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Case Report

Managing aplastic anaemia in pregnancy: a unique obstetric challenge

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ABSTRACT

Aplastic anaemia during pregnancy poses a unique obstetric challenge due to its rarity and potential life-threatening consequences. This article explores the intricate management of aplastic anaemia in pregnancy through a detailed case report of a 23-year-old primigravida. Despite the absence of clear guidelines, the patient successfully navigated pregnancy with multiple transfusions, presenting a compelling example of obstetric and neonatal success. The discussion digs into the complex relationship between pregnancy and aplastic anaemia, emphasizing the need for a multidisciplinary approach and careful decision-making to balance maternal and foetal well-being. The conclusion highlights the importance of a comprehensive strategy, including vigilant transfusion techniques, foetal growth monitoring, and delivery planning at tertiary centres.

Keywords: Aplastic anaemia, Pregnancy complications, Obstetrical challenges, Haematological disorders

INTRODUCTION

Aplastic anaemia (AA) is defined by the presence of pancytopenia with hypocellular bone marrow in the absence of an abnormal infiltrate or bone marrow fibrosis.¹ It is a rare yet life-threatening condition and has significant pregnancy-related consequences. Its natural history throughout the pregnancy is not well known.² Due to the rarity of this condition during pregnancy, there is a lack of information regarding its management. Treatment options include supportive care (packed cell and platelet transfusion), immunosuppressive therapy, and allogenic stem cell transplantation (which is contraindicated in pregnancy).³ For the management of aplastic anaemia during pregnancy, there is no consensus on the best supportive care, treatment plan, or even clear guidelines.

Here, we present a case of 23-year-old primigravida with aplastic anaemia managed with multiple transfusions throughout the pregnancy with a successful obstetric and neonatal outcome.

CASE REPORT

A 23-year-old primigravida presented to us at 23 weeks of gestation with chief complaints of easy fatigability for 6 months, which was insidious in onset and increased gradually disabling daily activities. She had received 6 doses of iron sucrose (200 mg) injections and 2 pints of packed red blood cells (PRBC) at 10 weeks of gestation in a local hospital in view of low haemoglobin levels. The examination in our institution revealed pallor and tachycardia of 115 bpm. Upon lab evaluation, Hb was found to be 2.8 g/dl, the leucocyte count was $3.9 \times 10^3/\mu\text{l}$ and platelet count was $12 \times 10^3/\mu\text{l}$. She was admitted to the ICU and received 2-pint PRBC and 4 random donor platelets (RDPs). Autoimmune workup done was negative for antiphospholipid antibody (APLA), antineutrophilic cytoplasmic antibody (ANCA) and antinuclear antibody (ANA). Anti-platelet antibody also found to be negative. Renal and liver function tests were within normal limits. Vitamin B12 and folic acid levels were also satisfactory. In view of refractory anaemia bone marrow biopsy done which was suggestive of aplastic anaemia (Figures 1 to 5).

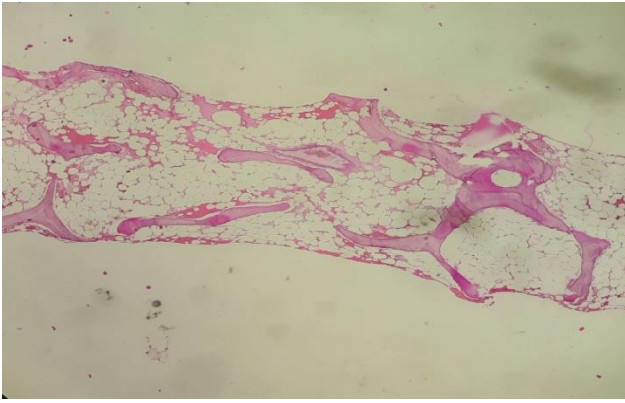


Figure 1: Bone marrow biopsy showing markedly hypocellular marrow spaces (H&E stain 40X).

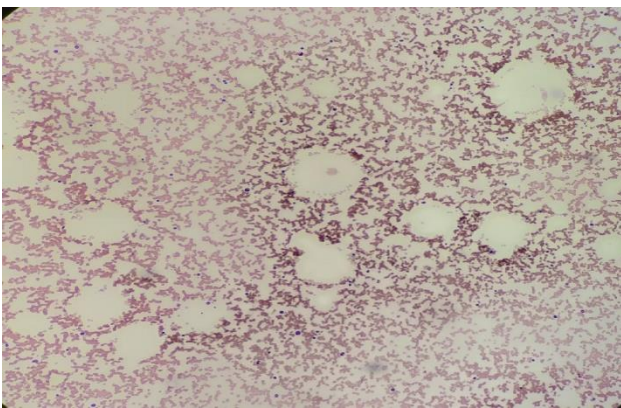


Figure 2: Bone marrow aspirate showing poor cell trails (Leishman stain 200X).

