Original Research Article

Morphological changes in placentas of normal and high risk pregnancies - 2 years study in MGM hospital

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International Archives of Integrated Medicine, Vol. 4, Issue 5, May, 2017.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 29-04-2017 **Accepted on:** 04-05-2017
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: Samala Raghuram Mohan, Jyosna, Sandhya Anil, S. Chandra Sekhar. Morphological changes in placentas of normal and high risk pregnancies - 2 years study in MGM hospital. IAIM, 2017; 4(5): 61-78.

Abstract

Background: Placenta-related disorders of pregnancy are almost unique to human species. These disorders, which affect around a third of pregnancies, primarily include miscarriage and pre-eclampsia.

Aim and objectives: The aim of the present study was to examine the morphological changes in placentas of normal and high risk pregnancies, and to evaluate the fetal outcome in these cases, which in turn will improve the quality of placental diagnosis. The objectives were to know the extent of the gross and microscopic placental changes that occur in normal pregnancies and to study the placentas of high risk pregnancies.

Materials and methods: A study of 132 placentas in normal and high risk pregnancies was conducted in the Department of Pathology, Kakatiya Medical College, MGM Hospital, Warangal, from 1st September 2014 to 31st August 2016. The materials included in this study were placentas from thirty females with normal pregnancies (controls) and one hundred thirteen with high risk pregnancies (cases) comprising anemia (27), pregnancy-induced hypertension (38), intrauterine growth retardation (15), diabetes mellitus (13), and twins (9).

Results: In high risk cases, women in the age group 21-25 years were 66.6% in anemia, 60.5% in PIH, and 60% in IUGR 69.2% in diabetes mellitus, and 33.3% in twin pregnancies. Only a few cases

were seen in the age group of 26-30 in both controls and cases. Seventy percent of the controls belonged to primigravida and 30% were multiparous women. In high risk cases, 66.6% were primigravida and 33.3% multipara. Majority of the multiparous mothers were seen in anemia constituting 48.1%. High risk cases exhibited exaggerated changes, except for calcification, which was seen equally in both the groups. Infarction was seen in 50% cases of PIH. Single case of retroplacental hematoma was also observed in PIH. Subchorionic fibrinoid was seen in 53.4% of IUGR placentas. Maternal floor infarction was seen in 4 cases of PIH and 3 cases of IUGR placentas. Subchorionic hematoma was seen in 1 case of IUGR placenta. IUGR placentas were small in size and weight for the gestational age and twin placentas were of diamniotic dichorionic type. The most prominent microscopic features were syncytiotrophoblastic knot formation and calcification. The high risk groups were showing prominent infarcts, fibrinoid necrosis, and stromal fibrosis more than those of control group.

Conclusion: The present study has highlighted the importance of examination of placenta in normal as well as high risk pregnancies. The placental changes are essential to correlate the fetal outcome, as it provides the information for the cause of death. Hence, it has an effective role in planning prenatal monitoring of a future pregnancy.

Key words

Placental pathology, High risk pregnancies, Placental Infarcts.

Introduction

Placenta helps in exchange of gases, metabolic products between mother and fetus, removal of wastes from the fetal blood into maternal circulation. Study of placenta gives information of mother and infant. The fetus, placenta and the mother form a complete triad of dynamic equilibrium in reproduction. Placenta is the most accessible and readily evaluable component of the triad.

Placenta-related disorders of pregnancy are almost unique to human species. These disorders, which affect around a third of pregnancies, primarily include miscarriage and pre-eclampsia. Changes in human lifestyle, such as delayed childbirth and hypercaloric diets, may have increased the global incidence of placenta-related disorders over the last few decades. Any disturbance in the maternal health will affect the placenta, which in turn leads to decreased placental perfusion and cause fetal morbidity and mortality. Disturbance in the placental function itself affects the fetal growth. Hence, the study of changes that occur in the placenta and the extent of lesions will help in the assessment of the degree of fetal insult in utero. Standardization of diagnostic criteria and increased inputs in placental pathology will improve the quality of diagnosis.

Various studies on placenta have been conducted in the past in India and abroad. This work is essentially undertaken to examine the histopathological changes in placentas and their correlation in high risk pregnancies that are commonly encountered in this institute, and to compare with the observations made earlier.

Aim and objectives

The aim of the present study was to examine the morphological changes in placentas of normal and high risk pregnancies, and to evaluate the fetal outcome in these cases, which in turn will improve the quality of placental diagnosis.

The objectives were

- To know the extent of the gross and microscopic placental changes that occurs in normal pregnancies.
- To study the placentas of high risk pregnancies.
- To correlate the placental features with that of fetal outcome.

Materials and methods

A study of placental pathology in normal and high risk pregnancies was conducted in the Department of Pathology, Kakatiya Medical College, MGM Hospital, Warangal, from 1st September 2014 to 31st August 2016.

The materials included in this study were placentas from thirty females with normal pregnancies (controls) and one hundred thirteen with high risk pregnancies (cases) comprising anemia (27), pregnancy-induced hypertension (38), intrauterine growth retardation (15), diabetes mellitus (13), and twins (9).

Relevant clinical data regarding maternal age, gravida, any significant maternal disorders, blood pressure monitoring, investigations advised (hemoglobin estimation, random or fasting blood sugar estimation, urine for proteins) in appropriate patients, weight and sex of the baby, whether live birth or still birth etc. were noted.

Clinical criteria for selection of patients were:

- Mothers with two minimum blood pressure readings of more than 140/90 mm Hg at least 4 hours apart, associated with the presence of proteinuria more than 0.5gm/dl in a previously normotensive women and pedal edema were categorised into pre-eclampsia. In a pre-existing hypertension, a rise of at least 20 mm Hg of diastolic blood pressure during pregnancy is accepted as pre-eclampsia.
- Anemia in pregnant women was taken as less than 10 gm/dl of haemoglobin.
- Diabetic mothers were mainly of gestational diabetes with raised levels of random and fasting blood sugar and glycosuria.
- IUGR or small for date (SFD) babies are those whose birth weights are disproportionately low for gestational age (10th percentile) or 2 SD.

Placentas were collected in separate clean plastic containers. They were placed in 10% formalin for one day for fixation. Taking all the precautionary measures, the specimens were examined with regard to the following features:

The size, shape, weight of placentas was noted. Membranes were looked for any change in the colour and site of rupture of membranes along with the number of cotyledons, presence of infarction, calcification etc. The length of the umbilical cord, insertion, external surface, number of vessels and site of insertion of umbilical cord were noted. The placental disc is weighed by removing the membranes and cord. Examination of twin placenta was same as for the normal one, but 5 placentas were treated separately. In diamniotic dichorionic placentas, bits were given from the septum and its site of attachment to the chorionic plate, in the form of 'T' zone/ section.

The placentas were cut into thin slices of one centimetre thickness from the maternal surface towards fetal surface. Tissues were taken from the following placental sites and fixed in 10% formalin overnight followed by processing and subjected for microscopic examination:

- Two bits from the umbilical cord (close to the placental disc and at the fetal end)
- One bit from the membrane roll
- A minimum of two bits from the placental disc
- If any gross pathologic lesions seen, further bits were taken from those areas

The sections were cut in 5 micron-thickness and fixed on the glass slide with egg albumin, stained with Hematoxylin and Eosin, and studied for the microscopic changes. Special staining was done using Periodic Acid Schiff stain for confirming basement membrane thickening.

Results

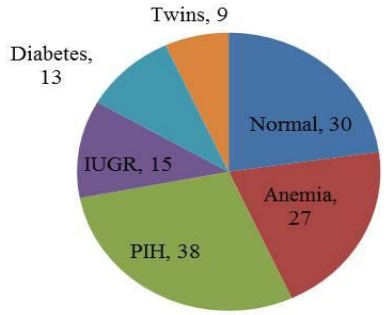
A study of placental pathology in high risk pregnancies was conducted in the Department of Pathology for a period of two years, from

September 2014 to August 2016, in Kakatiya Medical College, MGM Hospital, Warangal.

