

Histopathological Changes In Placentas Due To Pregnancy-Induced Hypertension And Gestational Diabetes Compared With Normal Term Placenta

Sawsan S. Hamzah¹, Ahmad Yar²

¹Department of Dentistry, Al- Farahidi University, Baghdad , Iraq.

²Sahiwal Medical College, Sahiwal, Pakistan

ABSTRACT

Hypertension is the most commonly known problem of restoration during pregnancy, with up to 10% of pregnancies confused. Gestational diabetes mellitus is described as the occurrence or for the first time in pregnancy of a change in glucose levels to varying degrees. There are medical problems when diabetics and hypertension worsen pregnancy and affect maternal health.

Objectives: Analysis of unexplained placenta changes in patients with concomitant hypertension and gestational diabetes caused by pregnancy.

Materials and Methods: This study included forty placentas, twenty were collected from uncomplicated normotensive pregnant ladies, and the rest (20 placentas) were collected from ladies with concomitant gestational diabetes and pregnancy-induced hypertension. Histological sections were prepared using routine haemotoxyline and eosin staining.

Results: The histomorphological study of placenta of patients with concomitant hypertension and diabetes mellitus showed a significant number of syncytial knots, stromal fibrosis, and the number of capillaries in terminal villi in Medium-sized diffusion areas of the vascular median cover.

Conclusion: Placenta examination is very important for the diagnosis of various pathological conditions, mechanisms are still far from well understood, but there is a common consensus that the pathological level depends on the type of diabetes and hypertension during pregnancy. This study provides an opinion of previous studies with a great association placental changes in patients with concomitant pregnancy-induced hypertension and diabetes. The clinical appearance and the magnitude of placental pathological changes were strongly associated. There is a wide range of microscopical changes noted as increased the numbers of the syncytial ganglia, fibrosis and the number of capillaries in the peripheral appendages

Keywords: Calcifications, Fibrinoid necrosis , infarction, Number of capillaries in terminal villi, Placental, Stromal fibrosis, Syncytial knots

Correspondence:

SAWSAN S. HAMZAH

Department OF Dentistry , Al- Farahidi University, Baghdad , Iraq.

*Corresponding author: Sawsan S. Hamzah email-address:

sawsan.sahib2020@gmail.com

INTRODUCTION

The fetus' survival and well-being depend on a primary organ, the placenta. The placenta is important for promoting pregnancy and promoting the normal development and improvement of children^[1]. The word placenta comes from the Latin word plakos, which means 'cake,' or Greek plakenta, which means 'stage chunk,' which refers to the stage of its human appearance^[2]. The placenta is at the interface of the maternal and fetal route with the necessary potential for pregnancy^[3]. It is the most vital and important organ of life in the mother's womb, which includes notifications from the mother and the fetus to coordinate the fetus's request with its needs for supplies sent from the mother^[4]. It is essentially implied for a trade of supplements amongst maternal and fetal dissemination to guarantee an ideal situation for fetal development and improvement^[5,6]. It really plays a central role in the creation of the fetus along these lines. The placenta villi are located within the placenta in the function of the main units, where the fetal blood is segregated from the mother's space in the space between the villi space by vascular layers at the top of the swollen blood vessels of the fetus^[7]. From the histological point of view, it is possible to refer to the term placenta by the presence of large numbers of villi and cellular nodes. These groups are the nuclei of the trophoblast created in groups leaving areas of the delicate cytoplasm without a median core^[8]. Examination of the placenta and umbilical line is imperative to recognize what had happened to hatchling in the gestational period^[9]. In present days,

hypertension is the most widely recognized restorative issue experienced amid pregnancy, representing up to 10% of pregnancies^[10,11]. According to the classification of the American College of Obstetricians and Gynæcologists (2019), High blood pressure disorders in pregnancy, a common term that includes chronic high blood pressure as well as pre-eclampsia, The most important causes of diseases and mortality are chronic hypertension with superimposed preeclamping and gestational hypertension, complications of up to 10 per cent of deliveries^[12-14]. The blood pressure related to pregnancy is determined based on the bases in which the first time high blood pressure was determined^[15].

