

Quantitative Correlation between Abruptio Placentae and Normal Term Placental Morphology

¹Dr. Nusra Rahman, MBBS, MD Anatomy, Assistant Professor, Department of Anatomy, J. N. Medical College, A.M.U., Aligarh.

²Dr. M Tariq Zaidi, MBBS, MS Anatomy, MS Surgery, Professor, Department of Anatomy, J. N. Medical College, AMU, Aligarh.

³Dr. Shaista M. Vasenwala, MBBS, MD Pathology, Professor, Department of Pathology, J. N. Medical College, AMU, Aligarh.

⁴Dr. Nafis A. Farooqui, MBBS, MS Anatomy, Professor, Department of Anatomy, J. N. Medical College, A.M.U., Aligarh.

Corresponding Author: Dr. Nusra Rahman, Assistant Professor, Department of Anatomy, J. N. Medical College, A.M.U., Aligarh, 202002, UP, India.

How to citation this article: Dr. Nusra Rahman, Dr. M Tariq Zaidi, Dr. Shaista M. Vasenwala, Dr. Nafis A. Farooqui, “Quantitative Correlation between Abruptio Placentae and Normal Term Placental Morphology”, IJMACR- December - 2025, Volume – 8, Issue - 6, P. No. 98 – 104.

Open Access Article: © 2025 Dr. Nusra Rahman, et al. This is an open access journal and article distributed under the terms of the creative common's attribution license (<http://creativecommons.org/licenses/by/4.0>). Which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Placental abruption is an obstetric complication where the placental lining has separated from uterus, clinically presenting as vaginal bleeding in the latter half of pregnancy. In spite of its high incidence, there is lack of study on the anatomical basis of abruption. The present study was aimed at the quantitative analysis of the gross and histological features of the abruptio placenta and term human placenta. Forty five placentae comprising 30 from abruptio and 15 from normal patients were collected as study and control groups respectively. The placentae were fixed with 10% formalin. Gross morphological variables (placental weight, thickness,

density) and histological features (cytotrophoblastic proliferation, syncytial knots, vasculosyncytial membrane, fibrinoid necrosis, basement membrane thickness, stromal fibrosis and infarction) were recorded and compared for both the groups. All statistical data were analyzed by using student's t-test. Gross morphological variables showed no statistically significant relationship. For each histological parameter 200 villi were counted in each section of the two quadrants. We found statistically significant increase in cytotrophoblastic cell proliferation, syncytial knots, fibrinoid necrosis, basement membrane thickness, stromal fibrosis and infarction in abruption cases than in

term placenta. Vasculosyncytial membrane was significantly reduced in abruptio cases than in term placenta. All these histological findings correlate well with the uteroplacental ischemia in abruptio placenta.

Keywords: Placenta, abruptio, cytotrophoblastic proliferation, syncytial knots, vasculosyncytial membrane, fibrinoid necrosis.

Introduction

Placental abruptio is an obstetric complication where the placental lining has separated from uterus, clinically presenting as vaginal bleeding in the second half of pregnancy.¹ Complications during pregnancy as abruptio placenta, gestational diabetes or hypertension are reflected in the placenta in a considerable way, both macroscopically and microscopically.² As a consequence the placenta has become a focus of increasing importance in this era of advanced obstetric management. In human beings, abruptio refers to abnormal separation after 20 weeks of gestation and prior to delivery. It complicates about 1% of pregnancies,³ with foetal mortality rate of 20 to 40% depending upon degree of separation.⁴ Risk factors for abruptio include prior abruptio, smoking, trauma, cocaine use, multifoetal gestation, hypertension, pre-eclampsia, thrombophilias, advanced maternal age, premature rupture of the membranes, intrauterine infections and hydramnios.⁵⁻¹³ The precise pathophysiology that leads to placental abruptio is unknown. Placenta shows histopathological changes in different disease entities. These changes are cytotrophoblastic proliferation, syncytial knots, infarction, fibrinoid necrosis, calcification, basement membrane thickening and vascular changes in spiral arteries.

Placental abruptio is one of the leading causes of perinatal death. In the patients, physical and mental trauma due to the loss of the foetus after nurturing it for 20 weeks causes distress. In spite of the high incidence of abruptio placenta there is a lack of study on the anatomical basis of abruptio. The present study was aimed at the quantitative analysis of the gross and histological features of the abruptio placenta and term human placenta.

