brought to distilled water for another 5 minutes. Sections were not allowed to dry from this point on.

2) To reduce non specific background staining due to endogenous peroxidase, the section was incubated in hydrogen peroxide for 10-15 minutes.

3) The sections were washed in phosphate buffer saline (PBS) 2 times.

4) Excess buffer was blotted off and the slides were dried around edges of the sections using pieces of cloth.

5) Pretreatment for epitope retrieval was done. Slides were placed in a coplin jar containing 10mM sodium citrate buffer, pH 6.0. It was covered with a vented plastic wrap and the jar was placed in a steamer for 20 min. The jar was taken out and the sections were left to cool in the jar for 20 minutes at room temperature. This was a very critical step.

6) Sections were then washed in PBS four times.
7) Excess buffer was blotted off slides and the slides were dried around edges of the sections using pieces of cloth.

8) Ultra V stock was applied and incubated for 5-10 minutes at room temperature to block non specific background staining.

9) Excess serum was drained or blotted off but without washing, the slides were dried around the edges of the sections using pieces of cloth.

10) Primary antibody (2 drops for each section) was added at this step and was incubated for 60 min.

11) Excess reagents were thrown off and slides were washed in PBS four times 2 minutes each.

12) The slides were dried around edges using pieces of cloth. Biotinylated goat anti-polyvalent serum was applied and incubated for 10 minutes in humidity chamber at room temperature.

13) Slides were washed in PBS four times 2 minutes each.

14) Slides were dried around edges of the sections using pieces of cloth. Streptavidin peroxidase was then applied and incubated for 10 minutes in humidity chamber at room temperature.

15) Slides were washed in PBS four times and then dried around edges of the sections with pieces of cloth.

16) DAB substrate system was prepared by combining 1-2 drops of DAB chromogen with each 1ml of DAB substrate. The mixture was applied to tissue sections and incubated for 5-15 minutes at room temperature.

17) Sections were rinsed well with distilled water.

18) Counter-staining of slides was done using 2 drops of Novocastra hematoxylin for 1-2 minutes. Slides were then washed in tap water until stain was blue then rinsed in distilled water.

19) Slides were dehydrated in ascending grades of ethanol 70%, 90% and 100% 5 minutes each.

20) Slides were cleared in xylene for two times 5 minutes each then Canada balsam was added to the slides and mounted with coverslip.
C- Results:

Dark brown deposits are considered positive.

**Transmission electron microscopic study:**

4-mm skin biopsies (striae) from selected cases before and after treatment by PDL and IPL were subjected to:

1- Fixation:

The skin (striae) specimens were first put in prepared 3% phosphate buffered glutaldehyde pH 7.3 for 3-4 hours and kept at room temperature.

Subsequently, samples were washed twice in the phosphate buffer for 10 minutes each. The specimens were then left overnight in the phosphate buffer at 4°C.

The fixed skin (striae) specimens were then post in 1% osmium tetroxide in phosphate buffer for 1-2 hours. The specimens should turn brownish black in color after post-fixation with osmium to ensure proper penetration of the fixative. The specimens were then rinsed three times in PBS and prepared for dehydration.

2- Dehydration:

Dehydration was accomplished at room temperature in graded ethanol (30%, 50%, 70%, 90% and 100%) two times in each grade 15 minutes each.

3- Clearing:

Clearing was done in propylene oxide for 15 minutes.

4- Infiltration and embedding:

Equal volumes of epon and acetone, were left on the specimen for one hour, facilitating the infiltration of the resin.

The hardness of the final block could be adjusted by the ratio of the two mixtures:

• Solution (B): Epon 100 ml and Methyl Nadic Anhydride [MNA] 89 ml.

The usual ratio used was 7 ml of solution B and 3ml of solution A which were mixed together with final addition of six drops of the accelerator Tridimethyl amino methyl phenol [DMP-30, Ciba Labs]. The final fixed specimens were disposed in beam capsule (BDH EM grade, England), a small drops of epon was placed at its bottom.

5- Polymerization:

This was done at 60°C for 24 hours.

6- Ultramicrotomy:

First semithin sections (1µm thick), were cut, collected on clean slides and left to dry on a hot plate. They were then stained with toludine blue 1% solution for one minute on the hot plate (60-100°C) (Dawes, 1980).

The sections were quickly rinsed with distilled water and dried on the hot plate. After drying, the sections were examined by the oil immersion power of the light microscope to ensure the presence of the required specimens in the cut sections.

Second, ultra thin sections (50-60 nm) thick, with a silver or gold color were cut. Sections were picked on grids and left to dry on a filter paper in Petri dish.

7- Staining:

Girds were stained by a double staining technique of uranyl acetate followed by lead citrate (Hayat, 1986). The grids were then washed in distilled water and dried on filter paper. Examination of the grids was done using Zeiss 100S transmission electron microscope 60 KV (Military Medical Academy).

Quantitative Morphometric Study

All quantitative morphometric estimations were done on histologic sections of the striae lesions using an image analyzer (Lecia Imaging System Ltd., Cambridge, England). Images were captured live on to the screen from sections under a light microscope (Olympus BX-40, Olympus Optical Co. Ltd., Japan) with an affixed video
Patients and Methods

camera (Panasonic Color CCTV camera, Matsushita Communication Industrial Co. Ltd., Japan). The video images were digitized using Leica Qwin which is a Leica's windows based image analysis tool kit fitted to an IBM compatible personal computer with a color monitor.

Quantitative Morphometric Study included:

1) Detection of the area percent occupied by blue color in Masson Trichrome stained sections. This was observed at X40 objective in 10 non overlapping fields for every specimen of all groups. The degree of color was fixed for all sections.

2) Detection of the area percent occupied by dark brown color in Orcein stained sections. This was observed at X40 objective in 10 non overlapping fields for every specimen of all groups. The degree of color was fixed for all sections.

3) Detection of the area percent occupied by dark blue color in Alcian blue stained sections. This was observed at X40 objective in 10 non overlapping fields for every specimen of all groups. The degree of color was fixed for all sections.

4) Detection of the area percent occupied by dark brown color in immunohistochemistry for detection of collagen I stained sections. This was observed at X40 objective in 10 non overlapping fields for every specimen of all groups. The degree of color was fixed for all sections.
Statistical Analysis:

- Data presented using tables and graphs percents were used to present qualitative data. Mean and standard deviation were used to present the quantitative data. Median was used to graphically present data.

- Chi square test and t test of significance were used to compare the changes between the two used methods. Paired t test was used to test the significance of change after treatment.

- Correlation coefficient was applied to test the correlation between the duration and the changes resulted after treatment.

- The Bivariate Correlations procedure computes Pearson's correlations coefficient, Spearman's rho, and Kendall's tau-b with their significance levels. Correlations measure how variables or rank orders are related. Before calculating a correlations coefficient, screen your data for outliers and evidence of a linear relationship. Pearson's correlations coefficient is a measure of linear association.

- Statistical evaluations were performed by Student t-test, Pearson correlations and Statistical Package for the Social Science; Inc., Chicago, IL, USA version 15 for Microsoft Windows (SPSS).


**RESULTS**

This study included thirty patients; twenty were treated by PDL on one side and IPL on the other side for comparison (66.7%), while three patients treated on both sides by PDL (10%) and seven patients treated on both sides by IPL (23.3%) as in table (7).

All patients included in the study were treated for five sessions with one month interval between sessions, 4-mm punch biopsy was taken before starting treatment and one month after the last treatment.

Comparison between the widths of the striae in patients treated with PDL before and after the treatment, showed there was a highly significant difference between them. The mean value of the width of the striae before PDL was 7.61±5.052 and after PDL was 5.904±4.628.

Comparison between the width in patients treated with IPL before and after the treatment, there was a highly significant difference between them. The mean value of the width of the striae before IPL was 6.33±4.029 and after IPL was 3.852±3.143 as shown in table (9) and Fig. (6).

**Table (9): Comparison between width of the striae before and after treatment with PDL and IPL**

<table>
<thead>
<tr>
<th>Striae Width</th>
<th>Mean</th>
<th>N</th>
<th>Std. Deviation</th>
<th>T-test</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre PDL</td>
<td>7.61</td>
<td>23</td>
<td>5.052</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post PDL</td>
<td>5.904</td>
<td>23</td>
<td>4.628</td>
<td>8.421</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pre IPL</td>
<td>6.33</td>
<td>27</td>
<td>4.029</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post IPL</td>
<td>3.852</td>
<td>27</td>
<td>3.143</td>
<td>5.041</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

(Student t-test: <0.001 = highly significant, <0.05 = significant)
Fig. (6): Comparison between width of the striae before and after treatment with PDL and IPL

Comparison between the different colors of the striae before and after treatment with PDL and IPL. We found that there were various degrees of colors of the striae including deep red, red, light red (pink) and white. The color of the striae in patients before treatment with PDL was; 7 patients had deep red striae, 11 patients had red striae and 5 patients had white striae. We found that the PDL treatment for five sessions changed the color of the striae in a very obvious manner. The striae of seven patients with deep red changed into five light red (pink) and two red. However, the striae of eleven patients had red color changed into 4 patients had normal color, six patients had light red (pink) and in one patient the color did not change and remained red as before PDL treatment. It should be pointed out that the color of the five patients with striae alba did not change after PDL treatment as shown in table (10).