Gestational Diabetes: Recently, the term pregnancy-related diabetes has been launched to include all cases of high blood sugar during pregnancy (GDM) which include; also pre-existing diabetes (PED). It also includes type 1 and 2 diabetes mellitus (T1DM) and (T2DM) before pregnancy^[11], GDM is identified as hyperglycemia and is recognized during first pregnancy^[16,17]. Gestational diabetes mellitus (GDM) is characterized by variable-degree glucose bigotry, first occurring during pregnancy^[18]. It is a vital problem, when diabetes confuses pregnancy and, also, hypertension, which affects maternal well-being, design, and placenta elements, may also endanger fetal regularity. The placenta is the extension between maternal-fetal exercises, considered a window through which maternal dysfunction and its effects on fetal prosperity can be understood^[19].

Histopathological Changes In Placentas Due To Pregnancy-Induced Hypertension And Gestational Diabetes Compared With Normal Term Placenta

MATERIALS AND METHODS

Samples

Forty cases were studied, divided as follows: twenty placentae from healthy mothers and twenty placentas from mothers with diabetes in addition to blood pressure. Samples were collected from the natural delivery room and the gynaecological operating room theatre of Babylon Maternity and Paediatric hospital.

Tissue Preparation

Twenty placentas were obtained from mothers suffering from concomitant pregnancy-induced hypertension and gestational diabetes. On the placenta surface, the location of the umbilical cord attachment is observed, and placenta surface samples are permitted through cross-wounds from the Centre of each placenta at a distance of 2 cm from the tissue and glued into 10 per cent of the formal saline. Tissues were treated in paraffin, and parts of 5 µm serial tissue were produced with rotator microtome assistance. Hematoxylin and eosin (H&E) stained the tissue portion for histopathological examinations. Ten random microscopic high-power fields (X400) were picked, the number of villi was Counting and studying in each area for the following criteria:

Villous Lesions:

1. Syncytial knots > 30% in 10 high power fields (HPF).
2. Fibrinoid necrosis > 5% in 10 High power fields.
3. Placental infarction > 5% in 10 High power fields.

Intervillous Space

1. Stromal fibrosis > 1% mild in field
>2 % moderate in field
> 3% sever in field
2. Presence of calcification.
3. Mean Number of capillaries in terminal villi in Ten High power fields (HPF).

Statistical Analysis

Student T-tests have been used to determine the statistical results. Hypotheses were tested at the significance level $\alpha = 0.05$, that is, the differences between the samples were considered significant at $p \leq 0.05$.

RESULT

A significant number of syncytial nodes, The histological studies of placentae in medium coat areas of the medium blood vessels in the combined diabetic and hypertensive population, have shown stromal fibrosis and capillary numbers in terminal villi. In contrast, control populations have standard histological characteristics, as shown in table 1.