Material and Methods

Patients were selected from outpatient and emergency department of Obstetrics and Gynaecology of our hospital. Forty five cases comprising of 30 cases of abruptio placenta and 15 cases of normal pregnancy were selected as study and control groups. After taking history of present illness, past illness and gynecological and obstetrics events, medical examination was performed and placenta with cord were collected for study.

Sample Collection and Processing of placenta

Following delivery, placenta were wiped to remove any clotted blood, umbilical cord and membranes was removed and then placenta was weighed. Gross morphological variables including placental weight, thickness and density was recorded for both the groups. The placenta were washed and fixed in 10% formalin solution after making longitudinal cuts to make sure that formalin penetrated well. Each placenta was divided but not cut into four quadrants and sample was collected from two randomly selected quadrants. Site of selection was from the centre of the maternal side of each quadrant. Tissue were then processed and stained with Haematoxylin and Eosin and Van gieson stains.

Histological study: For each parameter 200 villi were counted in each section of the two quadrants. Following histological features were recorded and studied.

1. Cytotrophoblastic proliferation
2. Syncytial knots
3. Vasculosyncytial membrane
4. Fibrinoid necrosis
5. Basement membrane thickness
6. Stromal fibrosis and
7. Infarction

All statistical data were analyzed by using student's t-test.

Observations and Results

Comparison of gross features of abruptio placenta and full term placenta

As illustrated in Table – 1, the mean placental weight of abruptio placentae and term placentas were 472 grams and 484 grams respectively. The mean thickness in abruptio placenta was observed as 2.038 cms while the same in full term placenta was 2.055 cms. A mean of 0.956 kgm^{-3} density was observed in abruptio placenta whereas 0.953 kgm^{-3} density was observed in full term placenta.

Comparison of histological features of abruptio placenta and full term placenta

Table – 2 showed that the mean percentage of cytotrophoblastic cells (Figure -2) was increased from 6.50 in term group to 16.53 in abruptio placentae group. The mean syncytial knot count (Figure - 1 & 3) in abruptio placenta and term groups was 41.80 and 6.10 respectively. The mean vasculosyncytial membrane (Figure - 1) in abruptio placenta was observed as 2.87 while there was an increase in mean vasculosyncytial membrane of 10.5 in full term placenta. An average of 1.6 fibrinoid necrosis (Figure - 4) was observed in

placentas of term group whereas 6.0 fibrinoid necrosis was observed in placentas from study group. The mean basement membrane thickness (Figure - 3) in study group was observed as 7.17 while there was a decrease in mean basement membrane thickness of 1.90 in full term placenta. An average of 1.60 stromal fibrosis (Figure - 5) was observed in placentas of term group whereas 11.10 stromal fibrosis was observed in placentas from study group. The average infarction in term placenta was observed as 1.77 while there was an increase in infarction of about 5.0 in abruptio placentae patients.

Discussion

The Present study was carried out at a tertiary care centre, which mainly receives referred patients from peripheral hospitals. Histomorphometric analysis of placentae of both the groups was done. The findings of present study are discussed here.

Table - 1 compares the placental weight, thickness and density of the term and abruptio placentae. However, the P value < 0.01 indicated insignificant relationship between placental weight, thickness and density. Even after far-reaching literature search, we have not found any study observing relationship between placental weight, thickness and density as a risk factor for placental abruption.

Our study showed a statistically significant increase in cytotrophoblastic cells proliferation, thus differentiating the abruptio from normal term pregnancy. Our histological findings are consistent with Fox^{15,16} and Wigglesworth.¹⁷ According to Fox¹⁵ the degree of cytotrophoblastic hyperplasia is related to the extent of the syncytial damage thus it serves as a rough quantitative index of the severity of the ischemia to which the villi have been subjected. Current study

showed a significant increase in syncytial knots in cases of abruption in comparison to normal term pregnancy. Kaminsky et. al.¹⁴ found about 80% increased syncytial knots formation in pre-eclamptic toxæmia which was consistent with our finding in placental abruption. Gensen¹⁸ reported that excessive syncytial knot formation was in response to overall reduction of foetal perfusion. According to Fox¹⁶, excess syncytial knot formation is a good index of the degree of reduction in villous perfusion. Our study showed a significant decrease in vasculosyncytial membrane in study group in comparison to control group. Tewari et. al.¹⁹ in their study also reported decreased (4%) vasculosyncytial membrane in cases of pre-eclamptic toxæmia which was consistent with our finding in placental abruption. Our findings are also consistent with the findings of Fox¹⁶ who found deficiency of vasculosyncytial membrane in mature placenta, associated with a high incidence of foetal hypoxia. In our study an increase in average of fibrinoid necrosis was observed in abruption, which was consistent with Majumdar et. al.,²⁰ observations in hypertensive patients. The above finding of increase in fibrinoid necrosis is consistent with decreased utero-placental blood flow. These abnormalities may predispose to ischemia and rupture of involved vessels,