The study group included 30 placentas from normal term pregnancies that were taken as controls, and 102 placentas from high risk pregnancies of both maternal and fetal causes, comprising cases of pregnancy-induced hypertension, Anemia, Intrauterine growth retardation, maternal diabetes, and twin pregnancies.

Total number cases which included 30 cases normal term pregnancies and 102 where high risk pregnancies (**Chart - 1**).

Chart – 1: Total No of cases.



Age distribution among the controls and cases was as per **Table - 1**. The mothers in the control group were in the range of 16-30 years in both groups. In high risk cases, women in the age group 21-25 years were 66.6% in anemia, 60.5% in PIH, and 60% in IUGR 69.2% in diabetes mellitus, and 33.3% in twin pregnancies. Only a few cases were seen in the age group of 26-30 in both controls and cases.

Parity of the mothers in controls and cases is represented in **Table - 2**. Seventy per cent of the controls belonged to primigravida and 30% were multiparous women. In high risk cases, 66.6% were primigravida and 33.3% multipara. Majority of the multiparous mothers were seen in anemia constituting 48.1%.

Table - 3 indicates the mode of delivery. All were normal vaginal deliveries except 17 cases of PIH, 7 cases of diabetes mellitus, 2 cases of twin pregnancies where lower segment caesarean section was performed.

Table – 1: Age groups of women.

Age group	**Normal** n (%)	**Anemia** n (%)	**PIH** n (%)	**IUGR** n (%)	**DM** n (%)	**Twins** n (%)
16-20 yr	13 (43.3)	4 (14.8)	12 (31.5)	5 (33.3)	_	3 (33.3)
21-25 yr	13 (43.3)	16 (66.6)	19 (60.5)	9 (60.0)	9 (69.2)	3 (33.3)
26-30 yr	4 (13.3)	4 (18.7)	2 (7.8)	1 (6.6)	4 (30.7)	3 (33.3)
Total	30	27	38	15	13	9

(PIH=*Pregnancy Induced Hypertension,* IUGR= *Intra-Uterine Growth Retardation,*DM= *Diabetes Mellitus*)

Table – 2: Parity of mothers.

Parity	**Normal** n (%)	**Anemia** n (%)	**PIH** n (%)	**IUGR** n (%)	**DM** n (%)	**Twins** n (%)
Primi	21 (70.0)	14 (51.8)	27 (71.0)	12 (80.0)	9 (69.2)	6 (66.6)
Multi	9 (30.0)	11 (48.1)	11 (28.9)	3 (20.0)	4 (30.7)	3 (33.3)
Total	30	27	38	15	13	9

Table – 3: Mode of delivery.

Delivery	**Normal**	**Anemia**	**PIH**	**IUGR**	**DM**	**Twins**
Normal Vaginal	30	27	21 (55.2%)	15	6(46.1)	7(77.7)
Caesarean	_	_	17 (44.7%)	_	7(53.8)	2(22.2)

The outcome of pregnancy and the weights of newborn are shown in **Table - 4** and **Table - 5** respectively. The babies of the control group were having normal birth weights ranging from 2.5-3.5 kg. Majority of high risk cases have low birth weight babies. In IUGR, all the babies were of low birth weight and were below 2.5 kg and intrauterine death was seen in 26.7%. In PIH, there was a decline in the range of birth weights and two were still births. A case of fetus papyraceus was observed in a twin pregnancy where the live born baby had normal birth weight, and the affected baby was compressed with only fetal remnants and a small atrophied placenta.

Table – 4: Outcome of pregnancy.

Pregnancy	**Live born** n (%)	**Still born** n (%)
Normal	30 (100)	_
Anemia	27 (100)	_
PIH	36 (94.7)	2 (5.2)
IUGR	11 (73.3)	4 (26.7)
DM	12 (92.1)	1(7.6)
Twins	8 (88.8)	1 (11.1)

Table – 5: Weight of babies.

Pregnancy	**< 2.5 kg** n (%)	**2.5 – 3 kg** n (%)	**> 3 kg** n (%)
Normal	_	11 (36.6)	19 (63.3)
Anemia	4 (14.8)	18 (66.6)	5 (18.5)
PIH	19 (50.0)	16 (42.1)	3 (7.8)
IUGR	15 (100)	_	_
DM	_	9 (69.2)	4 (30.7)
Twins	6 (66.6)	3 (33.3)	_

Table - 6 gives the information regarding the weight of placentas. The weight ranged from 250 gm to 590 gm. All the placentas in control groups were in 301-600 gm and 76.6% of them were between 401-600 gm. The placentas from the cases were slightly in the lower range compared to the controls. In IUGR, 53.4% of the placentas were in the range of 201-300 gm. indicating that the IUGR babies were affected mainly by low placental fetal perfusion ratio. 65.7% of placentas in PIH weighed between 301-400 gm.

Gross features of the placental changes are indicated in **Table - 7**. The normal placenta and its cut surfaces, and diagrams are shown in **Figures – 1 to 5**. The main lesions observed in our study included: subchorionic fibrinoid deposition (**Figure - 1**). Intervillous fibrinoid deposits (**Figure - 2**) were often multiple, several centimetres in greatest diameter, firm white plaques. Perivillous fibrinoid (**Figure - 3**) was seen in almost the entire placental disc including basal plate. Infarcts were seen as a pale to whitish lesions in the placental disc (**Figure - 4**). Control group showed subchorionic fibrinoid in 10%, intervillous fibrinoid and perivillous fibrinoid in 19.6%, infarcts in 6.6% and calcification in 26.6% of placentas and single case revealed retroplacental hematoma. High risk cases exhibited exaggerated changes, except for calcification, which was seen equally in both the groups. Infarction was seen in 50% cases of PIH. Single case of retroplacental hematoma (**Figure - 5**) was also observed in PIH. Subchorionic fibrinoid was seen in 53.4% of IUGR placentas.

Maternal floor infarction (**Figure - 6**) was seen in 4 cases of PIH and 3 cases of IUGR placentas. Subchorionic hematoma (**Figure - 7**) was seen in 1 case of IUGR placenta. IUGR placentas were small in size and weight for the gestational age (**Figure - 8**) and twin placentas were of diamniotic dichorionic type (**Figure - 9**).

Microscopic changes are listed in **Table - 8**. The normal villous structure and umbilical cord were seen in **Figure - 10** and **Figure - 11**. The placental changes included: trophoblastic lesions, stromal reactions, vascular and villous abnormalities in both controls and cases. The most prominent features were syncytiotrophoblastic knot formation and calcification. The high risk groups were showing prominent infarcts, fibrinoid necrosis, and stromal fibrosis more than those of control group.

Table – 6: Weight of placentas.

Pregnancy	**201 – 300 gm** n (%)	**301 – 400 gm** n (%)	**401 – 600 gm** n (%)	**Total**
Normal	_	7 (23.3)	23 (76.6)	30
Anemia	2 (7.4)	18 (66.6)	7 (25.9)	27
PIH	10 (26.3)	25 (65.7)	4 (10.5)	38
IUGR	8 (53.4)	5 (33.3)	2 (13.3)	15
DM	_	8 (61.5)	5 (38.4)	13
Twins	_	_	9 (100)	9

Table – 7: Gross features of placentas.

Pregnancy	**Sub chorionic fibrinoid** n (%)	**Inter villous fibrinoid** n (%)	**Peri villous fibrinoid** n (%)	**Infarct** n (%)	**Calcification** n (%)	**Retro-placental hematoma** n (%)
Normal	3 (10.0)	4 (13.0)	2 (6.6)	2 (6.6)	8 (26.6)	1 (3.3)
Anemia	5 (18.5)	4 (14.8)	6 (22.2)	12 (44.4)	8 (29.6)	_
PIH	9 (23.6)	7 (18.4)	10 (26.3)	19 (50.0)	22 (57.8)	1 (2.6)
IUGR	8 (53.4)	6 (40.0)	8 (53.4)	5 (33.3)	6 (40.0)	_
DM	4 (30.7)	9 (69.2)	4 (30.7)	9 (69.2)	4 (30.7)	_
Twins	3 (33.3)	3 (33.3)	_	3 (33.3)	3 (33.3)	_

Table – 8: Microscopic features of placentas.

