Placental changes associated with maternal anaemia

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SUMMARY

The present study aimed to assess the morphological and histological changes of placentas associated with maternal anaemia (mothers with Hb level <11 g/dl). The study was conducted in Bankura Sammilani Medical College, West Bengal, India for a period of six months. Placentas collected from cases of maternal anaemia (Hb < 11 g/dl) were forty (40) and those collected from control mothers were thirty (30). All the deliveries were at full term (37-42 weeks) and in the antenatal periods were without any complications or diseases. Macroscopic and microscopic analyses of the placentas were done and these findings were compared. Statistical analysis was performed by using t-test for comparing the mean values of fetal weights, placental weights, placental indices and placental volumes of the maternal anaemia group with those of control group. It was observed that the mean fetal weight of pregnancy with anaemia group was less than those of the control group. The mean placental weight and mean placental volume in pregnancy with the anaemia group were more than the control group. Also, the mean placental index of the maternal anaemia group was higher than that of the control group. Light microscopy revealed increased fibrin deposition, increased syncytial knotting, more avascular villi with incomplete trophoblastic lining, stromal fibrosis, intervillous haemorrhage and cytotrophoblastic cell proliferation in placentas of anaemic mothers as compared to those of the controls. We concluded that mater-

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nal anaemia resulted into bigger, heavier placentas and smaller fetuses, whereas placental morphological changes showed signs of chronic hypoxia and placental insufficiency.

Key words: Maternal anaemia – Placental weight – Fetal weight – Histology

INTRODUCTION

Anaemia in pregnancy is very common, but severe anaemia in pregnancy may have adverse effects on pregnancy, delivery and neonatal infants. Maternal anaemia is an independent risk factor for pre-term delivery and low birth weight (Levy et al., 2005). Anaemia is said to have occurred in pregnant women when the hemoglobin level is less than 11 g/dl (WHO, 2008). The intrauterine existence of the fetus is dependent on the placenta. It is elaborated by both maternal and fetal tissues to serve as an instrument of transfer of essential elements, i.e., nutrients and oxygen from mother to embryo, and the waste products of metabolism from embryo to the mother. Researchers like Barker et al. (1990), Godfrey et al. (1991), Lao and Tam (2000) have all concluded that maternal anaemia was associated with larger placental weights, reduced fetal birth weights, and higher ratios of placental weight: fetal weight.

The observations of Dhall (1994) were in contradiction to these; she reported that in anaemia the weight and volume of placenta decreased, though the result was statistically insignificant. According to Agarwal (1991), placental histology in maternal anaemia showed decreased villous vascularity and

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increased villous fibrosis. Joshi et al. (1996) reported villous hypovascularity, increased syncytial knots and fibrinoid necrosis in maternal anaemia. In spite of the extensive research on the effects of maternal anaemia on the fetus and the placenta, the observations were contradictory. The present study aimed to assess the fetal size, as well as other morphological and histological changes of placentae associated with maternal anaemia.

MATERIALS AND METHODS

The present study was carried out in Bankura Sammilani Medical College, West Bengal, India. Departmental and Institutional permission was taken to carry out the study. Seventy (70) full-term freshly delivered placentas were collected both after normal deliveries and Caesarean sections. Informed consents of the mothers were taken. Forty (40) placentas were collected from cases of maternal anaemia (Hb < 11 g/dl) and thirty (30) placentas were collected from mothers without anaemia (Hb >11 g/dl) and taken as controls. Gestational ages were established from Last Menstrual Periods, sometimes from USG reports.

The study was carried out for a period of six months; placentas were collected in first four months. All the deliveries, both control group and anaemic group, were at full term (37-42 weeks) and the antenatal periods were without any complications or diseases like pre-eclampsia or diabetes.

Birth weights of fetuses were noted from the Clinical sheets. The membranes were trimmed up to the margins of the placentas; umbilical cords were cut at about 5 cm from their insertions. Clots were removed. The placentas were dried with blotting paper. Then the following were recorded:

Weights of placentas were measured by means of an electronic weighing scale.

Volumes of fresh placentas were determined by water displacement technique.

Placental index was calculated by dividing placental weight from fetal weight.

From the placentas pieces of tissue were obtained: each piece included the entire thickness of the placenta, from maternal to fetal surfaces. The tissues were then fixed in 10% formalin solution, processed for wax-embedding and section-cutting (Carleton's Histological Technique, 1980). Seven-micrometer sections were cut and stained with Harris' Haematoxylin and Eosin stain and Masson's Trichrome stain. At least 10 sections were made from each placenta.

