# Maternal and fetal outcome in pregnancy with sickle cell disease in third trimester in central india: A retrospective study

Abstract

**Introduction:** Sickle cell disease is an uncommon cause of anemia and jaundice during pregnancy.however, SCD is common in parts of Maharashtra especially Vidarbha region. SCD in pregnancy can cause various maternal and fetal complications. This study was conducted to know maternal and fetal outcome in pregnancy with sickle cell disease in third trimester.

**Methodology:** This retrospective data was collected from hospital records of medical college located in central india for the period of one and half year from November 2019 to January 2021. During the study period, 42 women were admitted with pregnancy with sickle cell disease in third trimester. The baseline characteristics, frequency of sickle hemoglobin variants, and maternal and foetal outcome of pregnancy was collected.

**Results:** compiled data was analysed by simple descriptive statistcs and frequency tables. Majority were primigravida (59.52%). Type of sickle cell was determined on the basis of hb electrophoresis, out of 42 patients 36 had AS patern, 5 had SS patern SCD and 1 had As+b thal minor. Most common medical complication was anaemia [ 73.80%]and UTI 26.19%. Most common adverse obstetric outcome observed were IUGR/Oligohydramnios 42.85% and pre-eclampsia 21.42%. Majority of patients delivered by LSCS 42.85%. Adverse outcome in fetus were fetal distress 45.23% and meconium stained amniotic fluid 42.86%.

**Discussion:** due to hematological changes, extra demands, and sickle crisis, complications in both mother and fetus are more common in sickle cell anemia.

**Conclusions:** early detection and management of sickle cell anemia during pregnancy can reduce the adverse outcome in both mother and baby.



## Introduction

Sickle cell disease (SCD) is a group of inherited single gene autosomal recessive disorders caused by ‘sickle’ gene, which affects hemoglobin structure (Hb S). Sickle cell disease (SCD) is the commonest inherited single gene autosomal recessive disorders globally[14] The disease results from a single base A>T mutation in the triplet encoding the sixth residue of the β-globin chain, leading to a substitution of valine for glutamic acid and the abnormal haemoglobin S (HbS).The abnormal hemoglobin polymerises under hypoxic conditions, to form
rigid and fragile, sickle shaped red cells leading to hemolysis and vasoocclusion in microvasculatureHb S occurs commonly in populations previously

exposed to falciparum malaria, i.e., Africa, India and Saudi Arabia. In India, in the tribal belts of Madhya Pradesh, Chhattisgarh, Odisha, Andhra Pradesh and Maharashtra. In Maharashtra, the sickle gene is widespread in all the eastern districts, also known as the Vidarbha region, in the Satpura ranges in the north and in some parts of Marathawada. The prevalence of sickle cell carriers in different tribes varies from 0 to 35 per cent. The tribal groups with a high prevalence of HbS (20-35 %) include the *Bhils*, *Madias*, *Pawaras*, *Pardhans* and *Otkars*. It has also been estimated that Gadchiroli, Chandrapur, Nagpur, Bhandara, Yoetmal and Nandurbar districts would have more than 5000 cases of sickle cell anaemia.[15] There are little data on the maternal and perinatal outcomes of women with sickle cell disease in India, particularly in Maharshtra. A prospective study from Orissa showed that neonatal outcomes such as low birth weight, perinatal mortality rate, admissions to the neonatal care unit, intrauterine growth retardation and preterm births were significantly higher in sickle cell anemia mothers with successful pregnancies being achieved in 84.44 per cent of case.[16] Maternal and perinatal outcomes were also evaluated retrospectively from patients' case files in women with sickle cell disease in a tribal population in Madhya Pradesh. There were 25 deliveries to women with sickle cell disease and preeclampsia and disseminated intravascular coagulation were common problems. There was no maternal mortality; however, there were five intrauterine foetal deaths and one early neonatal death.[17] SCD is common and our centre receives many referrals. Maternal and fetal outcome in pregnant patients with sickle cell disease in third trimester pregnancies in women with sickle cell anemia and its complication was studied.

## Patient and methods

The case records of all 42 patients were studied retrospectively. All case papers were from November 2019 to January 2021. type of sickle cell was determined on the basis of hemoglobin electrophoresis. Outcome of both mother and fetus/newborn in third trimester was studied in detail. Maternal outcome was studied from both medical and obstetric point of view. Data obtained was compiled and analyzed by simple descriptive methods like frequency and percentage.

## Results

Out of total 42 patients 25 were primigravida, among these 25 patients 4 had SS patern sickle cell disease, 20 had AS patern and 1 had AS patern plus beta thalesemia trait. 17 patients were multigravida with 1 patient having SS patern and rest 16 had AS patern. 73.80% (n=31) presnted as anaemia of which (n=5) were SS patern, (n=25) were AS patern and (n=1) was AS patern+ beta thalesemia minor, only one patient that is 2.38% (n=1) had sickle cell crisis. 26.19%(n=11) had urinary tract infection and 6.66% (n=3) had lower respiratory tract infection at the time of delivery.