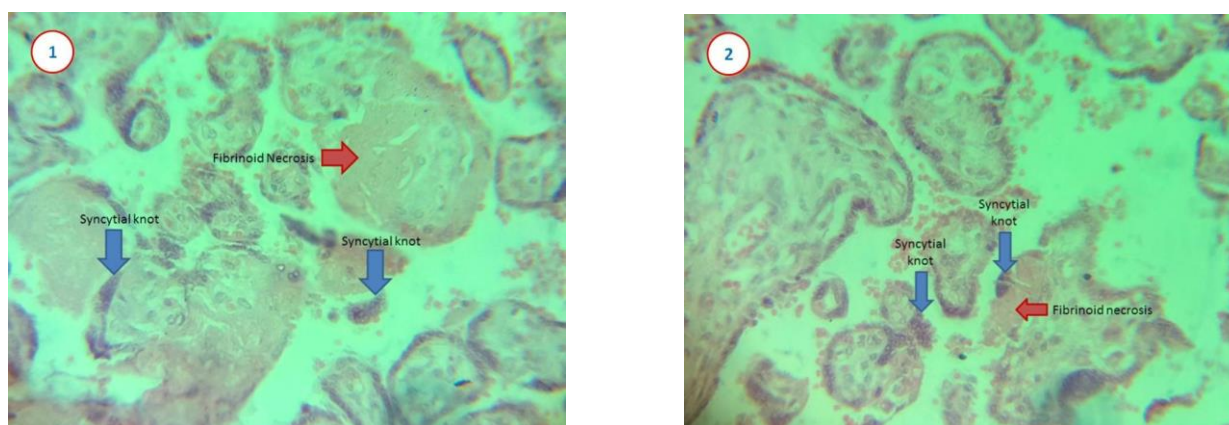
Table 1. Statistical study of the histology of placental villi review.

No.	Histological lesion	Control Mean± SD	Disease Group Mean± SD	P value
1	Syncytial knots	22.5+ 3.1	26.25+ 5.1	0.013
2	Fibrinoid necrosis	2.8+ 1.6	4.1+ 2.9	0.093
3	Placental infarction	0.1+0.3	0.2+ 0.6	0.428
4	Stromal fibrosis	0.4+ 0.5	2+ 0.7	< 0.000
5	Calcifications	0.2+ 0.61	0.2+ 0.69	1.000
6	Number of capillaries in terminal villi	2.3± 1.22	4.9± 1.54	< 0.000