Statistical analysis: Statistical analysis was performed by using *t*-test for comparing the mean values of fetal weights, placental weights, placental indices and placental volumes of maternal

anemia group with those of control group.

RESULTS

We observed that in placentas of the maternal anaemia group birth weights of fetuses ranged from 1250-3050 gm (average/ mean 2376.25 gm, mode 2250 gm). Weights of placentas in this group ranged from 220-500 gm (mean 382 gm, mode 450 gm). Mean placental index was calculated to be 0.15, mode being 0.13 (range 0.11-0.26). Placental volumes ranged from 170-500 ml (average/ mean 330.5 ml, mode 400 ml). In the control group of our study, the birth weights of fetuses ranged from 1750-3500 gm (mean 2595 gm, mode 2750 gm). Weights of placentas ranged from 250-500 gm (average 370.3 gm, mode 450 gm). Mean pla-

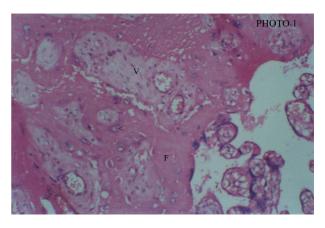


Fig. 1. Microphotograph of a group of villi (V) entrapped in perivillous fibrin deposits (F) showing degenerated trophoblastic lining, reduced number of capillaries within the villi, and few large extra-villous trophoblastic cells (X-cells) of "clear" variety (arrow) in a placenta associated with maternal anemia. Stain used: H & E, x400.

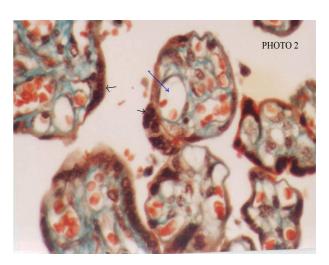


Fig. 2. Terminal chorionic villi showing markedly increased syncytial knots with higher numbers of nuclei (arrow), cytotrophoblasts within villous core, cytotrophoblasts lining the villi at places only and congested capillaries (blue arrow) are seen in a placenta associated with maternal anemia. Stain used: Masson's Trichrome, x400.

cental index was 0.13 and mode was 0.13 (range: 0.08-0.2). Placental volumes of control group ranged from 150-400 ml (mean 278 ml, mode 320 ml).

In the present study we observed that mean fetal weight in maternal anaemia group was less than that of control group (significantly less, p value -0.01). The mean placental weight of the maternal anaemia group was more than that of control group, though it was not statistically significant. The mean placental volume of maternal anaemia group of the present set was more than that of control group (significantly more, p value 0.008).

We calculated that the mean placental index of the maternal anaemia group was more than that of the control group (significantly more, *p* value - 0.008). The observations are enlisted in Table 1.

Histological findings

Light microscopic studies of the histological sections of placentas under different magnifications revealed variations in villous structures at different regions of the same placenta, and also from one placenta to that of the others. Structures of the basal plates, chorionic plates and intervillous spaces also varied. These variations were observed in both anaemic and control group of placentas.

In the placentas of maternal anaemia cases, there were wider regional variations of villous structures. In most of the placentas, syncytiotrophoblastic linings were more thinned out with considerable increase in size and numbers of syncytial knots (Fig. 2). Villous cytotrophoblast cells were noted to be increased in number. Fibrin deposits within the villi (intravillous fibrin deposition) and between the villi (perivillous/ intervillous fibrin deposition) were noted to be increased in these placentas as compared to the control placentas (Fig. 1). Trophoblastic lining degeneration of the stem villi were marked at places, they were replaced by perivillous fibrin deposits. Fig. 5 shows histological features of a control placenta in low power field.

Perivillous fibrin depositions were marked in the subchorionic areas, the entrapped villi were sclerosed, lacked syncytial lining and capillaries were either reduced in number or absent. Some fibrin masses contained groups or sheets of X-cells, which are extravillous large cytotrophoblasts. Areas of *infarction* were found to be extensive, characterized by villous crowding and increased num-

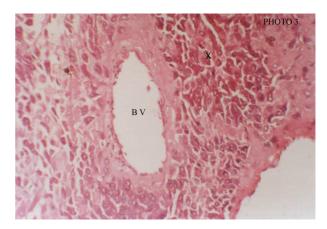


Fig. 3. Microphotograph of the basal plate of a placenta in maternal anemia showing plenty of large extra-villous trophoblastic cells (X-cells) (x) embedded in fibrinoid. Cross-section of an utero-placental blood vessel is seen (BV), its coats are replaced by fibrinoid. Stain used: H & E, x400.

ber of hypovascular villi.

