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Original Research Article

A Prospective Study on Histopathological Analysis of Placenta in IUGR pregnancies in comparison with non-IUGR cases in a Tertiary Care Hospital

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Abstract

Introduction: The placenta is a crucial organ and reflects the state of the mother and fetus.

Objectives: (1). To analyse the histopathological spectrum of placenta. (2). To compare the placental findings of IUGR and non-IUGR cases.

Materials and Methods: In this prospective study 105 placentas were included. Out of which 47 cases were associated with IUGR and the remaining 58 cases were considered as non- IUGR cases. Gross and microscopic examination was done.

Results: The gross findings of mean placental weight, diameter and thickness between IUGR and non- IUGR cases was statistically significant ($p<0.05$). The histopathological findings of ischemic necrosis, calcification, hyalinisation, chorangiosis, syncytial knots and chorioamnionitis were seen in higher number of IUGR cases significantly ($p<0.05$).

Conclusion: The pathological findings of placenta were seen more commonly in cases associated with IUGR.

Keywords: Placenta, Histopathology, IUGR.

Introduction

The placenta is a crucial organ for sustaining pregnancy and fostering fetus growth [1]. This essential organ is necessary for the embryo to survive inside the uterus. The state of the mother and fetus is reflected in the placenta.

Any pathological incident that affects the mother or the fetus will affect the placenta's usual function, leading to morphological and histopathological changes [2,3]. The role of placenta is to act as a functional unit between the mother and the fetus. The placenta serves as a record of occurrences during pregnancy [4].

The idea that a serious illness in adulthood results from improper intrauterine development may have a significant effect on public health initiatives for disease prevention [5].

Pathogenesis of preeclampsia, preterm births, abortions, IUGR, and intrauterine death have all been related to defective placentation [6,7]. Disorders that cause placental failure can occur repeatedly [8,9]. Finding these risk factors and correlating them with the clinical findings can be accomplished more successfully with more thorough pathological and morphological studies on the placenta.

Objectives:

1. To analyse the histopathological spectrum of placenta.
2. To compare the placental findings of IUGR and non-IUGR cases.

Materials and Methods

This prospective study was conducted over a period of one year. A total of 105 placenta specimens (preterm, term and post term gestations) with clinical details were received to the Department of Pathology, Kodagu Institute of Medical Sciences, Madikeri.

The placenta specimens received were from both vaginal and caesarean deliveries. These specimens were fixed in 10% formalin for 24 hours. The placenta was grossly examined and all the necessary measurements were recorded. The multiple tissue bits from representative areas were processed and embedded in paraffin wax. Sections of 4-5micron

thickness were taken and stained by Haematoxylin and Eosin (HE) for histopathological examination.

Inclusion Criteria: All placentas received in the Department of Pathology for histopathological examination

Exclusion criteria: Autolyzed specimens, twin gestation, fetuses with known chromosomal anomalies.

The various microscopic features were noted and compared between IUGR and non- IUGR placenta groups by use of relevant statistical parameters.

Results

In the study the mean maternal age was 26.1 years ranging between 18 to 45 years. The percentage of primigravidae comprised of 42.3%. At the time of delivery, the mean gestational age was 35.65 weeks. Among 105 cases, intrauterine fetal demise was seen in 9 cases and intrauterine growth retardation in 38 cases. The gross measurements of placenta in the IUGR and IUD cases were as follows- mean placental weight 306.4 grams, mean placental diameter was 15.32 cms and mean placental thickness at center was 2.36cms. 46.6% of placenta had eccentric umblical cord insertion and the remaining 53.4% showed central umblical cord insertion. The gross measurements of placenta in non-IUGR cases were as follows- mean placental weight 389.6 grams, mean placental diameter was 17.62 cms and mean placental thickness at center was 1.75cms. 53.4% of placenta had eccentric umblical cord insertion and the remaining 46.6% showed central umblical cord insertion.

The placenta weight, diameter and thickness between the IUGR and non-IUGR groups showed significant statistical difference with p value of 0.038, 0.0001 and 0.0001 respectively but the insertion of umblical cord did not vary significantly. The gross measurements of all the placentas are shown in the Table 1.

Table 1: Gross Measurements

Gross measurements	IUGR	NON-IUGR	p value
mean placental weight	306.4 grams	389.9 grams	0.038
mean placental diameter	15.32 cms	17.62 cms	0.0001
mean placental thickness	2.36 cms	1.75 cms	0.0001
eccentric umblical cord insertion	46.6%	53.4%	0.572

The microscopic findings of all the 105 placentas are shown in the Table 2 and Fig 1.

