International Journal of Medical and Health Sciences

Journal Home Page: http://www.ijmhs.net ISSN:2277-4505

Original article

Histological Changes in the Placentae from Severe Anaemic Mothers

Rohini M[1], Yogesh AS[*2], Goyal M[3], Praveen Kurrey[4]

[1]Senior Resident, Department of Anatomy, All India Institute of Medical Sciences, New Delhi.
[2]Assistant Professor, Department of Anatomy, Sri Aurobindo Medical College and Postgraduate Institute, Indore, MP
[3]Professor, Department of Anatomy, Pt. J.N.M. Medical College, Raipur, Chhattisgarh
[4]Assistant Professor, Department of Anatomy, Chirayu Medical College and Hospital, Bhopal, M.P.

ABSTRACT

The information provided from the pathological assessment of the placenta may provide important clinical information for both the mother and the neonate. Anaemia in pregnancy is a well recognized obstetric hazard, observed more frequently in developing countries. The present study was undertaken to analyze placental changes in the anaemia with a view to assess the significance of villous abnormalities by histopathological methods. Placentae from thirty mothers with uncomplicated pregnancy as control group and thirty mothers with severe anaemia as study group were studied. It was found that mothers with severe anaemia had smaller placentae. Gross examination of placentae revealed presence of foci of calcification and infarction. On light microscopic examination, the striking villous abnormalities were observed in the study group which included increased syncytial knot formation, stromal fibrosis, fibrinoid necrosis, medial coat proliferation of foetal blood vessels leading to obliterative endarteritis, intervillous haemorrhage, calcification, hyalinisation of the villi and cytotrophoblastic cell proliferation. These findings were on higher side in the study group than in the control group. The histomorphological findings of placenta in anaemic mothers might be an adaptation to maternal hypoxia.

KEYWORDS: Placenta, Maternal anaemia, Histology, Placental villi

INTRODUCTION

Placenta is the idiosyncratic feature of eutharian mammals. It is one of the most important first growing organs, which is responsible for bonding between the mother and the foetus in all aspects. As placenta serves to maintain a maternal-foetal interference for the exchange of blood gases, nutrients and wastes; normal foetal growth and survival depends on its proper development [1]. Also it acts as a metabolic and endocrine organ of supreme importance, both in the maintenance of pregnancy and development of the foetus. It also represents the only point of contact between maternal and foetal tissues and plays a dominant role in the immunological acceptance of the foetal graft by the mother. Thus it should not be considered as inactive conduit for fetomaternal transfer [2, 3, 4]. After delivery, if the placenta is examined minutely, it provides much insight into the prenatal health of the baby and the mother.

Severe anaemia during pregnancy is a potentially hazardous haematological disorder. It is associated with late abortions, prematurity, low birth weight and stillbirths [3, 5]. The sum of which is increased perinatal loss. The global prevalence of anaemia in pregnancy is 55.9% [6]. Anaemia in pregnancy is a well recognized observed more frequently in developing countries. In India incidence of anaemia in pregnancy has been noted as high as 40-80%. About 4-16% of maternal deaths are due to anaemia. It also increases the maternal, foetal and neonatal mortality & morbidity significantly [7]. The severity of anaemia among expectant mothers is judged by the criteria suggested by WHO [6]. According to this, anaemia is classified as mild degree (9-11 gms%), moderate (7-9 gms%), severe (4-7gms%) and very severe (< 4 gms%). The present study was undertaken to analyze placental changes in the anaemia with a view to assess the significance of villous abnormalities by histomorphological methods because these changes serve as a guide to the duration and severity of disease.

MATERIALS AND METHODS

For the present study sixty full term placentae were taken from mothers who delivered either vaginally or by caesarian section, from the Department of Obstetrics and Gynaecology of tertiary care hospital. Out of these, thirty placentae were of mothers with uncomplicated pregnancy (control group) and thirty were of those with moderate to severe anaemia (study group). Out of thirty placentae of anaemic mothers, 21 placentae were from patients of severe anaemia (Hb < 7gm %) and 9 were from patients of moderate anaemia (Hb < 7-9 gm %). In both groups, mothers were examined clinically (for height, weight, blood pressure, pulse, anaemia, jaundice etc.) along with recording of their medical history (history of past illness, history of previous child birth etc). Their investigation reports were checked (blood sugar, urea, creatinine, haemoglobin levels, urine for albumin, and pus cells along with ophthalmoscopic examinations).