Features	**Normal** n (%)	**Anemia** n (%)	**PIH** n (%)	**IUGR** n (%)	**DM** n (%)	**Twins** n (%)
Syncytial knots	6 (20.0)	20(81.4)	27(71.0)	8(53.4)	9(69.2)	3(33.3)
Cyto-trophoblastic proliferation	5 (16.6)	15(59.2)	24(64.5)	7(46.6)	9(69.2)	3(33.3)
Basement Membrane thickening	4 (13.3)	15(59.2)	21(55.2)	7(46.6)	4(30.7)	3(33.3)
Fibrinoid necrosis	3 (10.0)	7 (25.9)	11 (28.9)	8(53.4)	4(30.7)	3(33.3)
Intervillous fibrinoid	2 (6.6)	5 (18.5)	8(21.0)	7(46.6)	4(30.7)	3(33.3)
Infarction	2 (6.6)	12(44.4)	19(50.0)	5(33.3)	4(30.7)	3(33.3)
Calcification	8 (26.6)	15(55.5)	23(60.5)	6 (40.0)	4(30.7)	6(66.6)
Inflammation	-	9 (33.3)	6(15.7)	4(26.6)	9(69.2)	-

Syncytiotrophoblastic knots (**Figures – 12, 13**) were seen in 20% of the normal placentas and were significantly increased in high risk cases, anemia (81.4%), PIH (71%), and IUGR (53.4%).

Cytotrophoblastic hyperplasia (**Figure - 14**) was observed in 16.6% of normal pregnancies and 63.1% in PIH and 69.2% diabetes, 59.2% in anemia, 46.6% in IUGR, and 33.3% in twin placentas. Trophoblastic basement membrane thickening (**Figure - 15**) was seen in 13.3% of normal pregnancies and it was increased in high risk group constituting 59.2% in anemia, 55.2%

in PIH, 46.6% in IUGR, and 30.7% in diabetes and 33.3% in twins.

Figure – 1: Subchorionic fibrinoid seen as brownish masses.

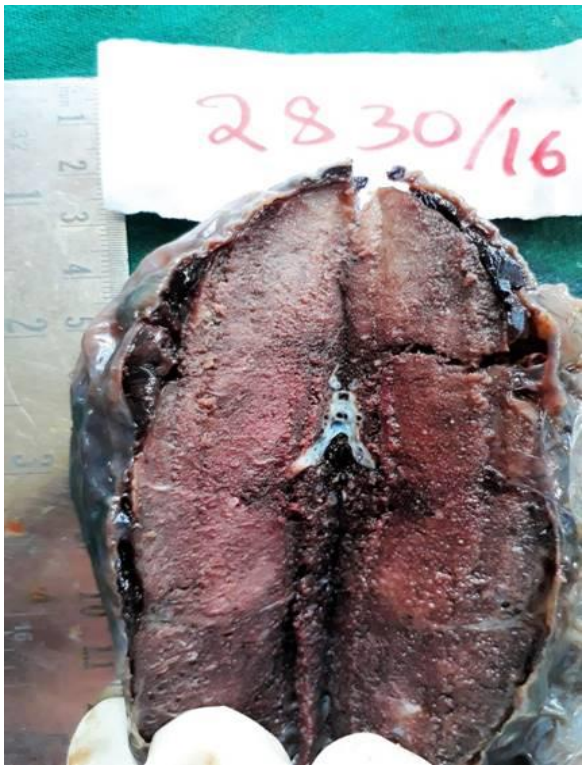


Figure – 2: Placenta showing multiple pale to yellowish nodular intervillous fibrinoid lesions.

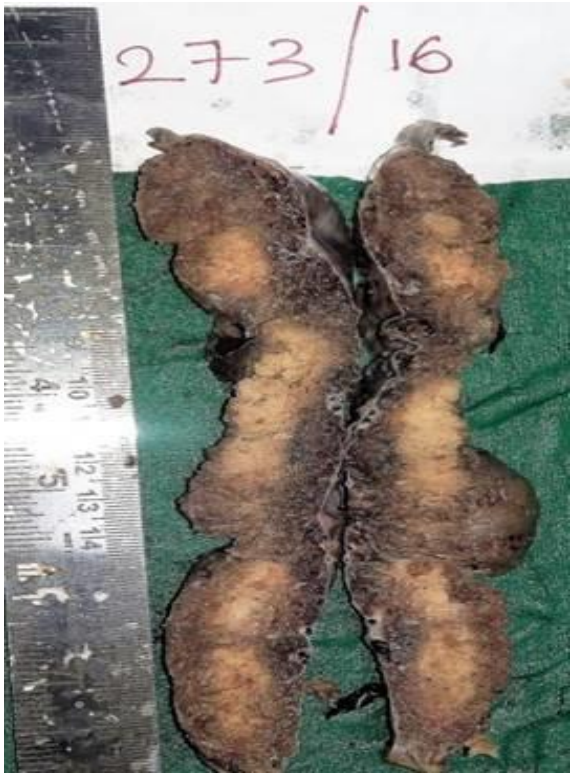


Figure – 3: Perivillous fibrinoid is a firm irregular yellowish white nodular area.

Figure – 4: Cut surface of placenta with infarction.

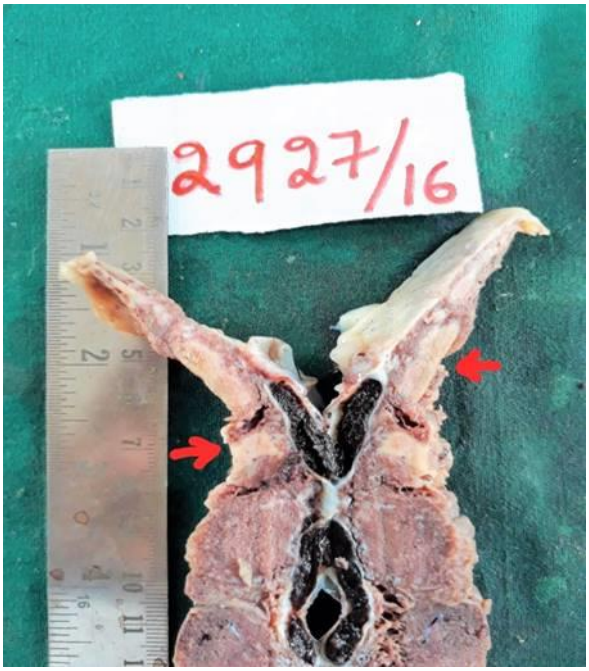


Figure – 5: Cut surface placenta with Retroplacental hematoma.

Figure – 6: Placenta with maternal floor infarct is large firm pale whitsh gritty area on maternal surface.

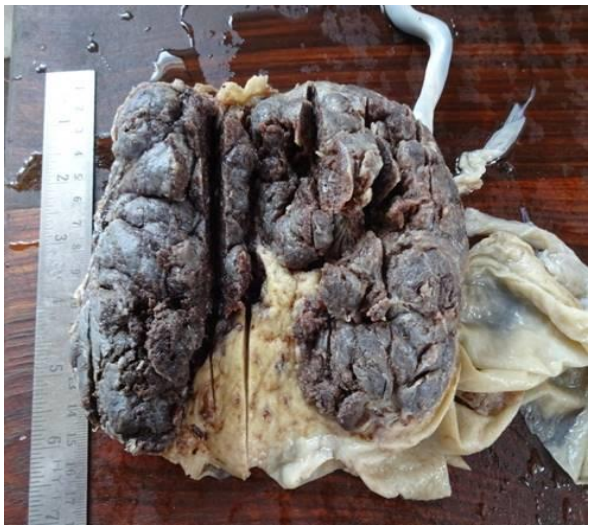


Figure – 7: Placenta with subchorionic hematoma.

