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Case Series

Obstetrics and Gynaecology Section

Pregnancy Outcomes among Antenatal Women with Sickle Cell Anaemia: A Case Series

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ABSTRACT

Sickle Cell Anaemia (SCA) is a common and severe form of an inherited blood disorder known as Sickle Cell Disease (SCD). SCD is a group of autosomal recessive disorders characterised by point mutation resulting in the formation of structurally defective Haemoglobin (Hb), called Haemoglobin S (HbS). SCD is clinically heterogeneous, with variability in manifestation ranging from being asymptomatic to a severe crisis that can be fatal. Pregnancies complicated by SCA are high-risk due to the disease's heterogeneous manifestations and propensity for maternal and foetal complications. This case series aimed to understand associated maternal and foetal outcomes at a tertiary care hospital. Women presented with a spectrum of clinical presentations from uncomplicated deliveries to cases involving severe acute and chronic sequelae. Preterm birth before 37 weeks, low birth weight, and Neonatal Intensive Care Unit (NICU) admissions were common, reflecting known risks of growth restriction and prematurity. Previous adverse outcomes such as stillbirth and spontaneous abortion highlighted the threat to maternal and perinatal mortality. Acute sickle cell crises and splenic sequestration occurred. Advanced maternal age and comorbidities such as asthma exacerbated the disease burden. Most of the participants suffered from recurrent adverse outcomes with subsequent gestations. As most of the participants belonged to backward communities, targeted screening could help with earlier identification and counselling to reduce disease incidence in vulnerable populations.

Keywords: Haematologic pregnancy complications, Low birth weight infant, Neonatal intensive care, Premature births

INTRODUCTION

Sickle Cell Anaemia (SCA) is a common and severe form of an inherited blood disorder known as Sickle Cell Disease (SCD). SCD is a group of autosomal recessive disorders characterised by point mutations, resulting in the formation of structurally defective Haemoglobin (Hb), called Haemoglobin S (HbS). SCD is clinically heterogeneous, with variability in manifestation, ranging from being asymptomatic to a severe crisis that can be fatal [1,2]. It is a disease of tremendous clinical variability to the extent that clinical presentation is seldom similar between any two individuals [2,3].

Over 300,000 babies are born with SCD annually. Approximately 90% of these births take place in Low and Middle-income Countries (LMICs). The total number of births of babies with SCD has increased globally by 13.7% [4,5].

While SCA arises from the same genetic mutation in all those affected, there is significant variability in its clinical manifestations between individuals [6]. Common presentations include vaso-occlusive crises, acute chest syndrome, and haemolytic anaemia, though the frequency, severity, and specific symptoms experienced differ greatly [7]. Some experience mild disease, while others suffer from severe, life-threatening complications regularly.

This heterogeneity is also apparent in pregnancies complicated by SCA [8]. Studies have shown elevated risk of maternal complications, such as anaemia, infection, preeclampsia and foetal growth restriction in affected mothers [9,10]. Their infants also have higher odds of low birth weight, preterm birth and respiratory issues [11].

The present case series aimed to address the maternal and foetal outcomes among SCA affected pregnant women over two years.

CASE SERIES

The case series included eight patients presenting with SCA. According to the World Health Organisation (WHO), anaemia is defined as a Hb concentration of less than 12.0 g/dL [12]. As part of the hospital's standard treatment protocol, all the women were subjected to haemogram and peripheral smear. Women found to be sickling positive were tested by Hb electrophoresis to confirm the diagnosis of sickle cell homozygous state or trait.

All cases were studied for demographic parameters (age, locality), booking status in current pregnancy and Period of Gestation (POG). All the relevant laboratory parameters, including haemogram and Hb electrophoresis, Liver Function Tests (LFT), and Ultrasonography (USG) of the abdomen were assessed. All complications during the antenatal, intranatal and postnatal periods were noted. Parameters related to SCD, like the occurrence of sickle crisis (flow of blood is blocked to an area because of the sickled cells) [13], need for hospitalisation, prophylactic blood transfusions, total requirement of blood and its components in the antenatal, intranatal and postnatal periods were noted. Maternal outcomes, such as duration of pregnancy, mode of delivery, details regarding any abnormality during pregnancy, and abnormal course in the labour were noted. Neonatal outcomes, such as birth weight, and Neonatal Intensive Care Unit (NICU) admission/premature baby unit admission were noted.

