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Perinatal Outcome in Sickle Cell Anemia

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Summary:

This study included 104 prospective cases who were sickling positive. Their perinatal outcome was compared with similar number of non-sicklers who delivered just subsequent to the delivery of each sickler. Amongst sicklers 41.35% were 'SS' type and 58.65% were 'AS' type. The perinatal loss was 31.73% in sicklers and 10.57% in non-sicklers. There was increased incidence of low birth weight, still births and neonatal deaths in sicklers.

One should screen all pregnant women with anemia to detect haemoglobinopathy so that early, aggressive and comprehensive care would improve perinatal outcome.

Introduction:

Sickle cell haemoglobinopathy is a condition where there is abnormality in b haemoglobin chain. Association of this condition is not uncommon in pregnancy. World wide trends towards increasing survival of children with sickle cell anemia into adulthood is attributed to the improvement in state of art of medicine, obstetric and perinatal care (Powars et al, 1986). Due to lack of knowledge and attitude towards hospital delivery, this is not true in rural India. Available literature has shown low prevalence rate of sickle cell disease in different studies because of higher death rates of sicklers in childhood as well as reduced fertility in sickle cell disease. However, the prevalence of sickle cell trait has been reported to be 10-30% in many tribes in India (Roy Choudhary, 1984). The present study was undertaken to find out the prevalence of sickle cell disease and trait amongst the pregnant women and perinatal outcome in sicklers in our area.

Material and Methods:

This study was done in the department of Obstetrics and Gynaecology, MGIMS, Sevagram. There were 870 antenatal cases who were screened for sickling. Blood samples from these patients were subjected to cellulose acetate paper electrophoresis to find out type ('SS', 'AS' or 'AA') of haemoglobin. Of these 870 patients 104, were sickling positive giving an incidence of 11.95%. The perinatal outcome of sickling positive cases which comprised of study group was analysed. A similar number (104) of non-sicklers who delivered just subsequent to delivery of each sickler served as control.

Observations:

There were 104 sicklers. The haemoglobin electrophoresis of these cases revealed that 61 (58.65%) cases were heterozygous (AS type) and 43 (41.35%) were homozygous (SS type). In these cases degree of anemia

was analysed depending on their haemoglobin make up. This showed there were 32 cases (30.77%) in 'SS' type and 9 cases (8.65%) in 'AS' type who had haemoglobin less than 6 gm percent (Table – I).

Table I
Haemoglobin Percentage in different groups

Haemoglobin Percent	Sicklers n=104 (100%)			Non-sicklers n=104 (100%)
	SS	AS	Total	AA
<6	32	9	41	11
	(30.77)	(8.65)	(39.42)	(10.57)
6-9	8	30	38	27
	(7.39)	(28.85)	(36.53)	(25.96)
>9	3	22	25	66
	(2.88)	(21.15)	(24.03)	(63.46)
	43	61	104	104
	(41.35)	(58.65)	(100)	(100)

Figures in paranthesis show percentage.

Amongst the sicklers total perinatal loss (still birth + early neonatal loss) was 33 (31.73%) as compared to that in non-sicklers where it was 11 (10.57%). Further analysis of perinatal outcome had shown that in homozygous sicklers there were 6 (5.76%) still births and 74 (13.46%) early neonatal deaths. In heterozygous sicklers the still births and early neonatal deaths were 3 (2.88%) and 10 (9.61%) respectively. Similar analysis in non-sicklers showed 3 (2.88%) still births and 8 (7.69%) early neonatal losses. (Table II).

Table II
Neonatal outcome in sicklers & non-sicklers

Neonatal outcome	Sicklers n=104 (100%)			Non-sicklers n=104 (100%)
	SS	AS	Total	AA
Full term	18	45	63	89
live birth	(17.30)	(43.26)	(60.57)	(85.57)
Preterm	5	3	8	4
live birth	(4.80)	(2.88)	(7.69)	(3.84)
Still birth	6	3	9	3
	(5.76)	(2.88)	(8.65)	(2.88)
Early neonatal	14	10	24	8
Death	(13.46)	(9.61)	(23.07)	(7.69)
	43	61	104	104
	(41.35)	(58.65)	(100)	(100)

$p<0.001$

Figures in paranthesis show percentage.

It was observed that babies with birth weight below 2 kgs were 16 (15.38%) in sicklers as compared 6 (5.76%) in non-sicklers. Birth weight of more than 2.5 kgs was observed in homozygous sicklers in 9 cases (8.65%) as compared to 44 (42.30%) in heterozygous sicklers (Table III).