Figure – 8: Placenta of IUGR baby. The dimensions are grossly reduced and are small for term pregnancy.

Figure – 9: Twin placenta showing two umbilical cords, two placental discs on microscopy showed Dichorionic Diamniotic type.

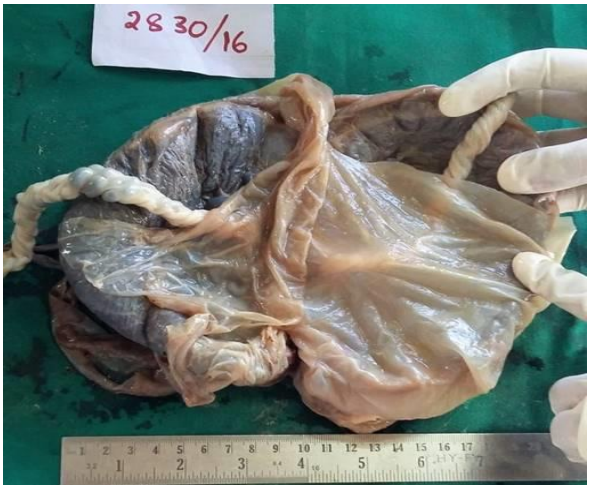


Figure – 10: Normal placental villi lining single layer of cytotrophoblast and a few syncytiotrophoblasts with fetal and maternal elements (H&E, 100X).

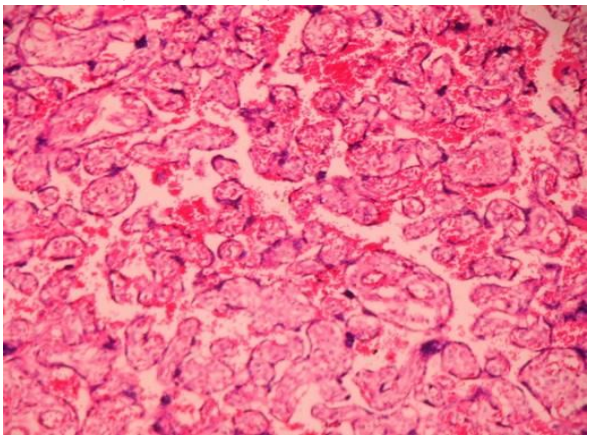


Figure – 11: Normal umbilical cord showing two arteries and one vein (H&E, 40X).

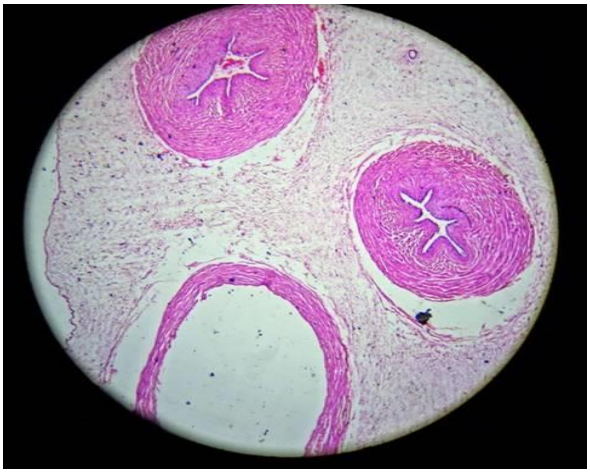


Figure – 12: Microscopic picture showing villi lined by syncytiotrophoblastic knots (H&E, 100X).

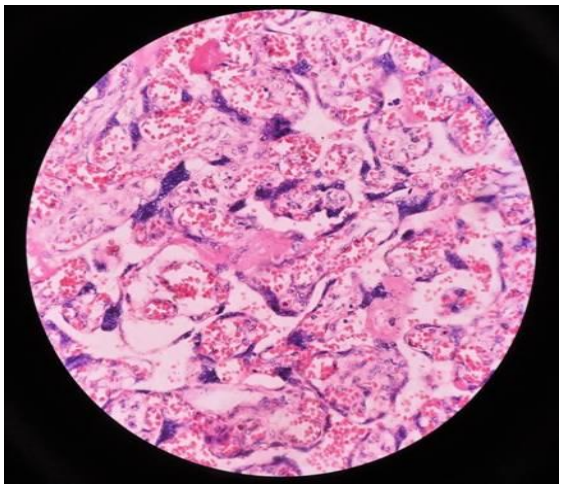


Figure – 13: Section showing syncytiotrophoblastic knots (H&E, 400X).

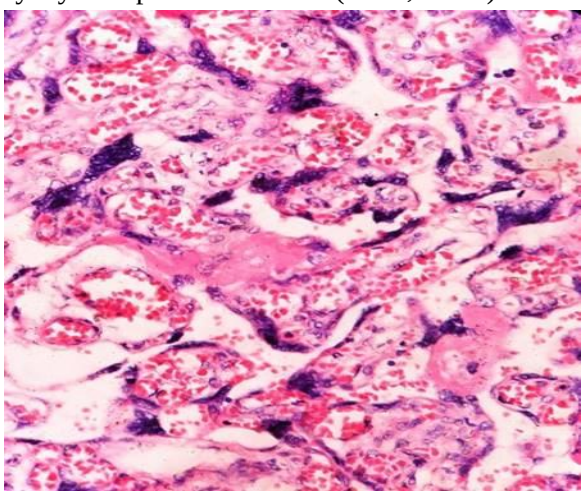


Figure – 14: Section shows villi with cytotrophoblastic hyperplasia (H&E, 400X).

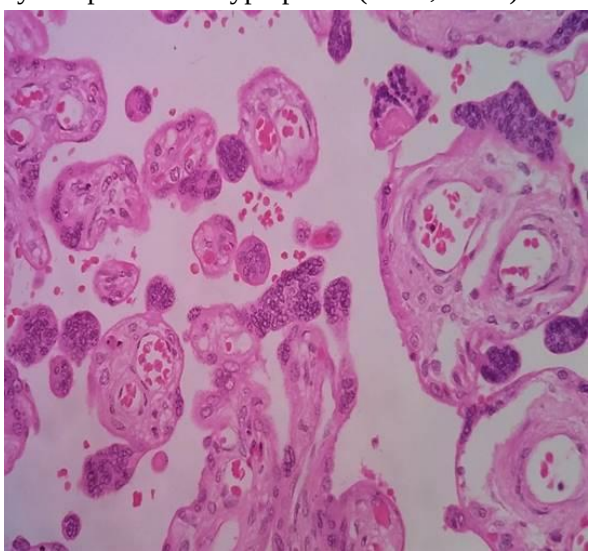
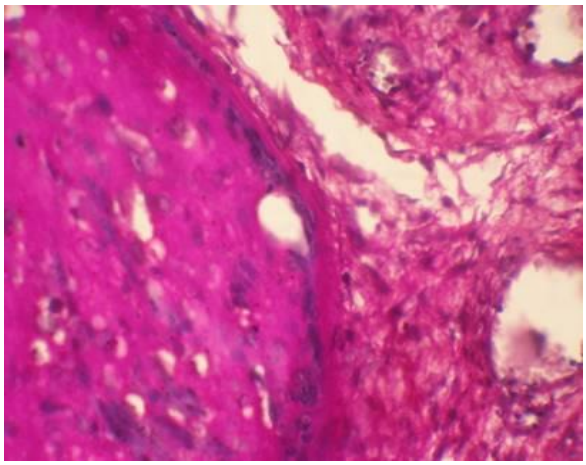


Figure – 15: Photomicrograph showing high power view of basement membrane thickening (PAS, 400X).



Intervillous fibrinoid (**Figure - 16**) was noted in 46.6% cases of IUGR, 33% in twins and 30.7 in diabetes, 21% in PIH, whereas in normal placentas, it was only 6.6%. Intra-villous fibrinoid deposits (**Figure - 17**) and perivillous fibrinoid deposits (**Figure – 18, 19**) were seen in 8 cases of IUGR.