Table (10): Comparison between the color of striae before and after PDL

<table>
<thead>
<tr>
<th>Color After PDL</th>
<th>Color Before</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Deep Red</td>
<td>Red</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Light Red</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Red</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>White</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>11</td>
</tr>
</tbody>
</table>
On the other hand, the color of striae before treatment with IPL was; 7 patients had deep red striae, 14 patients had red striae and 6 patients had white striae. We found that the IPL treatment for five sessions changed the color of the striae in also a very obvious manner. The striae of seven patients with deep red changed into three light red (pink) and four red. However, the 14 patients who had red color changed into 5 patients with normal color, seven patients with light red (pink) and in two patients the color did not change and remained red as before IPL treatment. While, the color of the six patients with striae alba did not change after IPL treatment as shown in table (11). The percentage of color improvement of striae was 73.9% after PDL and 70.4% after IPL while no changes in color of striae was 26.1% after PDL and 29.6% after IPL. While, there was no statistical significant difference between both lines of treatment as shown in table (12).

Table (11): Comparison between the color of striae before and after IPL

<table>
<thead>
<tr>
<th>Color after IPL</th>
<th>Color Before</th>
<th>Deep Red</th>
<th>Red</th>
<th>White</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Light Red</td>
<td></td>
<td>3</td>
<td>7</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Red</td>
<td></td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>White</td>
<td></td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>7</td>
<td>14</td>
<td>6</td>
<td>27</td>
</tr>
</tbody>
</table>

Table (12): Comparison between color changes of striae after ttt by PDL and IPL

<table>
<thead>
<tr>
<th></th>
<th>Improved</th>
<th>No Change</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Percent</td>
<td>Number</td>
</tr>
<tr>
<td>PDL</td>
<td>17</td>
<td>73.9</td>
<td>6</td>
</tr>
<tr>
<td>IPL</td>
<td>19</td>
<td>70.4</td>
<td>8</td>
</tr>
</tbody>
</table>

Chi square = 0.08    P>0.05 No statistically significant difference.

Comparing the skin texture of the striae before and after treatment with PDL and IPL, we noticed that there was a marked difference. This has been done through evaluation of skin texture parameters which includes; horizontal striations, the level of elevation of striae rubra, the depth of striae alba and over all skin improvement. The degrees of progress of improvement were; significant improvement, moderate improvement, mild improvement, slight improvement and no change. Comparing the
skin textures of the striae in patients treated with PDL before and after treatment, it was found that the degrees of progress of improvement were; significant improvement in 13%, moderate improvement in 34.8%, mild improvement in 26.1%, slight improvement in 17.4% and no change in 8.7% of patients as shown in table (13). While the degrees of improvement of skin texture in patients treated with IPL were significant improvement in 7.4%, moderate improvement in 40.7%, mild improvement in 25.9%, slight improvement in 18.5% and no change in 7.4% as shown in table (13) & Fig. (7-15).

**Table (13): Comparison between improvement of skin textures after PDL and IPL**

<table>
<thead>
<tr>
<th>Skin texture improvement</th>
<th>PDL</th>
<th></th>
<th>IPL</th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>score</td>
<td>No.</td>
<td>Percent</td>
<td>No.</td>
<td>Percent</td>
</tr>
<tr>
<td>Significant improvement</td>
<td>4</td>
<td>3</td>
<td>13.0</td>
<td>2</td>
<td>7.4</td>
</tr>
<tr>
<td>Moderate improvement</td>
<td>3</td>
<td>8</td>
<td>34.8</td>
<td>11</td>
<td>40.7</td>
</tr>
<tr>
<td>Mild improvement</td>
<td>2</td>
<td>6</td>
<td>26.1</td>
<td>7</td>
<td>25.9</td>
</tr>
<tr>
<td>Slight improvement</td>
<td>1</td>
<td>4</td>
<td>17.4</td>
<td>5</td>
<td>18.5</td>
</tr>
<tr>
<td>No change</td>
<td>0</td>
<td>2</td>
<td>8.7</td>
<td>2</td>
<td>7.4</td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td></td>
<td>100.0</td>
<td>27</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Before                                                                           After

Fig. (7) Striae rubra on the abdomen before and after treatment

Before                                                                           After

Fig. (8) Striae rubra on the abdomen before and after treatment
Fig. (9) Striae rubra before and after PDL (Left side) and IPL (Right side) of the abdomen (same patient)

Fig. (10): Striae rubra on the right thigh before and after IPL treatment
Results

Fig. (11) Striae rubra on the right thigh before and after IPL treatment

Fig. (12) Striae rubra on the breast before and after IPL treatment

Fig. (13) Striae rubra on the right thigh and leg before and after IPL treatment
When we evaluated the side effects which affected patients clinically after each session. We found that; there were no side effects in 12 patients (52.17%) after PDL treatment and 16 patients (59.25%) after IPL treatment as shown in table (15). All side effects occurred after first or second session and were well tolerated by all patients Fig. (16-19). They were transient and disappeared within 3 to 4 weeks either without treatment or with symptomatic treatment as topical antihistaminic for itching, glycerin for erythema; ice bags for pain and bleaching creams for hyperpigmentation.

Erythema only affected 2 patients (7.4%) with IPL. Erythema with pain affected one patient (4.3%) with PDL Fig. (17) and one patient (3.7%) with IPL. Erythema with oozing and itching affected one patient (3.7%) with IPL. Erythema with itching affected one patient (4.3%) with PDL. While, hyperpigmentation only affected 4 patients (17.4%) treated by PDL and 3 patients (11.1%) treated by IPL. Hyperpigmentation with pain affected one patient (4.3%) with PDL Fig. (18) and one patient (3.7%) with IPL. Hyperpigmentation with oozing and burn affected one
patient (3.7%) with IPL. Pain only affected one patient (4.3%) with PDL and two patients (7.4%) with IPL, while pain & itching affected 3 patients (13%) with PDL.

Table (14): Comparison between side effects with PDL and IPL

<table>
<thead>
<tr>
<th>Side effects</th>
<th>PDL</th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Percentage</td>
<td>No.</td>
</tr>
<tr>
<td>Erythema</td>
<td>2</td>
<td>7.4</td>
<td>2</td>
</tr>
<tr>
<td>Erythema, Pain</td>
<td>1</td>
<td>4.3</td>
<td>1</td>
</tr>
<tr>
<td>Erythema, Itching</td>
<td>1</td>
<td>4.3</td>
<td></td>
</tr>
<tr>
<td>Erythema, Itching, Oozing</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Hyperpigmentation</td>
<td>4</td>
<td>17.4</td>
<td>3</td>
</tr>
<tr>
<td>Hyperpigmentation, Pain</td>
<td>1</td>
<td>4.3</td>
<td>1</td>
</tr>
<tr>
<td>Hyperpigmentation, Oozing-burn</td>
<td>1</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>1</td>
<td>4.3</td>
<td>2</td>
</tr>
<tr>
<td>Pain, Itching</td>
<td>3</td>
<td>13.0</td>
<td></td>
</tr>
<tr>
<td>No side effects</td>
<td>12</td>
<td>52.2</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td></td>
<td>27</td>
</tr>
</tbody>
</table>

Fig. (16): Percentage of PDL side effects

Fig. (17) Temporary erythema after PDL treatment
Results

Fig. (18): Temporary hyperpigmentation after PDL treatment

Fig. (19): Percentage of IPL side effects

Histological results:

1- Hematoxylin and Eosin (H&E):

Examination of striae distensae rubra before treatment showed thinning of epidermis, flattened rete ridges within the papillary dermis and straightened basement membrane. It also revealed the presence of bundles of collagen fibers that were degenerated and fragmented associated with inflammatory cellular infiltration around blood vessels. Areas devoid of dermal connective tissue fibers were detected, Fig. (20A). Section from striae distensae rubra after treatment with PDL demonstrated thick epidermis, well formed rete ridges and thick bundles of collagen fibers within the underlying dermis denoting neocollagenesis in the reticular dermis, Fig. (20B).
On the other hand those treated with IPL showed well formed epidermis with straightened basement membrane and thick bundles of collagen fibers within the underlying dermis as well, Fig. (20C).

![Fig. (20) Striae Distensae Rubra (H & E X 200)](image)

- **Fig. (20A):** A photomicrograph of a section from striae distensae rubra before treatment showing fragmented bundles of connective tissue fibers (arrow), straightened basement membrane (wavy arrow), inflammatory cellular infiltration (arrow) around blood vessels (BV) and areas devoid of dermal connective fibers (star).

- **Fig. (20B):** A photomicrograph of a section from striae distensae rubra after treatment with PDL showing thick epidermis and thick bundles of connective tissue fibers (arrow) within the underlying dermis.