Table 2: Microscopic Features

Microscopic findings	IUGR (47)		NON-IUGR (58)		p value
	Frequency(n)	Percent(%)	Frequency(n)	Percent(%)	
No significant pathology	6	12.8	45	77.6	0.0001
Ischaemic changes/ infarct/ necrosis	41	87.2	13	22.4	0.0001
Calcification	23	48.9	8	13.8	0.0001
Chorangiosis	14	29.8	7	12.1	0.024
Hemorrhage	4	8.5	7	12.1	0.554
Hyalinisation	9	19.1	4	6.9	0.058
Syncytial knots	7	14.9	2	3.5	0.037
Chorioamnionitis	13	27.7	35	60.4	0.001
Funisitis	1	2.1	2	3.5	
Thickened blood vessels	5	10.6	1	1.7	

In the IUGR group, the predominant microscopic feature noted was ischemic necrosis (87.2%) which was followed by calcification (48.9%) and chorangiosis (29.8%). Other findings were hemorrhage (8.5%), hyalinisation (19.1%), syncytial knots (14.9%), chorioamnionitis (27.7%), funisitis (2.1%) and thickened blood vessels (10.6%). Whereas in the non- IUGR group, majority of the placentas did not show significant histopathological findings (77.6%).

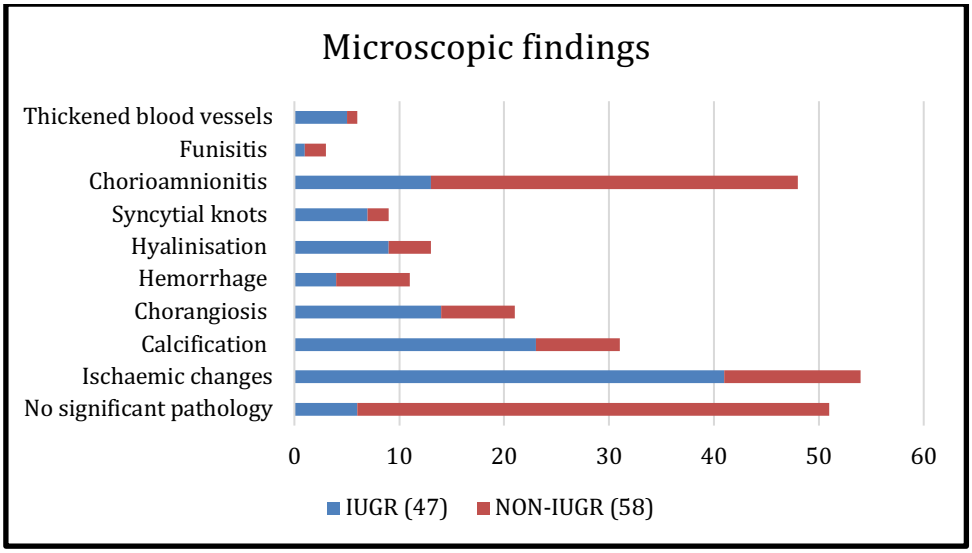

Figure 1: Frequency of microscopic findings of placenta in IUGR and NON-IUGR cases.

Figure 2: Gross specimen of placenta showing retroplacental clots.

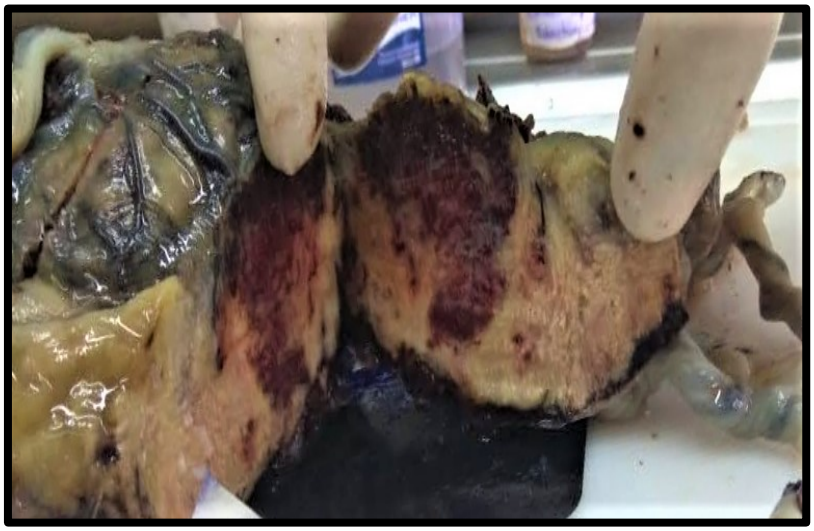

Figure 3: Gross specimen of placenta showing a large infarct.