thus causing placental abruption.²¹ The mean thickness of basement membrane was found to be significantly increased in abruptio placentæ. Our finding of increased basement membrane thickness was in accordance with the study of Sodhi et. al.,²² where the author demonstrated a significant thickened basement membrane in hypertensive patients. Present study showed a significant increase in stromal fibrosis in abruptio placentæ. Similar findings were reported by Majumdar et. al.²⁰ in the hypertensive patients. Sodhi et al.²² also demonstrated a similar change. In our study we noted a statistically significant increase in infarction rate in abruptio placentæ group. This finding is consistent with decreased utero-placental blood flow. Furthermore, Brosens and Renaer et. al.²³ also reported that increased placental infarction occurs due to hypoxia.

To conclude our histological findings, correlate well with the uterop-lacental ischemia in abruptio placentæ. Outcome of pregnancy can be improved with effective management and directing therapies to improve utero-placental perfusion. Furthermore, studying these variables can help to predict the risk of recurrence in subsequent pregnancies with the management strategies directed to prevent and treat them efficiently.

Table 1: Gross morphological features of placentæ in study (n=30) and control groups (n=15)

Gross Features	Abruptio Placenta (Mean \pm SD)	Term Placenta (Mean \pm SD)	Significant	P < 0.01
Placental wt. (grams)	472 \pm 29.21	484 \pm 19.55	Insignificant	
Thickness (cms)	2.038 \pm 0.0544	2.055 \pm 0.0562	Insignificant	
Density	0.956 \pm 0.014	0.953 \pm 0.0014	Insignificant	

Table 2: Histological features of placentae in study (n=30) and control groups (n=15)

Histological Features	Abruption placentae (Mean \pm SD)	Term Placenta (Mean \pm SD)	Significant P<0.01
Cytotrophoblastic cells proliferation	16.53 \pm 7.58	6.50 \pm 4.38	Significant P<0.01
Syncytial Knots	41.80 \pm 20.81	6.1 \pm 5.76	Significant P<0.001
Vasculosyncytial membranes	2.87 \pm 3.71	10.5 \pm 3.72	Significant P<0.01
Fibrinoid necrosis	6.0 \pm 4.17	1.6 \pm 0.84	Significant P<0.001
Basement membrane thickness	7.17 \pm 5.35	1.90 \pm 0.88	Significant P<0.01
Stromal fibrosis	11.10 \pm 8.66	1.6 \pm 0.7	Significant P<0.001
Infarction	5.0 \pm 4.38	1.6 \pm 0.7	Significant P<0.02

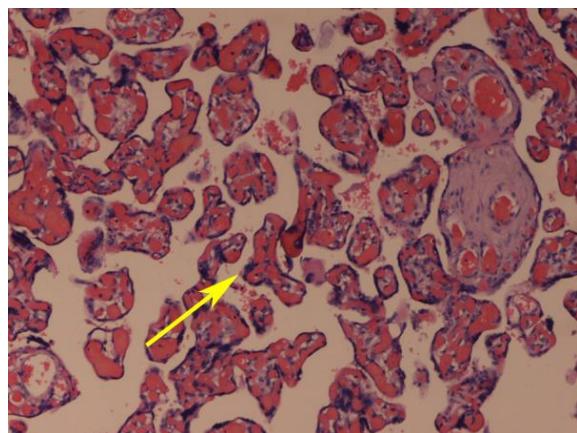
Legend Figures

Figure 1: Photomicrograph of placenta of control group showing mature small chorionic villi with syncytial knots – 20%, vasculosyncytial membrane present (H & E stain, 125X).

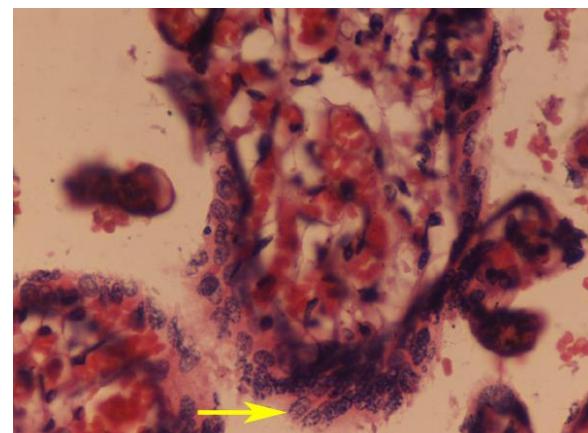


Figure 2: Photomicrograph showing polar cytotrophoblastic proliferation in chorionic villus (H & E stain, 500X).