- **Fig. (20C):** A photomicrograph of a section from striae distensae rubra after treatment with IPL showing epidermis with straightened basement membrane (arrow head) and thick bundles of connective tissue fibers (arrow).

Examination of sections of striae distensae alba before treatment revealed the presence of loose dermal connective tissue fibers, thinning and atrophy of epidermis, Fig. (21A). Striae distensae alba treated with PDL showed thick epidermis, loose connective tissue in the papillary dermis and denser in deep dermis with thick bundles of connective tissue fibers, Fig. (21B). IPL treated striae distensae alba showed dense dermal connective tissue with thick bundles of fibers and thick epidermis, Fig. (21C).
Fig. (21) Striae Distensae Alba (H & E X 200)

Fig. (21A): A photomicrograph of a section from striae distensae alba before treatment showing loose dermal connective tissue fibers (star).

Fig. (21B): A photomicrograph of a section from striae distensae alba after treatment with PDL showing epidermis, loose connective tissue in the papillary dermis (asterisk) and denser in deep dermis with thick bundles of connective tissue fibers (arrow head).

Fig. (21C): A photomicrograph of a section from striae distensae alba after treatment with IPL showing dense dermal connective tissue with thick bundles of fibers.

2- Masson Trichrome:

Striae distensae rubra before treatment revealed scanty amount of collagen which are thin fibers within the papillary dermis and thick bundles in the deep dermis, Fig. (22A). Following treatment by both PDL, Fig. (22B) and IPL, Fig. (22C) abundant amount of collagen was manifested.

Fig. (22) Striae Distensae Rubra (Masson Trichrome X 100)

Fig. (22A): A photomicrograph of a section from striae distensae rubra before treatment showing scanty amount of collagen which are thin fibers within the papillary dermis (arrow) and thick bundles in the deeper (asterisk).

Fig. (22B): A photomicrograph of a section from striae distensae rubra after treatment with PDL showing abundant collagen as thin fibers within the papillary dermis (arrow) and thick bundles in the deeper (asterisk).

Fig. (22C): A photomicrograph of a section from striae distensae rubra after treatment with IPL showing abundant collagen which are thin fibers within the papillary dermis (arrow) and thick intermingling bundles in the deep dermis with longitudinal sections(wavy arrow) and transverse ones (arrow head).

Also striae distensae alba before treatment showed scanty amount of collagen that was in the form of thin fibers within the papillary dermis and thick bundles in the deep dermis, Fig. (23A). The amount of which became abundant following both lines of treatments, Fig. (23B & 23C).
Fig. (23) Striae Distensae Alba (Masson Trichrome X 100)

Fig. (23A): A photomicrograph of a section from striae distensae alba before treatment showing scanty amount of collagen in the form of thin fibers within the papillary dermis (arrow) and thick bundles in the deep dermis (wavy arrow).

Fig. (23B): A photomicrograph of a section from striae distensae alba after treatment with PDL showing abundant collagen which are thin fibers within the papillary dermis (arrow) and thick bundles in the deep dermis (asterisk).

Fig. (23C): A photomicrograph of a section from striae distensae alba after treatment with IPL showing abundant collagen in the papillary dermis (arrow) and in the deep dermis (wavy arrow). Note areas devoid of blue staining for collagen (star).

3- Immunohistochemical staining with anti-collagen I α1 antibody:

Examination of striae distensae rubra before treatment immunostained by anti-collagen I revealed weak positive immunostaining for collagen type I within the dermis that appeared as thin fragments, Fig. (24A). The positive immunostaining was evidently stronger following treatment with PDL, Fig. (24B) as well as after treatment with IPL, Fig. (24C). It appeared as thin fibers in upper dermis and thick bundles in deep dermis in both.

The immunostaining with anti-collagen I of striae distensae alba before treatment revealed moderate positive immunostaining for collagen I within the dermis, Fig. (25A). This was changed into strong reaction following both lines of treatment. It appeared mostly as thick bundles in cases treated by PDL, Fig. (25B) and in those treated by IPL, Fig. (25C).
Fig. (24) Striae Distensae Rubra

(Collagen immunostaining with Novocastra hematoxylin counterstain X 200)

Fig. (24A): A photomicrograph of a section from striae distensae rubra before treatment showing weak positive immunostaining for collagen I α1 within the dermis that appears as thin fragments (arrow).

Fig. (24B): A photomicrograph of a section from striae distensae rubra after treatment with PDL showing strong positive immunostaining for collagen I α1 that appears as thin fibers in upper dermis (arrow head) and thick bundles in deep dermis (arrow).

Fig. (24C): A photomicrograph of a section from striae distensae rubra after treatment with IPL showing strong positive immunostaining for collagen I α1 that appears as thin fibers in upper dermis (arrow head) and thick bundles in deep dermis (arrow).

Fig. (25) Striae Distensae Alba

(Collagen immunostaining with Novocastra hematoxylin counterstain X 400)

Fig. (25A): A photomicrograph of a section from striae distensae alba before treatment showing moderate positive immunostaining for collagen I α1 within the dermis (arrow).

Fig. (25B): A photomicrograph of a section from striae distensae alba after treatment with PDL showing strong positive immunostaining for collagen I α1 within the dermis that appears mostly as thick bundles (arrow).

Fig. (25C): A photomicrograph of a section from striae distensae alba after treatment with IPL showing strong positive immunostaining for collagen I α1 within the upper and deep dermis that appears as thin filaments (arrow).
4- *Orcein:*

Sections from striae distensae rubra before treatment showed few elastic fibers that were sparse and fragmented within the dermis, Fig. (26A). Following treatment by PDL striae distensae rubra revealed elastic fibers more abundant throughout the reticular dermis, Fig. (26B). On the other hand treatment by IPL of striae distensae rubra revealed minimally increased elastic fibers, Fig. (26C).

Examination of striae distensae alba before treatment showed some elastic fibers within the dermis, Fig. (27A). This appearance changed to having abundant amount of elastic fibers following both lines of treatment within the reticular dermis, Fig. (27B & 27C).

![Fig. (26) Striae Distensae Rubra (Orcein X100)](image1)

**Fig. (26) Striae Distensae Rubra (Orcein X100)**

*Fig. (26A): A photomicrograph of a section from SD rubra before ttt showing few elastic fibers within the dermis (arrow).*

*Fig. (26B): A photomicrograph of a section from striae distensae rubra after treatment with PDL showing elastic fibers within the dermis (arrow).*

*Fig. (26C): A photomicrograph of a section from striae distensae rubra after treatment with IPL showing minimal amount of elastic fibers within the dermis (arrow).*

![Fig. (27) Striae Distensae Alba (Orcein X100)](image2)

**Fig. (27) Striae Distensae Alba (Orcein X100)**

*Fig. (27A): A photomicrograph of a section from striae distensae alba before ttt showing some elastic fibers within the dermis (arrow).*

*Fig. (27B): A photomicrograph of a section from striae distensae alba after treatment with PDL showing elastic fibers within the dermis (arrow).*

*Fig. (27C): A photomicrograph of a section from striae distensae alba after treatment with IPL showing elastic fibers within the dermis (arrow).*

5- *Alcian Blue:*

Before treatment striae distensae rubra showed weak & minimal positive Alcian blue reaction, Fig. (28A). Treatment of striae distensae rubra by PDL revealed
a moderate positive alcian blue reaction within the dermis, Fig. (28B) that became more abundant in those treated with IPL, Fig. (28C).

Striae distensae alba revealed both weak & minimal positive alcian blue reaction before treatment, Fig. (29A). Following treatment by both PDL, Fig. (29B) & IPL, Fig. (29C) the alcian blue positive reaction was detected to be moderate.

Fig. (28) Striae Distensae Rubra (Alcian blue X 200)

Fig. (28A): A photomicrograph of a section from striae distensae rubra before treatment showing weak and minimal positive Alcian blue reaction within the dermis (arrow).

Fig. (28B): A photomicrograph of a section from striae distensae rubra after treatment with PDL showing moderate positive Alcian blue reaction within the dermis (arrow).

Fig. (28C): A photomicrograph of a section from striae distensae rubra after treatment with IPL showing abundant positive Alcian blue reaction within the dermis (arrow).

Fig. (29) Striae Distensae Alba (Alcian blue X200)

Fig. (29A): A photomicrograph of a section from striae distensae Alba before treatment showing weak positive Alcian blue reaction within the dermis (arrow).

Fig. (29B): A photomicrograph of a section from striae distensae alba after treatment with PDL showing moderate positive Alcian blue reaction within the dermis (arrow).