Case 1

A 21-year-old multigravida (G2P1L1), from a tribal area was admitted with Hb of 3 g/dL at 39 weeks of gestation [Table/Fig-1]. She was unbooked. Her previous pregnancy was normal vaginal full-term delivery and the baby is alive and healthy. She had associated pre-eclampsia. She had a history of jaundice and joint pains on and off since childhood. During pregnancy, she had cough, expectoration, haemoptysis, and chest pain since the 8th month (chest crisis). Her investigations revealed normocytic hypochromic anaemia, moderate neutrophilia, and leucocytosis with sickling. Hb electrophoresis revealed sickle cell trait. On abdominal USG, she had a fatty liver, normal spleen and hepatomegaly. Her LFT was normal [Table/Fig-2].

In the present pregnancy, she had a normal vaginal delivery. She was transfused with five units of blood and was given three iron injections as soon as she was diagnosed as being anaemic. Her Hb at discharge was 8.6 g/dL [Table/Fig-3]. The birth weight was 2.5 kg [Table/Fig-4]. The baby was admitted to the NICU for meconium-stained liquor.

Section (LSCS) [Table/Fig-2]. The child is alive and healthy. There was a history of malaria in the 8th month [Table/Fig-5]. During the present pregnancy, she had a joint crisis and right hip joint pain. Her investigations had revealed normocytic hypochromic anaemia and neutrophilia with sickling. Her LFT was elevated. On USG, she had cholelithiasis and an absent spleen. Her Hb at admission was

Characteristics	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Age (years)	21	30	28	22	28	20	25	28
Locality	Rural	Urban	Rural	Urban	Rural	Urban	Urban	Rural
Booking status	Unbooked	Unbooked	Unbooked	Unbooked	Unbooked	Unbooked	Unbooked	Unbooked
Parity	G2P1L1	G3P2L1D1	G3P2L1D1	G2P1L1	G2L1	Primigravida	G2A1	G2P4L1D3
Period of Gestation (POG) at the time of admission (in weeks)	39	36	36	38	39	38	39	32

[Table/Fig-1]: Demographic features and history.

Characteristics	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Blood group	A	B	O	O	B	O	O	B
Haemogram	Normocytic hypochromic	Normocytic hypochromic	Normocytic hypochromic	Normocytic hypochromic	Normocytic hypochromic	Normocytic hypochromic	Microcytic hypochromic	Microcytic hypochromic
Hb electrophoresis	Sickle cell trait	HbSS* homozygous	HbSS* homozygous	HbSS* homozygous	HbSS* homozygous	HbSS* homozygous	Sickle cell trait	HbSS* homozygous
LFT's	Normal	Elevated	Elevated	Elevated	Elevated	Elevated	Normal	Elevated
Abdominal USG	Fatty liver, hepatomegaly, normal spleen	Cholelithiasis, absent spleen	Hepatomegaly, cholelithiasis, absent spleen	Hepatomegaly, ascites, cholelithiasis, absent spleen, left lower lobe consolidation	Cholelithiasis, absent spleen	Cholelithiasis, absent spleen	Cholelithiasis, normal spleen	Hepatomegaly, absent spleen

[Table/Fig-2]: Blood investigations and abdominal USG findings.
*HbSS: Haemoglobin SS

Treatment	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Hb at admission (g/dL)	3	10	7	8	9	8	8	7
No of units transfused	5	1	2	3	1	1	1	3
Total transfusions in life	5	4	4	6	1	1	1	5
Hb at discharge (g/dL)	8.6	9	8	8.3	9.2	8.2	8	8.6
Iron injection	3	-	-	-	-	-	-	-

[Table/Fig-3]: Haemoglobin at admission, haemoglobin at discharge and history of treatment received.