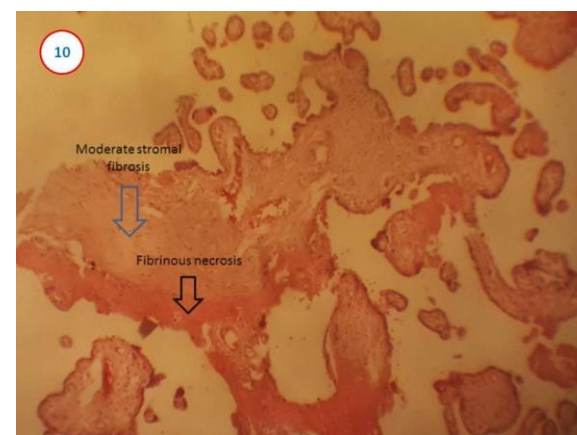
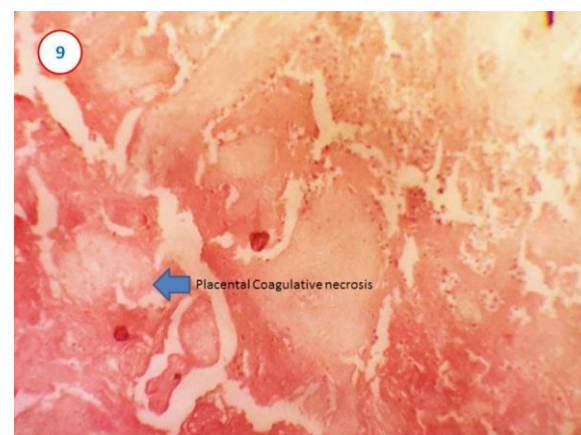
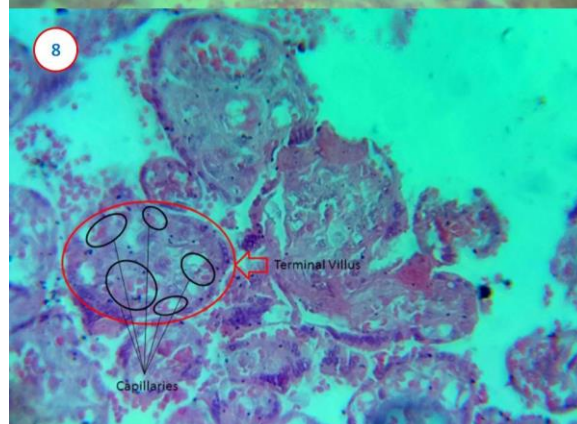
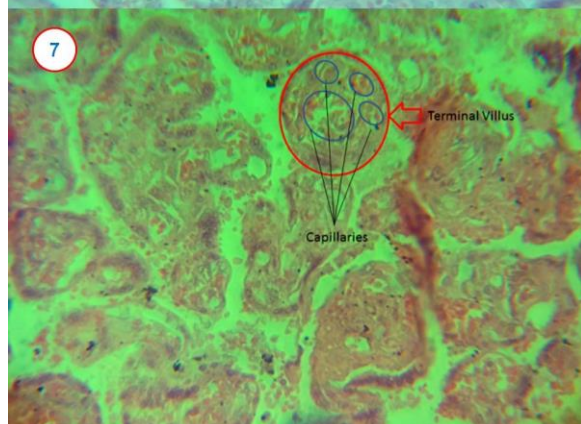
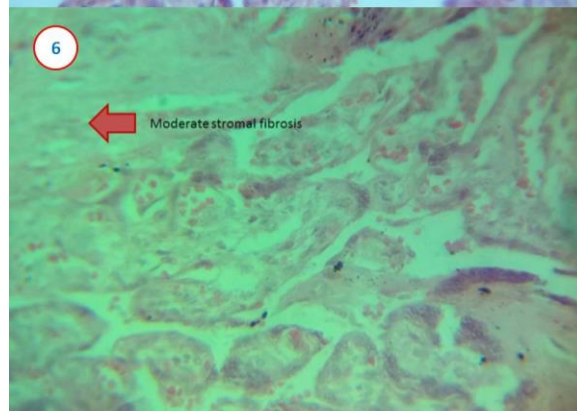
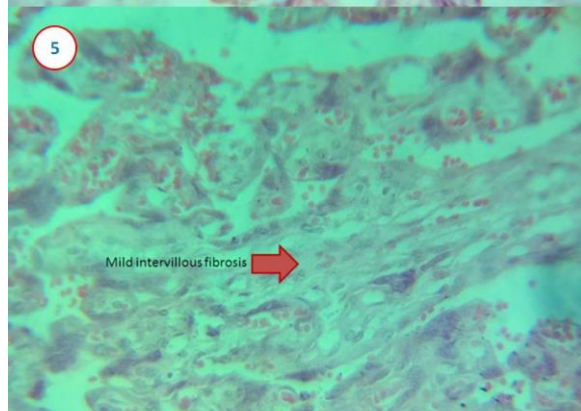
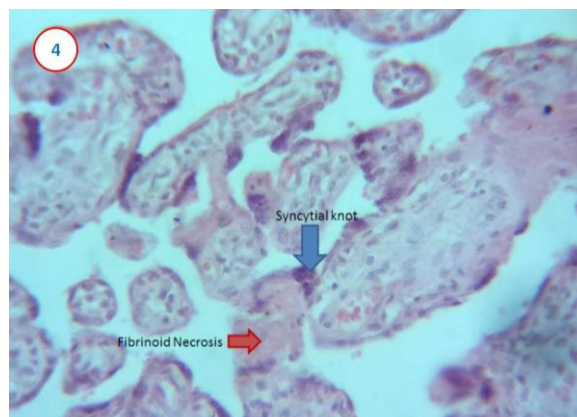
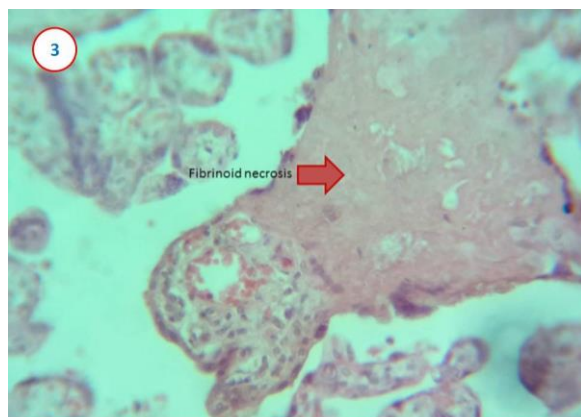
Significant $p \leq 0.05$