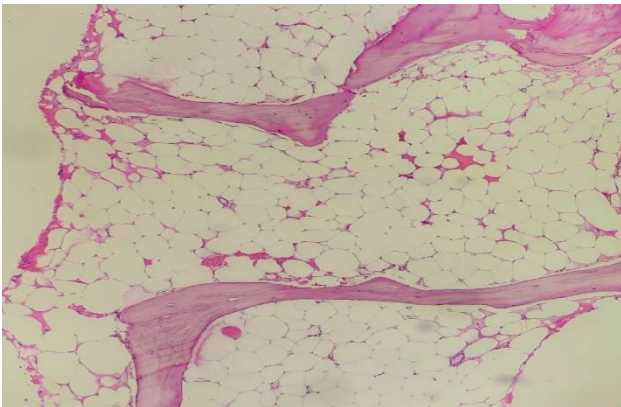


Figure 3: Bone marrow biopsy showing predominantly fat (H&E stain 200X).

As the patient was keen on pregnancy, she was planned to continue the pregnancy on transfusion support with a target of Hb >8 g/dl, platelet count >20×10³ μl and WBC >3.0×10³/μl. Throughout the pregnancy, she received a total of 8 pints of PRBC, 1 pint of single donor platelet (SDP), and 16 pints of RDP. However, the target could not be achieved despite multiple transfusions as shown in Table 1.

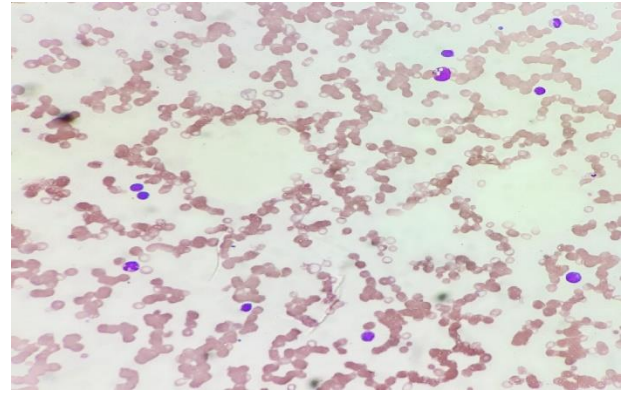


Figure 4: Bone marrow aspirate showing predominantly lymphocytes and few myeloid precursors (Leishman stain 200X).

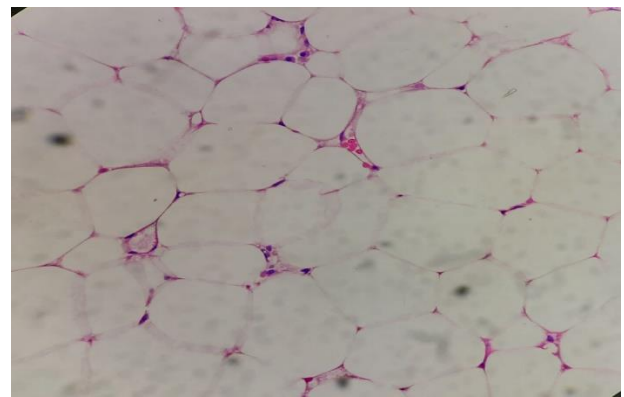


Figure 5: Bone marrow biopsy showing few pockets of lymphocytes (H&E stain 400X).

Table 1: Blood counts at different periods of gestation.

Period of gestation (weeks)	Haemoglobin (g/dl)	Platelet count (μl)	Leucocyte count (μl)
23	2.8	12×10 ³	3.9×10 ³
34	6.8	32×10 ³	3.3×10 ³
38	7.4	7×10 ³	3.4×10 ³

She had no complaints of bruising, spotting, or any abnormal bleeding throughout the pregnancy. The pregnancy progressed to term without any major complications, despite chronic anaemia periodic growth assessment by ultrasound showed foetal growth to be appropriate for gestation. At 39 weeks and 2 days of gestation, labour was induced with dinoprostone gel and was unsuccessful. Hence, patient was taken for caesarean section under general anaesthesia (in view of low platelet counts). She delivered a healthy female baby weighing 2660 grams. Pre op she received 1-pint PRBC, 10 pint of cryoprecipitates and 4-pint RDP. Postop 1-pint PRBC was transfused prophylactically. No postpartum haemorrhage or excessive bleeding was noted and post-operative period was uneventful.