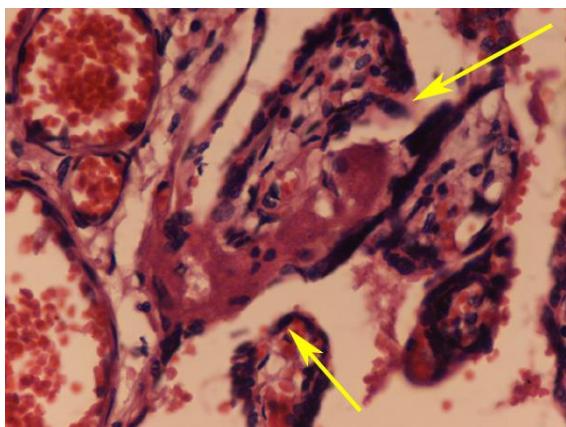


Figure 3: Photomicrograph showing chorionic villi with thickened basement membrane and increased syncytial knots 25% (H & E stain, 500X).

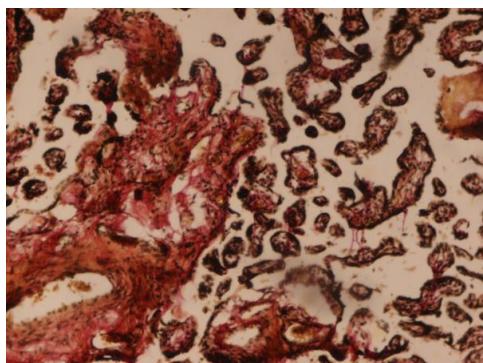


Figure 6: Photomicrograph showing (Retroplacental hemorrhage with hypertension) villi shows fibrosis & intervillus edema (V. G. stain, 125X).

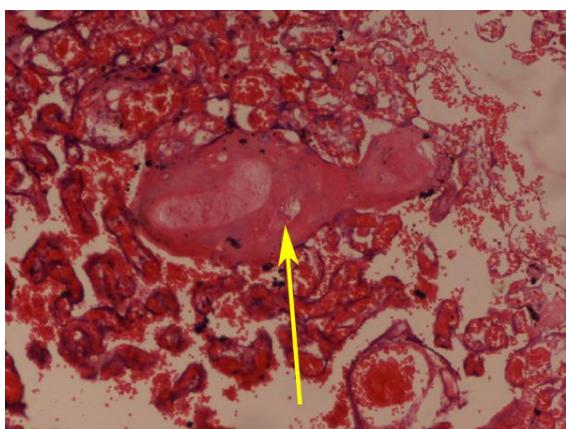


Figure 4: Photomicrograph showing fibrinoid necrosis of chorionic villus as homogenous eosinophilic material (H & E stain 125X).

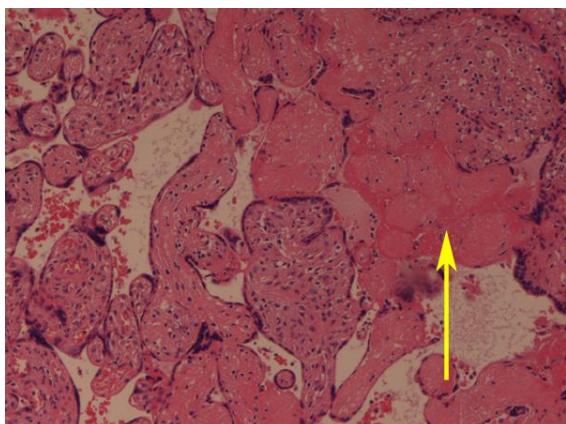


Figure 5: Photomicrograph showing chronic villitis, perivillitis with fibrosis of villi (H & E stain, 125X).

