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Haemoglobinopathies And Hemorrhagic Disorders In Pregnancy: A Clinical Case Series

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Abstract:
Haemoglobinopathies in pregnancy are a group of inherited disorders affecting haemoglobin structure or production, and they can significantly impact maternal and fetal outcomes. The most common types include sickle cell disease (SCD) and thalassemias (alpha and beta).These inherited disorders may lead to maternal complications like anemia, infections, pre-eclampsia, and thromboembolism, conditions like fetal growth restriction, preterm birth, and low birth weight can lead to serious health complications. Early identification through prenatal and neonatal screening plays a critical role in ensuring prompt and effective treatment.

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I. Introduction:

Haemoglobinopathies are inherited disorders affecting the structure or production of haemoglobin, the oxygen-carrying protein in red blood cells. The most common types are sickle cell disease and alpha- or beta-thalassaemia, which are more frequently seen in people of African, Mediterranean, Middle Eastern, and Southeast Asian origin. In pregnancy, these conditions present specific challenges, including chronic anemia, infection risk, and complications like pre-eclampsia, miscarriage, and preterm birth. Sickle cell disease may cause painful vaso-occlusive crises, while thalassaemia can lead to iron overload, especially in those needing frequent transfusions. These disorders also pose risks to the fetus, particularly if both parents are carriers, increasing the chance of the child inheriting a severe form. Therefore, early screening, genetic counselling, and prenatal diagnostic tests such as chorionic villous sampling (CVS) or amniocentesis are crucial for at-risk couples to make informed reproductive decisions.

Thrombocytopenia, or a low platelet count (below 150,000/µL), affects 7–10% of pregnant women. It can result from normal pregnancy changes or more serious conditions like preeclampsia, HELLP syndrome, or immune thrombocytopenic purpura (ITP). Identifying the cause is critical, as it impacts both maternal and fetal health. While mild cases may be physiological, pathological thrombocytopenia requires prompt diagnosis and management to prevent bleeding risks during and after delivery. Effective management of haemoglobinopathies in pregnancy requires a multidisciplinary approach involving obstetricians, hematologists, genetic counselors, and pediatric specialists. Careful monitoring, individualized treatment plans, and timely interventions are vital to optimize outcomes.

II. Case Report:

CASE 1-

A 32 year old female , Gravida 2, Para 1, Living 1 (prev LSCS) , came at 35.4 weeks of gestation with complaints of pain in abdomen, with per vaginal findings of fully dilated , fully effaced and station +1 , patient was immediately shifted to labour table, complications of vaginal birth after caesarean section (VBAC)

explained, consents taken and patient delivered vaginally. On admission haemoglobin (Hb) was 5 gm%, thus iron profile with Hb electrophoresis (HPLC) was sent. Two pint whole blood was transfused post-partum. HPLC report was s/o Beta-thalassemia trait. 48 hrs post transfusion Hb was 7.3gm% after which Injection Iron sucrose 3 doses on alternate days was given. Patient and baby were stable, thus discharged on post natal day 8 without any complications.

CASE 2-

A 28 year old female, G4P2L1D1A1 (prev 2 LSCS) came for ANC follow up at 34.2 weeks of gestation with K/c/o Beta thalassemia minor. All investigations were completed, revealing a hemoglobin level of 6.9 g/dL. Patient had already been transfused 1 pint whole blood and 1 dose of Injection ferric carboxymaltose in 6th month of pregnancy. Patient was admitted and another pint whole blood was transfused. Patient was advised weekly follow up. At 37.6 weeks of gestation patient was delivered by elective caesarean section, 1 whole blood was transfused pre-operatively. Post-operative, Hb was 6.3gm% , thus one more pint whole blood was transfused. Post transfusion CBC was done and Hb results were 8 gm%. The patient had no other complications, and was discharged on post-operative day 4.

CASE 3-

A 36 year old female, G2P1L1 (prev Normal Delivery), came for regular ANC follow up at 32.2 weeks of gestation. Patient came with CBC reports showing platelet count of 62,000/cumm. Patient was asymptomatic, and had no bleeding manifestations. Patient was a known case of Immune thrombocytopenia purpura (ITP) diagnosed during previous pregnancy. Patient was followed up in OPD with repeat CBC after one week in which platelets were 56,000/cumm. Weekly follow up was advised according to hematologist's opinion. Patient was admitted at 38 weeks of gestation and was delivered vaginally. On admission platelets were 50,000/cumm. Patient was monitored daily and did not show any signs of bleeding manifestations. There were no complications and patient was discharged on post natal day 4.

