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Original Research Article

To evaluate histopathological changes in placenta of IUGR

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ABSTRACT

Background & Method: The present study entitled "To evaluate Histopathological changes in Placenta of IUGR" was conducted in the Department of Obstetrics and Gynaecology. A total of 400 patients were studied. Out of them 200 were normal patients without IUGR and 200 with IUGR. These patients were evaluated with the help of semistructured proforma consisting of various socio-demographic and clinical variables.

Result: Number of LSCS deliveries in IUGR patients (59) is significantly more than control group (33). The number of syncytial knot formation (>10) in IUGR is 169 compared to 153 of control and hence is statistically insignificant. Cytotrophoblastic proliferation (>10) in IUGR (126) is significantly more than (92) in the control group. Number Of area fibrinoid necrosis in villi > 5 in IUGR group is 57 compared to control group (29) (significantly higher) No. of hylinized areas (> 5) in IUGR (101) is significantly higher than in control group (68).

Conclusion: Histopathological findings in low power field like syncytial knot formation, cytotrophoblastic cellular proliferation, calcification and hyalinisation of villi are also found as normal aging changes in placenta but it occur early and more frequently per low power field in IUGR group. To conclude, these morphological & histological findings of placenta are the etiological basis for Intra uterine growth restriction.

1. Introduction

The placenta was first recognised & venerated by early Egyptians, while it was the Greek physician, Diogenes of Apollona (480 B.C.) Describe the placental functions.

The placenta comprise of chorionic plate on the fetal side, basal plate on the maternal side while stem villi reaching out between these plates, and intervillous spaces between the stem villi is loaded up with the maternal blood.[1] The human placenta at term is a neighborhood, circle like thickening of the membranous sac that is accomplished by dividing the films into two separate sheets, the chorionic plate and the basal plate. The two sheets encase the intervillous spaces, as cover and bottom.[2] The intervillous space is perfused with maternal blood, which courses, straight around the trophoblastic surfaces of the placental villi. The villi are perplexing tree-like projections of the chorionic plate into the intervillous space. Inside the villi, fetal veins are available which are associated with the fetal circulatory framework. At the placental edge, the intervillous space is annihilated so the chorionic and the basal plate intertwine with one another and accordingly structure the chorion leaves.[3]

Intrauterine development limitation (IUGR) is definitely not a particular illness substance in essence, yet rather a sign of numerous conceivable fetal and maternal disorders.[4] Since clinical administration, advising, and extreme result are to a great extent subject to the

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etiology, it is significant for the clinician to find out the particular reason for development disappointment. There is a solid relationship between IUGR, chromosomal issues, and intrinsic mutations. Babies with chromosomal issues, including trisomy 13, 18, and 21 are regularly development limited, and infants with other autosomal anomalies (different cancellations and ring chromosome structure adjustments) likewise have problematic development. In spite of the fact that sex chromosome problems are often deadly, those embryos who endure might be development confined upon entering the world. Those irregularities most regularly connected with helpless development were trisomy 18 and anencephaly.

Generally, chromosomal problems and multifactorial inherent mutations are answerable for around 20% of IUGR babies, and that rate is considerably higher if development disappointment is recognized before 26 weeks' incubation or potentially is related with polyhydramnios.[5]

Maternal vascular sickness, with its related diminishing in uteroplacental perfusion, is accepted to represent 25–30% of all IUGR newborn children. It is the most widely recognized reason for IUGR in the nonanomalous infant.[6]

Beginning stage, extreme toxemia, and constant hypertension with superimposed toxemia typically have the most significant impact on fetal development. It is grounded that these issues are related with the inability to expand plasma volume and have huge and explicit placental pathology.[7]

2. Materials and Methods

The present study entitled "To evaluate Histopathological changes in Placenta of IUGR" was conducted in the Department of Obstetrics and Gynaecolog. A total of 400 patients were studied. Out of them 200 were normal patients without IUGR and 200 with IUGR. These patients were evaluated with the help of semistructured proforma consisting of various socio-demographic and clinical variables.

All patients in the study were hospitalised. IUGR was taken as baby weight less than 10th percentile of their gestational age. Preterm deliveries were not taken for study. Placenta with cord and membranes were collected immediately after delivery. Any abnormality of cord and membranes was noted. The placentae along with the umbilical cord were preserved in 10% formalin solution (in water).

2.1. Collection of placenta

Each placenta was collected soon after delivery from the labour room or from the operation theatre. Placenta as a whole was kept in formalin solution in a large container with snugly fitting lid. Then specimen was transported to research laboratory of Anatomy department.

