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Original Research Article

Histopathology of placenta in intrauterine growth restriction (IUGR)

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ABSTRACT

Background: Birth of healthy term baby depends on normal placenta. IUGR is a condition associated with placental insufficiency. There is a close relationship between IUGR and placental qualitative changes. The aim of the present study was to evaluate the morphological and histological changes in placentas of IUGR fetuses and in placentas of normal uncomplicated pregnancies and to determine the relationship that exists between morphological change and frequency of IUGR.
Methods: In a cross sectional study conducted in the department of Pathology, GMC Jammu, a total of 60 placenta were received, 30 placenta of IUGR fetus (group 1-case) and 30 placenta of uncomplicated pregnancy with normal single fetus (group 2-control). Exclusion criteria: Twin pregnancy, gestational hypertension, diabetes, congenital anomaly, antepartum hemorrhage and systemic disorder.
Results: Placental weights in IUGR group were significantly lower than control group. Average placental weight in IUGR group was 425 gms while in the control group (normal placenta) it was 550 gms. Infarction, intervillous thrombosis, chorionic villitis, hemorrhagic endovasculitis, placental intravascular thrombi, perivillous fibrin deposition, fibrinoid necrosis and villous edema were found to be more common in IUGR group (Group 1-case group) than Normal (Group 2- control group).
Conclusions: This study highlightened that significant pathological differences were found between the placentas of IUGR fetus and normal fetus. The gross and microscopic measurement of a placenta is a good way to get proper information about IUGR and helps in management of the pregnancy.

Keywords: Intrauterine growth retardation, Foetus infarction, Fibrinoid necrosis, Placenta, Thrombosis

INTRODUCTION

Intrauterine growth retardation (IUGR) is a failure to achieve the growth potential of a fetus that is determined by the genetic constitution and environmental influences endogenous to the pregnancy.

Fetal growth and viability depends on the maternal supply of nutrients and oxygen through the placenta into the umbilical circulation. Placental factors and hypoxemia are keys to intrauterine growth restriction (IUGR) and fetal death. IUGR is a condition associated with placental insufficiency.[1]

A close relationship between IUGR and placental qualitative changes was shown and the main pathological findings in placenta were prematurity and hypoplasia.[2]

In a histopathological evaluation of placenta in IUGR pregnancies, the weight of IUGR placenta was less than normal placenta. Infarction and intervillous fibrinoid deposition were higher in IUGR placenta. In addition, thickening of basal membrane and cytotrophoblast hyperplasia were more common among IUGR placenta. All the main histopathological findings pointed to placental blood flow reduction and fetal blood flow restriction.[3]

Information on placental abnormalities may reveal the presence of chronic fetal insults and allow their differentiation from acute (peripartum) stresses.[4]

The aim of the present study was to evaluate the morphological and histological changes in placentas of IUGR fetuses and in placentas of normal uncomplicated pregnancies and to determine the relationship that exists between morphological change and frequency of IUGR.

METHODS

This was cross sectional study conducted in the department of Pathology, GMC Jammu, India. A total of 60 placenta were received, 30 placentae of IUGR fetus (group 1-Case group) and 30 placentae of uncomplicated pregnancy with normal single fetus (group 2-Control group).

IUGR was defined on basis of an estimated fetal weight of less than the third percentile for gestational age, reduced amniotic fluid volume or Doppler USG of umbilical artery demonstrating absent end diastolic flow velocity and was established by serial ultrasonographic examination.

Inclusion criteria

Pregnant women between ages 19 to 37 years and gestational age 32 to 40 wks.

Exclusion criteria

Twin pregnancy, gestational hypertension, diabetes, congenital anomaly, antepartum hemorrhage and systemic disorder.

All the necessary information about pregnancy was collected from the record sheets and consent was also taken. After delivery, morphologic assessment of placenta like placental weight, cord insertion and number of umbilical vessels, color of maternal and fetal surface was done. All the placentas were fixed in 10% formaline for 24hours and processed for routine paraffin embedding. Multiple random samples were taken from placenta, two samples from umbilical cord, 2 samples from extra placental membranes. For microscopic examination, 3-4 microns thick sections were prepared and were stained with H and E stain.

RESULTS

Table 1 shows gross findings in IUGR and normal placentas. Placental weights in IUGR group were significantly lower than control group. Average placental weight in IUGR group was 425 gms while in the control group (normal placenta) it was 550gms.

Table 2 compares the microscopic findings between IUGR and normal placentas. Infarction was more common 53.3% in group 1 (case group), while only 6.6 % cases showed infarction in control group (Group 2).

Table 1: Gross findings in placentas.

Gross finding	Group 1 (IUGR Placenta)	Group 2 (Normal Placenta)
Umbilical cord inseration		
Central	22	10
Eccentric	08	20
No of abnormal umblicial vessels	01	0
Membrane inseration		
Marginal	27	30
Circumarginate	03	0

Table 2: Microscopic findings in the placentas.

