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# Balancing Blood and Bump: A Case Report on Aplastic Anemia in Pregnancy

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**Abstract:** ***Introduction**: Aplastic anemia refers to the syndrome of hematopoietic failure from injury leading to diminished or absent hematopoietic precursors in the bone marrow and resultant pancytopenia. During pregnancy it could be life-threatening for both mother and child. The only causal therapy for aplastic anemia is bone marrow transplantation, which is contraindicated during pregnancy because of potential embryo toxicity. Hence, Treatment options are erythrocytes and platelet transfusions and immunosuppressive therapy. **Case Presentation**: This is a case of a 30 year old G5P3L2D1SA1 who came to our OPD at 31weeks of gestation with severe anemia (Hb-6.2g/dl). Patient was diagnosed with Aplastic anemia 4 years back, (at P3L2D1, all FTND with cu-T insitu) and was lost to follow up. She was managed with multiple blood products transfusion, close consultation with Hematology and a maintenance therapy of immunosuppressants and thrombopoetin agonists that she responded to. This case report discusses on managing the complications that were encountered during her antenatal course and averting a catastrophic clinical deterioration using a multidisciplinary approach of management. **Conclusion**: Aplastic anemia when juxtaposed with pregnancy can be life-threatening. The seriousness depends on the degree of bone marrow suppression. During severe aplastic anemia or complications caused by the supportive therapy (erythrocyte and platelet transfusions and antibiotics) Anti-Thymocyte Globulin (ATG), methylprednisolone and/or Cyclosporine A could be started.*

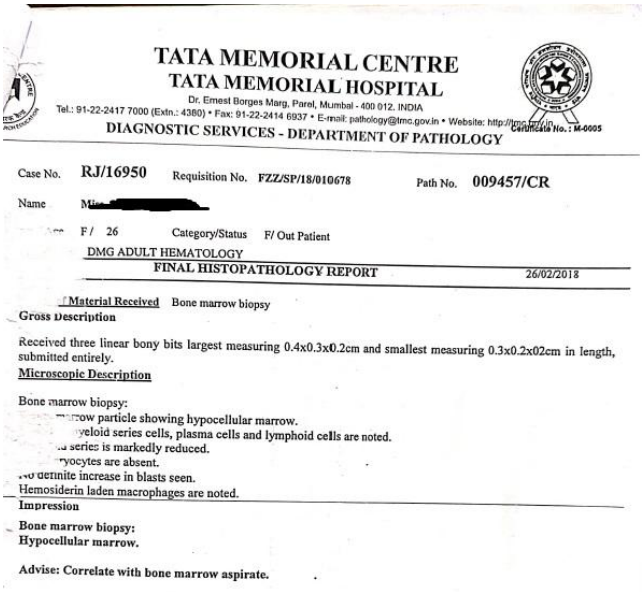
**Keywords:** Aplastic anemia, Pregnancy, Cyclosporine A, Anti-thymocyte globulin, Eltrombopag, Danazol

## 1. Introduction

Aplastic anemia refers to the syndrome of hematopoietic failure from injury/insult leading to diminished or absent hematopoietic precursors in the bone marrow and resultant pancytopenia. Depending on affected cell lines, aplastic anemia is associated with fatigue, bleeding due to thrombocytopenia and recurrent infections due to neutropenia. The diagnosis 'aplastic anemia' is confirmed by hypocellularity of the bone marrow. The remaining cells are morphologically unaffected without malignant infiltration. Potential triggers for the onset of aplastic anemia include T-cell mediated auto-immune disease, iatrogenic agents, viral infection etc. During pregnancy it could be life-threatening for both mother and child. The only causal therapy for aplastic anemia is bone marrow transplantation, which is contraindicated during pregnancy because of potential embryo toxicity. Treatment options are erythrocytes and platelet transfusions and immunosuppressive therapy. There are no clear guidelines for the management of aplastic anemia during pregnancy. Is immunosuppressive treatment more effective than supportive therapy consisting of erythrocytes and platelet transfusion and antibiotics?

## 2. Case Presentation

A 30 year old G5P3L2D1SA1 (Previous 3 Full Term normal deliveries) came to our OPD on at 31weeks 4 days of gestation by a scan of 10+0 weeks, currently with no complaints, with a report of Hb- 6.2g/dl. Patient was diagnosed with Aplastic anemia 4 years back at P3L2D1 (with cu-T insitu) when she was referred to KEM Hematology. Aplastic anemia was confirmed with bone marrow aspiration and biopsy reported by TATA hospital, and under further evaluation had PNH (Paroxysmal Nocturnal Hemoglobinuria) clone of 2.6% within neutrophils, negative for Fanconi anemia, and ICT DCT negative.



