Original Article

A MORPHOLOGICAL, HISTOLOGICAL STUDY OF PLACENTA IN NORMAL AND HYPERTENSIVE MOTHERS

Avinash chandra[1], R.K.Srivastava[2], Shirin Jahan[2], T Praveen[3], P Kiran Kumar [3]

1. Postgraduate, Department of Anatomy, Rama Medical College, Hospital and Research Centre, Kanpur
2. Professor, Department of Anatomy, Rama Medical College, Hospital and Research Centre, Kanpur
3. Tutor, Department of Anatomy, Rama Medical College, Hospital and Research Centre, Kanpur.

Abstract

The placenta is a unique organ, short lived by design. Its existence is essential for the survival of human embryo/foetus in the intra uterine environment. Pregnancy complications like hypertension (PIH) reflected in the placenta in a significant way both macroscopically and microscopically. The present study was carried out in 70 patients divided into two groups 50 normal, 20 cases of hypertensive. The placenta undergoes a variety of structural and functional changes as follows; placental weight, diameter and umbilical cord length were less in hypertensive placenta then normal placenta. In microscopic examination the hypertensive placenta showed increased syncytial knot & fibrinoid necrosis will lead to fetal distress syndrome due to hypoxia. **Results:** In 50% placentas Grade I increased syncytial knots were seen and 15% placentas showed Grade Il increased knots, 25% placentas showed decrease in syncytial knots, 10% placentas showed the syncytial knots comparable to those of normal placenta.The thinning of trophoblastic basement membrane was seen in 80% placentas and the basement membrane thickening was normal in 20% placentas. Crowding of villi and fibrin deposition was seen to be increased in 50% cases and 50% showed normal distribution.Thrombi deposition was significantly seen to be increased in 85% placentas.Thinning of the vessel wall was seen in 70% placentas. **Conclusion:** The mean weight, diameter of placenta& length of umbilical cord were low in hypertensive mother when compared to normotensive mother. On microscopic examination syncytial knot, fibrinoid necrosis, crowding of villi, thrombi & fibrin deposition of placenta were increased in hypertensive placenta when compared to normotensive placenta.Thinning of trophoblastic membrane & foetal vessel wall in hypertensive placenta.The above mentioned histo-morphological changes of placenta in hypertensive mothers leads to fetal distress syndrome due to hypoxia.

Key Words: Placenta, Pregnancy Induced Hypertension (PIH), Syncytial Knot, Intra Uterine Growth Retardation (IUGR), Fetal Distress syndrome.

Introduction

The placenta is a complex fetal organ that fulfills pleiotropic roles during fetal growth. The placenta is formed from elements of the membranes which surround the developing fetus as well as the uterine endometrium and provides the means for physiological exchange between the fetal and material circulations [1]. At various stages during development, the placenta performs a remarkable range of functions, until the fetal organs become functional [1]. These include gaseous exchange, excretion, and maintenance of homeostasis, hormone secretion, haemopoiesis and hepatic metabolic functions. Maternal or fetal disorders may have placental sequelae since the mother and fetus interface at this site[2]. Conversely, primary placental abnormalities can affect both maternal and fetal health. Thus, examination of the placenta may yield information on the impact of maternal disorders on the fetus or the cause of preterm delivery, fetal growth restriction, or neurodevelopment impairment[2].

Pregnancy complications like hypertension reflected in the placenta in a significant way both macroscopically and microscopically, the placenta undergoes a variety of structural and functional changes[2]. Their nature and extent depend on a range of variables including the quality of glycemic control achieved during the critical periods in placental development, the modality of treatment, and the time period of severe departures from excellent metabolic control of a non hypertension environment [1]. It has been recorded that the maternal utero-placental blood flow is decreased in pre-eclampsia[1], because there is maternal vasospasm. Maternal vasospasm leads to foetal hypoxia. Reduced maternal utero-placental blood flow leading indirectly to constriction of foetal stem arteries [2] has been associated with the changes seen in the placenta of pre-eclamptic women [3]. Calculated that 70% of the excess foetal deaths in women with hypertension are due to large placental infarcts[4]markedly small placental size and that histopathological changes related to confined placental mosaicism may be associated with inadequate placentation and hence with retroplacental ischaemia[5]. Foetal hypoxia is not uncommon near term and accordingly it may lead to foetal distress and foetal death. In recent years, it has been revealed that there is a clear relationship between confined placental mosaicism and foetal growth[6].

This study was conducted to find out histopathological changes in the placenta of normal and hypertensive mothers.