Table III
Relation of type of haemoglobin with body weight

Body weight In Kgs	Sicklers n=104 (100%)			Non-sicklers n=104 (100%)
	SS	AS	Total	AA
< 2	9	7	16	6
	(8.65)	(6.73)	(15.38)	(5.76)
2.1-2.5	25	10	35	15
	(24)	(9.61)	(33.65)	(14.22)
> 2.5	9	44	53	83
	(8.65)	(42.30)	(50.96)	(79.80)
	43	61	104	104
	(41.35)	(58.62)	(100%)	(100%)

Figures in paranthesis denote percentage.

Apgar score in 95 newborns of sicklers and 101 of non-sicklers (only for live births) is depicted in Table IV. The mode of delivery in all groups were more or less same. It was observed that low apgar score (less than 4) at 5 minutes was found in 7 (7.36%), 9 (9.47%) and 3 (2.9%) cases in 'SS', 'AS' and 'AA' types respectively.

Table IV
Relationship of Apgar score with type of haemoglobin.

Apgar Score	Sicklers n=95 (100%)			Non-sicklers n=101 (100%)
	SS	AS	Total	AA
< 4	7	9	16	3
	(7.36)	(9.47)	(16.84)	(2.97)
4-7	7	14	21	5
	(7.36)	(14.74)	(22.10)	(4.95)
> 7	20	38	58	93
	(21.05)	(40)	(61.05)	(92.07)
	34	61	95	101
	(35.78)	(64.21)	(100%)	(100%)

Figures in paranthesis denote percentage.

Discussion:

Sickle cell positive cases were 11.95% in present study. The incidence of heterozygous sicklers (AS) was found to be higher as compared to homozygous sicklers (SS) viz 58.65% against 41.35% respectively. This indicated 'AS' type to be commoner than 'SS' type. This is because of higher death rate of children of sickle cell disease as compared to sickle cell trait, as well as reduced fertility in those who survive (Donald, 1979; Swiet, 1995).

Stillbirth occurred in 8.65% & 2.88% cases in study and control group respectively. Of these 8.65% cases of stillbirth, 5.76% were of 'SS' type and 2.88% were of 'AS' type. Milner et al (1980) and Poddar et al (1986) found this to be 13% & 12.8% respectively in homozygous sicklers. The pathological basis of perinatal salvage suggested by various authors (Morrison – 1979, Perkins – 1971) is that sickling occurs in the arcuate arteries of uterus which would decrease myometrial and placental blood flow and thus slowdown placental fetal growth

and decrease maternal fetal exchange. Usually the late intrauterine death occurs concomitantly with severe maternal crisis, with evidence of placental sickling, infarction or both. Although possible, there have been no reported stillbirths due to sickling in Hb 'SS' fetus in utero. To prove, the placental pathology, histopathology should be mandatory (Roy Choudhury, 1984). Since in present series placental histopathology was not done it is difficult to opine.

The perinatal mortality (PNM) was nearly 3 times more in sicklers than in non-sicklers viz 23.07% against 7.69% respectively. Analysing the PNM amongst the type of sickler it was observed that the incidence is higher in SS type as compared to AS type (Table II).

The neonatal death was reported to be 5% by Poddar et al (1986) and 22% by Fort et al (1971) in homozygous sicklers, which could be due to low birth weight (LBW) children born to SS mothers. Additional fact could be lack of well equipped neonatal ICU attributing to PNM.

In the present study low birth weight babies were present in both SS and AS type. Brown et al (1972) had reported that women with sickle cell trait give birth to smaller babies than women free of the condition. This may be related to lack of oxygenation for developing fetus. We believe that the cause of IUGR and LBW seems to be multifactorial and anemia with sickling in maternal sinuses or placenta is contributory.

The apgar at 5 minutes was less than 4 in 7.36% of 'SS' type, 9.47% of 'AS' type and 2.97% of 'AA' type in the present study (Table – IV). The apgar score were identical in 'AS' and 'AA' types in the study of various authors (Mc Curdy, 1964; Perkins, 1971; Pritchard et al, 1976) suggesting that the baby is least affected in 'AS' type.

One should screen all cases of anemia to detect haemoglobinopathy and take extra care in its management. Pregnancy can be successfully completed in most women with major sickle haemoglobin abnormality with an expectation of nearly same outcome for mother and infant. It requires early, aggressive and comprehensive perinatal care which dramatically can improve perinatal outcome.

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