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CORONARY ARTERY OCCLUSION EXPLAINED BY MEANS OF A FRACTAL MODEL

BY

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Abstract. We prove through a fractal model that the blocking of the lumen of an absolutely healthy artery can happen as a result of the “stopping effect”, in the conditions of a normal sanguine circulation. Our fractal model was used for in vivo analyzes of ten clinical cases of patients with acute occlusive thrombus on an absolutely healthy artery. We present the two most relevant cases, with thrombus dimensions of 60 or more millimeters. Our theoretical results were verified by coronarography images.

Keywords: acute arterial occlusion; nonlinear dynamics; Bingham fluid; Scale Relativity Theory.

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1. Introduction

The acute arterial occlusion of an artery that has no significant preexistent lesions leads to dramatic consequences due to the lack of collateral substitutive circulation, as this kind of circulation usually develops within years, in the presence of hemodynamic significant stenosis (Hiatt *et al*., 2004).

Classical models which explain this phenomenon take into account the cracking of an intimal atheroma plaque, the activation of the pro-thrombogenic cascade through the denudation of the endothelium and the formation in certain circumstances of a completely occlusive thrombus (Badimon and Vilahur, 2014; Toney *et al*., 2014). At least one counterargument should be taken into consideration: why does an occlusive thrombus form so quickly in the absence of a stenosis, when the sanguine flux is unaltered? Why doesn't the "wash-out'' phenomenon appear?

Without contradicting these usual models, we will prove through a fractal model (Popa *et al*., 2015; Tesloianu *et al*., 2015) that the blocking of the lumen of an absolutely healthy artery can happen as a result of the "stopping effect" (even in the absence of the at least disputable cracked and non-protrusive atheroma plaque), in the conditions of a normal sanguine circulation.

2. Theoretical Model

If we consider blood a Bingham-type rheological fluid, then

$$\tau = \tau_0 + \eta \frac{dv}{dr} \tag{1}$$

where τ is the viscosity tangential unitary effort, τ_0 is the deformation tangential unitary effort, dv/dr is the velocity gradient with respect to the normal on the transversal section and η is the viscosity coefficient.

The mathematical procedure we used had the following steps:

i) determining the values of Reynolds' number for blood flow through the right coronary artery, using the following relation:

$$R_e = \frac{v_S D}{\eta} \tag{2}$$

where v_S is the minimum value of the average experimental systolic velocity of blood, D is the average experimental diameter of the right coronary artery, and η is the average kinetic viscosity coefficient of blood;

ii) determining the values of the loss coefficient of blood flow through the same artery, using Darcy's formula [6]:

$$\lambda = \frac{64}{\mathrm{R}_e} = \frac{64\eta}{v_S D} \tag{3}$$

iii) determining the values of the pressure loss for blood flow, using the following relation (Bar-Yam, 1997):

$$\Delta p = \lambda \frac{L}{D} \rho \frac{v_d^2}{2} = 32\eta\rho \frac{L}{D^2} \frac{v_d^2}{v_S} \tag{4}$$

where L is the average length of the experimental thrombus, ρ is the average experimental blood density, and v_d is the maximum value of blood's average experimental systolic velocity;

iv) determining the theoretical dimension of a right coronary artery thrombus, using the relation:

$$D_t = \frac{4\tau_0 L}{\Delta p} = \frac{1}{8} \frac{v_S \tau_0 D^2}{\eta\rho v_d^2} \tag{5}$$

where τ_0 is the average experimental deformation stress of blood (Axinte *et al*., 2014; Tesloianu *et al*., 2014).

3. Results

Our fractal model (Popa *et al.*, 2015; Tesloianu *et al*., 2015) was used for in vivo analyzes of ten clinical cases of patients with acute occlusive thrombus on an absolutely healthy artery. These cases were selected during a 2-year period (2013 – 2015). Patients with atrial fibrillation were excluded for preventing mismatch with thromboembolic acute coronary occlusion. Patients with patent foramen ovale (transesofageal echocardiodraphy study performed) were excluded in order to avoid a paradoxically coronary embolism. IVUS (intravascular ultrasound) or coronary angio CT were not performed for these patients; even if some irregularities could be seen at an angiography, it is clear that there are no significant ulcerated atheroma plaques or major signs of parietal atherosclerosis. Also, in patients older than fifty years an absolutely normal coronary wall is more likely a utopia. We had EKG holter monitoring in all patients for exclusion of paroxysmal atrial fibrillation.

We present here the two most relevant cases (Fig. 1), with thrombus dimensions of 60 or more millimeters (for the other eight cases, the thrombus dimensions were between 30 and 60 mm). Our theoretical results were verified by coronarography images.

