Cardiovascular Protections in Severely Impaired Hemostasis

To the Editor:

There is mounting evidence that severely impaired hemostasis is associated with a decreased risk of ischemic cardiovascular disease. In a study with long follow-up, hemophilic patients had an 80% reduction in the risk of fatal ischemic heart disease.1 In clinical practice, occurrence of cardiovascular disease is uncommon in patients with type 3 severe von Willebrand disease, a coagulation disorder in which the severity of bleeding may sometimes resemble that in patients with hemophilia.

However, Šrámek et al2 reported that patients with type 3 von Willebrand disease were not protected against the development of early atherosclerosis changes as assessed by measuring carotid and femoral intima-media thickness, thus suggesting that von Willebrand factor is not essential for the development of atherosclerosis. Therefore, in impaired hemostasis, other cardiovascular protective mechanisms could be involved.

An epidemiological study3 found that serum ferritin, a good measurement of body iron stores, emerged as one of the strongest risk factors for progression of carotid atherosclerosis as assessed by measuring carotid and femoral intima-media thickness, thus suggesting that von Willebrand factor is not essential for the development of atherosclerosis. Therefore, in impaired hemostasis, other cardiovascular protective mechanisms could be involved.

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Although ferritin levels have not been shown to be associated with carotid intima-media thickness,4,5 body iron stores may promote atherosclerosis at a stage beyond intima-media thickening. Thus, the protection against ischemic cardiovascular disease in individuals with impaired hemostasis might be related to the decrease of stored tissue iron caused by recurrent bleeding.

Luca Mascitelli, MD
Cardiology Service
Casa di Cura “Città di Udine”
Udine, Italy

Francesca Pezzetta, MD
Cardiology Service
Ospedale di Tolmezzo
Tolmezzo, Italy

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Luca Mascitelli and Francesca Pezzetta

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