Infarction (**Figure - 20**) was observed in 6.6% of normal pregnancies, 50% in PIH, 44.4% in anemia, 33% each in IUGR, twins and 30.7% in diabetes.

Fibrinoid necrosis (**Figure - 21**) was predominantly noted in IUGR placentas (53.4%), PIH (28.9%), anemia (25.9%), and in twins 33.3% and diabetes (30.7%).

Other microscopic findings like intervillous hemorrhage (**Figure - 22**), villous edema (**Figure - 23**), and immature villi (**Figure - 24**) were equally present in mild degree in both normal as well as high risk groups.

Calcification (**Figure - 25**) was common in both groups of placentas, and it accounted for 26.6%% in normal placentas, 60.5% in PIH, 55.5% in anemia, and 40% in IUGR.

Mild inflammation such as villitis (**Figure - 26**), chorioamnionitis (**Figures – 27, 28**) was seen in

anemia (33.3%), IUGR (26.6), and PIH (15.7%) but not found in normal group.

Figure – 16: Intervillous fibrinoid (H&E, 100X).

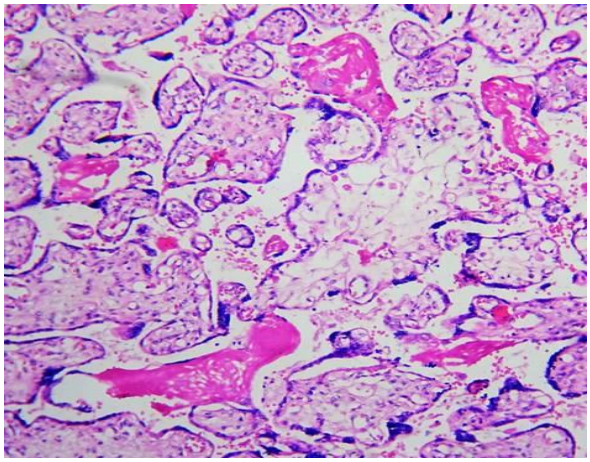


Figure – 17: Microscopic picture showing intravillous fibrinoid deposition (H&E, 100X).

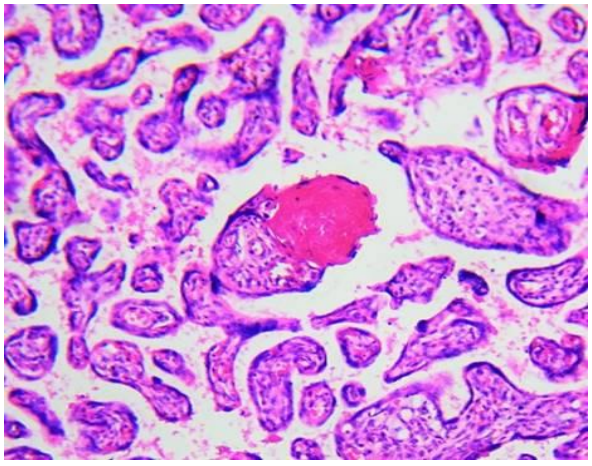


Figure – 18: Microscopic picture of perivillous fibrin deposition (H&E, 100X).

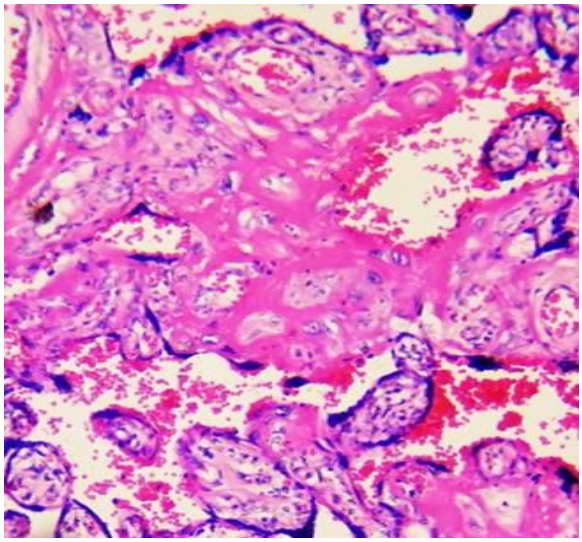


Figure – 19: Microscopic picture of perivillous fibrin deposition (H&E, 400X).

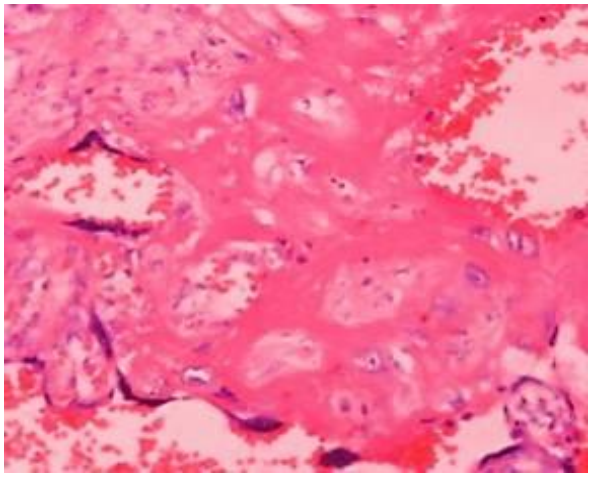


Figure – 20: Microphotograph of infarction with ghost like villi (H&E, 100X).

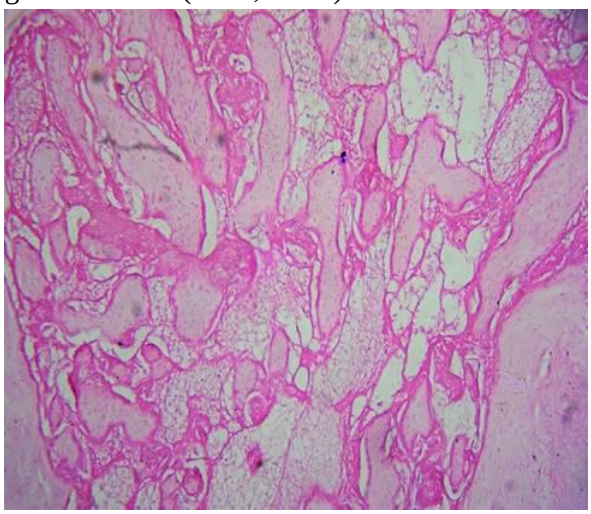


Figure – 21: Microphotograph showing fibrinoid necrosis.

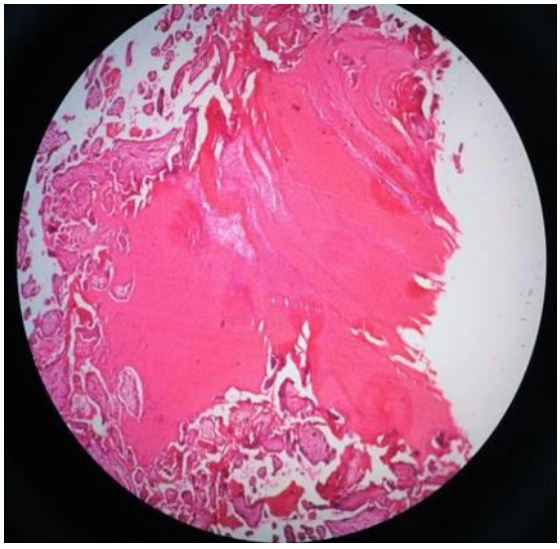


Figure – 22: Intervillous hemorrhage showing extensive blood filled areas (H&E, 100X).