Characteristics	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Associated preeclampsia	Present	Absent	Present	Present	Absent	Present	Present	Present
Mode of delivery	Vaginal delivery	Emergency LSCS	Vaginal delivery	Emergency LSCS	Emergency LSCS	Vaginal delivery	Vaginal delivery	Emergency LSCS
Maternal outcome¶	Poor	Poor	Poor	Poor	Poor	Poor	Poor	Poor
Obstetric complications	-	IUGR	IUGR	-	-	-	-	Preterm delivery
Neonatal outcome	Poor	Poor	Poor	Good	Poor	Good	Good	Poor
NICU admission	Admitted	Admitted	Admitted	-	Admitted	-	-	Admitted
Reason for NICU admission	Meconium-stained liquor	IUGR	Meconium-stained liquor	-	Meconium-stained liquor	-	-	Preterm HMD jaundice
APGAR score at birth	8-10	6-8-10	6-8-10	8-10	8-10	8-10	8-10	6-8-10
Birth weight (kg)	2.5	2.4	2.5	2.9	3.3	3	3.5	2.3
Hospitalisation (days)	14	16	30	45	10	10	6	36
Crisis among mothers	Chest crisis	Joint crisis	Jaundice	Chest crisis	Joint crisis	Joint crisis	Joint crisis	Chest crisis

[Table/Fig-4]: Course and outcome.
APGAR: Appearance, pulse, grimace, activity, respiration; HMD: Hyaline membrane disease; IUGR: Intrauterine growth restriction; LSCS: Lower segment caesarean section
¶Any medical complications observed during pregnancy is considered as poor outcome

Case 2

A 30-year-old multigravida G2P1L1 from an urban area was admitted at 36 weeks of gestation. The first pregnancy was a twin pregnancy and intrauterine death occurred at 7th month [Table/Fig-1]. During the second pregnancy, she had jaundice and was diagnosed with SCA in the 8th month. The child had foetal distress and was delivered at full term by emergency Lower Segment Caesarean

10 g/dL. She was transfused one unit of blood. Her Hb at discharge was 9 g/dL [Table/Fig-3]. The third (current) pregnancy was full-term. Elective LSCS was done for post C-section and Intrauterine Growth Restriction (IUGR) in the current pregnancy. The child is alive and healthy. The birth weight was 2.4 kg. Appearance, Pulse, Grimace, Activity, Respiration (APGAR) score was 6-8-10 [Table/Fig-4]. The baby was admitted to the NICU for IUGR.

Case No.	Clinical presentation	Prolonged hospitalisation (days)	Treatment
Case 1	Cough and expectoration with haemoptysis, chest pain since 8th month-chest crisis	14	Antibiotics
Case 2	Right hip joint pain-joint crisis	16	Paracetamol
Case 3	Fever, anaemia and jaundice, vivax malaria, skin blisters since 8th month	30	Falcigo and clindamycin
Case 4	Jaundice, postoperative high-grade fever and SOB-3, pneumonia, toxic hepatitis, congestive heart failure and pulmonary oedema-chest crisis	45	Hydroxyurea and antibiotics
Case 5	Recurrent joint pains-joint crisis	10	Symptomatic treatment
Case 6	Joint pains, malaria at her 8th month-joint crisis	10	Symptomatic treatment and Falcigo
Case 7	Jaundice and joint crisis	6	Symptomatic treatment
Case 8	Jaundice, fever, SOB-3, chest pain and palpitation, lower respiratory tract infection-chest crisis, joint crisis	36	Hydroxyurea and antibiotics

[Table/Fig-5]: Clinical presentation and treatment.
SOB: Shortness of breath

Case 3

A 28-year-old multigravida (G3P2L1D1) from a tribal area was admitted at 36 weeks of gestation [Table/Fig-1]. The first pregnancy ended in intrauterine death in the 8th month. There was a history of malaria and jaundice during the first pregnancy at the 8th month. She also had a history of gestational hypertension. The second pregnancy was a full-term, normal vaginal delivery. The child is alive and healthy. The mother was diagnosed with SCA during her third (current) pregnancy. Her investigations had revealed normocytic hypochromic anaemia and neutroleucocytosis with sickling. On Hb electrophoresis, she had an HbSS homozygous trait [Table/Fig-2]. Her LFT was elevated. Her USG had revealed hepatomegaly, cholelithiasis and absent spleen. Her Hb at admission was 7 g/dL. She was transfused two units of blood. Her Hb at discharge was 8 g/dL [Table/Fig-3]. The third (current) pregnancy was a full-term, normal vaginal delivery. The child is alive and healthy. The birth weight was 2.5 kg. APGAR score was 6-8-10. The baby was admitted to the NICU for meconium-stained liquor [Table/Fig-4].

