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Lack of Association between Recurrent Pregnancy Loss and Inherited Thrombophilia in Hispanic Patients from Colombia</PRE>.

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Abstract

Introduction: Several genetic defects of coagulation factors have been implicated as a possible cause of recurrent pregnancy loss in Caucasians. The role of inherited thrombophilia as a risk factor in populations of Hispanic origin affected with this clinical condition is unknown. To our knowledge, this is the first study conducted to evaluate this genetic predisposition in Hispanics.

Objective: To assess association between recurrent pregnancy loss and inherited thrombophilias: factor V G1691A (FV Leiden), prothrombin G20210A (FII G20210A), methylenetetrahydrofolate reductase C677T (MTHFR C677T), activated protein C resistance (APC resistance), and deficiencies of antithrombin III (AT-III) and protein C (PC).

Patients and methods: This ongoing case-control study investigates a tri-ethnic population of Hispanic origin from Medellin, Colombia. Inherited thrombophilia was studied in 76 recurrent pregnancy loss patients according to Sixth ACCP Consensus Conference on Antithrombotic Therapy (three or more miscarriages, and either second-trimester losses or gestational vascular complications). The control group included 117 healthy women (two or more children, and no more than one miscarriage). Polymorphisms were genotyped by PCR-RFLP. APC resistance and deficiencies of AT-III, and PC were evaluated using commercial kits (IL Test[™] APC[™] Resistance V, Antithrombin[™], and Proclot[™]). Sample

size of 100 patients and 200 controls was determined to have 80% statistical power to discriminate association.

Results: The prevalence of any inherited thrombophilia in this patient cohort was 17%, and 25% in controls (OR 1.16, CI 0.6–2.29). No statistically significant differences in any genetic thrombophilia frequency between patients and healthy controls were observed. FV Leiden and FII G20210A were both positive in one patient and one control (OR 1.55, CI 0–57.5, for both thrombophilic defects). In the patient group 13.2% homozygous carriers with MTHFR 677T were found, as compared to 22.2% among controls (OR 0.53, CI 0.22–1.25). The odds ratio for the association between recurrent pregnancy loss and APC resistance was 0.77 (CI 0.32–4.2). The inheritance of AT-III deficiency or PC deficiency was not associated with recurrent pregnancy loss. AT-III deficiency was not detected in patients and was found in only one control. Furthermore, one patient was defined as PC deficiency carrier while none were found in the control group.

Conclusion: Our preliminary results found no association between recurrent pregnancy loss and inherited thrombophilia in this population originated by admixture of Amerinds, Europeans, and Africans, such as the American population denominated Hispanic. Base on our current data analysis, we do not expect to find any association even with the planned larger sample size. This suggests that inherited thrombophilia might not play a main role in Hispanic populations affected with this clinical condition. Given these results, appears to be insufficient evidence to include inherited thrombophilia in the initial evaluation of recurrent pregnancy loss in this population group, and possibly Hispanic patients in America. We suggest it is important to look for other, more common, causes of recurrent miscarriage in the evaluation of this group of patients. These data suggest an important ethnic difference between this population and Caucasians.

Author notes

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