Microscopic findings	Group 1 (IUGR Placenta)	Group 2 (Normal Placenta)
Infarction	16(53.3%)	2(6.6%)
Thrombosis	6(20%)	0
Intervillous fibrinoid deposition		
5%	7(23.3%)	21(70%)
5-10%	10(33.3%)	7(23.3%)
>10%	13(43.3%)	2(6.6%)
Chorionic villitis	19(63.3%)	4(13.3%)
Chorioaminionitis	2(6.6%)	0
Haemorrhagic endovasculitis	11(36.6%)	1(3.3%)

There was no case of intravascular thrombi in Group 2 (control group), however 20% of placentas in the case group (Group 2) had placental intravascular thrombi.

Perivillous fibrin deposition was more common in Group 1-case group as compared to control group. In group 1-IUGR group, 13 cases (43.3%) showed 10% fibrin deposition while in control group-normal placenta, it was only 6.6% and most of the cases (21 cases) showed only 5% fibrin deposition.

Chorionic villitis occurred in 63.3% and 13.3% of group 1 and group 2 respectively. There was one case of haemorrhagic endovasculitis seen in the control group (group 2) as compared 36.6% of placentas in the case group (IUGR group) had haemorrhagic endovasculitis

DISCUSSION

The findings of the current study demonstrate several different placental histopathological lesions in IUGR related placenta. We excluded women with risk factor like gestational hypertension, diabetes, congenital anomaly, antepartum hemorrhage and systemic disorder.

This study provides important results in placentas of IUGR fetuses as well as compares it with normal placenta also. Placental weights in IUGR group were significantly lower than control group. Average placental weight in IUGR group was 425gms while in the control group (normal placenta) it was 550gms. Maulik et al, found placental weights to be 631g in the control group and 409g in the IUGR group with the differences being statistically significant.[5] The findings of both the studies are comparable.

The frequency of abnormal placental shapes like bilobate were more common in group 1 (case group-IUGR), while no such incidence was found among group 2 (control group-normal). In studies conducted by Benirschkeand Faye-Petersen and Fox and Maulik and Salafia et al. Circumvallate placenta, circummarginate, velamentous insertion of the cord and placenta previa were suggested as possible causes of IUGR.[5-9] However, Salafia *et al.* suggested that abnormal cord insertion rarely showed general fetal defects.

Placental infarction can be observed in normal pregnancies also. It is usually of no significance unless it affects more than 10-20% of the placental volume.[10] The existence of a relation between fetal hypoxia and placental infarction has been shown.[11] In the present study, placental infarction was detected in 53.3% (16 cases) in group 1-IUGR group and only 6.6% (2 cases) in group 2-normal fetus. The findings are comparable to a study by Sharma and Mardi where placental infarction on macroscopic and microscopic surfaces as well as ischemic necrosis was higher in the IUGR placenta compared to those normal.[2] Curtin also compared IUGR and normal placenta and found an association between the vascular reduction of fetoplacental and loss of functional placental tissue that these lesions being matched with IUGR.[3]

Intervillious fibrinoid deposition of less than 5% was observed more frequently (21 cases) in control group-group 2. In comparison to this, intervillious fibrinoid deposition of >10% was more common (13 cases) in the case group-group 2. Therefore, high amount of intervillous fibrinoid deposition is a pathological finding in IUGR-related placenta. The findings of Mardi and Sharma's study also correlates with our study. Fibrinoid necrosis and perivillous fibrin deposition are associated with IUGR, autoimmune processes, infection, toxic insult, a known abnormal host-placenta interaction, genetic disorders and confined placental mosaicism.[2]

It is reported that fetal thrombotic vasculopathy and fetal stem vessel thrombosis are common findings in women with adverse pregnancy outcomes.[12] In the present study, 6 cases (20%) in group 1-IUGR group had thromobosis/chorionic vessel occlusion. However, none of the placentas from healthy babies had placental intravascular thrombi.

In the present study, chronic villitis was seen in 19 cases (63.3%) of IUGR group. Similar findings were suggested in study by Redline and Pappin where they found the association between chronic villitis and growth restriction.[13]

In our study, chorioamnionitis (2 cases) and haemorrhagic endovasculitis (11 cases) were found to be significantly more common in IUGR related pregnancies. It is evident that the number of different types of lesions that are seen is far more strongly associated with fetal growth restriction or intrauterine death than the presence or severity of any one lesion.[14] It is more likely that accumulation of placental injury for a sufficient duration leads to IUGR and fetal death.[15]

Infarction, intervillous thrombosis, chorionic villitis, hemorrhagic endovasculitis, placental intravascular thrombi, perivillous fibrin deposition, fibrinoid necrosis and villous edema were found to be the types of lesions that cause a normal fetus to become growth restricted or die.[16]

CONCLUSION

In this study, significant differences were found between the IUGR and normal group regarding the pathologic changes, macroscopic infarction, and microscopic infarction, thrombosis and tissue ischemia. The results reported here indicate that a relationship exists between morphological changes in the placentas of IUGR fetuses and intrauterine death fetuses. The gross and microscopic measurement of a placenta is more objective and seems to offer a good way to get proper information about IUGR.

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