Fig. (29C): A photomicrograph of a section from striae distensae alba after treatment with IPL showing moderate positive Alcian blue reaction within the dermis (arrow).
As regards the use of special stains in order to detect the histological and immunological changes by the two lines of therapy. When we used Masson Trichrome stain, it was found that collagen percentage was before PDL (15.17 ± 9.553) and after PDL (28.09 ± 8.096), which means that the increase in collagen was highly significant (P=0.000) by PDL. Moreover, collagen percentage was before IPL (26.34 ± 7.779) and after IPL (35.05 ± 8.774), this means that the increase in collagen was significant (P=0.004) by IPL as shown in table (15) & figure (30). It should be pointed out that when we used immunohistological marker for collagen type I. It was found that collagen percentage before PDL was (22.53 ± 7.281) and after PDL was (49.50 ± 18.629), which means also that the increase in collagen and specially collagen type I was highly significant (P=0.000) by PDL. However, when examined the percentage of collagen type I before IPL which was (22.78 ± 7.889) and after IPL was (26.15 ± 13.243), this means that collagen type I was insignificantly increased after IPL (P = 0.193) as in table (15) & figure (31).

<table>
<thead>
<tr>
<th>Type of Stains</th>
<th>Mean ± Std. Deviation</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masson Trichrome (Collagen)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Masson before PDL</td>
<td>15.17 ± 9.553</td>
<td>0.000</td>
</tr>
<tr>
<td>Masson after PDL</td>
<td>28.09 ± 8.096</td>
<td></td>
</tr>
<tr>
<td>Masson before IPL</td>
<td>26.34 ± 7.779</td>
<td></td>
</tr>
<tr>
<td>Masson after IPL</td>
<td>35.05 ± 8.774</td>
<td>0.004</td>
</tr>
<tr>
<td>Collagen (anti-collagen I) Immunohistochemical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collagen before PDL</td>
<td>22.53 ± 7.281</td>
<td></td>
</tr>
<tr>
<td>Collagen after PDL</td>
<td>49.50 ± 18.629</td>
<td>0.000</td>
</tr>
<tr>
<td>Collagen before IPL</td>
<td>22.78 ± 7.889</td>
<td></td>
</tr>
<tr>
<td>Collagen after IPL</td>
<td>26.15 ± 13.243</td>
<td>0.193</td>
</tr>
<tr>
<td>Orcein (Elastic Fibers)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Orcein before PDL</td>
<td>5.70 ± 2.359</td>
<td></td>
</tr>
<tr>
<td>Orcein after PDL</td>
<td>6.21 ± 1.723</td>
<td>0.668</td>
</tr>
<tr>
<td>Orcein before IPL</td>
<td>5.05 ± 2.260</td>
<td>0.181</td>
</tr>
<tr>
<td>Orcein after IPL</td>
<td>5.76 ± 8.13</td>
<td></td>
</tr>
<tr>
<td>Alcian blue (Ground Substance)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcian before PDL</td>
<td>39.14 ± 16.819</td>
<td></td>
</tr>
<tr>
<td>Alcian after PDL</td>
<td>42.10 ± 17.965</td>
<td>0.263</td>
</tr>
<tr>
<td>Alcian before IPL</td>
<td>41.74 ± 13.016</td>
<td></td>
</tr>
<tr>
<td>Alcian after IPL</td>
<td>41.80 ± 14.131</td>
<td>0.913</td>
</tr>
</tbody>
</table>

(Student t-test: <0.001 = highly significant, <0.05 = significant)
Fig. (30): Shows Collagen changes by Masson Trichrome Stain

Fig. (31): Collagen type I changes after PDL and IPL

When we used Alcian blue stain, it was found that the percentage of ground substance was before PDL (39.14 ± 16.819) and after PDL (42.10 ± 17.965), which means that the increase in ground substance was insignificant (P=0.263) by PDL. Moreover, ground substance percentage was before IPL (41.74 ± 13.016) and after IPL (41.80 ± 14.131), this means that the increase in ground substance was insignificant (P=0.913) by IPL as shown in table (15) & figure (32). As regards Orcein stain for detection of elastic fibers, it was found that the percentage of elastic fibers was before PDL (5.70 ± 2.359) and after PDL (6.21 ± 1.723), which means that the increase in elastic fibers was insignificant (P=0.668) by PDL. Moreover, the percentage of elastic fibers was before IPL (5.05 ± 2.260) and after IPL (5.76 ± 0.813), this means that the increase in elastic fibers was insignificant (P=0.181) by IPL as shown in table (15) & figure (33).
Results

Fig. (32): Shows ground substance changes by Alcian blue Stain

![Fig. (32)](image)

Fig. (33): Shows elastic changes by Orcein Stain

![Fig. (33)](image)

When we compared between striae rubra and alba after treatment by PDL or IPL by special stains. We found that there was insignificant correlation in all different stains in both rubra and alba as shown in table (16). When we evaluate the difference between striae rubra (3.3± 43.55) and striae alba (46.1± 62.21) after treatment by PDL using Alcian blue stain, P- value was (0.082) which means it was insignificant; the same stain used also to compare between striae rubra (2.1 ± 33.56) and striae alba (2.1 ± 19.37) after treatment by IPL, P- value was also insignificant (0.999). This means that there was insignificant difference in the ground substance between striae rubra and alba after treatment by PDL or IPL as shown in table (16).
Table (16): Special stains after PDL and IPL in striae rubra and alba

<table>
<thead>
<tr>
<th>Type of Stains</th>
<th>Mean ± Std. Deviation</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Masson Trichrome (Collagen)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After PDL Striae Rubra</td>
<td>251.715 ± 300.908</td>
<td>0.055</td>
</tr>
<tr>
<td>After PDL Striae Alba</td>
<td>32.355 ± 40.741</td>
<td></td>
</tr>
<tr>
<td>After IPL Striae Rubra</td>
<td>29.530 ± 26.368</td>
<td>0.802</td>
</tr>
<tr>
<td>After IPL Striae Alba</td>
<td>35.463 ± 20.248</td>
<td></td>
</tr>
<tr>
<td><strong>Collagen (anti-collagen I)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After PDL Striae Rubra</td>
<td>158.744 ± 142.855</td>
<td></td>
</tr>
<tr>
<td>After PDL Striae Alba</td>
<td>120.136 ± 109.631</td>
<td>0.571</td>
</tr>
<tr>
<td>After IPL Striae Rubra</td>
<td>6.180 ± 61.727</td>
<td>0.149</td>
</tr>
<tr>
<td>After IPL Striae Alba</td>
<td>42.906 ± 46.172</td>
<td></td>
</tr>
<tr>
<td><strong>Orcein (Elastic Fibers)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After PDL Striae Rubra</td>
<td>43.433 ± 69.824</td>
<td>0.317</td>
</tr>
<tr>
<td>After PDL Striae Alba</td>
<td>40.419 ± 22.393</td>
<td></td>
</tr>
<tr>
<td>After IPL Striae Rubra</td>
<td>17.719 ± 24.031</td>
<td>0.239</td>
</tr>
<tr>
<td>After IPL Striae Alba</td>
<td>12.481 ± 15.246</td>
<td></td>
</tr>
<tr>
<td><strong>Alcian blue (Ground Substance)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After PDL Striae Rubra</td>
<td>3.3 ± 43.55</td>
<td>0.082</td>
</tr>
<tr>
<td>After PDL Striae Alba</td>
<td>4.1 ± 62.21</td>
<td></td>
</tr>
<tr>
<td>After IPL Striae Rubra</td>
<td>2.1 ± 33.56</td>
<td>0.999</td>
</tr>
<tr>
<td>After IPL Striae Alba</td>
<td>2.1 ± 19.37</td>
<td></td>
</tr>
</tbody>
</table>

(Student t-test: <0.001 = highly significant, <0.05 = significant)

When we used the Masson Trichrome stain to evaluate the collagen between striae rubra (251.715 ± 300.908) and striae alba (32.355 ± 40.741) after treatment by PDL, the P-value was (0.055) and also when we compared between striae rubra (29.530 ± 26.368) and striae alba (35.463 ± 20.248) after treatment by IPL, the P-value was (0.802). This means that there was insignificant difference in collagen content in the dermis between striae rubra and alba after treatment by PDL or IPL as shown in table (16).

The evaluation of collagen type I using immunohistochemical staining to compare between striae rubra (158.744 ± 142.855) and striae alba (120.136 ± 109.631) after treatment by PDL, the P-value was insignificant (0.571); also when we compared between striae rubra (6.180 ± 61.727) and striae alba (42.906 ± 46.172) after treatment by IPL, the P-value was insignificant too (0.149); which means that there was insignificant difference in collagen type I between striae rubra and alba after treatment by PDL or IPL as shown in table (16).

As regarding the evaluation of elastic fibers content in the striae using Orcein stain after treatment by PDL, striae rubra (43.433 ± 69.824) and striae alba (40.419 ± 22.393), the P-value was insignificant (0.317) between them; also after IPL,
striae rubra (17.719 ± 24.031) and striae alba (12.481 ± 15.246), the P-value was insignificant too (0.239); which means that there was insignificant difference in elastic fibers between striae rubra and alba after treatment by PDL or IPL as shown in table (16).

