STUDY OF GROSS AND HISTOLOGICAL FEATURES OF PLACENTA IN INTRAUTERINE GROWTH RETARDATION

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ABSTRACT

Fetal growth retardation is most commonly caused by failure of the placenta to meet the increasing demands for oxygen and substrate of the developing fetus. Intra uterine growth retardation is common occurrence in Indian setup. Recent literature suggests that placental causes are more common than the maternal causes in intrauterine growth retardation.

Gross and histological study of placenta can help us to understand the pathophysiology of placental involvement. This is warranted specially in those cases of intrauterine growth retardation which are not confounded by maternal causes.

Fifty five placentae [30 from cases of intrauterine growth retardation and 25 from normal (control)] were utilized for the study. Fetal weights, placental weights and placental dimensions were measured. Tissue for histological examination was obtained from: i) Umbilical cord ii) membranes and iii) three zones in placenta. The tissues were processed and stained with Haematoxlyin and Eosin. Tissues were microscopically studied for villous and intervillous lesions utilizing various criteria. Microscopic findings were: i) increased fibrinoid necrosis (46.7%), increased perivillous fibrinoid deposition (16.7%), increased syncytial knots (60%) and increased placental infarction (1.8%). Macroscopically there was significant decrease in placental weight, fetal weight, diameter and thickness of placenta.

The findings of the present study document comparatively higher incidence of fibrinoid necrosis and perivillous fibrinoid deposition. These findings underscore the predominant role of placental causes in cases of idiopathic intrauterine growth retardation.

Key words: Chorionic villous, fibrinoid necrosis, infarction, placenta, syncytial knots.

INTRODUCTION

Intrauterine growth retardation (IUGR) constitutes an important clinical problem associated with increased perinatal morbidity (Brodsky D and Christou H)[1], higher incidence of neuro-developmental impairment (Blair E and Stanley F)[2] and increased risk of adult disease, such as diabetes and cardiovascular disease(Hales C et al. and Barker DJP et al.).[3,4] Thus, the adverse consequences of altered fetal growth are not limited to the perinatal period. The concept that an adult disease is consequence of faulty intrauterine development may have a profound impact on public health strategies for the prevention of major illnesses. Currently, no specific strategies for prevention, treatment and intervention are available in cases of altered fetal growth. In order to make significant progress in this area, a better understanding of the underlying pathophysiological mechanisms is needed(Jansson T and Powell TL).[5] Placenta is a documentation of events of gestational life (Altshuler G).[6] The placenta is one of the readily available of all human organs. However, all of us know that abnormality in the placenta may have a casual relationship to abnormal fetal outcome (Beaconsfield R and Birdwood G).[7]

In India, 30% of the babies born at term are small for dates. Under nutririon and toxemia of pregnancy are considered to be important maternal causes for this (Park K).[8] However there are cases of IUGR with no documented history of maternal and fetal factors. Earlier such cases were called as idiopathic IUGR. Recent literature suggests primary placental involvement in such cases (Cetin I and Alvino G).[9]

Literature review suggests that few studies, dating back to 7th and 8th decades of previous century, have studied placental involvement in cases of IUGR (Mallik G et al., Althshuler G et al., Mirchandani J et al., Bhatia A et al.).[10,11,12,13,14] and even fewer have studied idiopathic IUGR (Cetin I and Alvino

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G).[9] The present study was undertaken to observe various macroscopic and microscopic changes in placenta in cases of idiopathic IUGR.

MATERIAL AND METHODS

Fifty five placentas were collected from Obstetrics and Gynecology department and studied at Anatomy department of a general public hospital after obtaining due consent from the patients. Out of 55, thirty placentas were obtained from documented cases of IUGR (birth weight less than 2500gms) with no history of confounding maternal and foetal factors. Twenty five placentas for control group were obtained from normal term pregnancies without any complications. The birth weights of new born babies were taken and foetoplacental ratio was calculated in each case. The general shapes of placentas were assessed. The collected placentas were weighed by trimming the membranes and umbilical cord. Then the diameters and thickness of placentas were noted. The position of insertion of umbilical cord on the fetal surface of placenta was observed. Transverse cuts were made through the maternal surface at a distance of 1-2 cm in bread loaf manner and examined for the pale areas. All placentas were immersed in 10% formalin overnight and examined on the next day. For each placenta, blocks containing cord, membrane and full thickness of villous tissue were prepared. Whole thickness villous tissue blocks were obtained from three zones, i)central zone ii) peripheral zone and iii) intermediate zone between the first two zones, so as to include all areas of placentae. The tissues were processed and stained with Haematoxlyin and Eosin. Microscopic study of placenta was carried out utilizing a set of standard criteria for villous and intervillous lesions (Mallik G et al.).[10]. For studying these criteria 8 random micrscopic fields were chosen and 100 villi were counted in each field and studied for the presence of following criteria:

Villous lesions

a) Syncytial knots >30% in one field

b) Fibrinoid necrosis >5% in one field

c) Placental infarction >5% in one field

Intervillous space

a) Perivillous fibrinoid deposition >5% in one field

b) Presence of calcification.