Out of 42 patients 9.52%(n=4) had previous perinatal loss, previous abortion was seen in 19.04% (n=8), gestational diabetes in 9.52% (n=4), IUGR/Oligohydramnios was seen in 42.85% (n=18), pre-eclampsia in 21.42% (n=9), gestational hypertention was seen in 9.42% (n=4), eclampsia in 2.38% (n=1), abruption was seen in 9.52% (n=4), preterm labour in 16.66% (n=7), LSCS was done in 42.85% (n=18), 57.14% (n=24) delivered vaginally.

Out of the 42 women 41 had live births and one patient had still birth. The still birth was fresh still birth and was associated with thick meconium and severe IUGR with birth weight of 1.9 kg at term.
The most common adverse outcome were fetal distress 45.23%(n=19) and meconium stained liquor 42.86%(n=18). Out of total 18 newborns with meconium stained liquor 4 i.e. 9.52% developed meconium aspiration syndrome. Total 9 newborns required NICU admission.
Prematurity was noted in 21.42%(n=9) and 38.09% (n=16) newborn were low birth weight that is there birth weight was below 2.5 kg.

Table 1: Distribution of type of sickle cell disease and parity

| Type of sickle cell | Primigravida | Multigravida | total |
|---|---|---|---|
| Ss patern | 4 | 1 | 5 |
| As patern | 20 | 16 | 36 |
| As+b thal minor | 1 | - | 1 |
| | 25 | 17 | 42 |

Table 2: Medical complications

| Complication | SS | AS | AS+ b thalesemia minor | total | percentage |
|---|---|---|---|---|---|
| Anaemia | 5 | 25 | 1 | 31 | 73.80% |
| Hemolytic crisis | 1 | - | | 1 | 2.38% |
| UTI | 3 | 7 | 1 | 11 | 26.19% |
| LRTI | 1 | 2 | | 3 | 7.14% |

Table 3: Mode of delivery

| Mode of delivery | SS | AS | AS+b thalesemia minor | total | percentage |
|---|---|---|---|---|---|
| LSCS | 1 | 16 | 1 | 18 | 42.85 |
| Vaginal delivery | 4 | 20 | - | 24 | 57.14 |

Table 4: Obstetric outcome

| Outcome | SS | AS | AS+ b thalesemia minor | Total | Percentage |
|---|---|---|---|---|---|
| Previous perinatal loss | 0 | 4 | 0 | 4 | 9.52% |
| Previous spontanious | 1 | 7 | 0 | 8 | 19.04% |

| | | | | | |
|---|---|---|---|---|---|
| abortion | | | | | |
| GDM | 0 | 4 | 0 | 4 | 9.52% |
| IUGR/OLIGO | 3 | 15 | 0 | 18 | 42.85% |
| Pre-eclampsia | 2 | 6 | 1 | 9 | 21.42% |
| Gestational hypertention | 1 | 3 | - | 4 | 9.52% |
| Eclampsia | 0 | 1 | - | 1 | 2.38% |
| Abruption | 1 | 3 | - | 4 | 9.52% |
| Preterm labour | 2 | 5 | - | 7 | 16.66% |

Table 5: Fetal outcome

| Outcome | SS | AS | AS+b thal trait | Total | Percentage |
|---|---|---|---|---|---|
| Prematurity | 2 | 7 | 0 | 9 | 21.42% |
| LBW < 2.5 kg | 3 | 13 | 0 | 16 | 38.09% |
| FD | 3 | 16 | 0 | 19 | 45.23% |
| Meconium | 3 | 15 | 0 | 18 | 42.86% |
| Meconium aspiration symdrome | 1 | 3 | 0 | 4 | 9.52% |
| NICU admission | 3 | 6 | 0 | 9 | 21.42% |
| Perinatal death | 0 | 1 | 0 | 1 | 2.38% |

Discussion

Vidarbha region is one of the high prevalence region for sicle cell disease. In Maharashtra the prevalence of SCD varries in different tribes from 0-35%. The screening test for sickle cell is done in all pregnant women irrespective of level of hemoglobin because if both mother and father are sickle cell trait, baby may have sickle cell disease. With proper medical care more number of females with sickle cell disease are reaching the child bearing age. Thus more number of pregnant females with sickle cell disease or sickle cell trait are reporting to the hospital. In our institution every pregnant female is screened for sickle cell disease if screening is positive hemoglobin electrophoresis is done to diagnose the type of sickle cell disease. In SCD at low-oxygen conditions polymerization of the abnormal hemoglobin occurs leading to formation of rigid and fragile sickle shaped red cells. This leads to increased episodes of vaso occlusive crisis, acute chest syndrome and pregnancy related complications [RCOG guideline 61]. According to Villers *et al*[3], maternal morbidity was increased due to increased rates of caesarean section,
Pregnancy related events like pre eclampsia, abruption and pulmonary complications, hypertension, and infection.