The placentae were collected soon after the delivery and cleaned keeping one centimetre long umbilical cord. Weight of the placenta and neonate was noted. Perfusion of placentae with 10% formalin through umbilical vessels was followed by immersion of the placenta in a jar containing 10% formalin for 48 hours. After that gross examination of the placentae for presence of any infarction, calcification and retro placental clots was done. Tissues each of 2×2 cm were taken from following placental sites and processed for histological observations for light microscopic studies. Tissues were taken from histological assessment from the following placental sites: near the attachment of umbilical cord, margin and centre of the placenta. It was also examined for the presence of calcified and infracted area if any. Slides were stained with Haematoxylin and Eosin (H & E) for general scrutiny and to study the histology of placenta. Slides were also assessed using Masson's Trichrome for fibrosis. In light microscopic examination of the placental villi were screened for counting of number of syncytial knots per 100 villi, fibrinoid necrosis, stromal fibrosis, medial coat proliferation of foetal blood vessels, intervillous haemorrhage, hyalinised villi, cytotrophoblastic cellular proliferation and calcification.

RESULTS

In the present study, mean weight of the placenta was significantly lower in anaemic groups (410.00 gms) than in the control group (462.16 gms) [Table 1]. On the gross examination of the placentae, calcification was found more common in placentae of anaemic cases (63.33%) than that of control group (26.66%). The areas of infarction in placentae of control group (3%) were observed significantly less than that in the anaemic cases (56.67 %) [Table1].

In light microscopic examination of the placental villi showed significant changes in the study group. The number of areas of Syncytial knots in

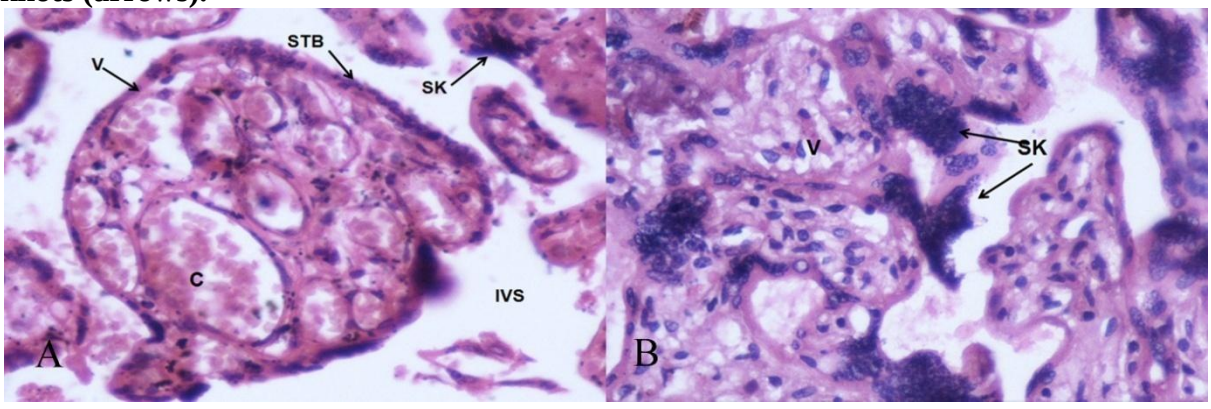
anemic group (62.83/100 villi) was significantly increased than in control group (26.93/100 villi) [Fig-1]. The number of areas of fibrinoid necrosis in anaemic group (10.03/ 100 villi) was also found to be increase than in control group (3.51/100 villi) [Fig-2A]. Stromal fibrosis and medial coat proliferation of foetal blood vessels were also increased in anaemic group (53.33% and 36.66% cases) than that in control group (23.33% and 10% cases respectively) [Fig-2B & 3A). Intervillous haemorrhage was absent in control group but observed in more than half of the anaemic cases (63.33%) [Fig-3B]. Hyalinised villi and calcification were found more commonly in anaemic group (73.33% and 46.66% cases) than that in control group (36.66% and 36.66% cases respectively). The mean area of cytotrophoblastic proliferation was less in anemic group (3.29) than in control group (3.46), but statistically nonsignificant [Table 1].