Comparison of placenta morphological characteristics in many classes

Figure [1] Histological changes in concomitant pregnancy - Induced Hypertension and gestational diabetics



Histopathological Changes In Placentas Due To Pregnancy-Induced Hypertension And Gestational Diabetes Compared With Normal Term Placenta



Discussion

Placental pathology was of almost zero scientific esteem if there was no clinical evidence for examination^[20]. Be

Histopathological Changes In Placentas Due To Pregnancy-Induced Hypertension And Gestational Diabetes Compared With Normal Term Placenta

that as it may, Placental pathology in the obstetric suite now plays an important role, and there are sections that are substantially represented in medico-legal cases^[21-24]. The placental analysis of all live births is not supported by adequate evidence^[25]. Known benefits of placenta screening are clarifying pathological features, and improved management of subsequent pregnancy by diagnosing a pathological condition which will be in danger of recurrence, or is often prevented or treated.

The present research was believed to study different pathological changes in hypertensive diabetic placenta relative to placentas from non-hypertensive diabetic pregnancy show in Figure[1].

Syncytial knots: A syncytial tie, fundamentally characterized under the light magnifying lens, is characterized as the critical grouping of syncytial cores on the terminal villi surface^[26,27]. In this research, histological results showed a substantial increase ($p \leq 0.05$) in the development of syncytial nodes in placental villi show in Figure [1]. A noteworthy increment in syncytial hitch development in placental villi demonstrates the unsettling influence in the hormonal elements, which may most likely prompt adjusted morphometry of placenta bringing about Pregnancy Indicate Hypertensive in the mother^[28,29]. The relationship of expanded syncytial ties in patients with pre-eclampsia is notable^[30-32]. This finding is similar to another research, which indicates a large ($p \leq 0.05$) number of syncytial nodes in the hypertensive group relative to the control group^[33-35]. In addition, hypertensive diabetic placentas have more syncytial knots, compared to placentas from non-hypertensive diabetic pregnancies^[36].

Stromal villus fibrosis: It is particularly basic in preterm placentas with intense rising diseases; potentially in light of the fact that the Premature villus looser stroma encourages amassing of extravascular liquid. The hidden reason for villous fibrosis is as yet indistinct. One hypothesis holds that collagen generation might be fortified by the expanded halfway weight of oxygen intervillous. Because Oxygen diffusion from the mother's space to the stroma, it is in the face of insufficient absorption of capillary embryos, due to the weak connection of oxygen to the excess tree, which may stimulate collagen synthesis as a result of increased oxygen content in the stroma^[37]. But the 2010 Fermat study^[38] indicated that placenta infections through the maternal bloodstream increased absolute diabetes. He suggested that such blood infections are typically related to villous stroma chronic inflammation and subsequent fibrosis. In this study, there was a significant increase ($p < 0.000$) in stromal villus fibrosis attributed to Poor oxygenation of the excess tree, may stimulate collagen synthesis and increase oxygen in the stroma in mother complaining of concomitant pregnancy-induced hypertensive and gestational diabetics.

Another report observed significant when ($p < 0.05$) In the hypertensive group, stromal fibrosis as in the control group^[33]. Another study also noted placenta tissue and its significant association with diseases. It is observed in diabetes patients compared to patients increased with blood pressure and less in ordinary patients^[39]. Diabetes is also associated with stromal fibrosis^[40,41].