**Table (17): Correlation between duration of striae and Collagen and Alcian stains after PDL and IPL**

<table>
<thead>
<tr>
<th></th>
<th>Pearson Correlation</th>
<th>P value</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcian PDL</td>
<td>-.125</td>
<td>.579</td>
<td>22</td>
</tr>
<tr>
<td>Alcian IPL</td>
<td>-.285</td>
<td>.158</td>
<td>26</td>
</tr>
<tr>
<td>Collagen PDL</td>
<td>.053</td>
<td>.835</td>
<td>18</td>
</tr>
<tr>
<td>Collagen IPL</td>
<td>.540**</td>
<td>.005</td>
<td>25</td>
</tr>
</tbody>
</table>

(Pearson Correlation: Significant = ≥0.5)

When we used Pearson correlation, it was found that there was a significant correlation between the duration of striae and the increase of collagen by IPL (.540) as in table (17). However, there was insignificant correlation between duration of striae and ground substance improvement by alcian blue stains after treatment by PDL and IPL.

**Electron microscopic results:**

Examination of specimens from striae distensae rubra by the electron microscope revealed widely spaced collagen bundles. Both longitudinal and transverse sections of them were detected with fibroblast in between Fig. (34). Treatment by PDL was associated by thickening of collagen bundles Fig. (35). Thickened collagen bundles with characteristic striations was also achieved in cases treated by IPL Fig. (36).

Also specimens from striae distensae alba showed similarly widely spaced thin collagen bundles Fig. (37). Following treatment with PDL relative thickening was
observed with scanty matrix in between Fig. (38). The same findings were also detectable following treatment with IPL Fig. (39).

Fig. (34): A photomicrograph of a section from striae distensae rubra before treatment showing widely spaced collagen bundles both longitudinal (L) & transverse sections (T). Note the fibroblast (F) between the collagen. (TEM X 4000)

Fig. (35): A photomicrograph of a section from striae distensae rubra after treatment with PDL showing thickened collagen bundles both longitudinal (L) & transverse sections (T). (TEM X 4000)

Fig. (36): A photomicrograph of a section from striae distensae rubra after treatment with IPL showing thickened collagen bundles with thin characteristic striations (star). (TEM X 4000)
Fig. (37): A photomicrograph of a section from striae distensae alba before treatment showing widely spaced thin collagen bundles both longitudinal (L.) & transverse sections (T). Note fibroblast (F). (TEM X 4000)

Fig. (38): A photomicrograph of a section from striae distensae alba after treatment with PDL showing relatively thickened collagen bundles (arrow) with less abundant matrix in between (star). (TEM X 4000)

Fig. (39): A photomicrograph of a section from striae distensae alba after treatment with IPL showing relatively thickened collagen bundles (arrow). (TEM X 4000).
Discussion
DISCUSSION

Striae distensae (SD) is a common disfiguring cutaneous condition occurs in pregnant women and obese persons with rapid weight change, it is accepted that a combination of; genetic factors, endocrine alterations and mechanical stretching of skin plays a significant role in its development. Histological examination of SD revealed epidermal thinning, flattened rete ridges and fragmentation with degeneration of the collagen and elastic fibers. The classic anatomical sites affected include the abdomen and breast for pregnancy-related striae, the outer thighs or lumbosacral regions for adolescent boys, and the buttocks, thighs, upper arms, and breast for adolescent girls.

Striae in this study were mostly found within; the thighs in 30%, axillae, forearms and arms in 26.66%, abdomen in 16.7%, buttocks in 10%, breast and submammary in 10%, and legs in 6.7% and this distribution was comparable to that found in a study done by Chang et al. (2004), where Striae were more common on thighs (35%), abdomen (25%), buttocks (15%), arms (10%), axillae (10%) and less common on legs (5%).

On the contrary the areas affected with SD in the current study were different than those reported by Cho and his coworkers in (2006) where 77.1% of 48 Korean girls aged 15-17 had SD within buttocks which were the most prevalent area of striae development, followed by the thighs and calves. The difference in areas between the current study and Cho et al. (2006) might be because of the difference in age and race of patients and also other predisposing factors such as pregnancy and steroid intake.

The occurrence of SD in the current study was due to pregnancy in 25% of patients, which includes 10% with increase weight gain and 15% with proportionate increase of weight. It is known that more than 90% of pregnant women develop SD due to a combination of hormonal factors (e.g. adrenocortical hormones, estrogen and relaxin), along with increased lateral stress on connective tissue (Lawley and Yancey, 2003).

In 2009 Maia and her coworkers reported that striae in younger women were more frequently observed, in those who gained more weight during pregnancy and/or
those who had babies with higher birth weight which suggested that increased maternal age could be a protecting factor against striae during pregnancy.

Estrogen is known to play a major role in skin organization such as elasticity, water holding capacity, vascularity and pigmentation. The increase in hormonal receptor expression such as estrogen, androgen and glucocorticoid receptors in the skin with SD, suggests that regions that undergo greater mechanical stretching of the skin may express greater hormonal receptor activity. This activity may influence the metabolism of the extracellular matrix, causing the formation of SD. Alterations in hormone receptors occur within a well-defined time period during the formation of SD; however, there are differences in the functionality of hormone receptors during different stages in the development of the lesions. This may represent an initial step towards an understanding of the pathophysiology of SD (Cordeiro et al., 2010).

Moreover, the triggering cause of striae gravidarum besides mechanical stress could be the lower serum relaxin levels in patients led to decreased elasticity of the connective tissue (Lurie et al., 2011).

The therapeutic options for striae are numerous; they include the use of topical tretinoin, microdermoabrasion, IPL and various lasers such as 308 nm excimer laser, 577 nm copper bromide laser, 585 nm PDL and fractional lasers e.g. Erbium Glass (1550 nm), Erbium YAG (2940 nm) and CO2 (10600 nm). Although, that the management of striae has been a source of curiosity as well as frustration for both clinician and researcher, the use of laser therapy and IPL has been the most frequently therapeutic modalities for the treatment of striae with promising results (Hernandez-Pérez et al., 2002).

In the current study a trial to evaluate the effects of both PDL and IPL treatments on the clinical and histopathological pictures of patients who were suffering from SD was done. Concerning the width of striae; it is known that striae with greater width have more disfiguring clinical picture. There were a few studies evaluated the consequence of therapy on the width of striae. It was reported that the use of 0.1% topical tretinoin (retinoic acid) could reduce the length and width of pregnancy-related striae but failed to increase collagen or elastin content, and they were only effective for early-stage striae. They could not explain their findings and
they added that the processes which are responsible for the clinical improvement remain unknown (Kang et al., 1996).

In 2005 Luis-Montoya and his coworkers found that the width of striae could be improved by using subcision or with 0.1% tretinoin cream, but they added that 21.5% of the striae treated with subcision had necrosis in the site of incision. So, it is not recommend doing subcision as a line of treatment for striae. Moreover, a study of Yang and Lee (2011) was done on the use of non-ablative 1,550 nm fractional Er: Glass laser and ablative fractional CO₂ laser resurfacing fractional laser on the width of SD. They showed that both lines of treatment were effective and safe and neither treatment showed any greater clinical improvement than the other one.

In the current study, comparing the outcome of both treatments on the width of SD, showed that both PDL and IPL produced a highly significant improvement (P <0.001 and 0.001), although that PDL showed a slightly better response. In order to explain this response, a comparative analysis that measures the degree of the width regression versus the duration of SD, was found that early striae with short duration which has small width always showed superior improvement with both PDL and IPL. However, late SD with long duration frequently demonstrates less improvement with both PDL and IPL. These results agree with the findings of Hassan and Soleiman in 2006, who demonstrated that IPL was effective in the treatment of twenty patients with striae. Ten sessions of double-pass IPL were performed on each of the patients once weekly. Data concerning the morphological features includes the length and width of each of the treated striae showed clinical and microscopical improvement with significant differences in the post treatment length and width of treated striae (P <0.01).

On the other hand, the considerable erythema which is usually detected in striae rubra is known to be due to dilated venules and perivascular lymphocytic and eosinophilic infiltration (Hassan and Soleiman, 2006). PDL and IPL output pulse and spectral profiles are selective treatment of vessels in vascular lesions (Weiss et al., 2011). So they might be a good choice in treatment of striae rubra.

It was found that both PDL and IPL treatments diminished the erythema gradually. According to its degree of redness, the percentage of change of striae reddish coloration ranged between 73.9% and 70.4% after treatment. However, striae
alba did not reveal any change of color. This result confirms the findings of Jiménez and his coworkers (2003) when they noticed that the degree of erythema was markedly diminished after PDL in patients with striae rubra.

As it is known that the tensile properties of skin determine some important physical attributes of skin elasticity, striae showed markedly different properties regarding its skin distensibility values. Evaluations of the tensile properties and functions of skin are complementary particularly when the connective tissue is abnormal (Pierard et al., 1999).

In a study done by Kim et al. in 2008, skin elasticity was found to be partially normalized after fractional photothermolysis laser with wavelength 1550-nm. In the current study both PDL and IPL treatment modalities had positive effects on the skin texture and the rate of improvement was almost equal with both treatments. In another study done by Nehal et al. in 1999, on five patients with SD; they showed a slight overall improvement in the appearance and surface texture of the striae following multiple PDL treatments in three patients. Histologic comparison of pre- and post-treatment biopsy specimens failed to reveal normalization of skin architecture.