Table 1: Comparative analysis of Histolomorphology of placantae from control and anaemic mothers

Parameters	**Control group** (N = 30)	**Anaemic group** (N = 30)	**p-value**
Placental weight (gms)*	462.16 ± 66.27	410.00 ± 61.81	0.0026
Areas of calcification$	08 (26.66%)	19 (63.33%)	0.0464
Mean areas of infarction$	01 (03%)	17 (56.67%)	0.0012
Syncytial knots per 100 villi*	26.93 ± 10.49	62.83 ± 19.14	< 0.0001
Fibrinoid necrosis*	3.73 ± 1.99	10.03 ± 3.51	< 0.0001
Stromal fibrosis$	07 (23.33%)	16 (53.33%)	0.0455
Medial coat proliferation$	03 (10.00%)	11 (36.66%)	0.0005
Intervillous haemorrhage$	00	19 (63.33%)	< 0.0001
Hyalinised areas$	04 (13.33%)	22 (73.33%)	0.0022
Calcification$	11(36.66%)	14 (46.66%)	0.1275
Cytotrophoblastic cellular proliferation*	3.46 ± 1.04	3.29 ± 1.09	0.5390

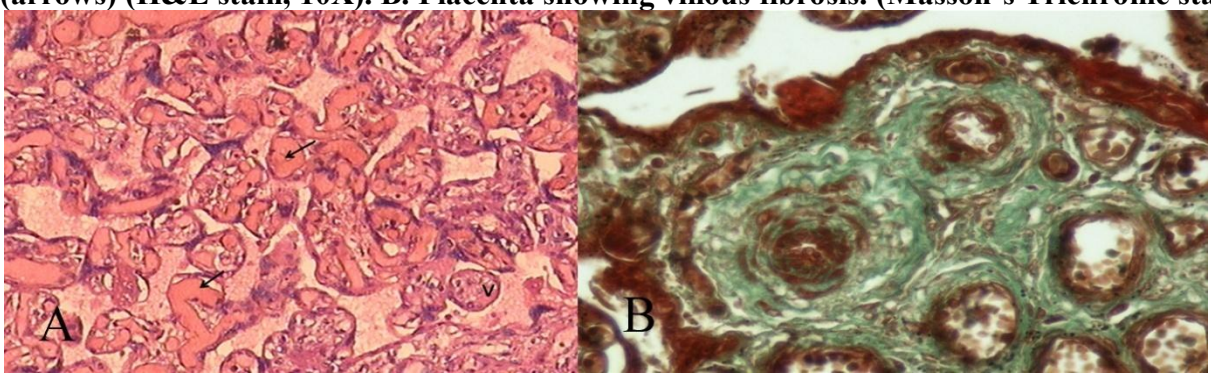
*unpaired 't' test was applied for these parameters ,$ chi square test was applied for these parameters ,
Significant change if p value is < 0.05

Figure-1: Histology of Placenta. (H & E Stain, 40X) A: Placenta from Control group with well vascularised chorionic villi. B. Placenta from severe anaemic mothers showing increased syncytial knots (arrows).