Material and Methods

Histopathological examination of placenta was done on cases obtained from the Obstetrics & Gynaecology, Department of Rama Medical College, Hospital & Research Center, Kanpur with patient consent. Duration of study was one year.

Study was carried out in 70 patients divided into two groups 50 normal, 20 cases of hypertensive will be studied & their results will be compared .The age range of these mothers varied from 20 years to 38 years, primi and para 2 to 3 and belonged to rural and urban population.

Results

The present study includes 70 placentas which were grouped under two categories normal and hypertensive. The placenta was considered hypertension of the patient whose blood pressure was in the range 130/90 – 180/140 mmHz.

Table 1

Category	Placental weight (Gms)weight -Range & mean	Placental Diameter-range &mean (Cms)	Umbilical cord length (Cms)
Normal (n=50)	450-550 (503)	15-20(16.0)	20-56(40)
Hypertensive(n=20)	350 - 450(410)	10-25(15.8)	26-44(33.3)

The placental parenchymal tissue examined showed presence of syncytial knots. Fibrinoid deposits were found on villous surfaces. The fibrinoid necrosis, trophoblastic basement membrane thickening villous oedema and crowding of villi was in the normal range.

- Three vessels were seen in the cord with no thickening of vessel wall and thrombus was not seen.
- The membrane showed no signs of inflammation.
- In 50% placentas Grade I increased syncytial knots were seen and 15% placentas showed Grade Il increased knots, 25% placentas showed decrease in syncytial knots, 10% placentas showed the syncytial knots comparable to those of normal placenta
- Fibrinoid necrosis was increased in all placentas.
- The thinning of trophoblastic basement membrane was seen in 80% placentas and the basement membrane thickening was normal in 20% placentas.
- Villous stromal fibrosis was increased.
- No villous oedema was seen.
- Crowding of villi and fibrin deposition was seen to be increased in 50% cases and 50% showed normal distribution.
- Thrombi deposition was significantly seen to be increased in 85% placentas.
- Thinning of the vessel wall was seen in 70% placentas.
- Membrane inflammation was not seen.

Discussion

The present study deals with the morphohistological study of placentas grouped under two categories normal and hypertensive. The total 70 placentas were studied in the above two categories. The study revealed that the mean weight of placenta was found least in the hypertensive cases as shown in Table-I. The mean weight of the normal placenta in the present study was found as 503 gm in normal gestation

Table -2 Microscopic Findings of Normal Placenta

Total N = 50	Normal	Increased	Decreased
Placenta	No. of cases %	No. of cases %	No. of cases %
1.Syncytial knots	50(100%)	_	_
2.Fibrinoid necrosis	50(100%)	_	_
3.Trophoblastic basement membrane thickening	50(100%)	_	_
4.Villous stromal fibrosis	50(100%)	_	_
5.Villous oedema	50(100%)	_	_
6.Crowding of villi	50(100%)	_	_
7.Fibrin deposition	50(100%)	_	_
Cord			
1.No of vessels	50(100%)	_	_
2.Thrombi	_		
3.Thickening of vessel wall	50(100%)	_	_
Membrane			
1.Inflammation	_	_	_

Table-3: Microscopic Findings of Hypertensive Placenta

N = 20	Normal	Increased		Decreased	
Placenta	No.of cases(%)	No.of cases %	No.of cases %	No.of cases %	No.of cases%
1.Syncytial knots	2 (10%)	10 (50%)	3 (15%)	5(25%)	-
2.Fibrinoid necrosis	-	20(100%)	-	-	-
3.Trophoblastic basement membrane thickening	4(20%)	-	-	12(60%)	4(20%)
4.Villous stromal fibrosis	7(35%)	13(65%)	-	-	-
5.Villous oedema	20(100%)		-	-	-
6.Crowding of villi	10(50%)	10(50%)	-	-	-
7.Fibrin deposition	10(50%)	10(50%)	-	-	-
Cord					
1.No of vessels	20(100%)	-	-	-	-
2.Thrombi	3(15%)	11(55%)	6(30%)	-	-
3.Thickening of vessel wall	-	6(30%)	-	14(70%)	-
Membrane					
1.Inflammation	-	-	-	-	-