In 2008 Stotland and his coworkers reported that twenty patients with SD received a total of 6 treatments using a 1550-nm, erbium-doped fiber laser with 2 to 3 weeks in between treatments, showed an improvement in texture of 26% to 50% in 50% of patients. The treatments were well tolerated by all patients with a majority experienced transient post-treatment erythema and edema.

The selection of the PDL and IPL devices applied in this study was based on their demonstrated efficacy in the treatment of scars as well as their preservation of the stratum corneum for enhanced safety and expedited skin recovery after treatment. Using hematoxylin and eosin stains as well as collagen, elastin and ground substances stains, the recovery of skin was demonstrated in the form of the increase in the rete ridges and increase epidermal thickness together with the normal appearing collagen content through the abundance of thin collagen fibers within the papillary dermis and thick intermingling collagen bundles in the deep dermis with longitudinal and transverse sections which supports the results of McDaniel and his colleagues (2007). Also, confirms the findings of Hassan and Soleiman (2006) on the efficacy of IPL on SD; in which the epidermal thickness was increased significantly on
average from 0.17 to 0.5 mm. They noticed that the dermal thickness showed the most important improvement at the end of treatment which can be clearly attributed to the increase in collagen fibers, as the post-treatment collagen acquired a more fibrillar aspect and took up more pink stain.

Furthermore, despite the fact that most of histologic findings of treated striae are objectively evaluated, they still correlate with the individually assessed clinical findings, including the pre- and post-treatment of striae. These microscopic parameters provide the cellular explanation for the clinical improvement including, improvement in the epidermal atrophy, dermal elastosis and quality of the collagen fibers. Also, **Suh and his colleagues in 2007** added that PDL increased collagen fibers in all the cases of SD and two thirds of the cases had increase in the elastic fibers. They added that the findings are likely to be responsible for the decreased striae width and improved their appearance.

As it was indicated that collagen and elastic fibers are considered the major components that are affected within the skin during the formation of striae. In **2008 Lie and his colleagues** mentioned that nonablative dermal remodeling has generated great interest among both laser surgeons and patients. Evidence indicated that dermal collagen formation is the key mechanism of action for the nonablative techniques.

Moreover, **Bak et al. (2009)** reported that when fractional photothermolysis (1550 nm) was used to treat SD (alba) with pulse energy of 30 mJ, and eight passes at intervals of 4 weeks for two sessions; six of the 22 patients (27%) showed good to excellent clinical improvement from baseline, whereas the other 16 (63%) showed various degrees of improvement. Skin biopsy revealed that average epidermal thickness and dermal thickness were greater than at baseline. It should be added that in **2004 Alexiades-Armenakas and his colleagues** mentioned that the whitish color of striae alba could be corrected up to 70% with the use of 308-nm excimer laser; which is safe and effective in pigment correction of hypopigmented scars and striae alba.

In **2003, Moody and Hruza** studied with ultrasound new collagen formation after nonablative laser irradiation. A single treatment with a 585-nm pulsed dye laser appeared to increase dermal collagen which could be assessed with noninvasive cutaneous ultrasound and that increase in dermal collagen was the cornerstone of nonablative dermal remodeling with laser therapy. They revealed that light has
relatively small component of delivered energy that is absorbed in the blood vessels and the remainder induces non-specific heating of the entire irradiated fields.

Considerable progress has been made in the understanding of the molecular events regulating new collagen formation after the application of PDL and IPL, as pointed out by Yu and his colleagues in 2006 that lower fluence (3 J/cm²) PDL increased the collagen production in fibroblasts in vitro by up-regulating the type I procollagen (α1, α2) mRNA expression, transforming growth factor-beta (TGF-β1) and significantly increased the level of hydroxyproline in culture supernatants (collagen production in fibroblast). TGF-β1 plays an important role in collagen production and exerts its biological effects on collagen production by a family of intracellular signaling molecules. These data suggested that lower fluence PDL (3 J/cm²) could stimulate new collagen production through procollagen gene and TGF-β1 receptors expressions. In 2003 Jimenez and her colleagues found that collagen per gram of dry weight of sampled SD tissue was increased with PDL using hydroxyproline assay.

Aiming in this study to evaluate the level of tissue expression of collagen after treatment with PDL and IPL in different types of striae and its correlation with the clinical improvement observed in patients. The current study found that collagen was increased in a statistically significant manner by both PDL and IPL (P <0.001 & 0.004) demonstrated by Masson Trichrome stain, which was higher with PDL treatment. Therefore, these results demonstrate that the increased collagen formation within striae after PDL and IPL treatment were considerably related to pathogenesis of striae which subsequently improved clinically as regards the width and texture of striae. This confirms the findings of Suh and his coworkers (2007) that 585 nm PDL increased the fibroblastic activity resulting in an increased production of collagen and elastic fibers.

Furthermore, the use of immunohistological marker for collagen type I; PDL only showed a significant increase of collagen type I (P <0.001), while IPL did not reveal a significant change (P = 0.193). It should be added that the consequence of stimulation of the fibroblast activity by PDL to express collagen type I was more than IPL, and this could be explained by the findings of Bak and his colleagues (2009) and Yu and his colleagues (2006) concerning the immunoreactivity of procollagen
type I which was increased after treatment with PDL. Also, the current study supports the work of Wong and his coworkers in 2009 when they mentioned that IPL is involved in the increase of extracellular matrix construction of fibroblasts by upregulating the gene expressions of collagen type III and TGF-betal. However, neither collagen type I nor fibronectin gene were expressed with IPL exposure. So, it is advisable to further study the effect of both PDL and IPL on collagen type III in case of SD.

On the other hand, when elastic fibers and ground substances were evaluated using Orcein and Alcian blue; they failed to show any statistical significant correlation between PDL and IPL before and after treatment.

Furthermore, these special stains failed also to show any significant difference in collagen, elastic fibers and ground substances between striae rubra and striae alba as a result of either PDL or IPL treatment. Additionally, the current study showed that about 52.2% to 59.3% of patients did not suffer from any permanent side effects with both PDL and IPL respectively.

Most of the patients tolerated both devices as; transient erythema (8.7% with PDL and 14.8% with IPL) which was resolved within two to seven days after treatment, pain (26.1% with PDL and 14.8% with IPL) and itching were transient (17.4% with PDL and 3.7% with IPL) and they were relieved within a few hours after treatment.

Hyperpigmentation was found in 21.7% of the patients treated with PDL and 18.5% of the patients treated with IPL. This was lower than hyperpigmentation measured by Suh et al. in 2007 which was 25%, and was reported in darker skinned Asian patients with PDL. The current study included patients with skin type III (53.3%) and skin type IV (46.7%) and hyperpigmentation was mostly found in skin type IV.

Also, Jiménez and her colleagues in 2003 found that extreme caution must be taken in treating patients with skin type V and VI with PDL as it resulted in post-inflammatory hyperpigmentation and they recommended that the PDL treatment of skin type VI should be avoided even with the use of low fluences.
Conclusions
CONCLUSION

Striae distensae (SD) is a common skin condition which can cause significant distress without any medical problems. Both PDL and IPL can enhance the clinical picture including striae width, color and texture through collagen stimulation. Collagen expression was found to be significantly increased after both PDL and IPL.

Although that PDL expressed collagen type I in a highly significant manner more than IPL which may be explained through modulation of its genes. Striae rubra gave a superior response with both PDL and IPL than striae alba which was evaluated clinically, although the histological changes could not be verified. It seems that both lines of treatments are promising therapeutic modalities for striae with minimal side-effects.
SUMMARY

Striae distensae (SD), also known as stretch marks are a common disfiguring condition associated with continuous and progressive stretching of the skin which is usually parallel to the resting tension lines.

The aetiology of the striae are poorly understood, they may develop as a result of stress rupture of the connective tissue framework or develop as a result of many other causes. Moreover, genetic factors play an important role in the development of striae, the expression of procollagen and fibronectin gene is markedly decreased in SD, also there is a decrease in $\alpha$ (I) and $\alpha$ (III) procollagen mRNA levels in SD compared with normal skin.

Both systemic and local glucocorticoid therapy can produce cutaneous atrophy by a dose related pharmacological effect. The mode of action of corticosteroids is through inhibition of collagen gene expression which occurs at transcriptional, translational and post-translational levels. Steroids also known to inhibit the formation of glycosaminoglycans.

Oral contraceptive pills and hormonal effects in pregnancy play a role in developing SD. Oral contraceptive pills increase cortisol level in blood which leads to the development of SD. In pregnancy hormonal effect and stretch factor, both lead to the appearance of SD.

In the past, SD were observed with many debilitating conditions such as tuberculosis, typhoid fever, Marfan syndrome and diabetes mellitus. In nutritionally deprived states, there is an increased risk of deficiency of ascorbic acid essential for normal collagen synthesis as a required co-factor for hydroxylation of proline and lysine, however, the basic aetiology is not known.