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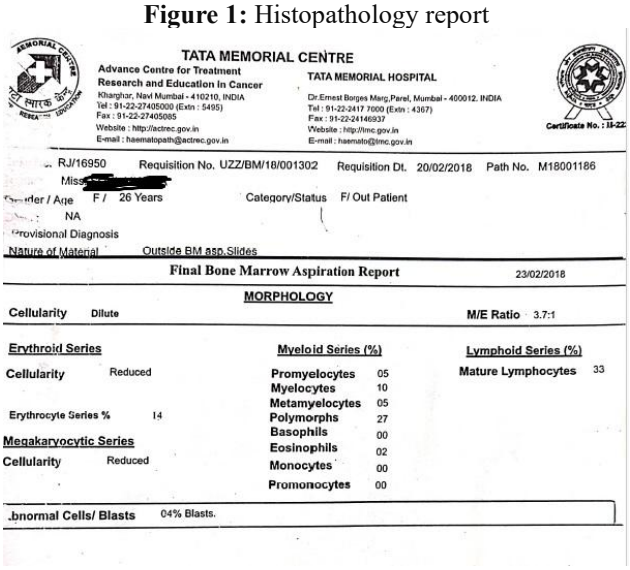
DIAGNOSTIC SERVICES - DEPARTMENT OF PATHOLOGY

Case No. RJ/16950 Requisition No. FZZ/SP/18/010678 Path No. 009457/CR
Name M[redacted]
F / 26 Category/Status F/ Out Patient
DMG ADULT HEMATOLOGY
FINAL HISTOPATHOLOGY REPORT 26/02/2018

Material Received Bone marrow biopsy
Gross Description
Received three linear bony bits largest measuring 0.4x0.3x0.2cm and smallest measuring 0.3x0.2x02cm in length, submitted entirely.
Microscopic Description
Bone marrow biopsy:
[illegible]row particle showing hypocellular marrow.
[illegible]yeloid series cells, plasma cells and lymphoid cells are noted.
[illegible] series is markedly reduced.
[illegible]ryocytes are absent.
[illegible] definite increase in blasts seen.
Hemosiderin laden macrophages are noted.
Impression
Bone marrow biopsy:
Hypocellular marrow.
Advise: Correlate with bone marrow aspirate.

**Figure 1:** Histopathology report



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RJ/16950 Requisition No. UZZ/BM/18/001302 Requisition Dt. 20/02/2018 Path No. M18001186
Miss[redacted]
[illegible]der / Age F / 26 Years Category/Status F/ Out Patient
NA
[illegible]rovisional Diagnosis
Nature of Material Outside BM asp.Slides
Final Bone Marrow Aspiration Report 23/02/2018
MORPHOLOGY
Cellularity Dilute M/E Ratio 3.7:1

| Erythroid Series | | Myeloid Series (%) | | Lymphoid Series (%) | |
|---|---|---|---|---|---|
| Cellularity | Reduced | Promyelocytes | 05 | Mature Lymphocytes | 33 |
| | | Myelocytes | 10 | | |
| | | Metamyelocytes | 05 | | |
| Erythrocyte Series % | 14 | Polymorphs | 27 | | |
| | | Basophils | 00 | | |
| Megakaryocytic Series | | Eosinophils | 02 | | |
| Cellularity | Reduced | Monocytes | 00 | | |
| | | Promonocytes | 00 | | |

[illegible]bnormal Cells/ Blasts 04% Blasts.