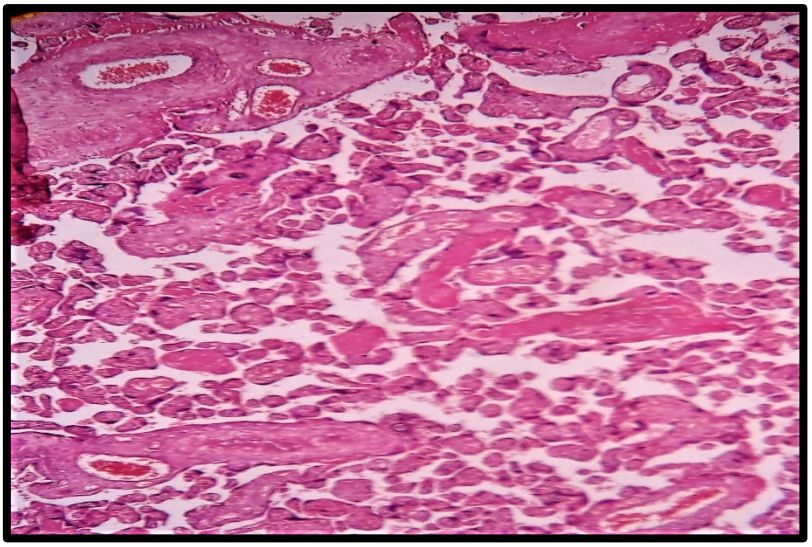

Figure 4: Microscopic image of placenta showing thickened blood vessels. HE stain (400X)

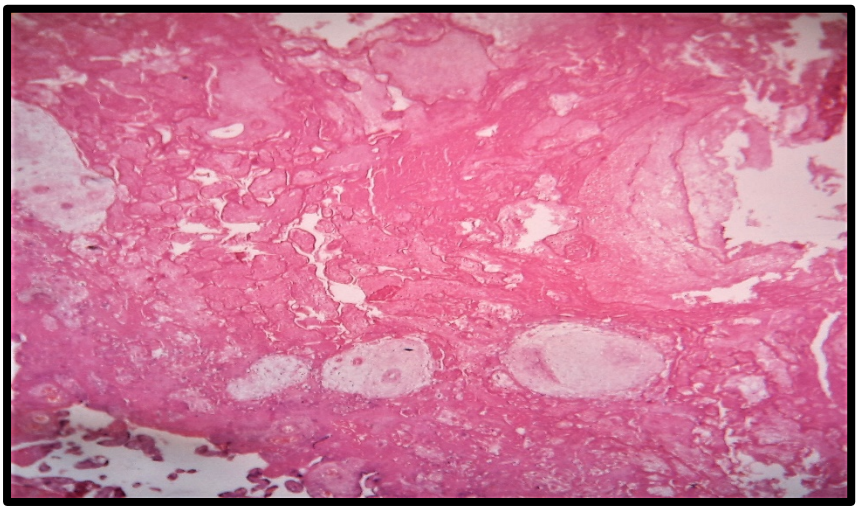

Figure 5: Microscopic image of placenta showing infarcted area. HE stain (400X)

