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Editorial

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Short-Term Prognosis of Juvenile Myocardial Infarction: Role of Plasma Viscosity

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In our early research[1] regarding the hemorheological pattern in patients with acute myocardial infarction (AMI) with a mean age of 61.45 ± 10.99 years, we showed that the major hemorheological parameters were almost normalized 2 weeks after the acute event. In the last decade, we focused on hemorheological parameters in juvenile myocardial infarction (JMI), defined as AMI in patients aged ≤45 years, in the "Sicilian study on juvenile myocardial infarction".[2,3]

Juvenile myocardial infarction is responsible for 2% to 10% of all cases with AMI in different surveys.[4,5] Juvenile myocardial infarction presents a typical pattern of risk factors[6,7] and shows clinical, angiographic, and prognostic characteristics.[5] Regarding risk factors, cigarette smoking is by far the most common, followed by family history of coronary artery disease and hypercholesterolemia, while arterial hypertension and diabetes mellitus are less frequent. Juvenile myocardial infarction may be associated with the use of oral contraceptives, pregnancy, or cocaine abuse as well as with congenital coronary artery abnormalities. Concerning the clinical picture, patients with JMI reach hospital earlier than older patients, enhancing the effectiveness of revascularization procedures and treatment of complications. In JMI, the absence of coronary stenosis is often demonstrated or just 1 coronary vessel is affected; 2- or 3-vessel disease is infrequent. Generally, patients with JMI have a lower incidence of complications, such as early and late heart failure, angina, reinfarction, and atrioventricular block; mortality during hospitalization and after 6 months is significantly reduced.

In the Sicilian study, we observed a pattern of inflammatory polymorphisms in patients with JMI.[8] A higher prevalence of proinflammatory polymorphisms (SNP A2080G of pyrin gene, SNP Gly670Arg of PECAM gene, C1019T of Cx 37 gene, and SNP G1059C of PCR gene) and a lower prevalence of anti-inflammatory polymorphisms (Asp299Gly of TLR4 gene, SNP – 1082 G/A of IL10 gene, CCR5Δ32) were present.

We revisited plasma viscosity (PV) in our survey of patients with JMI and considered 2 aspects in particular. The first is the potential role played by PV in the dynamics of myocardial microcirculation (namely, in the phenomenon of coronary slow flow) that may be secondary to AMI[9-11]; the second aspect was life expectancy.

Plasma viscosity is dependent on the plasma protein concentration, although the contribution of different proteins (fibrinogen, α_2-macroglobulin, immunoglobulins, haptoglobin, and ceruloplasmin) differs in relation to their molecular size and shape; the plasma protein composition can also change due to pathophysiological processes.[12] The PV plays a pivotal role, together with erythrocyte deformability and platelets, in microcirculatory blood flow.[13]

In this editorial, we describe the behavior of PV in 120 patients with JMI (109 men; mean age 39.4 ± 5.8 years); the time interval between AMI onset and the first hemorheological evaluation was 13 ± 7 days. Using fasting venous blood, we measured PV at the shear rate of 450 s^{-1} using the cone-and-plate viscometer Wells-Brookfield mod ½ LVT (Middleboro, Massachusetts). We reexamined this parameter 3 (n = 83) and 12 (n = 70) months after AMI.

At the initial stage, PV was increased compared to controls (CS 1.259 ± 0.125 vs JMI 1.519 ± 0.108 mPa- s, $P < .001$). The PV did not differ between ST-segment elevation myocardial infarction (STEMI) and non-STEMI as well as in 3 subgroups of the patients with JMI subdivided according to the number of cardiovascular risk factors (39 had 0 or 1 risk factor, 39 had 2 risk factors, and 42 had ≥ 3). Coronary angiography was performed in 103 patients; no significant coronary stenosis was demonstrated in 23, 46 had a single vessel disease, and 34 had multi-vessel disease. The PV did not differ between the 3 subgroups. At 3 and 12 months after AMI, PV was persistently increased compared to controls (at 3 months 1.466 ± 0.119 mPa- s and at 12 months 1.475 ± 0.009 mPa-s, respectively).