**Figure 2:** Bone Marrow Aspiration report

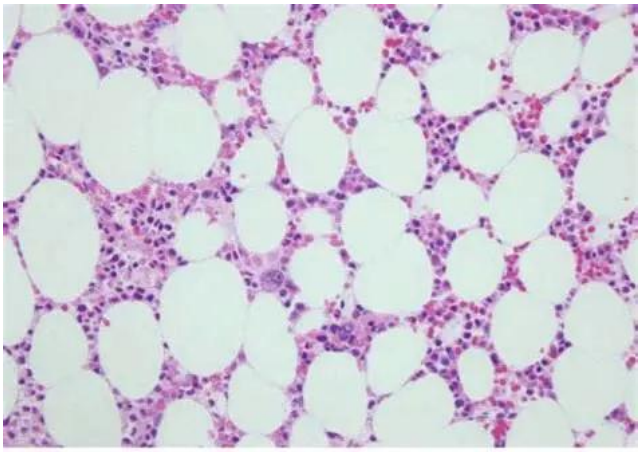

**Figure 3:** Hypocellular Bone marrow

4 years back she was initially started on Danazol and Cyclosporine but did not respond. She then underwent 3 cycles of treatment with Anti Thymocyte Globulin (ATG), 47 vials over 4 days, along with steroids, followed by maintenance immunosuppression with Tb Cyclosporin A 150mg bd with adequate antivirals and antifungals, regular blood transfusions. Patient had an menorrhagia in the following year for which Gyneac was reference taken, Copper-T IUCD removed and started on Tb. Tranexamic acid till her menorrhagia stopped. Tb. Danazol 100mg bd maintenance added to Cyclosporin A for adequate control. This continued for next three years with regular blood Cyclosporin A levels, blood transfusions and follow ups. In the following year when patient came with UPT Positive and 2 months of Amenorrhoea – Danazol was stopped immediately on account of it being fetotoxic, Cyclosporin A was continued. In the next follow up a few months later, patient came after spontaneous abortion, UPT negative status was confirmed and Danazol was restarted, Cyclosporin A continued. Patient was then lost to follow up, came to Hematology department of KEM Hospital 2 months back at 5 months ANC. They continued Tab. Cyclosporine 100mg bd, stopped danazol again and transfused 4 pints PCV. Her laboratory reports on Complete Hemogram were Hb- 7.3g/dl, WBC- 4000/mm3, Platelets- 37,000/mm3. At this point she was referred to our ANC OPD for registration, further evaluation and management at G5P3L2D1, previous 3 FTNDs at BD/BS ?/31+4 (10+0).

She was also a known case of Hepatitis B infection, HbSag positive detected 4 years back. Disease had progressed into Chronic Liver disease with cirrhosis, portal hypertension, esophageal varices status post Esophageal Variceal Ligation. She was started on Tb. Tenofovir and Tb. Propranolol. Her HBV DNA levels were 2400, latest HB Doppler showed Portal Cavernoma, Portal SVT, and Splenomegaly.

Patient was admitted for safe confinement. A repeat Paroxysmal nocturnal hemoglobinuria (PNH) profile was sent and found to be negative for clones following which she was started on Eltrombopag, a thrombopoetin receptor agonist used to treat thrombocytopenia in aplastic anemia after taking due consent of being potentially harmful to the fetus as the benefits outweigh the unknown, potentially harmful risks to the fetus. Eltrombopag 50mg od started and stepped up to 150 mg od with regular liver enzymes monitoring. Hb maintained above 8g/dl and platelet count over 50,000/mm3 with regular blood product transfusions. Gastroenterology review taken for Hepatitis B infection with Chronic Liver disease with cirrhosis, portal hypertension, esophageal varices s/p Esophageal Variceal Ligation, and maintenance therapy of Tb. Propranolol 40mg od and Tb. Tenofovir 300mg od continued. Under this maintenance Pregnancy continued till term to deliver vaginally a baby of 2.5kg. Postnatally patient hemodynamically stable. Blood products transfused to bring her parameter under normal limits. Patient was discharged on a maintenance therapy of Cyclosporin A, Danazol and Eltrombopag along with Tenofovir and Propranolol.

Contraceptive counselling was advised was patient's husband was counselled to undergo Vasectomy which was successful.

## 3. Discussion

In young non-pregnant patients first choice therapy for aplastic anemia is allogenic stem cell transplantation with a five-year survival of 70 to 80%. However, stem cell transplantation is not feasible during pregnancy because of the teratogenic effects of the immunotherapy and radiotherapy for the unborn child. Pregnancy termination to start bone marrow transplantation was not recommended because of the relatively good prognosis for both mother and child. During pregnancy supportive therapy with erythrocyte and platelet transfusions is a widely used, reasonable alternative. There is literature available of potential triggers for aplastic anemia that suggest that an acute attack of hepatis may precipitate aplastic anemia. Since our case has hepatitis B infection first diagnosed 4 years back, it may be a potential trigger, although deficient documentation of her initial disease course makes this diagnosis difficult.

## 4. Conclusion

Aplastic anemia in pregnancy can be life-threatening to mother and fetus due to severe reduction in erythrocytes, leucocytes and thrombocytes, which, if not treated adequagely can cause complications like postpartum hemorrhage, shock, cardiovascular failure in mother and intrauterine growth reduction, low birth weight etc in labour. As described in this case, adequate maintenance with immunosuppressants and thrombopoetin agonists with supportive blood product transfusions proved to be successful in a healthy baby and healthy mother post delivery.

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