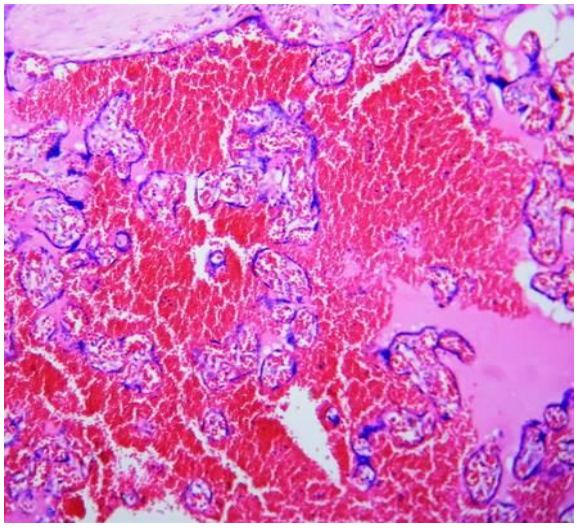


Figure – 23: Large villous structures with loose stroma in case of villous edema (H&E, 100X).

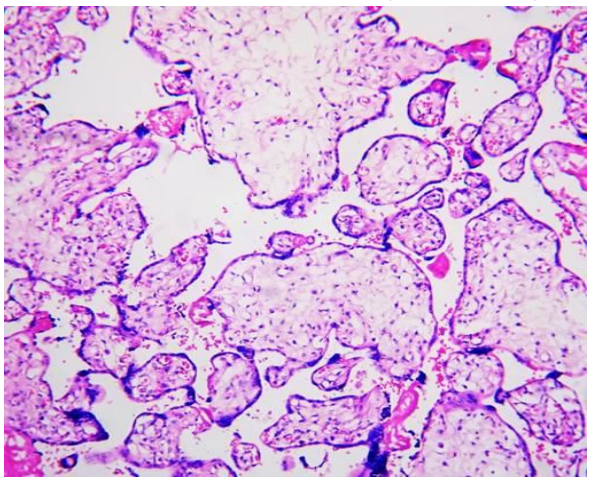


Figure – 24: Photomicrograph showing immature villi (H&E, 100X).

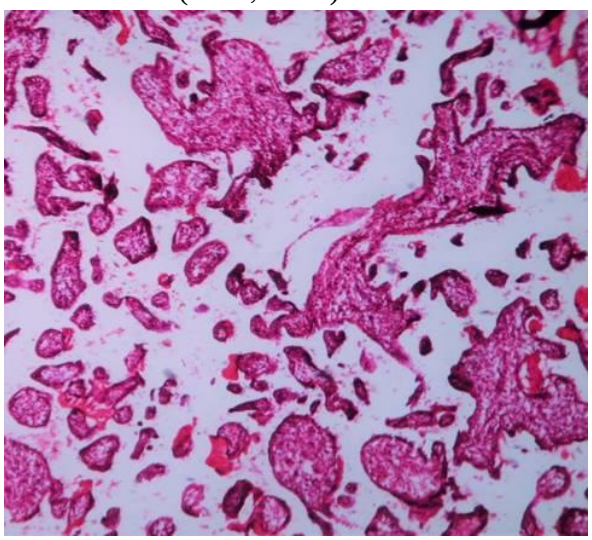


Figure – 25: Microphotograph showing calcium deposits in placental disc (H&E, 400X).

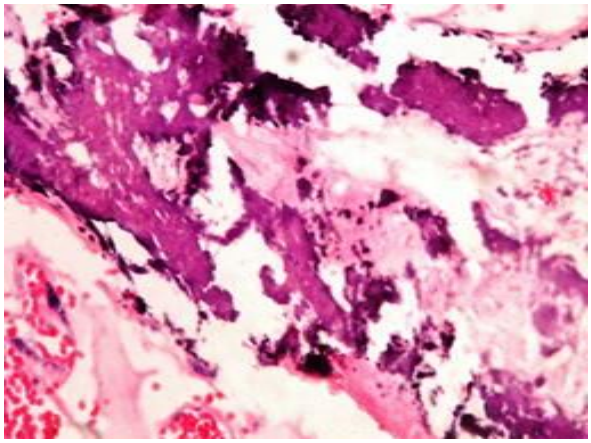


Figure – 26: Microphotograph showing villi with dense inflammatory villi distorting the villous architecture (H&E, 400X).

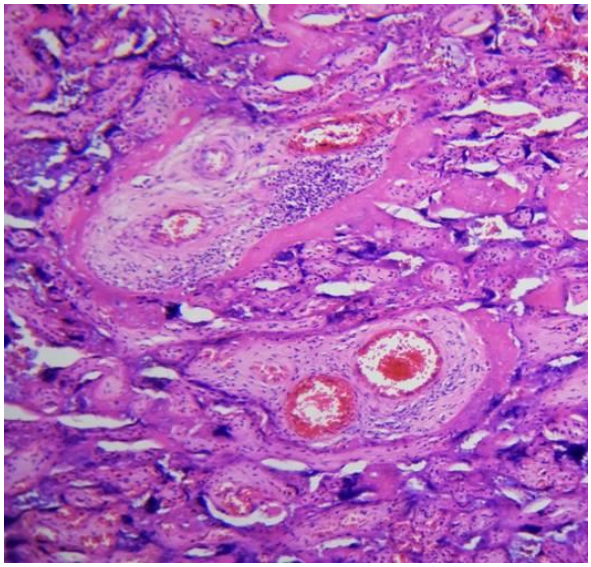


Figure – 27: Section showing chorioamnionitis (H&E, 100X).

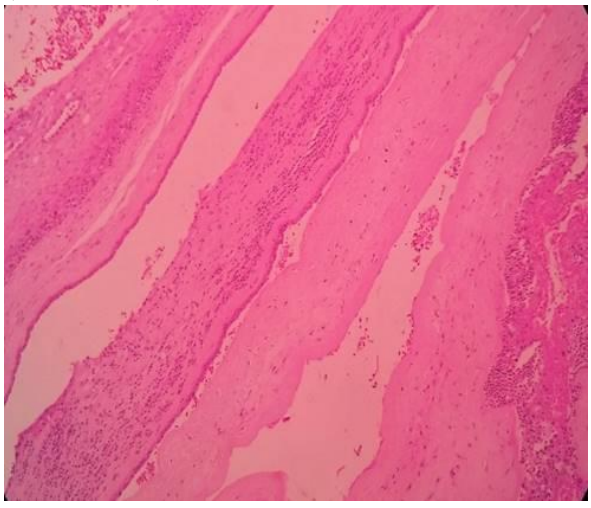


Figure – 28: Inflammtory infiltrate composed of neutrophils and esinophils in the membrane (H&E, 400X).

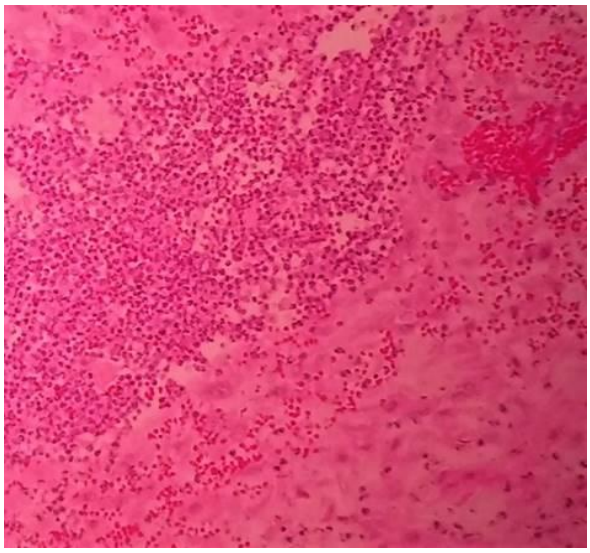


Figure – 29: Microphotograph showing Diamniotic Dichorionic type of twin placenta (H&E, 100X).