period whereas Greenhill (1955)[7] has described the placental weight as 500 gm which is near to our finding. Walker (1954)[8] described the weight of placenta as 480 gm in 40-41 weeks pregnancy. Wong (1966)[9] studied 60 placentas, 30 from normal and 30 hypertensive and observed that the average weight of normal placenta was found 463 gm and average weight of hypertensive placenta was 339 gm which clearly shows the downward trend as regards the weight of placenta from normal to hypertensive cases. Fox et al (1973)[5] described the placenta as the mirror of the perinatal period. Kalo et al, (1979)[10] Kher and Zawar (1981)[3], and Bhatia et al, (1981)[11] have reported reduced placental weight in cases of higher grades of hypertension, while placentae from cases of mild pre-eclampsia were reported to weigh within normal range. The study of Udainia et al (2001)[12] found the mean placental weight of 435.63 gm in mild hypertension and 371.43 gm in severe hypertension. Londhe et al (2011)[13] also observed that placental weights were lower in cases of pre-eclampsia than in controls. It endorses the finding of' present study which revealed the average weight of placenta in normal cases was 503 gm and in hypertensive cases was 410 gm.

Krishna M, et al (1979)[14] stated placental weight is significantly reduced in pregnancy hypertension and this reduction in weight was due to decrease in villi number.

Diameter of Placenta

The study revealed that there was decrease in the maximum diameter of placenta in hypertensive placentas as shown in Table–1. Patten. B.M (1953)[4], Scott (1960) [2] and Brews (1963)[15] stated the range of placental diameter from 15.20 cm. Greenhill (1955)[7] observed the range as 15-18 cm, whereas Potter et al (1940)[16], Stieve (1941)[17] & Dodd (1947)[18] observed the diameter as 16-20 cm. The findings of present study in normal were nearer to previous work performed. It also revealed that in normal cases it varied from 14-20 cm, average being 16 cms. In the hypertensive cases it varied from 10- 25 cm average diameter being 15.8 cm. and in the diabetic placenta it varied from 13-20 cm, average diameter being 16.4 cm.

Length of Umbilical Cord

The study revealed the mean cord length was least in case of hypertensive placentas shown in Table-3. Arey (1962)[19] stated the length of umbilical cord varied from 12.5-15.0 cm. The minimum length in his observation was nearer to that in the present study whereas maximum range has got much difference. Though he had observed the average length from 45-60 cm. Walker et al (1954)[8] mentioned the average length of umbilical cord is about 54cm which again falls in the range mentioned by Arey (1962)[19]. Though Brews(1963)[15] stated that the length of umbilical cord at term varies from 12.5-150 cm but he had mentioned that the average range from 40-50 cm. As regards the diameter of umbilical cord the present study revealed no significant variation in all the three categories under study villi number of vessels in the umbilical cord was three, its insertion on placenta was central in all

placentas studied irrespective of various categories. The length of umbilical cord in normal placenta ranged from 20-56 cm average being 40 cm. The umbilical cord length in hypertensive placenta varied from 26-44 cm, average being 33.3 cm. The umbilical cord length in diabetic placenta varied from 40-50 cm, average being 45 cm. Short cord that is less than 32 cm resulted in cord traction during the second stage of labour, causing delay in delivery and even cord rupture.

As regards the foetal and maternal surface, they were found to be normal in all placentas studied.

Microscopic Examination of Placenta

Yang IIX (1993)[6] observed that the villous immaturity, proliferation of small fetal vessels, syncytial knots increased in light microscopic examination. He had also mentioned that morphohistometric investigations showed that changes of placenta of gestational diabetes were significant.

The study revealed that on microscopic examination, the hypertensive placenta showed grade I increase in syncytial knots in 50% cases and grade II increase in 70% cases whereas 22% cases should decrease in syncytial knots as compared to 11% which were considered to the normal.

Fibrinoid necrosis was increased in all hypertensive placentas studied. Sala NA et al (1982)[21] also reported fibrinoid degeneration which was more frequent in venous regions. They suggested that it may be due to hypoxia. Mardi K et al (2003)[1] had observed significant increase in incidence of infarction, intervillous fibrin deposition. The hypertensive placentas also showed thinning of basement membrane in 78% cases and basement membrane showed normal feature in 22% cases Increase in villous stroma) fibrosis was seen in about 67% hypertensive placenta. There was no villous oedema in any of the hypertensive placenta, crowding of villi and fibrin deposition was increased in 50% and normal in rest 50%.

Thrombus was significantly increased in 85% hypertensive placenta. The vessel wall were thinned in 72% cases. Inflammation was not seen in membrane in any of the hypertensive placenta.

