Unusual etiology of acute myocardial infarction – factor V Leiden mutation

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Factor V Leiden is the most common cause of inherited thrombophilia in Caucasian populations, accounting for 40-50% of cases. The prothrombin gene mutation, deficiencies in protein S, protein C, and antithrombin account for most of the remaining cases.

Clinically, the condition is characterized by the presence of deep venous thrombosis, with or without associated pulmonary embolism, and an increased risk of thrombosis of the cerebral veins. No relationship has been established between the presence of factor V Leiden and arterial thrombosis, but this association is present with antiphospholipid syndrome.

We present a 30 y/o gentleman, with no cardiovascular risk factors, no drug abuse and no recent emotional stress was admitted to the hospital complaining of acute chest pain started 3 hours before admission. A diagnosis of acute anterior STEMI was made based on EKG findings (figure 1) and primary PCI was done. His physical examination showed regular S1, S2 and S4 and the rest of the examination was normal.

Echocardiography showed akinsia at apex and mid anteroseptal with large apical LV thrombus, no valvular lesions. (figure 1). The angiogram showed normal RCA, CX coronary arteries and total occluded of mid LAD (figure 1).

Intervention was done with manual large thrombus removal then IVUS screen showed no significant residual LAD lesion. Lab tests were significant for positive heterozygous factor V Leiden mutation.

Myocardial infarctions with normal (or near-normal) coronary arteries account for 1% to 12% of cases. A possible mechanism is occlusion of the vessel lumen by thrombus that is subsequently rapidly lysed. Etiological factors that have been reported include cocaine use, embolism, coronary endothelial dysfunction and hypercoagulable states.

We presented a case of acute myocardial infarction induced by arterial thrombosis with normal coronary arteries, has a large LV thrombus and the document etiology of thrombosis was heterozygous factor V Leiden mutation.

An association between factor V Leiden and arterial disease remains controversial, although there appears to be a weak but significant association between premature myocardial infarction (ie, first event before age 45) and factor V Leiden. There

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are a number of studies, including two meta-analyses, which have generally been unable to find an increased prevalence of the mutation in patients with myocardial infarction (MI). However, it is not clear whether there is a small risk that is amplified considerably when there are additional coronary risk factors present.

In a case control study in young women (18 to 44 years of age), the factor V Leiden mutation was associated with a 2.4-fold increase in risk of MI after adjustment for age, this increase in risk was limited to current smokers.

In a second study, factor V Leiden was found in 12 percent of young patients (mean age 44 years) with MI and normal coronary angiography, in 4.5 percent of age- and sex-matched patients with MI and significant coronary artery disease, and 5 percent of normal controls (p=0.02).

In the third study, the case-control study involved 1210 patients who had survived a first myocardial infarction at an age of <45, presence of eight prothrombotic gene polymorphisms, including factor V Leiden, was not associated with an increased or decreased risk of premature MI.

According to the previous data, the association between factor V Leiden and arterial disease need more prospective studies, and the real fact that we face these cases more frequently in our daily practice (myocardial infarction induced by arterial thrombosis with normal or near normal coronary artery) and in few cases there is no clear explanation about the etiology of disease.

References
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