Comments: "Association between Hemogram Parameters and Coronary Collateral Development in Subjects with Non-ST Elevation Myocardial Infarction"

Mehmet Ali Kosekli[1]

1. AIB University, Medical Faculty, Bolu, Turkey
Email: kosekli@gmail.com

http://dx.doi.org/10.1590/1806-9282.66.4.559

The severity of atherosclerotic heart diseases can be predicted by coronary collateral circulation, which is a consequence of coronary collateral development (CCD). Subjects who suffer from coronary artery disease with adequate CCD can benefit from an alternative blood supply of collateral vessels[1]. Cardiac morbidity and mortality rates are lower in patients with sufficient CCD compared to those without adequate CCD, since these collateral vessels make a natural arterial bypass[2].

Laboratory and clinical determinants of CCD are a matter of debate. While statin use has been supposed to be related to better CCD[3], increased inflammatory markers, such as c-reactive protein, are suggested to be associated with poor CCD[4]. Interestingly, anemia was also related to better development of coronary collateral circulation independent from erythropoietin level[5].

In the article by Sincer et al.[6], the authors reported that elevated platelet distribution width (PDW) was associated with better development of coronary collateral vessels. PDW is a marker of thrombocyte activation and refers to the size variability of circulating platelets. It has a potential role in the pathogenesis of myocardial infarction[7]. Moreover, the authors found that it was associated with type 2 diabetes mellitus and diabetic nephropathy[8]. Sincer et al.[6] evaluated the grade of CCD by Rentrop grading, in which grade 0 and 1 were classified as insufficient CCD, and grade 2 and 3 were classified as sufficient CCD. Their study design and interpretation of the results are justified. Increased PDW, as a result of increased platelet activation, could promote angiogenesis and better development of coronary artery vessels.

In conclusion, due to its nature of being easy to assess and repeat, increased PDW could be a promising predictor of CCD in patients with coronary heart disease.

REFERENCES

1. Ajayi NO, Vanker EA, Satyapal KS. Coronary artery dominance dependent collateral development in the human heart. Folia Morphol (Warsz). 2017;76(2):191-6.
2. Kodama K, Kusuoka H, Sakai A, Adachi T, Hasegawa S, Ueda Y, et al. Collateral channels that develop after an acute myocardial infarction prevent subsequent left ventricular dilation. J Am Coll Cardiol. 1996;27(5):1133-9.
3. Pourati I, Kimmelstiel C, Rand W, Karas RH. Statin use is associated with enhanced collateralization of severely diseased coronary arteries. Am Heart J. 2003;146(5):876-81.
4. Gulec S, Ozdemir AO, Maradit-Kremers H, Dincer I, Atmaca Y, Erol C. Elevated levels of C-reactive protein are associated with impaired coronary collateral development. Eur J Clin Invest. 2006;36(6):369-75.
5. Özyüncü N, Gülec S, Özdöl Ç, Candemir B, Ongun A, Tulunay Kaya C, et al. Impact of serum erythropoietin level on collateral vessel development in patients with coronary artery disease. Anatol J Cardiol. 2017;17(5):386-91.
6. Sincer I, Mansiroglu A, Gunes Y, Aktas G, Kocak M. Association between hemogram parameters and coronary collateral development in subjects with non ST elevation myocardial infarction. Rev Assoc Med Bras. 2020;66(2):160-165
7. Cetin M, Bakirci EM, Baysal E, Tasolar H, Balli M, Cakici M, et al. Increased platelet distribution width is associated with ST-segment elevation myocardial infarction and thrombolysis failure. Angiology. 2014;65(8):737-43.
8. Atak BM, Duman TT, Aktas G, Kocak MZ, Savli H. Platelet distribution width is associated with type 2 diabetes mellitus and diabetic nephropathy and neuropathy. National J Health Sci. 2018;3(3):95-8.