Reshetnikova et al (1995)[22] concluded that there was reduction rather then enlargement of placental villous tree in cases of maternal hypertension because placenta adapts through thinning of villous membrane so that diluting capacity was maintained at normal levels although Mirdi K et al (2003)[1] observed increase in basement membrane thickening placentas.

Conclusion

The following conclusions were drawn from the present study

1. The mean weight, diameter of placenta& length of umbilical cord were low in hypertensive mother when compared to normotensive mother.

2. On microscopic examination syncytial knot, fibrinoid necrosis, crowding of villi, thrombi & fibrin deposition of placenta were

increased in hypertensive placenta when compared to normotensive placenta.

3. Thinning of trophoblastic membrane & foetal vessel wall in hypertensive placenta.

The above mentioned Histo-morphological changes of placenta in hypertensive mothers leads to fetal distress syndrome due to hypoxia.

References

1. K Mardi et al. Histopathological evaluation of placentas in JUGR pregnancies . Indian J Pathol Microbiol. 2003; Oct; 46(4):551-4.
2. Scott, J.S, The placental septa 3. Obset & Gynec. Brit. Emp.(1960); 65: 774.777.
3. Kher AV and Zawar AP. Study of placental pathology in toxemia of pregnancy and its fetal complications, Indian Journal of Pathology and Microbiology 1981;24 :245.
4. Patten. B.M, Human Embroylogy. 2nd Ed. Marple Prcss. Co., York. PA.1953: Page:153-162.
5. Fox H (1997). Pathology of the Placenta. 2nd edition London, United Kingdom: WB Saunders Company Ltd.
6. Yang IX. Placental pathology in gcstational diabetes. Zhonghua FtJ Chan ke Za Zhi. 1993; 28 (12): 714-6
7. Greenhill J.P, Obstetrics eleventh Ed. (1955), W. Saunders Co.., Philadelphia and London
8. Walker E.H. discussion Cold Spr. Harb Syin Quant Biol, 1954 ;19:39.
9. Wong, Latour .J.P.A. Arm. Jour. Of Obst. & Gynec. 1966; 94 (7):942.
10. Kalo J, Levin S and Rosin N. Correlation between pathological findings of the placenta and clinical entities of the mother and fetus. Journal of Obstetrics and Gynaecology of India1979 ;29: 340.
11. Bhatia A, Sharma SD and Jalnawalla SF. A comparative study of placental pathology and fetal outcome. Indian Journal of Pathology and Microbiology1981; 24 :277.
12. Udainia A and Jain ML. Morphological study of placenta in pregnancy induced hypertension with its clinical relevance. Journal of Anatomical Society of India 2001;50(1): 24-27.
13. Londhe Pradeep S and Mane Abhay B . Morphometric study of placenta and its correlation in normal and hypertensive pregnancies. International Journal of Pharma and Biosciences2011; 4(2) :429-437.
14. Krishna M, Agarwal KN. Effect of pregnancy anaemia on cellular growth in the human placenta. Acta paediatr Scand. 1979 Nov; 68 (6): 899 —901.
15. Brews, M. (I 963), Holland & Brews Manual of Obstetrics 12th ed. 1:45 Clay A and Bourne A William Heinemann Medical book Ltd, London.
16. Potter, E.L., Adair, F.L. in: Fetal and Neonatal Death: A Survey of the Incidence, Etiology, and Anatomic Manifestations of the Conditions

Producing Death of the Fetus in Utero and the Infant in the Early Days of Life. The University of Chicago Press, Chicago; 1940.

17. Stieve, H; I ie entwick king and M nschlichcn Placenta Ztschr Mikrani. Forsch 1941;48:287
18. Dodds The area of chorionic villi in full term placenta Anat rec 1947;24: 287-294.
19. Arey, L.B. (1962) Developmental Anatomy 6th E.D. pp 137.
20. Brews, M. (I 963), Holland & Brews Manual of Obstetrics 12th ed. 1:45 Clay A and Bourne A William Heinemann Medical book Ltd, London.
21. Sala MA, et al. Regional variatioH in the frequency of fibrinoid degeneration in human term placenta. Z Geburtshilfe Perinatol. 1982 Apr- May; 186(2): 80-1.
22. Reshetnikova OS, Burton (U. Placental histomorphometry arid morphinetric diffusiiig capacity of the villous membranc in pregnancies complicated by maternal iron deficiency anaemia. AMJ Obstect. Gynecol. 1995 Sep; 173 (3PT 1): 724-7.

Corresponding author
Dr.Avneesh Chandra
Postgraduate
Department of anatomy, Rama Medical College, Hospital and Research Centre, Kanpur