VII.7. PHYSICAL MODEL OF THE HAEMODINAMIC EFFECTS AROUND THE OBSTRUCTING PLAQUE IN THE ACUTE CORONARY SYNDROMES TAKE ACCOUNT OF THE STENOSIS GEOMETRY

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Abstract

Selective coronary angiography with quantitative parameter assessment of the vessel stenosis geometry, which leads to acute coronary syndrome, was performed in 205 patients undergoing acute phase of myocardial infarction or unstable angina pectoris. Data for physicomechanical properties of the arterial vessels, blood and blood flow were used. The range of pressure variations accompanying the most frequently encountered ruptures of the atherosclerotic plaques was analyzed with physical model. It suggests that in 'lock' type plaques the ratio between the pressure on the wall of the normal vessel and that in the section outside the stenosis is considerably increased, especially in cases of enlarged vessel gauge. The results obtained show that the wall pressure closely to the plaque of a stenosed vessel with normal diameter of 3.5 mm exceeds 25 times the pressure within a normal vessel segment.

Introduction

The impact of the complicated atherosclerosis plaque on the pathogenesis of the acute coronary syndromes is well known (*Shah P.K., 1997; Foo R.S.Y, D.P. De Dono*, *2000*). The inhomogeneous plaque morphology is the reason for irregularly stress distribution in the area of the modified vessel wall (*Falk E., 1991; Fuster M., L. Badimon et J. Badimon, 1992*). This irregularity is in turn prerequisite for plaque rupture during abrupt changes in the regional haemodynamics or even in normal cardiac cycle. Using modeling of the vessel wall stress, *Richardson P., M. Davis et G. Born (1989)* interpret why in 2/3 of the incidents, the intima rupture occurs at the border between the normal endothelium and the pathologically changed one covering the atherosclerosis plaque. The aim of the present study is to contribute to a better understanding of the stress distribution within the most frequently occurring ruptures in coronary vessels using hydrodynamic model taking in consideration the stenosis geometry.

Model

The leading edge of the pulse wave of the blood flow in coronary vessel may be interpreted as fluid carrying with constant velocity through cylindrical tube. The hydrodynamic resistance related to unit vessel length is equivalent to the pressure on unit area of the vessel wall and is given by the expression (*Lightfoot E., 1974*):

$$P_1 = \lambda(L/d_1)(\rho V^2_m/2), \quad (1)$$

where (see Fig. 1) L is the vessel segment of interest subjected to the blood flow pressure, d_1 is the diameter of the non-changed vessel, V_m represents the mean volume velocity of the carried blood, ρ is the relative blood density (equal to 1.005, *Fry D.L., 1968*), λ is non-dimensional coefficient depending on the Reynolds' (Re) number by the relation $\lambda = 0.032 + 0.221/Re^{0.237}$.

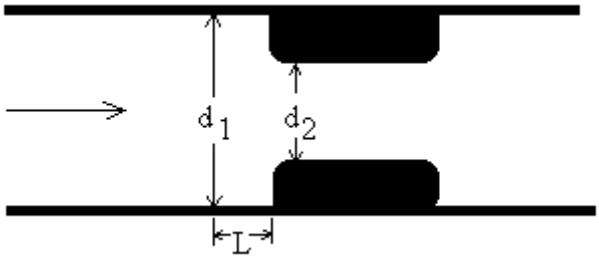

Fig. 1. Schematic image of vessel segment with plaque and the measured geometric distances.

The value of the local hydrodynamic resistance is determined by

$$P_2 = \xi(\rho V^2_m/2), \quad (2)$$

where ξ is the coefficient of the local resistance for a given vessel cross-section. In case of abrupt narrowing of the vessel lumen, ξ depends on the ratio of the cross-sections in the narrow (S_2) and the wide (S_1) part:

$$\xi = f(S_2/S_1) = f(d_2^2/d_1^2). \quad (3)$$

The values of ξ for a given ratio of cross-sections (or diameters) may be taken from tables (see for example *Lightfoot E., 1974*).

The conditions meeting the expression (3) lead to a border case of the plaque geometry – rectangular ('lock') profile of the wall plaque looking it at the side of the undamaged wall segment (Fig. 1). In this instance the expressions (1) and (2) allow the degree determination of the ongoing (pulsing) pressure increasing on the vessel wall in the close proximity of the narrowing. It represents the ratio of the pressures P_1 in the normal vessel and P_2 immediately after the narrowing:

$$P_2/P_1 = (\xi/\lambda).(d_1/ L). \quad (4)$$

Results

We used data obtained from 205 patients subjected to selective coronary angeography immediately after acute phase of myocardial infarct (112 cases) and unstable stenocardia III class according to *Braunwald E., 1989* (93 cases). The study was performed in the Clinic of Cardiology at the Medical University of Sofia. The group includes 162 men and 43 women with a middle age of 60.1±11.9 and 63.5±9.8 years, respectively. The diameters d_1 of the unchanged segment just close to the plaque and d_2 of the residual lumen of the stenosis segment are measured in all of the stenosis vessels. Since d_1 depends on the kind of the coronary vessels, their quantitative distribution is given in Table 1. Obviously, over 95% of the cases manifest a stenosis, which is relative to arterial vessels with diameter of their unchanged parts in the range of 1.5 through 6 mm (mean value 3.3±0.4 mm). The results have a good coincidence with these obtained by other researchers (see for example *Shefer S.E. et al, 2000; Drouet L., 2002*). For vessels with such diameter mean value, Re=200 and λ=0.066.