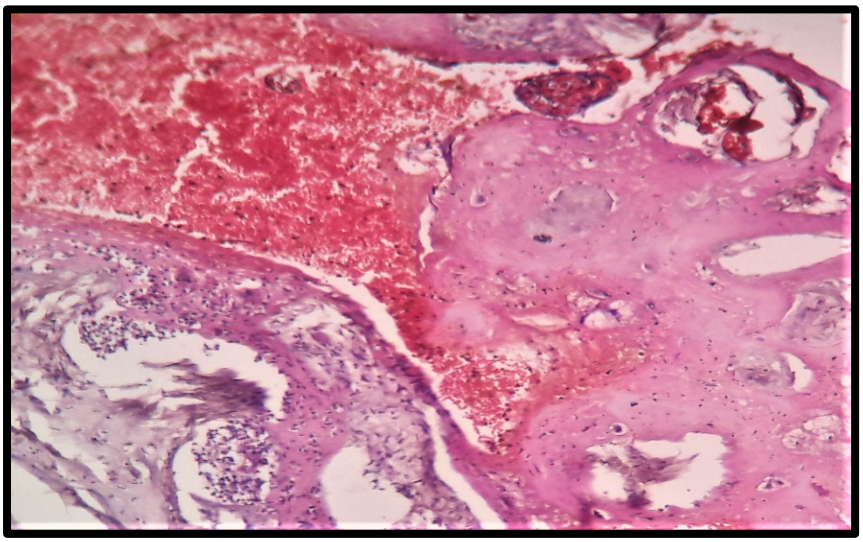

Figure 6: Microscopic image of placenta showing area of hemorrhage and hyalinisation. HE stain (400X)

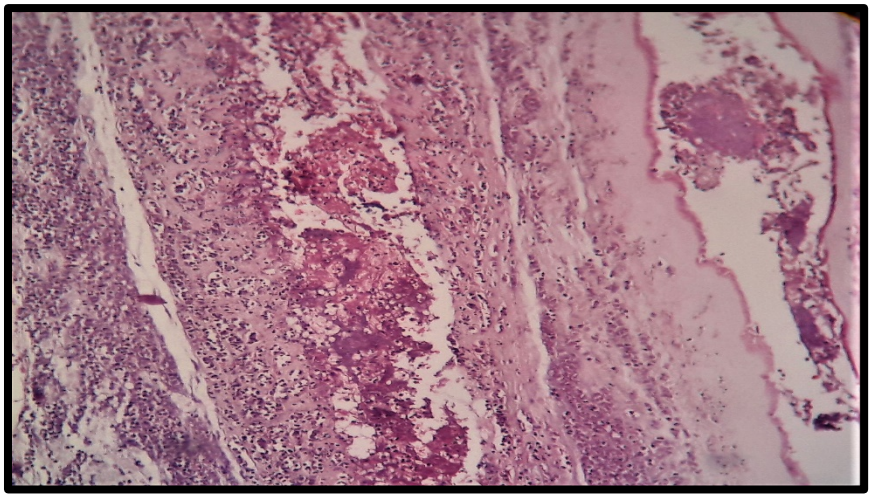

Figure 7: Microscopic image showing chorioamnionitis. HE stain (400X)

Discussion

IUGR can result by multiple factors related to maternal, placental, or fetal elements. Maternal factors that contribute to IUGR include preeclampsia, hypertension, diabetes mellitus, thrombophilias, extreme malnutrition, chronic renal disease, tobacco and other drug abuse, and poor obstetric history in general. Fetal factors include chromosomal anomalies (including confined placental mosaicism), congenital malformations, and multiple gestation10. Placental lesions associated with IUGR include: (1) vascular lesions that reduce maternal blood flow (decidual vasculopathy or chronic abruption), (2) vascular lesions that reduce fetal blood flow, (fetal thrombotic vasculopathy, large chorangiomas, or umbilical cord abnormalities), and (3) lesions that greatly reduce the amount of functional placenta (extensive chronic villitis, massive perivillous fibrin deposits, maternal floor infarction, or multiple infarcts [11]. Hence detailed evaluation of placenta is a very important aspect in assessing the cause of IUGR.

A request for a pathologic investigation of the placenta should include a synopsis of the clinical history relevant to the evaluation, together with the rationale for submission and specific inquiries– the mother's gravidity, parity, details concerning previous pregnancies, underlying maternal disease, antepartum course, labor and deliver, and the infant's gestational age, weight, and Apgar scores. In our study the gross findings with respect to mean placental weight, thickness and diameter showed statistical significance of p value <0.05 between the IUGR and non-IUGR cases. This was similar to the findings in the studies by Gupta N et al [12], Jakó M et al [13] and Kotgirwar S et al [14]. The most frequent causes of abnormally small placentas are fetal growth restriction and chronic uteroplacental underperfusion, which are brought on by maternal conditions linked to vascular disease [15]. One case of single umblical artery was documented in our study. One common and significant cord anomaly is the existence of a single umbilical artery (SUA). Although there is a well-established link between fetal abnormalities and a single umbilical artery, this link is not unique to any one organ or defect. Any organ system could be impacted, and numerous abnormalities are common. If an infant survives the neonatal period with a single artery cord and no discernible defects at birth, it is rare that further serious abnormalities would be found later [16].

