Pregnancy, Antithrombin III Deficiency and Venous Thrombosis: Report of Another Case

A. Alessandro Megha
G. Giuseppe Finzi
T. Tullio Poli
C. Cesare Manotti
A. Anton Giulio Dettori

Department of Medicine, 5th Medical Division, Regional Hospital of Parma, Italy
Prof. A.G. Dettori, 5a Divisione Medica, Ospedale Regionale I-43100 Parma (Italy)

Congenital antithrombin III (AT III) deficiency, either quantitative or qualitative, significantly increases the state of hypercoagulability characteristic of pregnancy (from 0.02 to 0.3% of ‘normal’ pregnancy to 70% and over) [1-3]. In pregnant women with AT III deficiency, prophylactic measures must consider risks and benefits of various medical treatments [4, 5].

A young woman was admitted to our Department for a sudden occlusion of the proximal deep venous stream of the right lower limb, and a contralateral recurrence within a few days, during the tenth week of pregnancy. Diagnosis was made by strain-gauge plethysmography [6]. We found she had a type I congenital AT III defect (AT III activity: 35%; AT III antigenic level: 40%), never diagnosed before.

We treated the patient with continuous intravenous infusion of sodium heparin (35,000-40,000 IU/ day, according to the aPTT ratio) and intravenous human AT III concentrate (2,000 IU followed by 1,000 IU/day, in order to keep AT III haematic level steadily above 60%). At the end of the 13th week of pregnancy, heparin and AT III infusions were discontinued, and acenocoumarol was administered per os, at a dosage sufficient to maintain an adequate deagulation level (INR above 3). The patient was then discharged.

Two weeks before the expected date of delivery, the patient was hospitalized again. Atenocoumarol was stopped, and replaced by sodium heparin, through continuous intravenous infusion (40,000 IU/ day). Fifteen days later the patient went into labour; sodium heparin was discontinued, and 12,000 IU of AT III were administered; prophylactic subcutaneous calcium heparin (5,000 IU every 12 h) was started; 6 h later caesarean section was performed, because of initial signs of fetal suffering. A male baby in good health was delivered (he has AT III congenital deficiency, type 1).

The mother was treated with AT III infusion for another 12 days, at a dosage of 1,000 IU/day, without discontinuing prophylactic subcutaneous calcium heparin. Eight days after delivery, acenocoumarol therapy was started again. We had neither bleeding nor thromboembolic complications, and believe that such a treatment program may be recommended.

References