Is increased homocysteine level a false trail or an accomplice to saphenous venous graft degeneration?

Although previous data revealed superiority of arterial graft to saphenous venous graft (SVG) in terms of late patency, a recent large-scale study showed comparable 5-year outcomes with arterial and SVG conduits as second graft (1). Therefore, any exploration of causative factors affecting occlusive disease in SVG is clinically highly relevant.

The article in this issue of The Anatolian Journal of Cardiology entitled "Plasma homocysteine levels are related to medium-term venous graft degeneration in coronary artery bypass graft patients" by Balogh et al. (2) shows results of a study regarding association of homocysteine (Hcy) levels with progression of graft disease in patients who underwent coronary artery bypass graft surgery >1 year prior to index angiography. It was concluded that Hcy levels are independent predictors of progression of graft disease, suggesting that Hcy should be taken into consideration for prognosis of these patients.

As the authors emphasized, accelerated graft disease in SVGs is associated through interactions of systemic and local factors. Systemic risk factors [i.e., cigarette smoking, hyperlipidemia, triglyceridemia, and lipoprotein (a)] have been already reported to play a role in the formation of diffuse graft atheromas (3). Based on some data, elevated blood Hcy level is also a possible promoter of SVG degeneration.

The assessment of potential role of a certain element in a multifactorial process can be challenging. In theory, all participating factors must be explored and should be incorporated into the statistical evaluation as explanatory variables. A limitation of the study described in the article by Balogh et al. (2) is that this goal was not completely achieved because local flow conditions were not analyzed as a contributor.

It had been previously reported that flow interference due to competitive flow through native coronary artery (4) and discrepancy between run-off tract and graft diameter is prone to development of SVG disease. Shear stress associated with low flow velocity in the graft (5) was proposed as an important factor in the pathological process. On the other hand, it is generally accepted that in cases of high flow condition, SVG can remain patent in very long term (6). This observation is well documented in the article in this issue in the representative case (Fig. 2), which shows presence of different degrees of stenosis in different grafts in the same patient (2). It also serves proof that the same risk profile in 1 patient can result different outcomes in SVGs according to local flow conditions.

In previous studies it has been reported that endothelial cells of SVGs subjected to arterial flow triggered release of atheroprotective vasoreactive mediators such as nitric oxide and prostaglandin (7, 8), which could support the conclusion of the authors: "Long-term SVG degeneration shows correlation with the elevated plasma total Hcy … while in cases with intact SVGs, the beneficial local flow conditions may protect the grafts from degeneration," (2).

Nonetheless, confirmation of this hypothesis requires further investigations with larger patient populations incorporating analysis of local flow conditions in SVGs.

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