Pregnancy in a patient with essential thrombocytosis

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Summary

An insurgent case of pregnancy in a patient in whom essential thrombocytosis was diagnosed five years earlier is described. Pregnancy was confirmed and therapy with platelet aggregation inhibitor was introduced. The pregnancy reached full term notwithstanding a positive result of the “Triple Test” during the 15th week of gestation. A histology exam of the placenta revealed an ischemic lesion. We retain that platelet aggregation inhibitor therapy remains an important aid in eliminating the risk of thrombosis determined by the presence of two conditions that are predisposed to these risks, such as pregnancy and essential thrombocytosis.

Key words: Essential thrombocythemia; Pregnancy; Antiaggregant therapy; Myeloproliferative disorder; Thrombocytosis.

Introduction

Essential thrombocytosis is a disease that, as a casual report of hematochemical routine tests, has been increasingly diagnosed in young asymptomatic patients in the last few years [5].

This fact explains why the association of this pathology with pregnancy is more frequently observed nowadays than it was in past years. Indeed, as thromboembolism complications frequently occur in essential thrombocytosis [8], and represent possible manifestations during pregnancy due to intrinsic changes in hematostatic mechanisms, it can be understood how the two conditions, if associated, can increase such a risk and how consistent antiaggregation thrombocyte therapy can reduce it [3].

We report here the case of a twenty-nine year old woman to whom an essential thrombocytosis was casually diagnosed five years before she became pregnant. The thrombocytosis was always asymptomatic and any pharmacological treatment, until conception, was used.

Case report

Essential thrombocytosis is a myeloproliferative disorder characterized by a persistent increase in the absolute number of thrombocytes (generally over 600x10⁹/L) caused by proliferation of a single staminal cell.

The clinical manifestations of this pathology occurring more frequently are thromboembolic, bleeder and vasomotor symptoms, but, as reported by some studies, it is often diagnosed casually in patients who are asymptomatic. Patients normally more affected by this pathology are in middle-advanced age, but today there has been an increase of peak in young subjects [7].

In our patient, essential thrombocytosis was diagnosed in 1989 as a result of a routine hematoochemical test in which a number of thrombocytes 829x10⁹/L, as well as the presence of neutrophilic leukocytosis (WBC 11.0x10⁹/L with 89% neutrophilis) then reduced, were discovered.

Medullary aspiration revealed hyperplasia of negakaryocytes. The osteomedullary biopsy revealed an abundance of white corpuscles in addition to a diffused increase in the number of megakaryocytes. No sign of fibrosis was revealed however, The karyotype test showed no chromosme aberration in the tested cells and was poarticularly unsuccessful in the search for Philadelphia chromosome. The leukocyte alkaline phosphatase, hemorrhagic time, prothrombin time, fibrinogenopenia and PTT all resulted normal. The hemocytometer revealed no anemia.

The thrombocyte aggregation changed and the dosage of thrombocyte endonucleotides was at the normal lower limits. It was decided that no therapy would be undertaken. The woman remained asymptomatic with a high number of blood platelets untilte the pregnancy was established in February 1994. The woman last menstruated on December 30th 1993.

When the pregnancy was established a therapy with acetylsalicic acid at a dosage of 100 mg a day was started. During the first months of pregnancy the number of thrombocytes remained high. After the first term (three months) the number of thrombocytes reduced considerably to values ranging from 450 to 600x10⁹/L.

During the pregnancy two important events occurred. Around the fifteenth week of gestation the Triple Test (dosage assessment of alphafetoprotein, estriol, chorionic gonadotropin) resulted positive. Consequently an amnecocentesis was performed. This however resulted negative.

The pregnancy then ran its course until the thirty fifth week when the scan showed a slowing down of the growth of the foetus. Despite this, the pregnancy ended happily. The woman gave birth as scheduled, to a normal baby weighing 3.000 Kg. The placenta histologic test showed an ischemic lesion. Puerperium was normal and an increased number of blood platelets was observed. These values, and those of the leukocytes returned to similar levels of those observed before the pregnancy.

Discussion

We have observed a young woman who had never taken any pharmacological therapy; her pregnancy, ana logically to what can be seen from recent studies, produ-
ced a positive reduction of thrombocytes probably due to
dilution [6].

Secondly, the evidence of a placenta infarct emphasizes
the importance of maintainancy consistent antiaggregation
therapy during the whole course of pregnancy in
order to at least reduce the possibility and gravity of the
thromboembolic phenomena [1, 2, 4, 9].

This information is further strengthened by recent
studies which point out a sharp reduction in the number
of abotions as well as a greater number of living and
normal fetus at birth in subjects treated with antiaggregating
thrombocyte medicines compared to those who did
not undergo any treatment [10].

We can then conclude that, although pregnancy and
essential thrombocytosis, both have thrombocytosis
potentiality, they can determine a fetus-placenta patho-
logy. Appropriate antiaggregating thrombocyte therapy
can reduce such a risk and make a normal pregnancy pos-
sible.

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