Basal plates were noted to have fibrinoid deposits, presence of increased number of X-cells. The uteroplacental blood vessels were noted to be lined with X- cells at places. Their tunica media were replaced by fibrinoid. (Fig. 3).

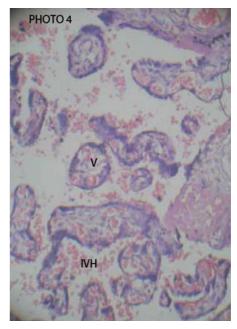


Fig. 4. Microphotograph of a placenta in maternal anaemia showing marked intervillous haemorrhage (IVH). One villus (V) is marked. Stain used: H & E, x100.

Table 1. Mean fetal weights, placental weights, volumes and placental indices of control and maternal anaemia group

Number of placentas		Fetal weight (mean)		Placental weight (mean)		PI volume (mean)		PI index (mean)	
Control	Maternal Anaemia	Control	Maternal Anaemia	Control	Maternal Anaemia	Control	Maternal Anaemia	Control	Maternal Anaemia
		2595 gm	2376.25 gm	370.3 gm	382 gm	278 ml	330.5 ml	0.13	0.15
30	40	P value 0.0	01 (significant)	P value 0.4 (not significant)		P value 0.008 (significant)		P value 0.008 (significant)	

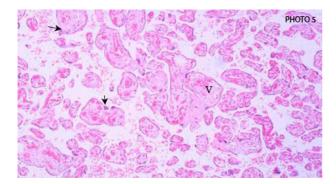


Fig. 5. Microphotograph of normal term placenta. Villi (v) are seen with syncytial knots, though less in number than anaemic placentas, (arrow) and capillaries are present within villi. Stain used: H & E, x100.

Intervillous haemorrhage was absent in control group but observed in the anaemic cases (Fig. 4).

Chorionic plates were lined by Langhan's layer of fibrinoid towards the intervillous spaces. In most of the placentae, the thicknesses of these layers were extensive with a large number of villi entrapped in them. The villi had incomplete syncytial lining and less vascularity. Large numbers of X-cells were also seen entrapped in the fibrin.

DISCUSSION

In the present study we observed that the mean fetal weight in the maternal anaemia group was less than that of control group. The mean placental weight and mean placental volume of the maternal anaemia group was more than that of control group. The mean placental index of maternal anaemia group was higher than that of the control group. Our observation was similar to those of other researchers (Beischer et al., 1970; Barker et al., 1990; Godfrey et al., 1991). Beischer et al. (1970) associated large placental weight with severe maternal anaemia. Barker et al. (1990) observed that larger placental weights and higher placental ratios were related to low maternal hemoglobin. Godfrey et al. (1991) concluded that anaemia in pregnancy was associated with larger placental weights, and higher ratios of placental weight: fetal birth weight, while fetal birth weights were reduced. Dhall (1994) observed that the weight and volume of placenta was reduced in the anaemic group but the difference was statistical insignificant.

Lao and Tam (2000) concluded that placental size increased relative to infant size in pregnancies complicated by anaemia, but whether this phenomenon reflected actual placental hypertrophy or failure of fetal growth to keep up with placental growth remains to be determined.

In the present study, light microscopy of placentae of maternal anaemia showed villi without complete trophoblastic lining, increased intravillous and perivillous fibrin deposition, increased syncytial knotting and more avascular villi as compared to

those of the controls. Similar histological changes were observed by Mehrota et al. (1972), who observed increased villous fibrosis and syncytial knotting in placentas of anaemic mothers.

Dhall (1994) also found significant increase in the number of syncytial knots in these placentas. Villous hypovascularity, increased syncytial knots and fibrinoid necrosis were also noted by Joshi et al. (1996). These features suggest hypoxia and reduced perfusion. Perivillous fibrin depositions were marked in the subchorionic areas and near the basal plates in some anaemic placentas. The entrapped villi were sclerosed, lacked syncytial lining and many were avascular. Some fibrin masses contained groups or sheets of X-cells. These perivillous fibrin depositions might be acting as a barrier between fetal and maternal circulation, thereby reducing the transfer of the essential nutrients to the fetus, these probably resulted into chronic placental insufficiency; thus causing smaller fetuses.

In the light of our observations we conclude that maternal anaemia resulted into decrease in birth weight of fetuses, and bigger placentas with increased weight and volume. Placental index is increased – this may be an evidence of compensatory hypertrophy. Placental histological changes show the signs of chronic hypoxic stress and placental insufficiency.

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