CASE 4-

A 23 year old female, primigravida, at term gestation came to the OPD with complaints of abdominal pain. Patient's Hb on admission was 7.5gm%. Patient had no history of blood/blood products transfusion during antenatal period. Patient was transfused with one pint whole blood. For further evaluation, anemia profile was sent along with HPLC before transfusion. HPLC report was s/o Compound heterozygous for sickle cell trait and Beta thalassemia. Patient delivered vaginally and post-delivery one pint whole blood was transfused. No complications were noted hence patient was discharged on post natal day 4.

CASE 5-

A 37 year old female , G3P2L2 (prev 2 ND) came at 26.1 weeks of gestation referred from local PHC in view of severe anemia with HB 4gm%. Patient came with complaints of chest pain and breathlessness. On examination, patient was severely pale, had tachypnea and the oxygen saturation (SpO2) on room air was 84%. Fetal heart sound was not heard on Doppler and Intra uterine fetal death was confirmed on Ultrasonography. Patient was but on O2 by mask at 10L of O2 and admitted in the ICU. 2DEcho was done which was s/o 20 - 30 % ejection fraction and cardiomyopathy. Along with routine investigations, anemia profile and HPLC was sent. HPLC was s/o sickle cell anemia with iron deficiency. Patient could not maintain on O2 by mask thus was shifted to Non-invasive positive pressure ventilation. Massive transfusion protocol was initiated. Despite this, patient could not maintain saturation and was intubated. Patient could not maintain saturation on 100% FiO2 and collapsed on day 3 of admission with blood pressure and pulse non recordable. Cardiopulmonary resuscitation was initiated as per protocol. Return of spontaneous circulation was not achieved and patient was pronounced deceased.

III. Observations:

TABLE 1-

	CASE 1	**CASE 2**	**CASE 3**	**CASE 4**	**CASE 5**
Period of Gestation (weeks)	38.2	34.2	32.2	37.6	26.1
Type of haemoglobinopathy (HPLC)	BetaThalassemia Trait	Beta Thalassemia Minor	Immune thrombocytopenic purpura	Heterozygous sickle cell with Beta thalassemia	Sickle cell anemia
HB on admission (gm%)	5	6.9	12.6	7.3	4
Platelets on admission	1.2	1.8	62,000	2.4	1.0

(per cumm)					
Previous child HPLC status	-	Negative	-	-	IUFD
Blood transfusion in current antenatal period	-	1pint whole blood and 1 dose Inj. ferric carboxymaltose	None	None	None
Mode of delivery	Vaginal delivery	LSCS	Vaginal Delivery	Vaginal delivery	-
Transfusions required post Delivery	3 pint whole blood	1 pint whole blood	None	1 pint whole blood	-
Transfusion required during delivery	1 pint whole blood	1 pint whole blood	none	None	-
Husband Blood Status	Not done	negative	negative	Negative	Negative

TABLE 2-

	CASE 1	**CASE 2**	**CASE 3**	**CASE 4**	**CASE 5**
Sr Iron(50-170 mcg/dl)	50	76	136	30 (low)	15 (low)
TIBC(250-450mcg/dl)	454	560 (raised)	356	670 (raised)	725 (raised)
Transferrin(200-400mg/dl)	150 (low)	154	333	110 (low)	134
Serumferritin(6.24-137ng/dl)	6.2	7.8	120	6.2	3.5 (low)
ReticulocyteCount(0.5-2.5%)	0.6%	0.5%	1.4%	0.2%	3.4%
Peripheral smear for Cell type	Hypochromic Microcytic anemia	Hypochromic Microcytic anemia	Normal	Sickling of RBCs with hypochromic microcytic anemia	Sickling of RBCs with hypochromic microcytic anemia
HPLC	HbA2	HbA2	HbA0	HbA2, HbS	HbS
Vit B12(180-514pg/dl)	344	570	590	680	560

IV. Discussion:

Sickle cell disease (SCD), thalassemia, and other haemoglobinopathies, though rare, significantly increase maternal and fetal risks during pregnancy due to complications like anemia, vaso-occlusion, and organ dysfunction. These conditions are linked to higher rates of preeclampsia, preterm birth, fetal growth restriction, and stillbirth. Thalassemia, particularly in transfusion-dependent patients, also introduces risks related to iron overload and cardiac complications.Early diagnosis through preconception and antenatal screening is crucial for timely intervention. Genetic counselling and partner testing allow for informed reproductive decisions and risk assessment.

V. Conclusion:

Timely diagnosis of haemoglobinopathies is critical, as unmanaged can lead to severe obstetric and perinatal outcomes.Integrating haemoglobinopathy screening into preconception and early antenatal care protocols can facilitate early identification, genetic counselling, and targeted management strategies. The implementation of standardized protocols is essential to optimize pregnancy outcomes in women with haemoglobinopathies.

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