In the current study, highly significant difference was noted with regards to the frequency of infarcts or ischemic changes on microscopy between the IUGR and uncomplicated cases showing p<0.001. Concordant observations were seen in Gupta N et al [12], Kaur G et al [17], Günyeli et al [18] and Kotgirwar S et al [14] studies. Chronic infarcts are distinguished by focal or multifocal abundance of fibrinoid material, while acute infarcts are easily recognized by the ghostly appearance of the villi. Shrunken, sclerotic villi with many condensed syncytial knots are signs of chronic placental ischemia [19].

A study by Saragade P et al [20] found the prevelance of calcifications in IUGR placentas statistically significant in comparison to normal gestation. This concurred with our present study where significant p value of <0.001 was seen and other studies by Nigam JS et al [21], Dhabhai P et al [22], Goswami P et al [23] and Goswami PR [24]. Many placentas with higher levels of fibrin or fibrinoid material acquire calcification. The majority of normal term placentas have moderate amounts of calcium, however hypoxic preterm babies may also have significant and widespread calcification. Mothers who smoke extensively and primigravidas may have higher levels of placental calcification [25]. We observed increased incidence of microscopic feature of syncytial knots in IUGR pregnancies showing statistical significance of p<0.05 in our study. This was in agreement with studies done by Sheela PV et al [26], Gupta N et al

[12] and Kaur G et al [17]. Other signs of uteroplacental underperfusion, including as decidual arteriopathy, placental infarction, and low placental weight, frequently coincide with increased syncytial knots [27].

Our study reported hyalinisation and chorangiosis in a greater number of placentas with IUGR when compared to non-IUGR cases which was significant statistically ($p<0.05$). This was also seen in study by Gupta N et al [12]. Karmakar MK [28] also reported increased incidence of hyalinisation in PIH induced IUGR cases.

Other studies by Kaur G et al [17] and Jain K et al [29] found significant number of IUGR cases with leucocyte infiltration compared to the control group. This observation was discordant in our study, where chorioamnionitis was seen predominantly in non-IUGR cases. Similar findings were seen in Sheela PV et al [25].

We also reported histopathological features of hemorrhage, funisitis and thickened blood vessels in the current study which were statistically insignificant.

It is not necessary for all of the different microscopic placental response patterns to be present at the same time; it is unclear why some patterns, even in cases with severe overall disease, occur while others do not.

Conclusion

Our study emphasises placental assessment to become routine part of obstetric care and to be routinely sent for histopathological analysis. The placenta from every newborn sent for examination deserves careful attention from the pathologist. All the pathologists have to be trained to analyse placenta. The outcome of future pregnancies can be improved by integrating placental analysis as regular component in patient care.

