An Uneventful Pregnancy and Delivery in a Patient with Chronic Myeloid Leukemia

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ABSTRACT
The concomitant occurrence of chronic myeloid leukemia and pregnancy is estimated to be one in 75000-100000 pregnancies annually. We present a case of 28 years old lady with 36 weeks of gestation diagnosed as chronic myeloid leukemia in chronic phase. Her pregnancy and delivery was uneventful. She breast fed the baby for 3 weeks before initiating treatment. Close vigilance and relevant investigations are of paramount importance in recognizing this extremely rare disease during pregnancy.

Keywords: Pregnancy, Chronic myeloid leukemia, Delivery.

INTRODUCTION
Chronic myeloid leukemia (CML) is a myeloproliferative disorder that originates in an abnormal pluripotent bone marrow stem cell and is consistently associated with the Philadelphia (Ph) chromosome and/or the BCR/ABL fusion gene. The disease is biphasic or triphasic characterized by an initial indolent chronic phase (CML-CP) followed by one or both of the aggressive transformed stages, accelerated phase (CML-AP) and the blast phase (CML-BP). The incidence of chronic myeloid leukemia in pregnancy is one is 75000-100000 pregnancies with a risk of leukostasis and placental insufficiency with consequent low birth weight, fetal prematurity and increased mortality. We report a case of a 28 years lady with 36 weeks of gestation in chronic phase of CML delivering a healthy male baby.

CASE HISTORY
A 28 years old woman, gravida 3, parity 2 and living 2 weeks with 36 weeks of gestation presented with labor pains. Her antenatal period was uneventful. Menstrual cycles were regular. Routine hemogram revealed a total leukocyte count of 1,03,000 cells/cumm, hemoglobin 11.7 g/dl, platelets 2,67,000 cells/cumm, neutrophils 64%, myelocytes 10%, metamyelocytes 6%, myeloblasts 2%, lymphocytes 14%, basophils 4% (Fig. 1). She was diagnosed as a case of CML-chronic phase in labor. She delivered a normal healthy male baby weighing 2.5 kg with Apgar score of 7 at 1 minute and 8 at 5 minutes with no abnormality and blood counts were normal. Repeat hemogram was done 24 hours after delivery which revealed, total leukocyte count 88,000 cells/cumm, hemoglobin 10.8 g/dl and platelets 3,60,000 cells/ cumm (Fig. 2). Ultrasonography of abdomen after delivery showed splenomegaly. Bone marrow examination revealed hypercellular bone marrow with increased granulopoiesis and 2% blasts. Cytogenetic study (46,XY, t(9;22) (q34;q11), bcr/abl positive), confirmed the diagnosis of chronic phase CML. The child was breastfed for 3 weeks before starting therapy for chronic phase CML.

DISCUSSION
Leukemia during pregnancy is very rare. CML accounts for less than 10% of all leukemias during pregnancy. CML is a clonal myeloproliferative disorder of a pluripotent stem cell, first described by John Hughes Bennett in 1845 at The Royal Infirmary of Edinburgh. Classically three phases of disease progression are recognized in CML: chronic phase (CP), accelerated phase (AP), and blast phase (BP). Frequently the diagnosis is made incidentally during a routine complete hemogram for unrelated reasons. Nearly 90% of patients with CML have their conditions diagnosed in the chronic phase as they have a competent immune system and may remain asymptomatic for prolonged period. Patients usually present with weakness, malaise and abdominal discomfort due to splenomegaly. The diagnosis is usually based on peripheral blood leukocytosis with basophilia, eosinophilia which is further confirmed on bone marrow examination and cytogenetic analysis.

Chronic myeloid leukemia was the first malignancy to have a specific chromosomal abnormality uniquely linked to it after the discovery of a minute chromosome now known as the Philadelphia (Ph) chromosome, later defined to result from a t(9;22) reciprocal chromosomal translocation.
There is no evidence that the behavior of chronic myeloid leukemia is altered in pregnancy. However, there is a risk of leukostasis and placental insufficiency with consequent low birth weight, fetal prematurity and increased mortality if CML is left untreated during pregnancy.

The management of CML in pregnancy involves prevention of placental insufficiency and other complications of hyperleukocytosis by control of maternal leukocyte count, while avoiding harmful fetal exposure to cytotoxic drugs. There have been few reports of successful treatment of CML during pregnancy by therapeutic agents like hydroxyurea, busulfan, interferon α and imatinib. Leukapheresis is also being tried in few cases. However there has been no systemic investigation of the effect of these therapeutic options in pregnancy.

As pregnancy does not alter the course of CML, the case can be managed by close observation without active intervention in the best interest of both the mother and fetus. However, if there is presence of symptoms, a rapidly increasing leukocyte count or emerging signs of a more advanced disease will mandate the need for immediate therapy.

REFERENCES

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