Case 4

A 22-year-old multigravida (G2P1L1) from an urban area was admitted at 38 weeks of gestation [Table/Fig-1]. The first pregnancy ended in neonatal death. She had a history of jaundice and chest crisis during the 8th month diagnosis. She also had a history of gestational hypertension. Her investigations revealed normocytic hypochromic anaemia, neutrophilic leucocytosis, and increased nucleated Red Blood Cells (RBCs) with sickling. On Hb electrophoresis, she had an HbSS homozygous trait [Table/Fig-2]. Her LFT was elevated. Her USG had revealed hepatomegaly, ascites, cholelithiasis, left lower lobe consolidation and absent spleen. Her Hb at admission was 8 g/dL. She was transfused three units of blood. Her Hb at discharge was 8.3 g/dL [Table/Fig-3]. The second pregnancy (current) was full-term. Emergency LSCS was done for post-LSCS. The child is alive and healthy. The birth weight was 2.9 kg. APGAR score was 8-10 [Table/Fig-4].

Case 5

A 28-year-old multigravida (G2A1) from a tribal area was admitted at 39 weeks of gestation [Table/Fig-1]. The first pregnancy ended in spontaneous abortion at 3rd month. She had a history of recurrent joint pains for 13 years. Her investigations had revealed normocytic hypochromic anaemia and neutrophilia with sickling. On Hb electrophoresis, she had an HbSS homozygous trait [Table/Fig-2]. Her LFT was elevated. Her USG had revealed cholelithiasis and an absent spleen. Her Hb at admission was 9 g/dL. She was transfused one unit of blood [Table/Fig-3]. Her Hb at discharge was 9.2 g/dL. The second (current) pregnancy was precious. Emergency LSCS was done at full-term for premature rupture of membranes (for 10 hours), meconium-stained liquor and foetal distress. The child was alive and healthy. The birth weight was 3.3 kg. APGAR score was 8-10 [Table/Fig-4]. The baby was admitted to the NICU.

Case 6

A 20-year-old primigravida from an urban area was admitted at 38 weeks of gestation [Table/Fig-1]. She had a history of malaria and pre-eclampsia. Her investigations had revealed normocytic hypochromic anaemia and neutrophilia with sickling. On Hb electrophoresis, she had an HbSS homozygous trait [Table/Fig-2]. Her LFT was elevated. Her USG had revealed cholelithiasis and an absent spleen. Her Hb at admission was 8 g/dL. She was transfused one unit of blood [Table/Fig-3]. Her Hb at discharge was 8.2 g/dL. The first (current) pregnancy resulted in full-term, normal vaginal delivery. The child is alive and healthy. The birth weight was 3 kg. APGAR score was 8-10 [Table/Fig-4].

Case 7

A 25-year-old multigravida (G2A1) from an urban area was admitted at 39 weeks of gestation [Table/Fig-1]. She had a history of Tuberculous Meningitis (TBM) and right hemiparesis at 18 years of age. The first pregnancy ended in spontaneous abortion at 2nd month. Her investigations had revealed microcytic hypochromic anaemia with sickling. On Hb electrophoresis, she had sickle cell trait [Table/Fig-2]. Her LFT was normal. Her USG had revealed cholelithiasis and a normal spleen. Her Hb at admission was 8 g/dL. She was transfused one unit of blood [Table/Fig-3]. Her Hb at discharge was 8 g/dL. The second (current) pregnancy was delivered at full-term by normal vaginal delivery. The child is alive and healthy. The birth weight was 3.5 kg. APGAR score was 8-10 [Table/Fig-4].

