

Study of Placenta in Sickle Cell Disorder

A.C. Shrivastava • Mrs. D.M. Bhat • Anita Assudanti • S. K. Bobhate
Dept. of Pathology, Govt. Medical College and Hospital, Nagpur.

Summary: The present study was undertaken to find out various pathological changes in placenta in patients with sickle cell disorder as compared to controls. We found increase in the incidence of fetal wastage in general and increase in placental infarction on gross examination which was more in patients with sickle cell disease than sickle cell trait. Microscopic findings included increase in infarction, syncytical knot formation, fibrinoid necrosis, presence of sickled RBCS in intervillous space, intervillous calcification and cytotrophoblastic proliferation.

Introduction

The placenta which has been a neglected organ previously is now being used as an instrument for research into basic cell biology (Beaconsfield 1979). The present study includes gross and microscopic examination of placenta in patients with sickle cell disorder as compared to normal controls and the effect of maternal sickling on the fetus via placenta.

Materials and Methods

Fifty eight cases from the Department of Obstetrics, Govt. Medical College and Hospital Nagpur, which included the placentas of cases delivered during the period Jan. 92 to Sept. 93, were studied. The cases were divided into two groups.

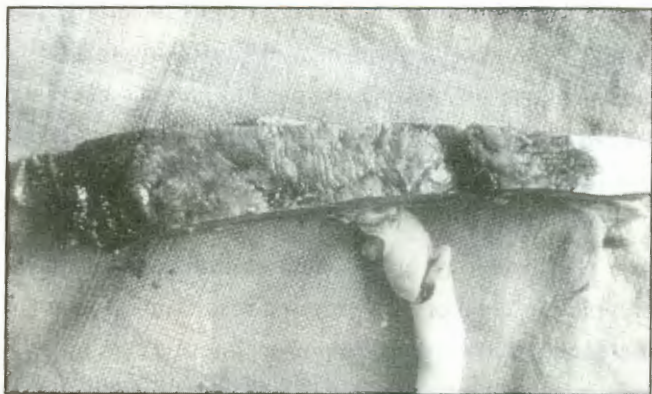
Group I : Control (Placentas from normal delivery.)
20 cases Baby wt > 2.5 kg.
Gestational Period > 37 wks.
Blood Pressure < 140/90mm. of Hg throughout Pregnancy.

Group II : Cases with sickle cell disorder
31 cases - 'AS' pattern (Sickle Cell Trait)
07 cases - 'SS' pattern (Sickle Disease)
38 Total cases.

Detailed clinical history and baby notes were taken.

Collection and Examination of placenta :

In both the groups fresh placentas were collected and examined. Inspection and gross examination of the placenta was done by using proforma by Benirschke (1961), Fixation was done in 10% formalin solution. Sections were taken from central and peripheral regions of the



Photograph showing old blood clot from a case with sickle cell disease

placenta, midportion of cord and membranes and grossly abnormal lesions if any (Benirschke, 1961). All sections were formalin fixed paraffin embedded and stained by routine H & E.

Results

Results are tabulated as following.

Table No. 1 : Shows distribution of age, parity and associated systemic disease.

Table I

Parameters	Group I	Group II
Age < 20	25%	7.89%
20 - 30	65%	86.85%
> 30	10%	5.26%
Parity		
Primiparous	40%	39.47%
Multiparous	60%	60.52%
Associated Systemic Disease		
Toxaemia of Pregnancy		18.42% (7)
Valvular Heart Disease		23.68% (9)
Tuberculosis		2.63% (1)

Table II

Parameters	Group I	Group II
Average Placental wt	455 gms	510 gms
Average fetal wt	2610 gms	2353 gms
Average fetoplacental weight ratio	5.73 : 1	4.69:1
Placental Infarction	60% (12)	92.10% (35)
Extent of Infarction	0.5% in all cases	0.5% (6 cases) 6-10% (24 cases) 11-15% 5 cases
		Meconium stained in 18.42% (7 cases)

Table No. II - Shows gross examination findings.

Table No. III - Shows histopathological findings of placentas in group 1 & group 2.

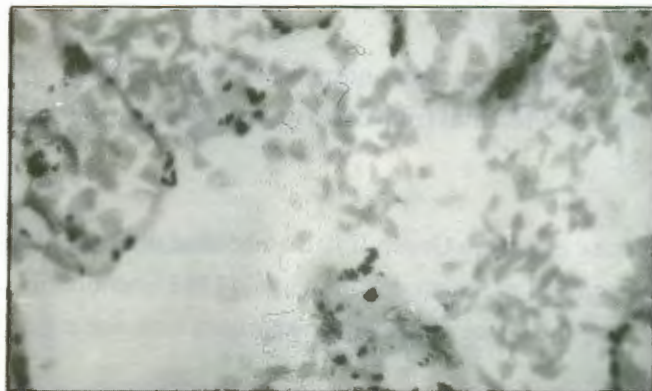
Maximum no. of patients from group 2 were Mahar by caste (71.05%) followed by Teli (15.78%) and Kunbi (13.15%)