Placental infarction is the production of chemical villi necrosis locally located areas that usually cause maternal perfusion in the intervillous space. In this study, a difference between groups of mothers was observed;

mothers having pregnancy hypertension and diabetes Compared to the category of controls. Another analysis has been found out a more common relationship in the placenta compared with the hypertensive mothers and or those having pre-eclampsia

Calcifications: The risk of calcification increases with an increase in the gestational period in the basal plate, which parenchyma, which typically occurs in the late and later placenta. Calcification can occur with or without other placental pathologies^[43]. The clinical meaning, if any, is generally not clear. No major variations between the two groups were found in this sample.

Fibrinoid necrosis: Fibrinoid degradation consists of the deposition of fibrinoid material, which is initially external to syncytiotrophoblast basement membranes^[44] as the foundation for bosselations and thus laminated sub-choral plaques, which change so much from one placenta to another^[45].

In this study, No big discrepancies between the two classes were observed. But according to Shams et al. (2012) Fibrinoid necrosis and placental hyalinization were significantly higher in mothers with diabetes and hypertension under light microscopy^[6].

Terminal Villi Number of Capillaries: In view of some assumptions and studies, it is clear the terminal villi growth is affected by the modification of the longitudinal growth boost the middle villi with that of their capillary loops. Between the two classes, there was a big difference. Another examination demonstrates the comparative outcome in the expanded number of villous vessels was frequently watched in diabetic mothers compared with the placenta control groups^[46].

Acknowledgments : For all patient that help me to complete this research

Ethics approval and consent to participate

The analysis is was conducted in compliance with the Declaration of Helsinki of 1975. by the Ethics Committee of Babylon maternity and Paediatric hospital based on the grounds that this form of study is human subject research, consent was obtained from the participants of the study .

Conflict of interest statement : I announce that I have no interest conflicts. No financial assistance has been received for this analysis.

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. Huppertz B, Kingdom J.C.P. The placenta and Foetal membranes In :Edmond DK editor. Dew hurts Text book of Gynaecology and Obstetrics. 7th ed. London: Blackel the publisher; 2007. p.19-25.
3. Desoye G, Hauguel-de Mouzon S. The human placenta in gestational diabetes mellitus. The insulin and cytokine network. Diabetes Care. 2007;30 Suppl 2:S120-S126.
4. Desoye G, Shafrir E The human placenta in diabetic pregnancy. Diabetes 1996; Rev 4: 70-89.
5. Chang KT. Pathological examination of the placenta: raison d'etre, clinical relevance and medico legal utility. Singapore Med J 2009; 50:1123-33.
6. Shams F, Rafique M, Samoo NA, Irfan R. Fibrinoid Necrosis and Hyalinization Observed in Normal Diabetic and Hypertensive Placentae. Journal of the

Histopathological Changes In Placentas Due To Pregnancy-Induced Hypertension And Gestational Diabetes Compared With Normal Term Placenta