Although the literature has described a low incidence of cardiovascular complications in JMI,[5] in our survey, follow-up carried out for as long as 18 months showed that 5 patients developed heart failure, 15 a new ischemic event (angina in 12

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and reinfarction in 3), and 4 had an ischemic event associated with heart failure. The drugs prescribed at hospital discharge were in agreement with current recommendations (antiplatelet agent in 96.69%, statin in 75.31%, and a beta-blocker in 64.45%); however, enrollment started in 2001 and at that time the use of 2 antiplatelet agents after AMI was not common.

Considering the number of patients who developed cardiovascular complications, we reexamined PV measured at the initial stage, in patients with JMI with (n = 24) and without (n = 74) cardiovascular events (CEs). The PV was initially higher in the subgroup that subsequently developed CE (JMI without CE 1.500 ± 0.107 vs JMI with CE 1.555 ± 0.094 mPa-s, $P < .05$).

Considering that several studies investigated the neutrophil to lymphocyte ratio (NLR) in patients with AMI not selected for age,[14,15] we subdivided our patients with JMI into 2 subgroups according to the median NLR value. In the subgroup with high NLR, PV was higher (JMI + low NLR 1.492 ± 0.097 vs JMI + high NLR 1.545 ± 0.113 mPa-s, $P < .01$, respectively).

While the subdivision of patients with JMI according to the NLR supports the role of PV as part of the acute-phase reaction,[16] its behavior in the patients subdivided according to short-term cardiovascular outcomes suggests an influence on coronary microcirculation. The regulation of myocardial microcirculation has been extensively analyzed,[17] and blood viscosity was shown to be linked to the hemodynamic profile in small and large vessels.[18] The association of a significantly increased PV with small-vessel occlusion was also described in ischemic cerebral disease.[19]

Some studies showed a link between PV and the risk of cardiovascular diseases[20,21] as well as a relationship between PV and the severity of coronary artery disease.[22,23] It has been recently observed that PV was positively associated with the incidence of cardiovascular and noncardiovascular death during the long-term follow-up of a male population.[24] Similar to what was described in patient with unstable angina,[25] transmural myocardial infarction,[26] and in no-reperfusion STEMI who had undergone primary coronary intervention,[27] PV also seemed to have a prognostic value in our survey. Therefore, the persistence of a hemorheological alteration can be detrimental to coronary hemodynamics, contributing to the phenomenon of secondary coronary slow flow.[9-11] Since the increase of PV in patients with JMI persisted after 12 months, monitoring the hemorheological profile and its possible response to drugs should be given further attention.

Declaration of Conflicting Interests

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References

1. Caimi G, Raineri A, Sarno A. Blood rheology in acute myocardial infarction. *Acta Diabetol*. 1982;37(6):401-409.
2. Caimi G, Hoffmann E, Montana M, et al. Haemorheological pattern in young adults with acute myocardial infarction. *Clin Hemorheol Microcirc*. 2003;29(1):11-18.
3. Caimi G, Valenti A, Lo Presti R. Acute myocardial infarction in young adults: evaluation of the haemorheological pattern at the initial stage, after 3 and 12 months. *Ann Ist Sup San*. 2007;43(2): 139-143.
4. Doughty M, Mehta R, Bruckman D, et al. Acute myocardial infarction in the young - The University of Michigan experience. *Am Heart J* 2002;143(1):56-62.
5. Imazio M, Bobbio M, Bergerone S, Barlera S, Maggioni AP. Clinical and epidemiological characteristics of juvenile myocardial infarction in Italy: the GISSI experience. *G Ital Cardiol*. 1998;28(5):505-512.
6. Choudhury L, Marsh JD. Myocardial infarction in young patients. *Am J Med*. 1999;107(3):254-261.
7. Friedlander Y, Arbogast P, Schwartz SM, et al. Family hystory as a risk factor for early onset myocardial infarction in young women. *Atheroschlerosis*. 2001;156(1):201-207.
8. Incalcaterra E, Caruso M, Balistreri CR, et al. Role of genetic polymorphisms in myocardial infarction at young age. *Clin Hemorheol Microcirc*. 2010;46(4):291-298.
9. Beltrame JF, Limaye SB, Wuttke RD, Horowitz JD. Coronary hemodynamic and metabolic studies of the coronary slow flow phenomenon. *Am Heart J*. 2003;146(1):84-90.
10. Fineschi M, Bravi A, Gori T. The "slow coronary flow" phenomenon: evidence of preserved coronary flow reserve despite increased resting microvascular resistances. *Int J Cardiol*. 2008; 127(3):358-361.
11. Leone MC, Gori T, Fineschi M. The coronary slow flow phenomenon: a new cardiac "Y" syndrome? *Clin Hemorheol Microcirc*. 2008;39(1-4):185-190.
12. Rampling MW. Compositional properties of blood. In: Baskurt OK, Hardeman MR, Rampling MW, Meiselman HJ, eds. *Handbook of Hemorheology and Hemodynamics*. Amsterdam, Netherlands: IOS Press; 2007: 34-44.
13. Lipowsky HH. Blood rheology aspects of the microcirculation. In: Baskurt OK, Hardeman MR, Rampling MW, Meiselman HJ, eds. *Handbook of Hemorheology and Hemodynamics*. Amsterdam, Netherlands: IOS Press; 2007: 307-321.
14. Balta S, Celik T, Mikhailidis DP, et al. The relation between atherosclerosis and the neutrophil-lymphocyte ratio [published online February 9, 2015]. *Clin Appl Thromb Hemost*. 2015.
15. Caimi G, Lo Presti R, Canino B, Ferrera E, Hopps E. Behaviour of the neutrophil to lymphocyte ratio in young subjects with acute myocardial infarction [published online September 25, 2015]. *Clin Hemorheol Microcirc*. 2015.
16. Késmárky G, Kenyeres P, Rábai M, Tóth K. Plasma viscosity: a forgotten variable. *Clin Hemorheol Microcirc*. 2008;39(1-4): 243-246.
17. Matschke K, Jung F. Regulation of the myocardial microcirculation. *Clin Hemorheol Microcirc*. 2008;39(1-4):265-279.

