

A Study on Morphology of Placenta in pregnancy Induced Hypertension in Wayanad, Kerala

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Abstract: Placenta is a discoid organ which is transient and is responsible for the proper growth and survival of the developing fetus. According to Park (2009) the hypertensive disorders are responsible for 5-8 % of all maternal deaths. A wide variation in placental size has been reported in Pregnancy induced hypertension (PIH) women. The study was done in 200 placentas, Out of the 200 placentas collected, 100 placentas were from uncomplicated full term deliveries and served as control group. Another 100 placentas were collected from PIH cases. The distribution of the weight of the placenta differs in normal and PIH cases. There is a reduction in the weight of the placenta in majority of PIH cases. The number of cotyledons decreased markedly in PIH cases and the attachment of umbilical cord is eccentric type in more number of PIH cases.

Keywords: Hypertensive, Placenta, Pregnancy, Pregnancy induced hypertension and Growth.

1. Introduction

Placenta is a discoid organ which is transient and is responsible for the proper growth and survival of the developing fetus [1]. It maintains the accurate record of the infant's prenatal experience [2]. In other words many prenatal facts can be provided by detail examination of the placenta. It forms the morphological record of anatomical condition, intrauterine events and intra partum events of gestation. According to Park (2009) the hypertensive disorders are responsible for 5-8 % of all maternal deaths. A wide variation in placental size has been reported in Pregnancy induced hypertension (PIH) women. Usually the placental size is described as smaller than normal but unduly large placentae have also been reported. The proper fetal growth is dependent on and determined by the weight of placenta [3, 4]. Abnormal placenta adversely affects the fetal outcome [5]. PIH is a common obstetrical condition in India, which results in increased risk of mortality and morbidity of the fetus [6]. Hypertension in pregnancy is associated with poor blood supply to the inter villous spaces of placenta [7]. The inadequate supply of blood to placenta leads to Placental Abruption, IUGR and Preterm Birth [8, 9, 10 and 11]. Such gross abnormality in the placenta will lead babies to reduced birth weight and is known to be more prone to acquire hypertension in later life [12].

2. Material and Methods

The study was done in the Department of Anatomy, DM-Wayanad Institute of Medical Sciences, Wayanad, Kerala and Srinivas institute of medical sciences, Mangalore. The specimen was obtained from the OBG Department in both the colleges. The study was done in 200 placentas, Out of the 200 placentas collected, 100 placentas were from uncomplicated full term deliveries and served as control group. Another 100 placentas were collected from PIH cases and served as study group. The age of the women varied from 20 to 35 years. The classification of the placenta was done by the Department of OBG and the patient personnel information was not provided.

The criteria adopted by the OBG Department for grouping of these cases were defined according to The International Society for the Study of Hypertension in Pregnancy Classification followed by the American College of Obstetrics and Gynecologists (ACOG, 1988). The alterations in blood pressure were observed on at least two different occasions, at least six hours apart.

The placentae were collected soon after delivery and kept in 10 percent formalin. Detailed gross examination of the placenta was done. The external features and any abnormalities in the placenta were noted. On the fetal surface the attachment of the umbilical cord was noted. On the maternal number of cotyledons was noted and the weight of the placenta was taken using standard weighing machine.

3. Results



Figure 1: placenta of study group showing less number of cotyledons and Retroplacental hemorrhage



Figure 2: placenta of control group case showing more number of cotyledons and no Retroplacental hemorrhage



Figure 3: centrally attached umbilical cord.



Figure 4: Eccentrically attached umbilical cord.

Table 1: Frequency of the distribution of the weight of placenta in Normal and PIH cases

Group	No of cases	Mean no. of cotyledons
Normal	100	16.6
PIH	100	12.32

Table 2: Frequency of number of cotyledons in Normal and PIH cases

Group	No of cases	Central	Eccentric
Normal	100	76	24
PIH	100	58	42

Table 3 Frequency of the attachment of the umbilical cord to the placenta in Normal and PIH cases. The distribution of the weight of the placenta differs in normal and PIH cases. There is a reduction in the weight of the placenta in majority of PIH cases. The number of cotyledons decreased markedly in PIH cases and the attachment of umbilical cord is eccentric type in more number of PIH cases.

4. Discussion

In the earlier studies by Nobis and Das, the pattern of cord insertion was central in 44.19%, eccentric in 42.17%, and battledore in 1.26% [13].

Our study is not in agreement with the study conducted by Nobis and Das. This might be a regional variation since the study was conducted in different populations.

The arrangement of intracotyledon vasculature is altered in hypertension resulting in low birth weight of the babies [14]. Heavy proteinuria increases the incidence of low birth weight babies in preeclampsia [15]. Also the reduction in the villous population will interfere with fetal nutrition and growth, leading to decrease in neonatal weight [16]. The hypertensive pregnancy leads to less number of cotyledons. Placental insufficiency is the cause of neonatal morbidity [17], which in turn might be the result of these observed alterations in placental weight.

