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Is Turkey a prothrombin gene mutation region similar to the Mediterranean countries?

To the Editor,

Myocardial infarction (MI) is a leading cause of morbidity and mortality worldwide (1). Acute MI generally develops following a critical narrowing of the coronary artery or a narrowing or complete occlusion of the coronary vessel by an acute plaque rupture (2). MI in young adults may be categorized into two groups as normal coronary artery anatomy and coronary artery disease (CAD) accompanied by various etiologies; moreover, conditions associated with hypercoagulopathy play a significant role in the pathophysiology of both groups (3).

We examined 68 patients (aged <45 years) with ACS and 69 healthy controls for hypercoagulable states in our institution between January 2008 and June 2010. We found a statistically significant difference between the groups for factor V Leiden (FVL), whereas there was no statistically significant difference for prothrombin gene mutation (P G20210A).

The two most common reasons of familial thrombophilia are P G20210A and FVL. P G20210A is frequently observed in Southern European countries and most notably in countries that have coast to the Mediterranean (4). Despite conflicting results, some studies have demonstrated that the combination of known risk factors and P G20210A is a risk factor for the development of arterial thrombus and ACS (5). In our study, there was no statistically significant difference between the patient and control groups (2.9% vs. 1.4%, p=0.551). P G20210A was found to be heterozygotic in three (2.2%) among a total of 137 cases. However, in the study by Akar et al. (6), P G20210A prevalence rate in Turkey was reported to be 6.2%, which is similar to the rate in Mediterranean countries; however, this finding is contradictory to our study findings. Despite being a Mediterranean country, Turkey is located right in the middle of three continents and has a distinctive geography. Therefore, FVL mutation prevalence rather than P G20210A may be more frequent, particularly in the Central Anatolian, Eastern Anatolian, and Black Sea Regions, which is similar to that observed in the Northern European countries.

Data regarding the association of FVL mutation with the development of CAD and ACS are conflicting. However, large studies investigating young patients with ACS have reported that FVL mutation was found to be statistically significant (7). Similarly, we found in our study that FVL mutation was statistically significant in the patient group compared with that in the control group (22.1% vs. 5.8%, p=0.006).

In conclusion, patients with ACS carrying FVL mutation might have a role in the pathophysiology of developing ACS. Furthermore, Turkey appears as a FVL mutation region rather than a P G20210A mutation region, which is similar to the Northern European countries, thereby opposing the known current literature. However, further prospective controlled studies in larger patient populations with careful analysis of other risk factors and mutations are required to understand the pathophysiological process of ACS.

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DOI:10.14744/AnatolJCardiol.2016.6565