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# Splenectomy for Thrombocytopenic Purpura in Pregnancy

## *Report of a Case*

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**H**EMORRHAGE during pregnancy is always a severe complication. Hemorrhage in pregnancy due to thrombocytopenic purpura becomes a most serious complication. Hemorrhage during pregnancy due to thrombocytopenic purpura that does not respond to medical treatment is a frightening prospect. The report below is the thirteenth of a case of splenectomy for thrombocytopenic purpura in a pregnant woman.<sup>1, 3, 10, 11</sup> It is the second such instance in which the platelet count fell to zero before splenectomy.<sup>9</sup>

Thrombocytopenic purpura is an unusual disease.<sup>2, 5, 12</sup> Its occurrence coincidental with pregnancy is even more unusual. Fewer than 100 cases have been recorded. The combination of these two conditions brings to the fore many questions which must largely be answered on the basis of each individual case.<sup>4, 6, 11</sup> The initiation of pregnancy, the actual onset of the thrombocytopenia, the amount of bleeding, and the response to therapy all have bearing on the management of the individual case.<sup>7-9</sup>

Our patient is one in whom the purpura developed during pregnancy. Severe epistaxis at Week 25 of gestation was the first clinical manifestation of the problem in this patient.

### CASE REPORT

The subject was a 22-year-old primigravida with no previous medical history that related to bleeding with the exception of a D&C at age 17. No blood studies were done at that time and there was no indication of the etiology of the bleeding, except that the endometrium was noted to be proliferative. There were no bleeding problems during early pregnancy.

Initial studies showed a hemoglobin level of 13 gm. and a hematocrit of 38%. The patient's blood type was A, Rh positive. Pregnancy was progressing normally until the patient had a severe nosebleed. Detailed examination of her nose showed a chronic perforation of the septum; the cause was unknown. The initial nosebleed responded to cauterization, but 48 hr. later the patient bled again. A postnasal pack was inserted; after it was removed the patient continued to bleed to an alarming degree. On hematologic examination the diagnosis of thrombocytopenia was established. Platelet counts were initially 22,000/cu. mm. The bleeding time was normal to slightly increased.

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## SPLENECTOMY IN PREGNANCY

The bone marrow showed megakaryocytic hyperplasia with immature forms that were not producing platelets. The bone marrow findings were interpreted as being compatible with idiopathic thrombocytopenic purpura.

Large doses of Prednisone were given in addition to small transfusions of fresh blood in specially prepared siliconized bags,<sup>4</sup> but the platelet count fell to zero. For several days careful examinations of peripheral blood smears failed to reveal any platelets on an entire film. Splenectomy was performed with the patient under general anesthesia, in spite of the technical problems associated with the presence of a 29-week-old fetus.

The postoperative course was uneventful. The platelets returned and rebounded. The values were 20,000 immediately after operation, 340,000 in 8 hr., and 800,000 in 4 days. The patient progressed normally with her pregnancy. In the fortieth week she had a spontaneous rupture of the membranes without labor. Labor was induced with intravenous oxytocin. The patient's general condition during labor was satisfactory and she was not given steroid replacement therapy. There was no bleeding and no difficulty with the induction of labor or childbirth. Platelet counts from the umbilical cord blood and, at a subsequent date, from the peripheral blood, were normal. At this writing, the infant (6 months old) shows no blood problem. The mother is also normal.

### DISCUSSION

If untreated, thrombocytopenic purpura tends to become worse during pregnancy. The more severe cases of thrombocytopenic purpura are the ones that occur primarily during pregnancy. Hemorrhage into the placental site is not a usual complication of thrombocytopenic purpura during late pregnancy. The frequency of premature separation of the placenta remains the same as in patients without thrombocytopenic purpura. The mode of delivery follows general obstetric principles. Postpartum hemorrhage is more frequent, occurring in 8% of cases, slightly above the average 6%.

Maternal mortality in association with thrombocytopenic purpura cannot be stated with certainty. It appears to be determined solely by the progress of the purpura itself,

and to have little relation to the presence or absence of pregnancy. However, there is increased fetal loss in patients who have splenectomy during pregnancy, approaching 25–30%. Clinical evidence of purpura is present in 65% of the babies born alive. In one series, only 5 infants were found to have normal platelet counts at the time of delivery. In term infants, abnormal platelet counts reverted to normal within 6 weeks without treatment.

As we review the work of others there is a marked tendency towards conservative management of thrombocytopenic purpura during pregnancy. Perhaps this tendency is due to rather convincing evidence that splenectomy is extremely hazardous to the fetus. Steroid and other medical management of thrombocytopenic purpura has greatly improved the outcome of the disease. However, there must be no hesitation to perform splenectomy if conditions so indicate. In spite of technical difficulties, splenectomy can and should be performed at any stage of pregnancy when the life of the mother is in danger.

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