Clinically striae pass through distinct stages; initially they appear as raised pink purple linear lesions without significant depression of the skin (rubra), but with time they mature to become paler, depressed and finally wrinkled (alba).
Striae usually appear on abdomen, lower back, upper arms and legs. Usually SD are a cosmetic problem, which could be prevented by limiting weight gain, a lot of exercise and intake of plenty of fluids.

Histologically, initial inflammatory changes are followed by a flattening and thinning of the epidermis due to underlying changes in the numbers and organization of collagen, fibrillin, and elastin fibers. Histologic studies of mature striae reveal stretched collagen fibers aligned parallel to the skin surface, followed by subsequent loss of collagen and increased flattening of rete ridges. Contributing to the atrophied appearance of striae are the reduced amounts of fibrillin surrounding the dermal-epidermal junction, reduced elastin in the papillary dermis and reorganization of elastin and fibrillin fibers in deep dermis.

Different methods have been used in the treatment of SD which include: topical treatment as tretinoin, microdermabration, chemical peeling, laser therapy, IPL, radiofrequency and cosmetic surgery. It has always been suggested that effective treatment of SD be instituted during the active stage.

In this study aiming to compare between Pulsed Dye Laser and Intense Pulsed Light in the treatment of striae distensae in relation to the degree of striae distensae clinically and histopathologically.

This study was carried out on thirty patients; twenty three with striae rubra and seven with striae alba. Patients were 29 females and one male, their ages ranged between 14 - 42 years with mean of age 22.77 ± 5.54.

According to Fitzpatrick skin type classification, 16 patients were skin type III and 14 patients were skin type IV. These striae were found within the thighs in 9 patients, axillae, arms and forearms in 8 patients, abdomen in 5 patients, buttocks in 3 patients, breast and sub mammary area in 3 patients, and legs in 2 patients.

There were various causes that led to the appearance of striae distensae in this group of patients including weight gain in 19 patients (63.33%) which were due to increase food intake (20%), steroid intake (30%), pregnancy (10%) and early adolescence with weight gain (3.33%). Weight loss was in 5 patients (16.66%), pregnancy with proportionate increase of weight was in 3 patients (10%) and steroid
intake without increase of weight was in 3 patients (10%). Twenty patients were treated with PDL on one side and IPL on the other side, while seven patients were treated on both sides by IPL and three patients were treated on both sides by PDL.

Patients were evaluated clinically before and one month after the last treatment by two fixed dermatologists. The width of the widest striae of each lesion was measured at baseline and at week 20. The difference in width was converted to the percentage of reduction from the baseline. Skin texture was evaluated using various parameters including: skin elasticity, horizontal striations, the level of elevation of striae rubra, the depth of striae alba and over all skin improvement. The degree of progress of improvement was graded from 0 to 5 according to De Angelis and his colleagues (2011). Digital photos were taken for patients before and one month after the treatments. Formalin-fixed, paraffin-embedded skin biopsies were cut into 5 µm thick sections and placed on glass slides from patients before and after treatments. They were stained with HX and E, Masson Trichrome for collagen, Orciein for elastic and Alcian blue for ground substances. Sections were also labeled with primary monoclonal antibody (anti-collagen I α1) for immuno-histochemical evaluation. Electron microscopic study was done in selected cases to evaluate the ultra-structural changes.

Results of this study based clinically on width of the widest striae and showed a highly significant difference before and after treatment by PDL and IPL; color improvement of striae was 73.9% after PDL and 70.4% after IPL; skin texture improvement of patients treated with PDL before and after treatment showed; significant improvement in 13%, moderate improvement in 34.8%, mild improvement in 26.1%, slight improvement in 17.4% and no change in 8.7% of patients. While the degrees of improvement of skin texture in patients treated with IPL were significant improvement in 7.4%, moderate improvement in 40.7%, mild improvement in 25.9%, slight improvement in 18.5% and no change in 7.4%. Histologically, using Masson Trichrome stain; collagen percentage was increased after ttt by PDL and IPL by the use of, by Alcian Blue stain; ground substance was increased and by Orcien stain; elastic fibers was increased after both lines of ttt. It should be pointed out that when we used immunohistological marker for collagen type I. it was found that collagen
percentage was increased specially collagen type I by both lines of treatment which was highly significant by PDL and insignificantly increased by IPL.

When we compared between striae rubra and alba after treatment by PDL or IPL by special stains, we found that there was insignificant correlation in all different stains in both rubra and alba. There was insignificant difference in collagen content in the dermis between striae rubra and alba after treatment by PDL or IPL and there was insignificant difference in collagen type I between striae rubra and alba after treatment by PDL or IPL.

**By electron microscope**, striae distensae rubra revealed widely spaced collagen bundles. Thickening of collagen bundles after treatment by PDL. Thickened collagen bundles with characteristic striations were also achieved in cases treated by IPL. Specimens from striae distensae alba showed similarly widely spaced but thin collagen bundles. Following treatment with PDL relative thickening was observed with scanty matrix in between. The same findings were also detectable following treatment with IPL.

So, both PDL and IPL can enhance the clinical picture including striae width, color and texture through collagen stimulation. Striae rubra gave a superior response with both PDL and IPL than striae alba. It seems that both lines of treatments are promising therapeutic modalities for striae with minimal side-effects.
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References


Arabic Summary
الملخص العربي

الخطوط التي تظهر على الجلد نتيجة شد مSEL جلد و عادة تكون موازية لخطوط السد المتواجدة في حالة السكون.

و تعتبر أسباب نشأة هذه الخطوط غير مفهومة وفيما تتشكل نتيجة الإجهاد في القياس التكويني للأنسجة الضامة أو تتشكل نتيجة لعوامل أخرى كبيرة، فالعوامل الوراثية تلعب دورا هاما في نشأة هذه الخطوط وقدرة جين البروكولاجين و الفيبرونيكليت عن تنويع تونوز مع هذه الخطوط و أيضا مستوى البروكولاجين ألفا (I) و (III) في الخطوط نيل بالمقارنة مع الأنسجة الطبيعية.

وي تعتبر العلاج بالكورتيزون الموضعي أو الجهازى يسبب إحداث ضمور جلدى تبعا للجرعة العلاجية، فالنظام الدفعي للكورتيزون هو عن طريق تشتت الجين الخاص بالكولاجين و أيضا من المعروف أن الكورتيزون يبحث صنع أحماض السكر.

أما عن أعراض منع الحمل و التأثير الهرموني للحمل لهم دورا هاما في نشأة هذه الخطوط.

فأعراض منع الحمل تؤدي إلى زيادة معدل الكورتيزون في الدم، الذي يؤدي إلى نشأة الخطوط كما أنه في الحمل لكل من التأثير الهرموني و عوامل السد تؤدى إلى ظهور هذه الخطوط.

في الماضي، تم ملاحظة هذه الخطوط الجلدية مع كثير من أمراض الضعف مثل الكنز و حمى التيفود و أيضا في متلازمة مارفان و مرض السكر كما أن في أمراض نقص التغذية التي تزيد خطورة نقص فيتامين سي الأساسية للعملية الطبيعية ل تصنيع الكولاجين كعامل مساعد للدورة البرولين و الليسين و لكن السبب الأساسي غير معروف حتى الآن.

و من الذاتية الإكلينيكية، تمر هذه الخطوط خلال مرحلة محددة حيث بداية تظهر كخطوط حمراء داكنة بدون انخفاض في الجلد (الحمراء) لكن مع الوقت تتضح لتحصى باهثة منخفضة و أخرى تتجعد (البيضاء).

هذه الخطوط عادة تظهر في أسفل الظهر، البطن، أعلى الأذن و الأرجل. و تكون مشكلة تجميلية و لكن من الممكن منعها بالمحافظة على الوزن و عمل تمارين رياضية كبيرة و تناول كمية كبيرة من السوائل.

يحدث في البداية تغيرات نتيجة لالتهاب ثم يتبعه تسخيخ و ترقق في الأدمه نتيجة تغيرات في عدد و نظام الكولاجين و الفيبرونيكليت و حزم الإيلاستين. مصطلحات وجد أن الخطوط الجلدية الناضجة تظهر فيها حزم الكولاجين مشدودة و مرتيب موازيه لسطح الجلد يتبعها فقد في الكولاجين و زيادة تسخيخ
السلسلة الشبكية تم بدأ الخطوط تضمن نتائج فئة كمية الفيبريللين حول الوصل بين البشرة و باطن الجلد، مع قله الإيلاستين في الطبقية العليا من باطن الجلد و إعادة منظومة الفيبريللين والإيلاستين في الطبقية السفلية من باطن الجلد.