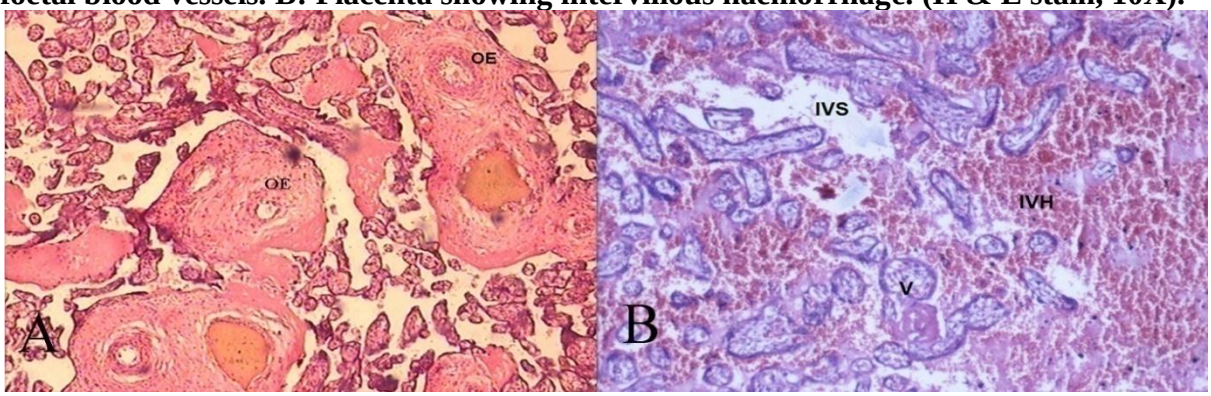
V: villus; STB: syncytiotrophoblast; SK: syncytial knots; IVS: intervillus space; C: capillary.

Figure-2: Placenta from severe anaemic mothers. A. Placenta with showing areas of fibrinoid necrosis (arrows) (H&E stain, 10X). B. Placenta showing villous fibrosis. (Masson's Trichrome stain, 40X).



V: villus.

Figure-3: Placenta from severe anaemic mothers. A. Placenta showing obliterative endarteritis of foetal blood vessels. B: Placenta showing intervillous haemorrhage. (H & E stain, 10X).



IVS: intervillous space; V: villus; OE: obliterative endarteritis; IVH: intervillous haemorrhage.

DISCUSSION

The chorionic villi are the functional unit of placenta. It provides oxygen and nourishment to foetus and also serves as excretory unit [1]. Thus it can be regarded equivalent to lung acini as well as renal glomeruli. The histological appearance of chorionic villi varies with the gestational age and with the stage of development and maturations of villous tree. Oxygen plays a vital role in the development of placenta, as it is known key factor in the regulation of cytotrophoblastic differentiation, proliferation and invasion in early pregnancy [8]. Anaemia in pregnancy is associated with increased incidence of both maternal and foetal morbidity and mortality [9]. Hypoxia is responsible for the placental changes in women with anaemia [10]. Beischer et al observed that the maternal anaemia was associated with placental hypertrophy [10]. Lao TT et al noted increase in the placental weight of anaemic patients contrary to the present study [11]. This might be due to that Lao has studied the placenta in mild anaemic mothers. Mild anaemia might have produced placental hypertrophy as explained by Beisher et al [10]. But in our study, severe anaemia produced small placentae. In earlier reports as well as in the present study, the gross examination of the placentae from anaemic mothers showed higher incidence of calcification

and infaction [12, 13]. This might be probably due to the decreased size of placentae.

Syncytial knots are consistently present, increasing with increasing gestational age, and can be used to evaluate villous maturity. Earlier study also showed increase in syncytial knots in low oxygen tension [14]. The probable reason behind the increased syncytial knots formation in the anaemic group may be explained by findings of Kristina et al (2009) who mentioned that increased syncytial knots are associated with conditions of uteroplacental malperfusion [15]. Increased syncytial knots in placentae in anaemia suggested that an attempt was being made to form new villi so as to increase an effective surface area for exchange [16]. Fibrinoid necrosis is seen as a nodular mass of homogenous acidophilic material in the villi. Fibrinoid necrosis has been considerd as a hallmark of an immunological reactions within the trophoblastic tissue [1]. In the present study, significantly increased fibrinoid necrosis in placentae of anemic mothers might be evolved due to degenerative changes in villous cytotrophoblasts [17].

