LETTERS TO THE EDITOR

On the Association Between Recurrent Venous Thromboembolic Disease and Hyperhomocysteinemia

To the Editor: Pulmonary embolism is generally caused by the migration of a thrombus located in the deep venous territory of the lower limbs. It is highly prevalent and has high morbidity and mortality. Recurrent venous thromboembolic disease (VTD) is a complex problem requiring an exhaustive multidisciplinary approach. The most common genetic risk factor for VTD affecting the Caucasian population is factor V Leiden, a genetic risk factor for VTD affecting the multidisciplinary approach. The most common problem requiring an exhaustive complex metabolic and hematologic workup.

The patient was a 57-year-old man who was an active smoker with no clinical or functional signs of chronic obstructive pulmonary disease. He reported a 3-day history of dyspnea and pleuritic chest pain in the left hemithorax, followed a few days later by pain in the right hemithorax. His clinical history included several recurring episodes of deep venous thrombosis and pulmonary thromboembolism 2 years before the present episode. He had been treated with oral anticoagulants, which he had stopped taking some months earlier. Examination showed the patient to be hemodynamically stable and cyanotic, breathless at rest, with a respiratory rate of 30 breaths/min. Heart sounds were normal; chest auscultation revealed a soft murmur in both hemithoraces, with coarse basilar sounds were normal; chest auscultation revealed a small pleural effusion. Computed tomography revealed shallow breathing, left basal pulmonary S1Q3T3 pattern in leads II-III. A chest radiograph revealed a condition associated with genetic causes and with deficiencies of folic acid and vitamin B₁₂. HHC has been considered a vascular risk factor (atherogenic and thrombogenic) ever since the studies by McCully in 1969 (mentioned in Fernández Miranda et al.), although much doubt currently surrounds this association. HHC is defined as a cause of VTD in adults, there being few studies on young people. Some studies have tried to establish a relationship between the C677T MTHFR polymorphism and the risk of developing VTD, although results have been inconsistent. The diagnosis in our patient, who had a history of recurrent VTD, was confirmed by the high level of homocysteine and the presence of the C677T MTHFR polymorphism, and both the response to treatment and the outcome were good. In young people, however, these abnormalities have not been established as a risk factor for recurrent VTD.

The recently published results of a meta-analysis involving different populations with VTD (including young patients) revealed a relationship between HHC and VTD. However, attempts to analyze the relationship between VTD and the C677T MTHFR polymorphism have revealed it to be weak; therefore, the mutation cannot be shown to be the risk factor for developing VTD. More light has been shed by the results of a prospective study by Frederiksen et al (cited in Fernández Miranda et al and Wald et al) with 9238 patients followed for 23 years, which did not reveal a greater risk of VTD among the homozygous population with HHC. Research currently under way might remove many doubts. Studies confirming that a reduction in homocysteine levels is associated with a fall in vascular morbidity and mortality are necessary.

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