The mean diameter of the residual lumen is d_2=0.79 mm. These diameters of the narrowing, as well as the most frequently encountered coronary vessel diameters from 2.9 to 3.7 mm allow the determination of ξ using the relation (3). Assuming that the pressure of the unaffected coronary vessel segment is measured at 1 mm in front of the narrowing (L=1 mm), the ratio of the pressures P_1 in normal vessel and P_2 immediately after the narrowing may be obtained by (4) – see Fig. 2.

Affected vessel	Number	%
Common trunk of left coronary artery	9	4.4
Left anterior descending artery	106	51.7
Artery circumflex	37	18.0
Right coronary artery	53	25.9

Table 1. Distribution of the vessel stenosis types in the group of 205 patients

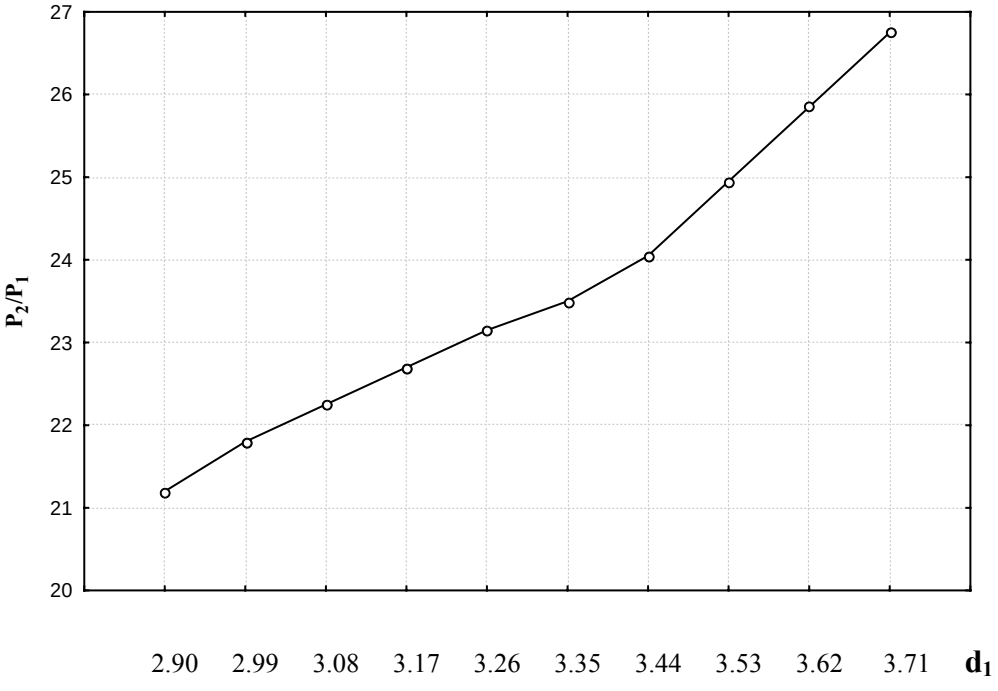

Fig. 2. Ratio of the vessel pressures segment immediately before (P1) and after (P2) the narrow with respect to coronary vessel diameter

Discussion and Conclusions

The references offer different hypotheses about the mechanisms, which provoke ruptures of the atherosclerotic plaques. Our results show that in cases of plaques with front, which is proper to the 'lock' type, the blood flow hydrodynamic characteristics in transition from normal to narrowed lumen change abruptly the forces operating on the vessel walls and the plaque front. These changes are particularly manifested at the systole beginning when the blood flow is still not broken off while the coronary vessel increases its circumferential stress

(*Fry D.L., 1968; Richardson P., M. Davis et G. Born, 1989).* For example, in case of vessel diameter about 3.5 mm the pressure on the plaque front exceeds 25 times the pressure on the vessel wall immediately before the plaque. One may expect that 'soft' plaques with detrited mass are considerably distorted in form on the side of the pulse wave and consequently are very often ruptured. *Shah P.K.* (*1997*) *and Foo R.S.Y. et D.P. De Dono* (*2000*) also accentuated on the predominated influence of the plaque content instead of its size when the risk of acute events have to be assessed. The contribution to the model of the vessel diameter has good arguments. The possibility of plaque transformation depends on the vessel wall pressure, which is determined in turn by perimeter (and consequently by the diameter) of the vessel (*Lightfoot E., 1974*). Longer perimeter leads to higher volume blood flow velocity. These agents determine more considerable increase of the pressure ratio in the transition area of the narrowing. Similar opinions based on practical monitoring are reported by *Von Birgelen C. et al* (*2000*) *and Drouet L.* (*2002*).

In conclusion, one may claim that the risk of rupture within coronary arterial plaques of 'lock' type increases with enlarging of the vessel size.

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