References

1. Udainia A, Jain ML. Morphological Study of placenta in pregnancy induced hypertension with its clinical relevance. J Anat Soc India. 2001; 50(1):24-7.
2. Hagirtai B, Marton T, Cox BM. Examination of the human placenta. Journal of Clinical Pathology. 2004; 57:785–92.
3. Hay WW Jr, Catz CS, Grave GD, Yaffe SJ. Workshop summary: Fetal growth: Its regulation and disorders. Pediatrics. 1999; 585–91.
4. Altshuler G. The placenta. In: Strenbergssed's Diagnostic Surgical Pathology. Philadephia: Lippincott Williams and Wilkins 1999; 2087-2107.
5. Jansson T, Powell TL. Human placental transport in altered fetal growth: Does the placenta function as a nutrient sensor? A review. Placenta. 2006; 27:92-7.
6. Redline RW. Placental pathology: a neglected link between basic disease mechanisms and untoward pregnancy outcome. Curr Opin Obstet Gynecol 1995; 7: 10-5.
7. Ness RB, Sibai BM. Shared and disparate components of the pathophysiologies of fetal growth restriction and preeclampsia. Am J Obstet Gynecol 2006; 195: 40-9.
8. Chaiworapongsa T, Romero R, Kusanovic JP, Savasan ZA, Kim SK, Mazaki-Tovi S, et al. Unexplained fetal death is associated with increased concentrations of anti-angiogenic factors in amniotic fluid. J Matern Fetal Neonatal Med 2010; 23: 794-805.
9. Canterino JC, Ananth CV, Smulian J, Harrigan JT, Vintzileos AM. Maternal age and risk of fetal death in singleton gestations: USA, 1995-2000. J Matern Fetal Neonatal Med 2004; 15: 193-7.
10. Wilkins-Haug L, Quade B et al (2006) Confined placental mosaicism as a risk factor among newborns with fetal growth restriction. Prenat Diagn 26:428–432
11. Sebire NJ, Sepulveda W (2008) Correlating placental pathology with ultrasound findings. J Clin Pathol 61:1276–1284
12. Gupta N, Khajuria A. Histomorphological Features of Placenta in Pregnancy Complicated with Intrauterine Growth Retardation. JK Science 2016; 18: 21-25.
13. Jakó M, Surányi A, Kaizer L, Németh G, Bártfai G. Maternal Hematological Parameters and Placental and Umbilical Cord Histopathology in Intrauterine Growth Restriction. Med Princ Pract 2019; 28:101–108.
14. Kotgirwar S, Ambiye M, Athavale S, Gupta V, Trivedi S. Study of gross and histological features of placenta in intrauterine growth retardation. J. Anat. Soc. India 2011; 60(1): 37-40.
15. Naeye RL. Do placental weights have clinical significance? Hum Pathol. 1987;18:387–391.
16. Raio L, Ghezzi F (1998) The clinical significance of antenatal detection of discordant umbilical arteries. Obstet Gynecol 91:86–91
17. Kaur G, Singh B, Kaur P, Kaur N, Khan E. Perinatal Outcome and Histopathological Evaluation of Placentas in Intrauterine Growth Restriction (IUGR) Pregnancies. International Journal of Medical Science and Clinical Inventions 2017; 4(11): 3293-3298.
18. Günyeli I, Erdemoğlu E, Ceylaner S, Zergeroğlu S, Mungan T. Histopathological analysis of the placental lesions in pregnancies complicated with IUGR and stillbirths in comparison with noncomplicated pregnancies. J Turkish-German Gynecol Assoc 2011; 12: 75-9.

19. Kaplan C. Fetal and maternal vascular lesions. Semin Diagn Pathol 2007;24:14-22.
20. Saragade P, Chaudhari R, Chakravarti A. Study of Histopathological findings of Placenta in Cases of Deliveries at Tertiary Health Care Institute. MVP Journal of Medical Sciences 2017; 4(2): 165–171.
21. Nigam JS, Misra V, Singh P, Singh PA, Chauhan S, Thakur B. Histopathological Study of Placentae in Low Birth Weight Babies in India. Annals of Medical and Health Sciences Research 2014; 4(2):79-83.
22. Dhabhai P, Ghanshyam G, Bapna N. Histological study of human placenta in normal and Pregnancy Induced Hypertension (PIH) cases. International Journal of Pharmaceutical Science Invention. 2013; 2(11):30–5.
23. Goswami P, Memon S, Pardeep K. Histological and radiological study of calcified placenta. IOSR Journal of Dental and Medical Sciences. 2013; 7(4):37–41.
24. Goswami PR, Shah SN. Placenta in normal and pregnancy induced hypertension in relation to its clinical significance: A gross study. International Journal of Scientific Study. 2016; 4(7):58–61.
25. Redline RW, Boyd T, Campbell V, et al. Maternal vascular underperfusion: nosology and reproducibility of placental reaction patterns. Pediatr Dev Pathol 2004;7:237-249.
26. Sheela PV, Sridevi M, Sujatha R, Saroja V. Placental Pathology in Intra Uterine Growth Retardation. Journal of Evolution of Medical and Dental Sciences 2015; 4(28): 4809-4815.
27. Naeye RL. Functionally important disorders of the placenta, umbilical cord and fetal membranes. Hum Pathol. 1987; 18:680–691.
28. Karmakar MK, Kar S, Kumar SM, Chattopadhyay SK, Vaid LK, Sen S. A study of histological changes of human placenta in rural population of eastern India. Int J Reprod Contracept Obstet Gynecol 2018; 7: 3280-7.
29. Jain K, Kavi V, Raghuveer CV, Sinha R. Placental pathology in pregnancy induced hypertension (PIH) with or without intrauterine growth restriction. Indian J Pathol Microbiol 2007; 50(3):533-537.