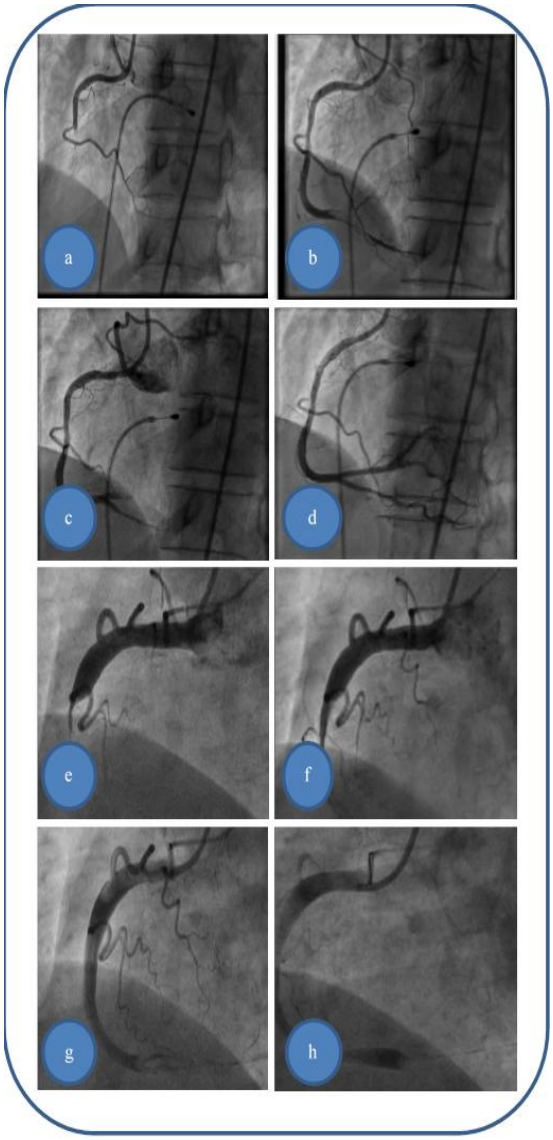

Fig. 1– Acute thrombus formation in apparently healthy artery with no evidence of plaque dissection like as a responsible lesion – different interventional approach stages: patient 1 (*a-d*), patient 2 (*e-f*).

i) Patient 1, 52 years old male patient, who was diagnosed with acute infer lateral ischemia; the coronary angiography revealed an acute occlusive thrombus (4-4.5 mm diameter and 60 – 80 mm length) at the junction between segments I and II of right coronary artery; after thrombus aspiration a distal thrombotic embolism appears with an apparently healthy artery (or possible

minimal lesion – no sign of plaque dissection) at the initial thrombus level; repeated thrombus aspiration at the level of secondary occlusion reveals the posterior descending branch and subsequently posterolateral branch; also, there was no evident coronary lesion responsible for the above stated pathological phenomena;

ii) Patient 2, 57 years old male patient who was diagnosed with acute inferior and poster lateral ischemia; coronary angiography revealed an acute occlusive thrombus extended from the beginning of right coronary arterysegment II to crux (4.5 – 5 mm diameter and approx. 80 – 100 mm length), possible with extension to right posterior descending artery and poster lateral branches; unsatisfying results in term of distal TIMI flow (0-1) but with no evidence of significant atherosclerotic disease at the level of culprit zone.

We present in Table 1 the average experimental parameters of blood flow through the right coronary artery, used in our study, and also the average theoretical parameters of blood flow through the right coronary artery, obtained using our theoretical model (Popa *et al*., 2015; Tesloianu *et al*., 2015).

Table 1
Average Experimental Parameters of Blood Flow Through the Right Coronary Artery for the Two Clinical Cases

Patient's age [years]	D_e [mm]	L [mm]	τ_0 [N/m^2]	v_d [cm/s]	v_S [cm/s]	ρ [kg/m^3]	η [m^2/s]
52	4	70	9/75 mm Hg	35 ± 11	24 ± 7	1060	3.04 x 10^{-6} at 36.5°C
57	5	90	7/83 mm Hg	35 ± 11	24 ± 7	1060	3.04 x 10^{-6} at 36.5°C
Observations			The method from (Sharif *et al*., 2015) was used	The method from (Sharif *et al*., 2015) was used	The method from (Malek *et al*., 1999) was used	The method from (Malek *et al*., 1999) was used	The method from (Sharif *et al*., 2015) was used

R_e	λ	Δp [N/m]	D_t [mm]
226	0.283	634	4.54
283	0.226	457	5.52

Legend: D – average experimental thrombus diameter; L – average experimental thrombus length; τ_0 – average experimental deformation stress as a function of diastolic pressure; v_d – average experimental diastolic velocity; v_S – average experimental systolic velocity; ρ – average experimental blood density; η – average experimental kinetic viscosity coefficient; R_e – Reynolds' number; λ – Darcy's loss coefficient; Δp – pressure loss; D_t – thrombus diameter determined using our model.

4. Conclusions

We can see a good conformity between the values from the theoretical model with the experimental/real estimated values (Hiatt *et al*., 2004; Tesloianu *et al*., 2015) in coronary angiography we found in the two cases presented above. Due to the fact that our model can be extrapolated to every cylindrical structure, in our opinion similar phenomena can occur, at least theoretically, in every artery of similar dimensions and hydrodynamic regimen (brain, kidney, splanchnic system etc.).

We note that the same model can also be applied, because of its theoretical implications, in engineering and materials science, in various domains, such as the ones described in (Agape *et al*., 2016; Agape *et al*., 2017; Gaiginschi and Agape, 2016; Gaiginschi *et al*., 2011; Gaiginschi *et al*., 2014a; Gaiginschi *et al*., 2014b; Gaiginschi *et al*., 2017; Vornicu *et al*., 2017).

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OCLUZIA ARTEREI CORONARIENE EXPLICATĂ PRIN INTERMEDIUL UNUI MODEL FRACTAL

(Rezumat)

Folosind un model fractal, se arată că ocluzia unei artere absolute sănătoase, în condițiile unei circulații sanguine normale, poate apărea ca urmare a acțiunii unui „opritor". Acest model a fost folosit pentru studierea in vivo a unui număr de 10 cazuri clinice de tromboză ocluzivă în artere absolute sănătoase. Prezentăm cele mai relevant două cazuri, cu dimensiuni ale trombusului de peste 60 mm. Rezultatele teoretice obținute sunt validate de imaginile angiografice.