Discussion

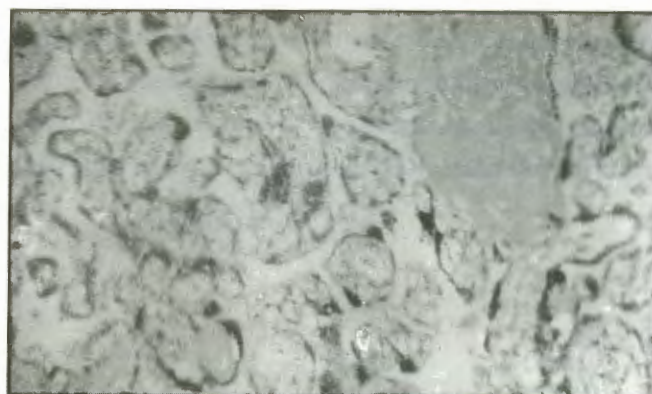
The present study was undertaken to find out various

Table III

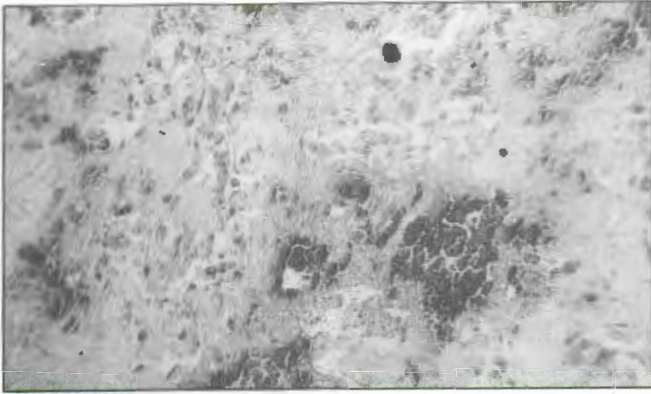
Parameters	Group I	Group II
Infarction	60% (12)	92.10% (35)
Syncytial Knot formation		
< 30%	70% (14)	7.8% (3)
30-50	30% (6)	76.31% (29)
> 50%		15.78% (6)
% of Villi showing vasculo Syncytial membrane		
<6%	10% (2)	31.87% (12)
>6%	90% (18)	68.42% (26)
% of Villi showing stromal fibrosis in > 2% villi		7.83% (3)
Cytotrophoblastic Proliferation	5% (1)	31.57% (12)
Calcification	55%(11)	86.84% (33)



Photomicrograph showing sickled red blood cells in the intervillous space from a case with sickle cell disease H & F x 200



Photomicrograph showing increased syncytial knotting and fibrinoid necrosis from a case with sickle cell trait. (H&E, x 200).



Photomicrograph showing villous and intervillous calcification from a case with sickle cell trait (H & E x 100)

pathological changes in the placenta in patients with sickle cell disorders as compared to controls. A total no. of 58 cases were studied and divided into two groups.

Group I : Placentas from normal deliveries (20)

Group II : Placentas from cases with sickle cell disorders (38)

Seven cases from Group II had associated diseases like toxemia of pregnancy. Susan et al (1983) mention increased incidence of toxemia in patients with sickle cell disease.

Eight cases from Group II (21.05%) developed sickle cell disease during pregnancy. (combination of vaso occlusive and hemolytic crises.)

The average placental weight, average fetal weight and fetoplacental weight ratio in the present study are comparable with similar findings of Beisher et al (1970) who found that the placental hypertrophy was associated with

maternal anaemia. There is increased incidence of fetal wastage in cases with sickle cell disorder (Anderson and Kay, 1966).

Gross examination of placenta revealed infarction varying from dark red to white. The extent of infarction varied depending upon whether the patient had sickle cell disease or trait. Infarction involved larger areas of placenta in cases with sickle cell disease.

Histological examination of placentas revealed changes in villi in patients with sickle cell disorders in the form of infarction, congestion and formation of ghost villi, increased syncytial knot formation as a response to trophoblastic ischaemia or hypoxia, increased fibrinoid necrosis, presence of sickled R B Cs in the intervillous space, areas of villous and intervillous calcification and cytotrophoblastic proliferation in response to hypoxia.

Thus the lesions of placenta affect the fetus in a number of ways hence gross and microscopic examination of placentas should be routinely done.

References

1. Anderson W.R. and MC Kay D.J. Amer Jour. Obst. Gynace. 95:1134, 1966
2. Beaconsfield, P. Placenta: A neglected experimental animal Oxford. Pergamon 1979
3. Beischer, N.A, R. Sivasambo, S. Vohra, Suporn Silpissarukasal and S. Reid. Jour. Obst. Gynace. Brit Common 77:398-409-1970
4. Benirschke, K: Amer. Jour. Obst. Gynaec. 84 : 1595, 1961.
5. Susan M.W. W. Studa, Jr. J.M. White, Brit. Jour. Obst. Gynace. 90:112, 1983.