Case 8

A 28-year-old multigravida G2L1 G2P4L1D3 from a tribal area was admitted at 32 weeks of gestation [Table/Fig-1]. She had a history of childhood asthma, tonsillectomy, and pre-eclampsia. She had undergone cholecystectomy for gallstones at 15 years of age. The first pregnancy ended in full-term stillbirth. There was a history of jaundice since 2nd month of first pregnancy. The baby had pathological jaundice. The second pregnancy was at full-term by normal vaginal delivery. But the baby died at 4th month due to jaundice. The third pregnancy ended in full-term stillbirth due to jaundice. The mother was diagnosed with SCA during the fourth pregnancy in the 8th month. It was full term, normal vaginal delivery with cerebral palsy child. Her investigations had revealed microcytic hypochromic anaemia with sickling. On Hb electrophoresis, she had HbSS homozygous trait [Table/Fig-2]. Her LFT was elevated. She had prolonged Prothrombin Time (PT), International Normalised Ratio (INR), and Activated Partial Thromboplastin Time (APTT). Her USG had revealed hepatomegaly and an absent spleen. The fifth (current) pregnancy was preterm, elective LSCS. It was done due to bad obstetric history. The child is alive and healthy. The birth weight was 2.3 kg. APGAR score was 6-8-10 [Table/Fig-4]. The baby was admitted to NICU for jaundice and preterm delivery.

The overall clinical presentation and treatment of all the cases are presented in [Table/Fig-5].

DISCUSSION

There has long been recognition that SCA manifests with significant clinical variability, both between patients and within the course of an individual's disease. However, the underlying basis for this heterogeneity is still under investigation [14]. A better understanding of what drives differences in SCA severity and complications could have important implications for improving risk stratification and personalised treatment approaches.

The pathogenesis of SCA stems from a single point mutation in the β-globin gene that results in the production of abnormal HbS [15]. Under conditions of low oxygen tension, HbS polymerises within RBCs, distorting their morphology into a rigid sickle shape. This sickling phenomenon drives the primary vascular occlusive events and downstream sequelae that underlie the multi-systemic manifestations of SCA [16]. The core pathogenic process of SCA causes impaired microcirculation. This occurs due to abnormal polymerisation of Hb within RBCs under hypoxic conditions, causing their distortion into rigid sickle shapes that obstruct blood flow [17].

Acute sickle cell-related complications were documented, including chest crisis, jaundice episodes, and gallbladder disease requiring surgery. The women also exhibited chronic organ damage, including splenic sequestration and hepatic dysfunction. Prior comorbid conditions, such as asthma and Tuberculosis (TB) further increased vulnerability. Together, these findings exemplify the multisystem involvement and clinical heterogeneity encompassed under the umbrella diagnosis of SCA.

This case series was done to understand both maternal and foetal outcomes associated with SCA-affected pregnancies receiving care. Existing literature primarily reported isolated case studies without a broader examination of trends [18,19].

A retrospective study done by Khalid N et al., examined the pregnancy outcomes among 28 women with SCD or trait. The high rates of pre-eclampsia, preterm labour, IUGR and oligohydramnios were found to be similar to the authors' findings. Regarding SCD manifestations, anaemia requiring transfusion and infections were also common, as was the need for blood management. Their mode of delivery and perinatal outcomes also reflected the authors' observations of increased operative vaginal and Caesarean rates driven by foetal distress, alongside NICU admissions and morbidity [20].

Although the case series had a smaller sample size, it could have subject selection bias. Larger multicentre studies employing standardised disease severity classification tools and detailed recording of comorbidities, socioeconomic factors, and other confounding variables are needed to better understand the natural history and outcomes of SCA.

CONCLUSION(S)

The present case series has identified increased morbidity in pregnant women with SCD and a few with sickle cell trait. The common maternal complications were anaemia and organ involvement, while prematurity and low birth weight was frequent neonatal issues. There is a need for public health measures to proactively screen those belonging to high-risk communities. Such screening could help raise awareness and allow counselling to help reduce the incidence of SCD among newborns from these vulnerable populations.

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