- College of Physicians and Surgeons, Pakistan 2012; Vol. 22 (12):769-772.
7. Calderon IM .Morphometric study of placental villi and vessels in women with mild hyperglycemia or gestational or overt diabetes. *Diabetes Res Clin Pract* 2007;78: 65-71.
 8. Jones CJ, Fox H .Syncytial knots and intervillous bridges in the human placenta: an ultrastructural study. *J Anat* 1977;124: 275-286.
 9. Burke CJ, TannenbergaE. Prenatal brain damage and Placental Infarction –An autopsy study. *Dev med child neurol.* 1995;37:555.
 10. –James M.Roert , MD,Chair; Phyllis A. The Task Force on Hypertension in Pregnancy. *Hypertension in Pregnancy.* ACOG 2013;vol 12:no 15.
 11. ACOG practice Bulletin.Diagnosis and management of Preeclampsia and Eclampsia *obstetgynaecol* 2002;99:159-167. doi: 10.1016/s0029-7844(01)01747-1.
 12. Bateman BT, Shaw KM, Kuklina EV, et al. Hypertension in women of reproductive age in the United States: NHANES 1999–2008. *PLoS ONE* 2012; 7(4): e36171. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
 13. American College of Obstetricians and Gynecologists; Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol* 2013; 122: 1122–1131. [[PubMed](#)] [[Google Scholar](#)]
 14. ACOG practice bulletin no. 202: gestational hypertension and preeclampsia. *Obstet Gynecol* 2019; 133: e1–e25. [[PubMed](#)] [[Google Scholar](#)]
 15. Butalia S, Audibert F, Cote AM, et al. Hypertension Canada's 2018 guidelines for the management of hypertension in pregnancy. *Can J Cardiol* 2018; 34(5): 526–531. [[PubMed](#)] [[Google Scholar](#)]
 16. Sacks DA, Metzger BE. Classification of diabetes in pregnancy: time to reassess the alphabet. *Obstet Gynecol* 2013; 121: 345-348 [PMID: 23344285 DOI: 10.1097/AOG.0b013e31827f09b5]
 17. Chamberlain C, McNamara B, Williams ED, Yore D, Oldenburg B, Oats J, Eades S. Diabetes in pregnancy among indigenous women in Australia, Canada, New Zealand and the United States. *Diabetes Metab Res Rev* 2013; 29: 241-256 [PMID: 23315909 DOI: 10.1002/dmrr.2389].
 18. Stuebe AM, Mantzoros C, Kleinman K, et al. Gestational glucose tolerance and maternal metabolic profile at 3 years postpartum. *Obstet Gynecol.* 2011 Nov. 118(5):1065-73.
 19. Rahman H, Khalil M, Ferdousi R, Uddin M, Chowdhury MM, Sultana SZ, Mannan S. Micro vascular changes in the placenta of Bangladeshi overt diabetic mothers and hypertensive diabetic mothers. *Journal of Bangladesh Society of Physiologist* 2006; 1: 27-34.
 20. Van Der Veen, F. & Fox. H. The human placenta in idiopathic intrauterine growth retardation: a light and electron microscopic study. *Placenta* 1983; 4, 65-78.
 21. Fox. H. (197%) The placenta in abnormalities and disorders of the fetus. In *The Pathology of the Placenta* W. B Saunders, London, p. 251.
 22. Fox H (1997). *Pathology of the Placenta.* 2nd edition London, United Kingdom: WB Saunders Company Ltd.
 23. Wigglesworth JS (). The Langhans layer in late pregnancy. A histological study of normal and abnormal cases. *Journal of Obstetrics and Gynaecology of British Commonwealth* 1962; 69 355.
 24. August P, Lindheimer M .D *Pathophysiology of Preeclampsia.* Hypertension 1995; Raven Press: New York. 2407-26.
 25. IfraSaeed, IramIqbal, RehmanSarfraz, Khadija Qamar, Shadab Ahmed Butt, SaffiaShaukat. Eclamptic Mothers with Reference to Vasculosyncytial Membrane Thickness and Syncytial Knot Formation *Journal of Rawalpindi Medical College (JPMC);*2012;16(1):51-54.
 26. Rogers BB, Momirova V, Dizon-Townson D, Wenstrom K, Samuels P, Sibai B, Spong C, Caritis SN, Sorokin Y, Miodovnik M, O'Sullivan MJ, Conway D, Wapner RJ. Avascular villi, increased syncytial knots, and hypervascular villi are associated with pregnancies complicated by factor V Leiden mutation. *Pediatr Dev Pathol.* 2010;13:341-7.
 27. Kristina Loukeris , Raanan Sela ,and Rebecca N. Baergen. Syncytial Knots as a Reflection of Placental Maturity: Reference Values for 20 to 40 Weeks' Gestational Age, Pediatric and Developmental Pathology 2010;13, 305–309.
 28. Teasdale F. Gestational changes in functional structure of the human placenta in relation to foetal growth. *Am JObstet.* 1980;137:560–2.
 29. *Ratnamala Siddheshware, Sunil S. Patil, Pradip W. Sambarey.* Clinical correlation with pathology of placenta in medical disorders of pregnancy and its comparison in normal pregnancy. *IJRCOG*2017; VOL6, NO.1.
 30. Cantle SJ, Kaufmann P, Luckhardt M, Schweikhart G. Interpretation of syncytial sprouts and bridges in the human placenta. *Placenta* 1987;8:221–234.
 31. Heazell AEP, Moll SJ, Jones CJP, Baker PN, Crocker IP. Formation of syncytial knots is increased by hyperoxia, hypoxia and reactive oxygen species. *Placenta* 2007;21:S33–S40.
 32. Deepalaxmi Salmani, Suja Purushothaman, Saligrama Chikkannasetty Somashekara, Ekambaram Gnanagurudasan, Kampli Sumangaladevi, Recapu Harikishan, and Muthinpala Venkateshwarareddy. Study of structural changes in placenta in pregnancy-induced hypertension. *J Nat Sci Biol Med.* 2014 Jul-Dec; 5(2): 352–355.
 33. Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A, A Study of Placenta In Normal and Hypertensive Pregnancies, *J.Anat.Soc. India* 2005; 54 (2) 1-9.
 34. Rath G, Garg K, Sood M. Insertion of umbilical cord to the placenta in hypertensive mothers. *J Anat Soc India,* 2000; 49 (2): 149-154.
 35. Udainia A, Bhagwat SS, Mehta CD. Relation between placental surface area, infarction and foetal distress in pregnancy induced hypertension with its clinical relevance. *J Anat Soc Ind* 2004, 53; 1: 27-30.
 36. Gian Carlo Di Renzo, Lois G.Jovanovic , Moshe Hod Oded . *Textbook of Diabetes and pregnancy* 2016, 3rd ed. Page. 94..
 37. Debra S, Heller, Vijay V joshi . *Handbook of placental pathology*2005; 85-90.