أما عن طرق العلاج التي يتم استخدامها لهذه الخطوط فهي كثيرة و تشمل التشريز الكيميائي و الصنفرة و العلاج الموضعي مثل التريتروين إبانواع كثيرة من البلازمنا و الضوء الموضعي الكثيف و جهاز الترددات (الراديو فركيونسي) وقد يلجأ المريض إلى الجراحات التجميلية مع علم أن العلاج الفعال يؤثر دائما على الخطوط الإنسانية الإنتقائية و هي في البداية.

كان الهدف من هذه الدراسة هو المقارنة بين الليزر البينيسي الصبغي و الضوء الموضعي الكثيف في علاج الخطوط الإنتقائية و علاجها بإدخال هذه الخطوط إلكليتيكيا و هستولوجيا، كذلك تم دراسة تأثير مختلف الصبغات الكولاجين والإيلاستين و الماء الأيضي (الجليكون أمينو جليكان) لتقييم إستجابة إنتان الخطوط.

شملت هذه الدراسة ثلاثون مريضا ثلاث و عشرون منهم يعانون من الخطوط الحمراء و سبعة من الخطوط البيضاء، تسعة و عشرون آثري و رجل واحد، تراوحت اعمارهم بين 14 إلى 42 عاما.

طبقا لتصنيف فيتزباتريك لأنواع الجلد، من المرضى جدد 14 مريض من نوع 3 و 16 جلدهم من نوع 4. أما أماكن تواجد هذه الخطوط، فإنه أخذ تسعه من المريض و تحت اليبس و الساعد و الأذرع و في ثمانية مريض و البطن في خمسة من المرضى أما في الثدي و تحت الثدي في ثلاثة مرضي و أرداف ثلاث منهم و أثاث من المرضى الخطوط الجلدية في أرجحهم.

فيما يتعلق بالأسباب التي أدت إلى ظهور هذه الخطوط في المرضى التي تشمل هذه الدراسة، فقد كانت مختلفة فمنها نتيجة زيادة الوزن (19 مريض) و قد قمنا بتقسيم هذه المجموعة إلى زيادة الوزن نتيجة زيادة في الأكال (6 مركسي) أو العلاج بالكورتيزون (9 مريض) أو الحال (3 مرضي) أو نتيجة المراهقة المبكرة و النمو المفاجئ (مرض واحد)، أما باقي المرضى فقسم منهم كانت ظهور الخطوط نتيجة النقص في الوزن و قد قسمنا هذه المجموعة إلى النقص السريع في الوزن نتيجة لرجيم قاسي (4 مريض) أو نقص الوزن نتيجة الإسراف في ممارسة التمارين الرياضية (مرض واحد)، و يبقى سته من المرضى ثلاث منهم ظهور الخطوط كنت نتيجة الحمل مع زيادة نسبية طفيفة جدا في الوزن و الثلاث الأخيرين نتيجة أخذ الكورتيزون من غير زيادة الوزن.

عشرون مريضا عولجا بالليزر النبضي الصبغي في جانب من الجسم و الضوء الموضعي الكثيف في الجانب الآخر، أما العشر الأخرين فسع ممنهم عولجا بالضوء الموضعي الكثيف في كلا الجانبين من الجسم و ثلاث عولجا بالليزر النبضي الصبغي في كلا الجانبين من الجسم و قد قروا إكلينياقب العلاج و بعد شهر من آخر جلسة للعلاج عن طريق اثنين من أطباء الأمراض الجلدية.
المتخصصين، وقد تم قياس عرض أعراض خط جلد موجود بالمرضى قبل العلاج و بعد عشرين اسبوعا من العلاج.

كذلك تم تقسيم بنية نسيج الجلد من خلال عدة مقومات منها مرونة الجلد و الخطوط العرضية الموجودة داخل الخط الجلدي و معدل ارتفاع الخط الأحمر و عمق الخط الأبيض و التحسن العام للجلد. أما درجة التحسن فقد تم قياسها تبعا لدرجات انجليز (2011).

و قد أُخذت صور فوتوغرافية لبعض المرضى قبل العلاج و بعدة أشهر، كما أُخذت عينات جلدية مقاس 4 مم للدراسة الهистولوجية قبل العلاج و بعدة أشهر حيث ثبتت بالفوريامين و طمرت في البالافين ثم طغعت إلى شرائح سماكة 5 ميكرومتر و وضع على شرائح زجاجية، صبغت الشريحة بالبرونزاكيديس و الإيروبين، بالماسون ترايكروم لفحص الكولاجين، الألومنيوم لفحص الأورساتين، الألمنين الزرقاء لفحص المادة الأرضية. و تم فحص الشريحة أيضًا بالتحليل المناعي الكيميائي للأنسجة باستخدام الجسم المضاد (ألفا كولاجين I، ألفا I)، كما تم استخدام الميكروسكوب الإلكتروني لدراسة بعض الحالات و تقسيم التغيرات التي حدثت في بناء الأنسجة.

اعتمدت نتائج هذه الدراسة إكلينيكيا على قياس عرض أعراض خط جلد موجود بالمرضى و وجد إختلافات إحصائية كبيرة ذات أهمية بين قبل العلاج و بعده سواء بالليزر النبضي الصبغي أو بالضوء الوهميسي الكثيف، التحسن في لون الخط الجلدي بعد العلاج بالليزر النبضي الصبغي و قد بلغ نسبة 73.9% ونسبة 70.4% بعد ضوء الوهميسي، أما عن التحسن في بنية نسيج الجلد بعد العلاج بالليزر النبضي الصبغي كانت كالآتي: تحسن ملحوظ بنسبة 13%- تحسن متوسط بنسبة 34.8%- تحسن معتدل بنسبة 26.1%- تحسن قليل بنسبة 17.4%- لا يوجد تحسن بنسبة 8.7% بينما التحسن في بنية نسيج الجلد بعد العلاج بالضوء الوهميسي الكثيف كانت تحسن ملحوظ بنسبة 6.4%- تحسن متوسط بنسبة 40.7%- تحسن معتدل بنسبة 25.9%- تحسن قليل بنسبة 18.5%- لا يوجد تحسن بنسبة 7.4%.

أما عن تقييم الأنسجة هستولوجيًا كان عن طريق الميكروسكوب الضوئي حيث وجد نسبة الكولاجين تزايد بعد العلاج بالليزر النبضي الصبغي و أيضا بعد الضوء الوهميسي الكثيف باستخدام صبغة الماسون ترايكروم، كما تزايدت نسبة المادة الأرضية باستخدام صبغة الألمنين الزرقاء و حزم الإيروبين تزايدت أيضا باستخدام الأورساتين بعد العلاج بكلا الجهازين.

و وكذلك وجد تزايد في نسبة الكولاجين و باخصا كولاجين I باستخدام التحليل المناعي الكيميائي للأنسجة. باستخدام الجسم المضاد أنتى كولاجين I و بعد العلاج بكلا الجهازين و لكن هذه الزيادة ظهر معها وجود أمومة إحصائيات كبيرة باستخدام الليزر النبضي الصبغي و عدم وجود أهميه إحصائيات باستخدام الضوء الوهميسي الكثيف.
و عند المقارنة بين الخط الجلدي الأحمر والأبيض بعد العلاج بكلا الجهازين وجدنا عدم وجود علاقة إحصائيه ذات أهميه بإستخدام كل الصبغات السابقه، كما وجدنا عدم وجود إختلاف إحصائي في حزم الكولاجين بين الخط الأحمر والخط الأبيض بعد العلاج بكلا الجهازين، أما بالنسبة للكولاجين فقد وجد أيضا عدم وجود أهميه إحصائيه بين الخط الأحمر والخط الأبيض بعد العلاج بكلا الجهازين.

و باستخدام الميكروسكوب الإلكتروني وجد أن الخطوط الحمراء تظهر على هيئة مساحه واسعه من حزم الكولاجين أما بعد العلاج فهذه الحزم سمكها يزداد بكلا الجهازين و لكن بخطوط مميزه داخل حزم الكولاجين عند استخدام الضوء الوميضي الكثيف، أما الخطوط البيضاء فهي أيضا تظهر بمساحات واسعه من حزم الكولاجين الرفيعه و بعد العلاج بكلا الجهازين هذه الحزم يزداد سمكها مع وجود نسيج ضئيل بينها.

و بذلك يستطيع كل من الليزر الوردي الصيفي و الضوء الوميضي الكثيف أن يحسن الشكل الإكلينيكي للخطوط الإنتفاخية من حيث عرض الخط الجلدي و لون الخط و بنية نسيج الجلد من خلال تحفيز الكولاجين علما بأن الخطوط الحمراء تسحب أفضل للعلاج باستخدام أي من الجهازين عن الخطوط البيضاء. و يتضح لنا ايضًا أن العلاج بأي من الجهازين هو وسيلة علاجيه ناجحه مصحوبة بقله من الأعراض الجانبية.
دراسة مقارنة بين الضوء الوميضي الكثيف والليزر النبضي الصغي في علاج الخطوط الإنتفاخية الإنبسطاوية

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دراسة مقارنة بين الضوء الوميضي الكثيف و الليزر النبضي الصغى في علاج الخطوط الإنتفاخية الإنبسطية

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