References

1. Ananth CV, Berkowitz GS, Savitz DA, Lapinski RH., Placental abruption and adverse perinatal outcomes. *JAMA*, 282:1646–51,1999.
2. Browne JCM. and Veall N., The maternal placental blood flow in normotensive and hypertensive women. *J. Obstet. Gynaecol. Br. Emp.* 60: 141-147, 1953
3. Sheiner E, Shoham-Vardi I, Hallak M, Hadar A, Gortzak-Uzan L, Katz M, et al. Placental abruption in term pregnancies: clinical significance and obstetric risk factors. *J Matern Fetal Neonatal Med*, 13:45–9, 2003.
4. Ananth CV, Wilcox AJ., Placental abruption and perinatal mortality in the United States. *Am J Epidemiol*, 153:332–7, 2001.
5. Sheiner E, Shoham-Vardi I, Hallak M, Hadar A, Gortzak-Uzan L, Katz M, et al., Placental abruption in term pregnancies: clinical significance and obstetric risk factors. *J Matern Fetal Neonatal Med*,13:45–9, 2003.
6. Salihu HM, Beken B, Aliyu MH, Rouse DJ, Kirby RS, Alexander GR., Perinatal mortality associated with abruptio placenta in singletons and multiples. *Am J Obstet Gynecol.*, 193:198–203, 2005.

7. Ananth CV, Savitz DA, Luther ER. Maternal cigarette smoking as a risk factor for placental abruption, placenta previa, and uterine bleeding in pregnancy. *Am J Epidemiol*, 144:881–9,1996.
8. Ananth CV, Savitz DA, Williams MA. Placental abruption and its association with hypertension and prolonged rupture of membranes: a methodologic review and meta-analysis. *Obstet Gynecol*, 88:309–18, 1996.
9. Ananth CV, Oyelese Y, Srinivas N, Yeo L, Vintzileos AM. Preterm premature rupture of membranes, intrauterine infection, and oligohydramnios: risk factors for placental abruption. *Obstet Gynecol* ,104:71–7, 2004.
10. Ananth CV, Savitz DA, Bowes WA Jr, Luther ER. Influence of hypertensive disorders and cigarette smoking on placental abruption and uterine bleeding during pregnancy. *Br J Obstet Gynaecol*, 104:572–8, 1997.
11. Ananth CV, Smulian JC, Demissie K, Vintzileos AM, Knuppel RA. Placental abruption among singleton and twin births in the United States: risk factor profiles. *Am J Epidemiol*, 153:771–8, 2001.
12. ACOG educational bulletin. Obstetric aspects of trauma management. Number 251, September 1998 (replaces Number 151, January 1991, and Number 161, November 1991). American College of Obstetricians and Gynecologists. *Int J Gynaecol Obstet*, 64:87–94, 1999.
13. Kupferminc MJ, Eldor A, Steinman N, Many A, Bar-Am A, Jaffa A, et al. Increased frequency of genetic thrombophilia in women with complications of pregnancy [published erratum appears in *N Engl J Med* 1999;341:384]. *N Engl J Med*,340:9–13, , 1999.
14. Kaminsky LM, Ananth CV, Prasad V, et al: The influence of maternal cigarette smoking on placental pathology in pregnancies complicated by abruption. *Am J Obstet Gynecol* 197:275.e1-275.e5, 2007
15. Fox H. The villous cytotrophoblast as an index of placental ischaemia. *J. Obstet Gynaecol J. Br. Common W.* 71: 885, 1964.
16. Fox H. The morphological basis of placental insufficiency. *J. obstet. Gynaec. India*, 25 (4): 441-450, 1975.
17. Wigglesworth JS,The gross and microscopic pathology of the prematurely delivered placenta. *J. Obstet Gynaecol Br Common W*, 69: 934-943, 1962.
18. Genset DR, Estimating the time of death of stillborn fetuses-II. A study of 71 stillborns. *Br. J. Obstet. Gynaecol*, 80: 585-592, 1992.
19. Tiwari K Tyagi SP, Saxena KF and Usmani BR., Ultrasonographic and histological studies of placenta in abnormal pregnancy cases. *J Obstet & Gynecol India* 47(2):119-126, 1997.
20. Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A, A study of placenta in normal and hypertensive pregnancies. *J. Anat. Soc. India* 54 (2), 34-38, 2005.
21. Salafia C.M, Pezzullo J.C, Simmens S, Minior V.K, and Vintzileos A.M, Placental pathologic features of preterm preeclampsia. *Am J Obstet Gynecol.* 173: 1097-105, 1995.
22. Sodhi S, Mohan H, Jaiswal T.S., Praveen S, Rathee S and Mohan, Placental pathology in pre-eclampsia syndrome. *Indian J. Pathol. Microbiol.* 33 : 1 ; 11-16, 1990.
23. Brosens I.A., Robertson W. B., Dixon H. G. The role of spiral arteries in pathogenesis of preeclampsia. *Obstet Gynecol Ann.* 1: 177-91, 1972.