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18. Marossy A, Svorc P, Kron I, Gresová S. Hemorheology and circulation. *Clin Hemorheol Microcirc*. 2009;42(4):239-258.
19. Grotemeyer KC, Kaiser R, Grotemeyer KH, Husstedt IW. Association of elevated plasma viscosity with small vessel occlusion in ischemic cerebral disease. *Thromb Res*. 2014;133(1):96-100.
20. Koenig W, Sund M, Filipiak B, Döring A, Löwel H, Ernst E. Plasma viscosity and the risk of coronary heart disease: results from the MONICA-Augsburg Cohort Study, 1984 to 1992. *Arterioscler Thromb Vasc Biol*. 1998;18(5):768-772.
21. Vayá A, Simó M, Santaolaria M, Carrasco P, Corella D. Plasma viscosity and related cardiovascular risk factors in a Spanish Mediterranean population. *Thromb Res*. 2007;120(4):489-495.
22. Junker R, Heinrich J, Ulbrich H, et al. Relationship between plasma viscosity and the severity of coronary heart disease. *Arterioscler Thromb Vasc Biol*. 1998;18(6):870-875.
23. Lowe GD, Drummond MM, Lorimer AR, et al. Relation between extent of coronary artery disease and blood viscosity. *BMJ*. 1980; 280(6215):673-674.
24. Patterson CC, Blankenberg S, Ben-Shlomo Y, et al. Which biomarkers are predictive specifically for cardiovascular or for non-cardiovascular mortality in men? Evidence from the Caerphilly Prospective Study (CaPS). *Int J Cardiol*. 2015;201:113-118.
25. Neumann FJ, Katus HA, Hoberg E, et al. Increased plasma viscosity and erythrocyte aggregation: indicators of an unfavourable clinical outcome in patients with unstable angina pectoris. *Br Heart J*. 1991;66(6):425-430.
26. Sargento L, Do Rosário HS, Perdigão C, Monteiro J, Saldanha C, Martins e Silva J. Long-term prognostic value of the hemorheological profile in transmural myocardial infarction survivors: 60-month clinical follow-up. *Rev Port Cardiol*. 2002;21(11): 1263-1275.
27. Wasilewski J, Turczyński B, Słowińska L, Kowalik V, Osadnik T, Poloński L. Haemorheological factors and myocardial reperfusion in patients with ST-elevation myocardial infarction undergoing primary coronary intervention. *Kardiol Pol*. 2007;65(7):778-785; discussion 786-787.

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