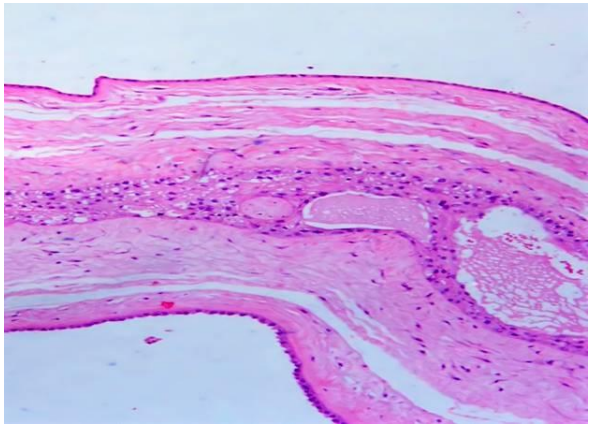
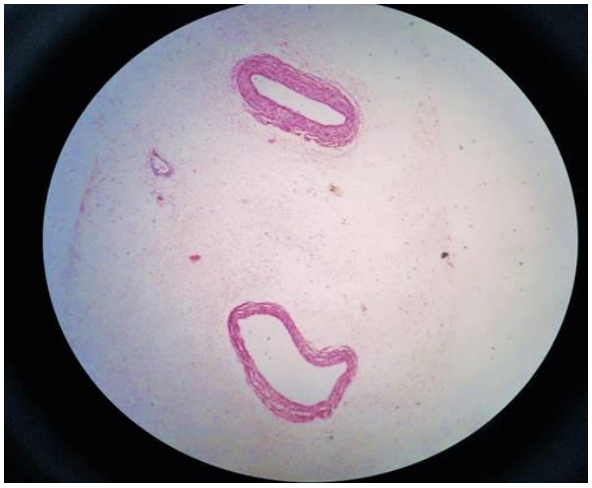


Figure – 30: Section showing umbilical cord with one artery and one vein (H&E, 40X).



All the twin placentas were of diamniotic dichorionic type (**Figure - 29**) with two amniotic membranes and two chorions, separated by scanty loose fibrovascular connective tissue.

In one case of diabetes, the umbilical cord showed one artery and one vein, but there were no gross abnormalities in the newborn (**Figure - 30**).

Discussion

A study of placental pathology in high risk pregnancies was undertaken for a period of two years in the Department of Pathology. The study group included 30 placentas from normal term pregnancies, and 102 placentas from high risk pregnancies of both maternal and fetal etiology, consisting of cases from anemia, pregnancy-induced hypertension, intrauterine growth retardation, maternal diabetes, and twin pregnancies.

Perivillous fibrinoid deposition was seen in 6.6% of controls in our study. It was a tan brown, granular material with irregular outlines and was located away from the maternal surface. It is considered to indicate local ischemic process. It was seen in 53.4% of IUGR and 22.2% in anemic cases.

Two cases of retroplacental hematoma was observed, one each in normal placenta (3.3%) and PIH (2.6%), the hematoma constituting 50% of the total placental volume. The outcome in these was a live born baby. Retroplacental hematoma causing adverse perinatal outcome is related to the size of the lesion and the severity of the accompanying disorder particularly pre-eclampsia, SLE and infarction. Macpherson [3] observed perinatal mortality in 3.9 per 1000 births in his clinicopathologic study, which showed 30% retro-placental hematoma.

One case of single umbilical artery was observed in diabetes in our study, and there were no gross fetal abnormalities. Only about 50% of cases with single umbilical artery had been associated

with congenital anomalies. Half of them had major structural or functional defects. Anomalies are often multiple, most frequently affecting genitourinary, musculoskeletal, cardiovascular, gastrointestinal and central nervous systems. An association with white race, diabetes mellitus, maternal hypertension and smoking had been reported. A single umbilical artery alone will not cause fetal hypoxia.

Rohini M [4] studied 30 cases of anemia, observed areas of infarction (56.67%) and calcification (63.33%) grossly which were higher when compared to control group (infarction 3% and calcification 26.66%). In the present study, infarction is 44.4% which is coinciding with the Rohini M study and calcification is 29.6% (**Table – 9**).

Table – 9: Gross findings in anemia.

Features	**Rohini M [4]** (n=30)	**Present study** (n=27)
Subchorionic fibrinoid	_	18.5%
Intervillous fibrinoid	_	14.8%
Perivillous fibrinoid	_	22.2%
Infarct	56.67%	44.4%
Calcification	63.33%	29.6%
Retroplacental hematoma	_	_

Mehendale [5], in a study of 43 cases of anemia, observed syncytiotrophoblastic knots in 88% of cases, whereas in the present study, it was 61% cases (**Table - 10**). The other findings included cytotrophoblastic proliferation, trophoblastic basement membrane thickening, and inflammation which were coinciding with the present study.

The decrease in the syncytiotrophoblastic knot formation may be due to variation in the degree of anemia. The knotting increases with low oxygen carrying capacity of the maternal blood which causes decreased functional capacity of the placenta. The theory of apoptosis of cytotrophoblasts leading to the formation of syncytiotrophoblast can be explained from this.

Table – 10: Comparison of microscopic findings in anemia.

Features	**Mehendale [5]** (n=43)	**Present study** (n=27)
Syncytial knots	88%	81.4%
Cytotrophoblastic proliferation	69.7%	59.2%
Basement Membrane Thickening	62.7%	59.2%
Fibrinoid necrosis	_	25.9%
Intervillous fibrinoid	_	18.5%
Infarction	44%	44.4%
Calcification	62.7%	55.5%
Inflammation	55.8%	33.3%

Narasimha A, et al. [6] studied 63 cases, observed subchorionic fibrinoid (9.52%) and Intervillous fibrinoid (47.6%). In present study subchorionic fibrinoid and Intervillous fibrinoid are 23.6% and 18.4%. There is variability of both changes when compared with the Narasimha, et al. as per **Table – 11**.

Table – 11: Gross findings in PIH.

Features	**Narasimha A [6]** (n=63)	**Present study** (n=)
Subchorionic fibrinoid	9.52%	23.6%
Intervillous fibrinoid	47.6%	18.4%
Perivillous fibrinoid	63.49%	26.3%
Infarct	41%	50%
Calcification	26.9%	57.8%
Retroplacental hematoma	1.1%	2.6 %

There is also variability in pervillous fibrinoid (26.3%) in the present study when compared with Narasimha A, et al. perivillous fibrinoid (63.49%).

Infarction in the Narasimha, et al. [6] grossly observed 41% and correlated with the present study 50%.

In Present study, calcification is 57.8% and compared with Narasimha study calcification 26.8%.

Retroplacental hematoma in the present study is 2.6% and correlated with the Narasimha, et al. study retroplacental hematoma (1.1%).

The present study showed the basement membrane thickening in 55.2% cases of pregnancy-induced hypertension (**Table - 12**). Fox [7] studied basement membrane thickening in 159 cases of PIH and observed thickening in no. of cases (52.6%) of cases. The study indicated that extensive thickening of villous trophoblastic basement membrane was principally the result of placental ischemia. This was suggested by the finding of a high incidence of basement membrane thickening in pre-eclamptic toxemia of pregnancy. The high incidence of hypoxic complications in babies of PIH implied that ischemia was due to the basement membrane thickening seen in these cases. The association between basement membrane thickening and Langhan's cell hyperplasia is a response to placental ischaemia [7].

Table – 12: Comparison of microscopic findings in pregnancy-induced hypertension.

Features	**Fox [7]** (n=159)	**Moldenhauer [8]** (n=52)	**Mehendale [5]** (n=18)	**Narasimha [6] (n=63)**	**Present study** (n=31)
Syncytial knots	_	_	87.5%	90.4%	71%
Cytotrophoblastic proliferation	_	_	87.5%	86%	63.1%
Basement Membrane thickening	52.6%	_	100%	95.23%	55.2%
Fibrinoid necrosis	_	_	_	97.8%	28.9%
Intervillous fibrinoid	_	19.2%	_	_	21%
Infarction	_	19.2%	87%	_	50%
Calcification	_	_	75%	_	60.5%
Inflammation	_	32.7%	12.5%	_	15.7%

There was a high variability of infarction in two different studies conducted by Mehendale [5] and Moldenhauer [8] separately and represented 87% and 19.2% respectively. The present study showed infarction in 50% cases of PIH.