DISCUSSION

Aplastic anaemia is a serious condition defined by pancytopenia, or hypocellularity of the bone marrow, in the lack of an underlying malignant or myeloproliferative disease. Acquired aplastic anaemia is more common.² Adult-onset aplastic anaemia can be genetic with late-onset symptoms, idiopathic (>80% of cases), or brought on by medications, infections (especially hepatitis), or other factors.

Ehrlich released the first aplastic anaemia report in 1888. His patient was pregnant and passed away from postpartum haemorrhage one month after giving birth.² It's still unknown how pregnancy and aplastic anaemia are related. Earlier studies had established no association between the disorders. Other studies support a clear link between pregnancy and aplastic anaemia, and some reviews even list pregnancy as a contributing factor to aplastic anaemia.³

Aplastic anaemia is known to cause antenatal complications like spontaneous miscarriage (16.7%), preterm birth (12.1%), intrauterine death (16.7%) and stillbirth (15.1%).¹ The two leading causes of death in pregnant women with aplastic anaemia are haemorrhage and sepsis. However, fortunately none of these complications were seen in our case.

The fundamentals of aplastic anaemia treatment during pregnancy involves identifying any underlying causes and treating cytopenia while limiting adverse effects on the mother and foetus.⁴ If a trigger factor for bone-marrow suppression, such as a drug reaction or infection, is found and the medicine cannot be stopped or the microorganism cannot be successfully treated, termination should be considered. Given the significant possibility of potentially fatal complications for both the mother and the foetus, pregnancy termination should also be taken into consideration for individuals with severe pancytopenia.² However, in our case even after elaborate counselling for termination of pregnancy, patient chose to continue the pregnancy.

Hematopoietic stem cell transplantation (HSCT) and the use of immunosuppressive regimens are two treatments advised for aplastic anaemia in the non-obstetric population.⁴ Supportive treatment throughout pregnancy with transfusions for haemoglobin >8 g/dl and platelet count >20×10³ is advised.⁵ There is minimal published experience with using antithymocyte globulin (ATG) during pregnancy.

As a reasonably safe medicine, ATG primarily causes allergic responses, vein irritability, nausea, vomiting, and diarrhoea as side effects. ATG has not been linked to any foetal adverse effects in human reports, and low birth weight may be due to coexisting disorders rather than drug toxicity.

If corticosteroids are administered, it is preferable to choose ones that cannot cross the placenta, such as prednisone, prednisolone, and hydrocortisone, to reduce exposure to the developing fetus's brain and the small risk of orofacial deformities.⁷ There is also a case of aplastic anaemia treated with Eltrombopag (thrombopoietin receptor agonist) which was continued throughout the pregnancy but as the efficacy and safety of eltrombopag in pregnancy has not yet been established, its routine use should be avoided.⁸

Most of the supportive treatments for aplastic anemias related to pregnancy is transfusion of blood products.³ However, this can result in problems such as hemochromatosis and—more seriously—HLA alloimmunization. Platelet-transfusion refractoriness (PTR) is brought on by alloantibodies against human platelets. HLA- and/or HPA-compatible platelet transfusions are advised if PTR is found in a pregnant patient who has undergone blood transfusions. The possibility of newborn thrombocytopenia in mothers who get platelet transfusions should also be considered in case of aplastic anemias. Maternal antibodies against HPAs that pass the placenta cause neonatal alloimmune thrombocytopenia.

The ideal delivery method is vaginal because, even in cases of severe thrombocytopenia, hemostasis may usually be attained with suitable uterine contractions following delivery. For vaginal delivery and cesarean delivery, respectively, a platelet counts of >20×10³ and >50×10³ is considered acceptable.⁸ Educating the patient about the condition, treatment options, and potential risks is essential for informed decision-making.

The decision-making process involves a careful balancing act, considering the health of the mother and the well-being of the fetus. It's crucial for the healthcare team to work closely with the patient to make informed decisions based on the individual circumstances. Each case is unique, and the approach may vary based on factors such as the severity of aplastic anemia, the gestational age, and the overall health of the mother and fetus.

CONCLUSION

Aplastic anemia needs a comprehensive multidisciplinary-team approach with an obstetric, hematological, anesthetic, and neonatal strategy to anticipate complications during the peripartum period. To prevent alloimmunization-related problems, cautious transfusion techniques are required. Strict fetal growth monitoring is advised throughout the pregnancy and delivery should be planned at a tertiary center with vaginal delivery being the ideal method.

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