Obliterative endarteritis of the fetal stem arteries is characterized by swelling and proliferation of intimal cells, together with thickening and reduplication of the basement membrane. In the present study, stromal fibrosis and medial coat proliferation of foetal blood vessels were also increased in anaemic group. This increase incidence of stromal fibrosis may be related to obliterative endarteritis which was found in placentae of anaemic group. This fibrosis and obliterative endarteritis may be a result of relative hypoxia [18]. Intervillous haemorrhage was also observed in placentae of anaemic group only by Rangnekar et al [12] There was significant increase in calcification in placentae of anaemic groups in comparison to control group [13]. Cytotrophoblastic cellular proliferation was significantly lower in anaemic group which might be also an adaptive response to decreased oxygen supply. [19]. All these changes in placentae of anaemic mothers may be the cause of worse foetal outcome and maternal hazards. This signify need of assessment of placenta at the time of delivery.

CONCLUSION:

Severe anaemia in pregnancy alters the placental histomorphology. To avoid the placental malformation, prompt treatment of anaemia is required.

REFERENCES

1. Fox H. Effect of hypoxia and trophoblast in organ culture. Am J Obs Gynaecol 1970; 107: 1058-1064.

2. Udainia A, Jain ML. Morphological study of placenta in Pregnancy Induced Hypertension with its clinical relevance. J Anat Soc Ind 2001; 50(1): 24-27.

3. Morgan RP. Immunology of term and preterm labor. Repro biol endocrinol 2003; 1: 122.

4. Errol RN, Danny JS, Susan JF. Implantation and survival of early pregnancy. N Engl J Med 2001; 345: 1400-1408.

5. Klebanoff MA, Shrono PH, Selby JU, Trachtenberg AI, Gaubard BI. Anaemia and Spontaneous preterm birth. Am J Obstet Gyenaecol 1991; 164: 59-63.

6. WHO Report. Technical Report 1989; 776: p 308-310.

7. Anaemia in pregnancy. By Dr Samar K Basu, Senior Consultant (Obst &Gynae). [Monograph on the internet] G M Modi Hospital. [Cited on 2012 Oct 8]. Available from: http://delhimedicalcouncil.nic.in/Anemiainpregnancy.pdf

8. Nabil Aziz. Recurrent Pregnancy Loss and Oxidative Stress. Humana Press, Springer Science Business Media, New York. 2013. p 131-141.

9. Breymann C. Iron deficiency and anaemia in pregnancy: modern aspects of diagnosis and therapy. Bld Cel Molecul Dis 2002; 29(3): 506-516.

10. Beischer NA, Sivasamboo R, Vohra S, Silpisornkosal S, Reid S. Placental hypertrophy in severe pregnancy anaemia. J Obs Gynaecol Br Common Wl 1970; 77: 398-340.

11. Lao TT, Wong WM. Placental ratio - its relationship with mild maternal anaemia. Placenta 1997; 18(7): 593-596.

12. Rangnekar AG, Darbari R. Placental changes in pregnancy anaemia, A study of one hundred cases. J Obs Gynaecol Ind 1993; 43(4): 473-478.

13. Rusia U, Kapor S, Madan N, Nair V, Sood Sk. Placental morphology and histochemistery in iron deficiency anaemia. Ind J Med Res 1998; 87: 468-474.

14. Aplin JD. Hypoxia and human placental development . J Clin Invest 2000; 105(5): 559-560.

15. Kristina L, Raanan S, Rebecca B. Syncytial knots as a Reflection of Placental Maturity: Reference values for 20 to 40 weeks gestational age. Pediat Develop Path 2009; 28; 28-37.

16. Capellini I. The evolutionary significance of placental interdigitation in mammalian reproduction: Contributions from comparative studies. Placenta 2012; 33 (10): 763-768.

17. Verma R, Mishra S, Kaul JM. Ultrastructural changes in the placental membrane in pregnancies associated with diabetes. Int J Morphol 2011; 29(4): 1398-1407.

18. Gersell DJ, Kraus FT. Diseases of the Placenta. In: Kurman RJ, Ellenson LH, Ronnett BM, editors. Blaustein's Path Female Genital Tract. 6th ed. 2011. Springer US. P-999-1073.

19. Shashi MM, Jain SK, Yadav M. Placenta: The Wonder Organ. Ind Acsd Forensic Med 2011; 33(2): 140-142.

*Corresponding author: Dr. Yogesh Sontakke

E-mail: dryogeshas@rediffmail.com