Hyperhomocysteinemia and Left Ventricular Thrombus

The Editor,

We read with great interest the article – Left ventricular mass: A tumor or a thrombus diagnostic dilemma, describing the narrated differential diagnosis.[1] Left ventricular thrombus formation is commonly observed in patients with myocardial infarction with systolic dysfunction. However, it becomes difficult to come to the certain diagnosis when such situation occurs in patients with absent history of coronary artery disease and normal ventricular function.[2] While considering various hypercoagulable conditions, hyperhomocysteinemia should be evaluated, especially in the Indian context.

Hyperhomocysteinemia is an important independent risk factor for cardiovascular morbid events.[3] The prevalence of hyperhomocysteinemia in Indian population is reported to be 52%–84%.[4] Incidence of homocystinuria, all over the world, varies between 1 in 50,000 and 1 in 200,000. Mutations in the genes for enzymes involved in homocysteine metabolism – cystathionine-beta-synthase, methylenetetrahydrofolate reductase, or methionine synthase – cause raised level of homocysteine in the blood.

In hyperhomocysteinemia, this amino acid undergoes auto-oxidation to produce oxygen-free radicals, which can promote atherosclerosis. It also inhibits the antithrombotic action of thrombomodulin. This in turn activates protein C by thrombin. Both these actions lead to thrombosis in patients with hyperhomocysteinemia. Hyperhomocysteinemia has been independently associated with left ventricular thrombus formation. This condition can also present as left atrial thrombus and left ventricular mass in patients with sinus rhythm.[5] Left ventricular thrombus formation has also been reported in patients with normal left ventricular dimensions and systolic function due to hypercoagulable states such as raised homocysteine level.[6] In study of patients with acute anterior myocardial infarction following thrombolytic therapy, hyperhomocysteinemia independently raises the risk for the development of left ventricular thrombus.[7] In prospective investigational study of 936 patients, hyperhomocysteinemia predicts cardiovascular mortality and low left ventricular ejection fraction in hypertensive patients independent of coronary artery disease and history of myocardial infarction. Such left ventricular systolic dysfunction itself becomes significant risk factor to develop left ventricular thrombus.[8]

It is essential to consider homocysteinemia as important differential diagnosis in patients with cardiac thrombosis. Thrombectomy can solve the problem temporarily; however, along with proper anticoagulation, such patients require administration of Vitamin B12, Vitamin B6, and folate regardless of the levels before treatment to reduce cardiovascular risk further. Folic acid and Vitamin B12 along with pyridoxine (100–500 mg/day) are prescribed. These vitamins provide clinical benefit by effectively reducing the homocysteine levels. Antiplatelet agents or anticoagulants can be suggested for secondary stroke prevention. Patients who are pyridoxine insensitive are advised low-methionine diet with betaine supplementation. Effectiveness of the treatment can be monitored by measuring homocysteine levels.[9] Early diagnosis of homocysteinemia and prophylactic dietary and medical care plays a key role in reducing complications and improving long-term prognosis.

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Conflicts of interest

There are no conflicts of interest.

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