The lean umbilical cords were observed in hypertensive pregnancy by ultra sonographic study [18]. But no significant changes have been observed in umbilical cord dimensions between the two groups in the study.

5. Conclusion

The study observed that, the morphological changes in the progress of pathology from gestational hypertension to preeclampsia, but there is no significant change in the dimensions of umbilical cord. Thus the study reveals that, when the degree of pathology progresses from gestational hypertension to preeclampsia, the insufficiency of placental circulation results, which in turn increases the fetal demands. The reduction in placental morphological parameters might be the reason for the reduction in baby growth in preeclampsia. Future scope lies in regional studies to know the problems occurring in the placenta of eclampsia and pre-eclampsia cases, which may ultimately result in finding the cause and bring down the fetal and maternal mortality.

Group	No of cases	200-300gms	301-400gms	401-500gms	501-600gms	601-700gms
Normal	100	2	7	28	48	15
PIH	100	25	36	21	16	2

References

- [1] Udainia A, Jain ML. Morphological study of placenta in pregnancy induced hypertension with its clinical relevance. *J Anat Soc India* 2001;50:24-7.
- [2] Benirshke K. The placenta: How to examine it and what you can learn. *Contemp Obstet Gynaecol* 1981;17:117-9.
- [3] Asgharnia M, Esmailpour N, Poorghorban M, Atrkar-Roshan Z. Placental weight and its association with maternal and neonatal characteristics. *Acta Medica Iranica*. 2008; 6(6):467-472.
- [4] Lo YF, Jeng MJ, Lee YS, Soong Wj, Hwang B. Placental weight and birth characteristics of healthy singleton new borns. *Acta Paediatr.Taiwan*. 2002 Jan-Feb; 43(1):21-25.
- [5] Eskild A, Vatten LJ. Do pregnancies with PE have small placentas? A population study of 317688 pregnancies with & without growth restriction in the offspring. *BJOG*. 2010; 117:1521-1526.
- [6] Jain L. Effect of pregnancy induced & chronic hypertension on pregnancy outcome. *J Perinatol*. 1997 Nov-Dec; 17(6):425-427.
- [7] Kaauaur K, Jouppila P, Kuikka J, Luotola H, Toivanen J, Rekonen A. Inter villous blood flow in normal & complicated late pregnancy measured by means of an inter villous 133XE method. *Acta obstetrica et Gynecologica Scandinavica*. 1980 Jan; 59(1): 7-10.
- [8] Naeye RL. Pregnancy hypertension, placental evidence of low uteroplacental blood flow & spontaneous premature delivery. *Human Pathology*. 1989; 20(5):441-444
- [9] Ananth CV, Peltier MR, Kinzler WL, Smulian JC, Vintzileos AM. Chronic hypertension & risk of placental abruption: Is the association modified by ischemic placental disease? *Am J Obstet Gynecol*. 2007 Sep; 197(3): 273.e 1-7.
- [10] Robert Resnik MD. Intra uterine growth restriction High- Risk pregnancy series: An expert view. *American college of obstetricians & Gynecologist*. 2002 Mar; 99(3): 490-496.
- [11] Tom KAB Eskes. Abruption placentae. A 'Classic' dedicated to Elizabeth Ramsey. *European Journal of Obstetrics & gynecology & reproductive biology*. 1997; 75:63-70.
- [12]. Johan Eriksson, Tom Forsen, Jaako Tuomilehto, Clive Osmond, David Barker. Fetal & Child hood growth and hypertension in adult life. *Hypertension*. 2000; 36: 790-794.
- [13] Nobis P, Das U. Placental morphology in hypertensive pregnancy. *J Obstet Gynecol* 1991;41:166-9.
- [14] Majumdar S, Dasguptha H, Bhattacharya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies. *J Anat Soc India* 2005;54:34-8.
- [15] Chakravorthy AP. Foetal and placental weight changes on normal pregnancy and pre-eclampsia. *J Obstet Gynaecol Br Commonw* 1967;74:247-53.
- [16] Fox H. The morphological basis of placental insufficiency. *J Obstet Gynaecol India* 1975;25:441-50.
- [17] Villar J, Carroli G, Wojdyla D. Preeclampsia, gestational hypertension & intrauterine growth restriction related or independent condition? *Am J Obstet Gynecol*. 2006; 194: 921-931.
- [18] Luigi Raio, Fabio Ghezzi, MD, Edoardo Di Naro, MD, Massimo Franchi, MD, Daniele Bolla MD, Henning Scneider MD. Altered sonographic umbilical cord morphometry in early onset preeclampsia. *Obstetrics & gynecology*. 2002 Aug; 100(2): 311-316. *IJBR* (2013) 04 (01).

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