OBSERVATIONS-

In the present study of 30 cases of IUGR, macroscopic and microscopic findings of placenta were as follows:

MACROSCOPIC

The mean placental and fetal weights in IUGR cases were 281 ± 24.69 gms and 1508 ± 377.98 gms which were significantly decreased ($p<0.001$) as compared to the control group (573 ±125.81 gms and 2626 ±321.46 gms) respectively. The fetoplacental ratio was 5:1 which was more than the control group (4.7: 1).The mean diameter and thickness were 13.56cms and 1.16cms which were decreased as compared to the control group (17.46 and 2.14cms) respectively. The umbilical cord was inserted eccentrically in 18 cases as compared to the 14 cases in the control group.

MICROSCOPIC

The control group.showed syncytial knots in moderate numbers and perivillous fibrinoid deposition in moderate quantity (fig .1). Fibrinoid necrosis was observed in only in few cases.

The Syncytial knots>30% were found in 60% cases which were more than the control group (44%) (fig.2). Fibrinoid necrosis>5% was found in 46.7% cases (fig 3).This was observed in significantly more number of cases ($p<0.01$) as compared to the control group where it was seen only in 8% cases. Placental infarction>5% was found in 1.8% cases which was absent in the control group. Perivillous fibrinoid deposition >5% was found in 16.7% (fig 4) as compared to 1.8% in control group. There was presence of calcification in 60% cases which was slightly more than the control group in 56% cases. Microscopic features were similar in all the three zones of placentae studied.

No pathological changes were observed in umbilical cord and membranes.

DISCUSSION

Evaluation of the placenta is extremely important in attempting to understand the pathophysiology of IUGR. Only with careful gross and histologic evaluation, along with clinical pathologic correlation, can the underlying causes and recurrence risks be understood (Tyson R and Barton C).[15]. There is great deal of debate going on whether placental inefficiency is a primary cause, contributory factor or a result of extraneous factors (infection,genetic,nutritional,etc.) in cases of IUGR(Cetin I et al, Althshuler G et al.).[9,11] Placental insufficiency, in some form or fashion, is associated

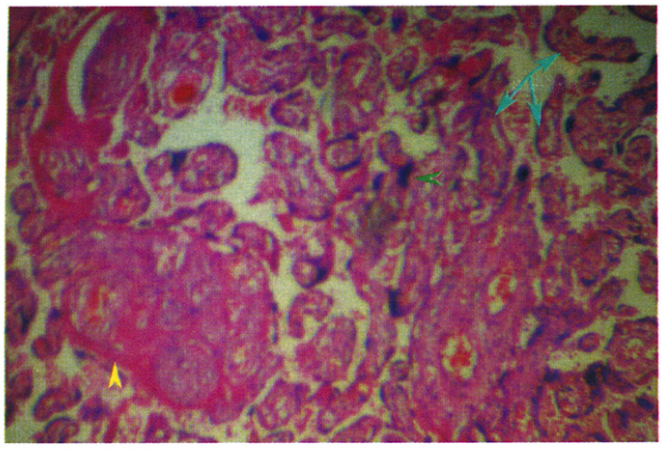

Fig. 1 - Showing section of tertiary chorionic villi (blue arrows) from control group.Syncytial knots are seen in moderate numbers i.e. < 30% in one field (green arrow head). Perivillous fibrinoid deposition seen in moderate quantity i.e. < 5% in one field (yellow arrow head). (H&E; 10X)

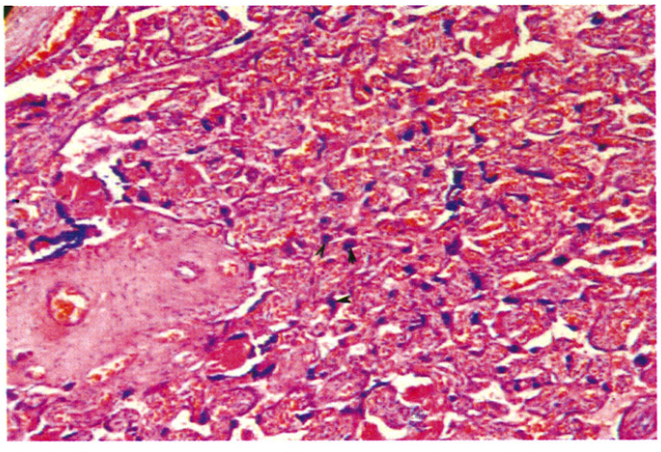

Fig.2- Showing sections of tertiary chorionic villi from cases of IUGR. Fibrinoid necrosis is seen (arrows) within the villi. (H&E; 45X)