2.2. Preparation for examination of placentae

Whole of the specimen of placenta was taken out from the large container and kept on a clean flat surface on the dissection table wrapped with a sheet of polythene. The specimen was washed well with normal saline and the membrane was trimmed off with a pair of sharp scissors near the margin. The specimen was soaked with blotting paper and excess blood clot was removed from the surface. The umbilical cord was cut at a distance of 2 cm from its attachment with placenta (Ameren and Dunhill, 1966).

2.3. Study designed

Prospective Study.

2.4. Inclusion criteria

1. Patients with baby birth weight of < 10th percentile of gestational age.
2. IUGR due to all causes.
3. Any age.
4. Informed consent.

2.5. Exclusion criteria

1. Preterm patients.
2. Lack of consent.
3. Damaged placenta.

3. Results

Number of LSCS deliveries in IUGR patients (59) is significantly more than control group (33).

The number of syncytial knot formation (>10) in IUGR is 169 compared to 153 of control and hence is statistically insignificant.

Cytotrophoblastic proliferation (>10) in IUGR (126) is significantly more than (92) in the control group.

Number of area fibrinoid necrosis in villi > 5 in IUGR group is 57 compared to control group (29) (significantly higher).

No. of hylinized areas (> 5) in IUGR (101) is significantly higher than in control group (68).

4. Discussion

Placenta being a fetal organ has a similar anxiety, to which the baby is uncovered. Subsequently any infection cycle influencing the mother and hatchling additionally enormously affects placenta. Ordinarily the placental morphology differs extensively during its short life span.[8] Modifications in placenta as a feature of "Maturing" marvel are most likely a piece of development measure and go connected at the hip with proceeded with development of placenta. Placenta develops till 37^{th} week and therefore youthful villi are seen even till term. Thus in the

Table 1: Mode of delivery

Outcome	Control		IUGR	
	No.	%	No.	%
LSCS	33	16.5	59	29.5
Vaginal	167	83.5	141	70.5
Total	200	100%	200	100%

(Chi square value 11.33, p value 0.0008)

Table 2: Number of syncytial knot formation

Syncytial knot formation	Control		IUGR	
	No.	%	No.	%
<10	42	21	31	15.5
10-25	152	76	158	79
>25	01	0.5	11	5.5
Total	200	100%	200	100%

Chi square value 9.99 and p .007

Table 3: Cytotrophoblastic proliferation

Cytotrophoblastic proliferation	Control		IUGR	
	No.	%	No.	%
<10	108	54	74	37
10-20	66	33	83	41.5
>20	26	13	43	21.5
Total	200	100%	200	100%

Chi square value 12.5 and p .002

Table 4: Number of areas of fibrinoid necrosis in villi

Number of area fibrinoid necrosis in villi	Control		IUGR	
	No.	%	No.	%
< 5	171	85.5	143	71.5
> 5	29	14.5	57	28.5
Total	200	100%	200	100%

Chi square value 11.6 and p .001

Table 5: Number of Areas of hylinized villi

Number of Area hylinized villi	Control		IUGR	
	No.	%	No.	%
< 5	132	66	99	49.5
> 5	68	34	101	50.5
Total	200	100%	200	100%

Chi square value 11.2 and p .001

examination on placenta Fox (1975) has focused on the significance of investigating the placental pathology quantitatively and has expressed that the significance of the injuries could be acknowledged just when surveyed in connection of fetal development and development.

The histology of placenta of moms of IUGR patients likewise shows huge expansion in syncytial tie development, cytotrophoblastic cell multiplication, stromal fibrosis, calcification and hyalinisation of villi in contrast with the control group.[9]

Exceptionally huge expansion in the rate of dead tissue, intervillous fibrin testimony, stromal fibrosis and syncytial hitching were found in IUGR placentas contrasted with full term typical placentas on tiny examination.[10,11] They additionally found as non-huge expansion in cytotrophoblastic cell multiplication.

Syncytial hitches development, cytotrophoblastic cell multiplication, stromal fibrosis, calcification and hyalinisation of villi is up managed by intra placental hypoxia and down controlled by expanding intra placental oxygen levels.[12] This likewise demonstrates a degenerative cycle as reaction to nearby hypoxia. This is viewed as a versatile marvel to decrease the dispersion distance from the intevillous space to the fetal vessels within the sight of

diminished oxygen pressure.[13,14] The discoveries of our investigation correspond to those of different creators.

5. Conclusion

Histopathological findings in low power field like syncytial knot formation, cytotrophoblastic cellular proliferation, calcification and hyalinisation of villi are also found as normal aging changes in placenta but it occur early and more frequently per low power field in IUGR group. To conclude, these morphological & histological findings of placenta are the etiological basis for Intra uterine growth restriction.

6. Source of Funding

None.

7. Conflict of Interest

The authors declare no conflict of interest.

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