ACUTE MYELOID LEUKEMIA IN PREGNANCY

by

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Introduction

The combination of acute myeloid leukemia and pregnancy has been reported infrequently. In the non-pregnant female population between ages 15 and 45, the estimated incidence of leukemia is 1 per 100,000. In pregnant women the incidence has been reported to be the same (Mitchell and Capazzi, 1982). Over the years, the reported fetal mortality in pregnancies complicated by acute leukemia has decreased from 60% in 1954 (Allan), to 33% in 1961 (Ask-Upmark). Due to the relative infrequency of this disease in pregnancy, more recent figures are unavailable.

The placenta permits transfer of normal (Maevis *et al*, 1958) and abnormal (Rigby *et al*, 1969) maternal cells to the fetus. The present report concerns a case of acute myelomonocytic leukemia where the possibility of transplacental transfer of malignant maternal cells was studied. A plan for the optimal management of labour in leukemic women with low platelet counts is outlined.

CASE REPORT

A 20 years old gravida 3, para 2 was booked for delivery and post-partum sterilization at 31 weeks of gestation, at the Department of Obstetrics. She was found to have pallor but the pregnancy was otherwise found to be normal. Routine investigations were done and she was asked to return after two weeks. At the second visit it was noted that her hemoglobin was 7.3 G/dL and that her peripheral blood smear showed anisocytosis and a normochromic normocytic anaemia.

At admission, the white cell count was 4,800/mm^3 with 54% neutrophils, 18% lymphocytes, 8% band forms, 2% metamyelocytes, 2% myelocytes, 1% promyelocytes and 13% myeloblasts. A sternal bone marrow aspiration showed 17% myeloblasts and 14% monoblasts. A diagnosis of acute myeloid leukemia—myelomonocytic variant was made. Her blood group was O Negative and the indirect Coomb's test was negative. Her bleeding parameters were found to be normal. The platelet count which on admission was 102,000/mm^3 dropped to 56,000/mm^3 on September 6 and 21,000/mm^3 on September 9. The patient, however, remained asymptomatic.

The cervix was found to be unfavourable for induction and ripened with intravenous oxytocin. When the cervix was found to be favourable membranes were artificially ruptured after which oxytocin infusion was continued. Four units of platelet—rich—concentrate were kept in readiness. Labour which was conducted under antibiotic cover, progressed rapidly and the patient delivered normally before the platelets could be transfused. Prophylactic ergometrine was given. The total blood loss at delivery was about 200 ml. Platelet-rich-concentrate was transfused immediately after delivery and the platelet count was 72,000mm/^{3}. The puerperium was uneventful but tubectomy was decided against. Both the patient and her husband were unwilling for chemotherapy and she was discharged on the fifth postnatal day.

Infant: The infant was a term, male, small for gestational age baby, weighing 2.3 kg. The Apgar Score at 1 minute was 9. Physical examination was unremarkable and the hospital course uneventful. Cord blood was collected at

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delivery and smears studied to detect the presence of abnormal cells. A peripheral blood smear was repeated at 6 hours and again at five days of age. None of the smears showed abnormal cells.

Placenta: The placenta weighed 390 G, measured 19 cms x 11 cms and was grossly normal. Sections of the placenta did not show abnormal cells in the intervillous spaces, chorionic villi or in the fetal capillaries. No malignant cells were seen in sections of the umbilical cord.

Discussion

The initial low haemoglobin indicates that the patient probably had the disease process at the time of booking. The six week delay in diagnosis was due to the normal peripheral blood smear at the first visit. The absence of symptoms and clinical signs, even with the low platelet count was unusual and also contributed to the delay in diagnosis. Post-partum hemorrhage has been found to occur in 15-19% of such cases (McLain, 1974). As post-partum hemorrhage was expected, platelet-rich-concentrate was arranged for, prophylactic Ergometrine given and operative interference kept to a minimum. With this low platelet count, she tolerated labour and delivery surprisingly well. In leukemic women with low platelet counts, straining efforts of the second stage of labour have been reported to cause intracranial and retinal hemorrhages (Allan, 1954) and it is recommended that elective forceps be applied. However, as the present patient was a third gravida, second stage was rapid. An episiotomy was avoided and this along with the prophylactic ergometrine reduced the blood loss to less than 200 ml. Ophthalmic examination in the postpartum period was normal in this case. In order to avoid excessive hemorrhage in women with low platelet counts the precautions outlined above seem to be adequate.

Leukemic cells have been identified in the intervillous sapces of the placenta, in the fetal capillaries of the chorionic villi and in the cord blood of the babies born to mothers with leukemia (Rigby *et al*, 1964). Cramblatt *et al* (1958) have reported a case where the baby born to a mother with acute lymphatic leukemia developed the same disease at nine months of age. However, in the present case no leukemic infiltrates could be identified in the placenta or the cord blood of the fetus. The peripheral blood of the baby remained free of leukemic cells till the last follow-up.

Growth retardation of the fetus has not been reported in leukemic mothers. In the present case there were no identifiable maternal factors which could have resulted in intra uterine growth retardation. The patient had previously delivered normal sized babies at term. In the absence of leukemic infiltrates in the placenta, the cause for the growth retardation of the fetus remains unexplained.

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