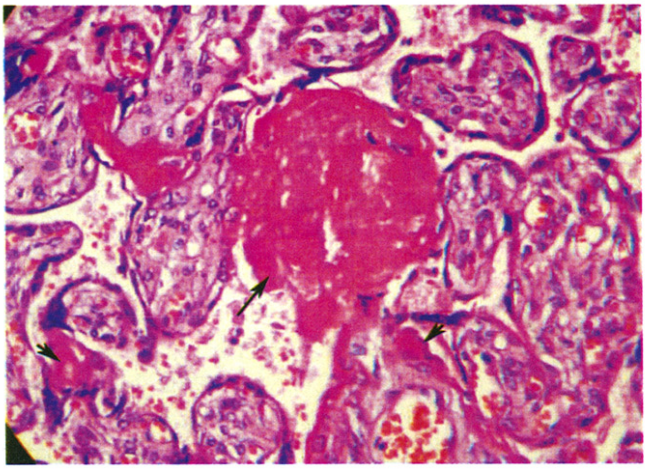

Fig.3- Section of placenta from cases of IUGR showing profuse fibrinoid deposition in the intervillous space. The normal structure of chorionic villi is also seen to be disrupted by extreme fibrinoid deposition. (H&E; 10X)

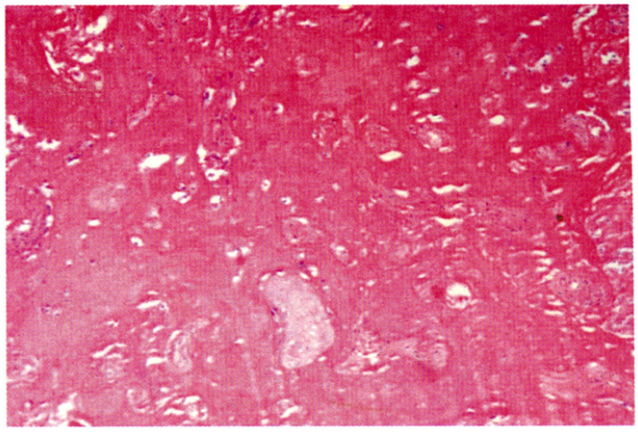

Fig.4- Showing sections of tertiary chorionic villi from cases of IUGR. Numerous syncytial knots are seen (arrow heads). (H&E; 10X)

with the majority of cases of IUGR(Hendrix N and Berghellla V).[16]

The placental dimensions found in the present study were significantly reduced as compared to the control group. Similar findings have been reported by Malik et al.(1968)[10]. Other workers have not considered these criteria in their studies. Fetal and placental weights were significantly reduced and were similar to findings reported in previous studies(Mallik G et al. , Althshuler G et al., Mirchandani J et al., Bhatia A et al. ,Fox H).[10,11,12,13,14,17]

Fibrinoid necrosis is well recognized as one of the hallmarks of immune attack on trophoblastic cell. The fibrinoid material in the affected villi contains a considerable quantity of immunoglobulins (Fox H).[17] Fibrinoid necrosis was observed in 50% cases in our study as compared to 32%-38% cases as reported by the other workers (Mallik G et al., Mirchandani J et al., Bhatia A et al.).[10,12,13,14] This difference may be attributed to selection of IUGR cases. In the present study we considered only idiopathic IUGR. The higher percentage of fibrinoid necrosis, as found in the present study, indicates primary placental involvement in cases of 'idiopathic IUGR'.

Perivillous fibrin deposition in intervillous space is a result of thrombosis of maternal blood. The villi embeded in this fibrin are not infarcted but are incapable of participating in any transfer activity. Ironically, such deposition of fibrin tends to develop in placentas with good maternal blood supply. The greater the blood flow, greater the turbulence & stasis and greater is the perivillous fibrin deposition(Fox H).[17] Earlier workers reported higher incidence of perivillous fibrin deposition (Mallik et al. - 36%,

Mirchandani et al. - 21%)[10,12]. Our study reported incidence of perivillous fibrin deposition in 16.7% cases. The reason for the variable findings and the existing ambiguity regarding pathophysiology of fibrin deposition calls for further studies.

Syncytial knots are indicators of compromise in fetal circulation Fox H.[17]Incidence of syncytial knots was higher in IUGR cases as compared to the control group. Similar findings have been reported by other workers (Mallik et al, Mirchandani et al).[10,12]

Placental infarction has been reported occasionally ranging from 0-10 % by different workers (Mallik et al, Mirchandani et al.).[10,12] except by Bhatia et al. (1981) who observed infarction in all 3 cases studied by them (Bhatia A et al .)[14]. Present study documents a very low incidence of placental infarction (1.8%) similar to other workers.

Authors did not find appreciable difference in prevalence of calcification in IUGR and control group. Previous studies have reported variable prevalence of calcification ranging from 8-100% [Gauri et al. (1968) - 8%, Bhatia et al. (1981) 100%, Mirchandani et al. (1978) - 29%] [10,12,14] . This difference may be attributed to subjective reporting and variable sample size.

CONCLUSIONS

The present study highlights some important facts:

1. Cases of IUGR with no confounding factors formed the basis of study.
2. Foetoplacental weights and placental dimensions were statistically significantly reduced.
3. Syncytial knots were observed in 60% cases.
4. Placental involvement was observed in more than two third cases (47% fibrinoid necrosis and 16.7% perivillous fibrinoid deposition). The findings of the present study differ from earlier studies in comparatively higher incidence of placental involvement.
5. These findings draw our attention to the predominant role of placental causes in cases of idiopathic intrauterine growth retardation.

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