The incidence of SCD SS pattern was 0.30% and of AS pattern was 1.98% the low incidence recorded may be because of undiagnosed asymptomatic sickle cell patients delivering in other centres. 100% patient with sickle disease SS pattern presented as Anaemia and the percentage reduced to 73.80% when AS pattern was included which corroborated with other studies[2]. Other medical conditions complicating pregnancy were hemolytic crisis, UTI and LRTI. From the 5 patients of SS pattern only one had hemolytic crisis, UTI was observed to be the most common complication i.e. in 26.19% in contrst to a study by Afolabj et al[12]. where there was no significant difference between SCD and normal women. Common organism isolated from the urine culture samples were klebsiella and e-coli, no patient developed pyelonephritis. This observation was similar to study by kavitha and hota et al[2]. LRTI was seen in 7.14% at the time of delivery.

SCD patients had a higher incidence of miscarriage 19.04% and perinatal loss 9.52%. This incidence was comparable to other study done by D'Couth S et al. In our study the proportion of SA pattern is more as comparred to other studies[2,3] . Major obstetric complication was IUGR with Oligohydramnios seen in 42.85% which is comparable to other studies in past[2] .

More than half of cases in this study were primigravida i.e. 25 out of 42. All the women of SS pattern had recieved blood transfusions during pregnancy and anaemia and anaemia was corrected, they still had medical and obstetric complications during pregnancy, this was also seen in the study by NGO et al[7]. In our study only 5 patients were of SS pattern from whome only 1 had the hemolytic crisis. There was no maternal mortality in our study similar to study by sun et al.

Apart from IUGR and oligo, other obtetrics complications were pre-eclampsia 21.42%, gestational hypertension 9.52% and eclamsia 2.38%. this rate of pre-eclampsia was more than the study by Al Jama et al[11]. This may be because of underlying renal disease, plcental ischemia or endothelial damage and hypertension. There was no increase in pre-eclampsia in SCD in studies by Afolabj et. Al[12] and Serjeant et al[9]. In this study 9.52% had abruption which was associated with pre-eclampsia. 16.66% had preterm labour which included the patients of pre-eclampsia.

Of all patients LSCS was done in 42.85% and 57.14% had vaginal delivery. The indications for LSCS were mostly meconium stained amniotic fluid, fetal distress, previous LSCS, CPD and uteroplcental insuficiency. Higher rate of caeserean delivery 42.85 was seen in our study which was similar to other studies in past[2, 13].

Mothers having sickle cell deisease had adverse outcome in their newborns because of utero-plcental insufficiency, alloimmunization, and opioid exposure. In our study the most common adverse outcome seen was fetal distress 45.23% and meconium stained amniotic fluid 42.86% which was similar to other studies in past[2,3]. Several studies have been documented to have results showing increase in IUGR, preterm delivery and stillbirths[2,3,4]. In our study IUGR/oilgo was seen in 42.85%, preterm delivery was seen in 21.42% and only one case of perinatal mortality was noted. Placental ischaemia

because of pre-eclampsia may be an important factor resulting in IUGR and oligo. Perinatal mortality seen was less because of less number of SS patern which have higher mortality rate[2,11,12].

Preterm deliveries were seen in 21.42% and low-birth weight was seen in 38.09% of newborn. The above findings were because of increased number of IUGR. Birth weight was <2.5 kg in 38.09% of newborns. Similar results were seen in studies done in past[2,10,11,13].

All the patients of SCD SS pattern had received blood transfusion during pregnancy for correction of anaemia. Routine blood transfusion are not recomended by the RCOG. Only indications for blood transfusion are aplastic crisis, bone crisis, hemolytic crisis (HbA1<20%, hematocrit< 25%0) and infetion. All patients were given folic acid 5mg and tablet sodamint thrice daily. Patients in whome iron studies showed iron deficiency were given oral iron supplementation.

Conclusion

In our study we have observed increased morbidity in pregnancy in women with SCD because of increased number of medical and obstetric complications. There is need for screening for SCD in each and every pregnant women in the high prevalence area so that early detection and intervention could be done to improve the outcome of both mother and baby. Couples should be counselled to undergo preconceptional testing for sickle cell disease, so that measures can be taken to give the pregnant women better antenatal care right from first trimester.Preconceptional counselling and testing will lead to antenatal diagnosis of SCD in fetus and action can be taken accordingly.

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