In our study, 60% of the infarcts in the high risk cases were centrally located. It is important to describe the location of the lesion, whether it is juxtabasal, central or subchorionic with in the placental disc. Infarcts are more significant, if they are central and more than 3cm in greatest diameter [9]. The most likely location for ischemic villous death in the event of decidual vascular occlusion is at the maternal surface. In most of our cases, infarction was associated with acute or chronic villous inflammation. Our study showed 15.7% of inflammatory lesions in 31 placentas affected by PIH. Inflammatory changes were not very prominent in the placentas affected by PIH but a few cases had been recorded in various studies. Chorioamnionitis was seen in

32.7% in 52 cases of PIH in the study of Moldenhauer [8], but it was noted 12.5% by Mehendale [5].

Chronic villitis can occur as specific or non-specific form. The specific form is part of hematogenous infection, predominantly of viral origin, and fetal outcome is related to fetal infection. In some cases, the degree of villous damage is extensive, without any apparent immediate perinatal fetal compromise. In most cases of chronic villitis, infections which cannot be demonstrated are included in villitis of unknown origin [9].

Calcification can be prominent but is not the characteristic feature in PIH. It is of dystrophic type, where the placenta is damaged due to the discordant blood pressure. It was observed in 60.5% placentas in our study whereas Mehendale [5] noted in 75% of cases of PIH.

Intervillous thrombosis was seen in 19.2% of cases of PIH in Moldenhauer [8] and it was recorded as 22.6% in our study. It is mainly due to the access of fetal erythrocytes into the maternal space and initiation of intervillous thrombosis at sites of chorionic villous damage and hemorrhage.

Narasimha A, et al. [6] studied fibrinoid necrosis in 97.8% cases. Fibrinoid necrosis of villi or intravillous fibrinoid deposition was seen in 28.9% cases of PIH and was observed beneath the syncytiotrophoblasts. It was observed in 10% of normal pregnancies. It is due to degeneration from the placental ageing or hypoxic damage to the trophoblasts.

Acute atherosis was seen in 78% cases in the study of Khong [1] in 33 cases of PIH. Acute atherosis was seen in vessels that had not undergone physiologic vascular changes. In established acute atherosis, perivascular mononuclear cell infiltrate and lipophages were observed within the vessel wall that had been destroyed by fibrinoid necrosis.

In 75 cases studied by Khong [1], only 15 cases of placenta and placental bed biopsy specimens were available for examination and all of them showed the changes. A clinico- histologic study of acute atherosis in late complicated pregnancy was undertaken by Khong [1]. According to him, the findings of acute atherosis in normotensive women whose pregnancies were complicated by small for gestation age infants had been disputed, but its confirmation gave further support to the impression that hypertension alone does not cause acute atherosis. It was also seen in immunologic fetomaternal reactions which might be major determinants of acute atherosis.

The study of Moldenhauer [8] hypothesised that placental lesions were more common in the placentas of women with pre-eclampsia and particularly at early gestational age.

Hemalatha [10] studied 75 cases of IUGR, observed grossly subchorionic fibrinoid 33% and it is 53.4% in the present study (**Table – 13**).

Table – 13: Gross findings in IUGR.

Features	**Hemalatha [10]** (n=75)	**Present study** (n=)
Subchorionic fibrinoid	33%	53.4%
Intervillous fibrinoid	36.7%	40%
Perivillous fibrinoid	_	53.4%
Infarct	22.7%	33.3%
Calcification	33%	40%
Retroplacental hematoma	33%	_

In the present study intervillous fibrinoid is 40%, infarction is 33.3% and calcification is 40% correlated with Hemalatha [10] study which showed 36.7%, 22.7% and 33% respectively.

Intrauterine growth retardation is associated with increased pathological changes in the placenta leading to decreased functional capacity. The

placentas are usually small in size and may be associated with infection.

Fibrinoid necrosis was the common finding observed in IUGR placentas and various authors mentioned the quantity of fibrinoid necrosis as 43% (Altshuler [11]), 48% (Mehendale [5]), and 40% in the present study. Syncytial knot formation was found in 48.6% of IUGR placentas in the study of Mehendale [5]. The present study showed fibrinoid necrosis in 53.4% placentas.

Inflammatory lesions are variable in placentas of IUGR and they depend on the duration between the rupture of membranes and the delivery. It was found in 25% of 63 cases studied by Altshuler [11], 12% in 75 cases studied by Kavita [12], and the highest, 70% reported by Mehendale [5]. The main reason for such a high range of lesion in the latter study was because of the selection of cases from the mothers suffering from fever during pregnancy. The inflammation of the placenta itself might be the cause of maternal fever causing decreased placental function, and finally leading to IUGR (**Table – 14**).

Table – 14: Comparison of microscopic findings in intrauterine growth retardation.

Features	**Altshuler [11]** (n=63)	**Mehendale [5]** (n=39)	**Kavita [12]** (n=75)	**Present study** (n=15)
Syncytial knots	_	48.6%	_	53.4%
Cytotrophoblastic proliferation	30%	62%	44%	46.6%
Basement Membrane thickening	_	56.7%	40%	46.6%
Fibrinoid necrosis	_	_	_	53.4%
Intervillous fibrinoid	_	_	48%	46.6%
Infarction	43%	48%	28%	33.3%
Calcification		38%	36%	40%
Inflammation	25%	70%	12%	26.6%

Intervillous thrombosis seen in 46.6% in our series was almost equal to that of Kavita [12] (48%).

The present study showed basement membrane thickening in 46.6% cases. It was observed in 56.7% and 40% of cases respectively by Mehendale [5] and Kavita [12].

Calcification was correlating in all studies of IUGR placentas by Mehendale [5], Kavita [12] and ours, and respectively it was 38%, 36% and 40%.

Cytotrophoblastic proliferation was variable in various studies and accounted for 30% in Altshuler, 62% in Mehendale, 44% in Kavita. The present study correlated with the latter (46.6%).

The outcome in the IUGR placenta is a low birth weight baby. Neonatal morbidity and mortality may also be high and are mainly due to inflammatory and / or morphological changes in the placenta.

Basement membrane thickening was observed in 23.5% cases of placentas of diabetic mothers in a study by Fox [7] in 27 cases. Tewari V, et al. [13] observed in 100% cases of placentas i.e. in all 30 cases. In our study, it was seen in 30.7% cases. Thickening of the basement membrane is a significant finding in diabetic mothers which affects the materno-fetal perfusion.

Tewari V, et al. [13] study revealed increased syncitial knots in 80 % cases as compared with normal placenta .The areas of fibrinoid necrosis also showed the same pattern (80%). In the present study syncitial knots is seen in 69.2% and fibrinoid necrosis in 30.7% (**Table – 15**).

Table – 15: Placental findings in Diabetes mellitus.

Features	**Fox [7]**	**Tewari V [13]** (n=30)	**Present study** (n=13)
Syncytial knots	_	80%	69.2%
Cytotrophoblastic proliferation	_	_	69.2%
Basement Membrane Thickening	23.5%	100%	30.7%
Fibrinoid necrosis	_	80%	30.7%
Intervillous fibrinoid	_	_	30.7%
Infarction	_	_	30.7%
Calcification	_	_	30.7%
Inflammation	_	_	_

Any lesion that occurs in singleton placenta can also be found in twin Placentas. The main challenge for the pathologist is to distinguish monochorionic diamniotic placenta and fused dichorionic diamniotic placenta [14]. This is achieved by observing the dividing membrane microscopically as was done in our cases, which were all of dichorionic diamniotic type.

Conclusion

The present study has highlighted the importance of examination of placenta in normal as well as high risk pregnancies. It serves as a very valuable diagnostic tool to understand and explain the pathophysiology of various conditions affecting the mother, child, or itself. Standardization of the method of placental examination is essential to achieve optimal benefits from the diagnostic reports [15]. The placental changes are essential to correlate the fetal outcome, as it provides the information for the cause of death. Hence it has an effective role in planning prenatal monitoring of a future pregnancy. A thorough and proper study of placenta coupled with complete clinical details, will certainly enrich our knowledge to ensure better maternal and neonatal care.

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