Histopathological Changes In Placentas Due To Pregnancy-Induced Hypertension And Gestational Diabetes Compared With Normal Term Placenta

38. Verma R . Cellular changes in the placenta in pregnancies complicated with diabetes. *Inter J Morohol* 2010; 28: 259-264.
39. Farrah Shams and Nawaz Samoo, Amount of Calcification and Fibrosis in Placentas: A Comparative Study from Hypertensive, Diabetic and Normal Pregnancies, *Journal of the Dow University of Health Sciences Karachi* 2015, Vol. 9 (1): 1-2.
40. Debras. , Ona Marie Faye- Petersein , Vjay V. Joshi. *Hand book of placenta pathology* .2005, 226.
41. Naeye, R., Maisels, M.J., Lorenz, R.P. and Botti, J.J.: The clinical significance of placental villous oedema. *Pediatrics* 71:588-594, 1983.
42. PubMedGoogle Scholar Pathak S, Hook E, Hackett G, Murdoch E, Sebire NJ, Jessop F, Lees C. Cord coiling, umbilical cord insertion and placental shape in an unselected cohort delivering at term: relationship with common obstetric outcomes. *Placenta*. 2010 Nov;31(11):963-8. doi: .1016/j.placenta.2010.08.004.
43. Kraus FT *et al.* *Atlas of Nontumor Pathology – Placental Pathology*. eds. American Registry of Pathology, Washington DC, 2004.
44. Joshi Vijay V. *Handbook of Placental pathology*. 1st Edition. Igaku-Shoin New York and Tokyo; 1994:1-10.
45. Peter Kaufmann , Kurt Benirschke , *pathology of human placenta* , 2nd ed. Springer Science and Business Media . 2013, 310.
46. K H Jácomo, W L Benedetti, M A Sala, H Alvarez. *Pathology of the trophoblast and fetal vessels of the placenta in maternal diabetes mellitus*, *Acta Diabetol Lat.* Sep-Dec 1976;13(5-6):216-35. doi: 10.1007/BF02581119.