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Quantitative Correlation between Abruptio Placentae and Normal Termed Placental Morphology

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

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

Introduction

Placental abruption 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 placentae, 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, abruption 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 abruption include prior abruption, 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 abruption 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 abruption 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 placentae there is a 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.

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 placentae 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 placentae with cord were collected for study.

Sample Collection and Processing of placenta

Following delivery, placentae 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 placentae 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 as1.77 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 Fox[15] 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 toxaemia which was consistent with our finding in placental abruption. Genset[18] reported that excessive syncytial knot formation was in response to overall reduction of foetal perfusion. According to Fox[16], 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 toxaemia which was consistent with our finding in placental abruption. Our findings are also consistent with the findings of Fox[16] 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] observation 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 placentae. 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 placentae. Similar findings were reported by Majumdar et. al.[20] in the hypertensive patients. Sodhi e.t al.[22] also demonstrated a similar change. In our study we noted a statistically significant increase in infarction rate in abruptio placentae 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 placentae. 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 placentae in study (n=30) and control groups (n=15)

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

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

Histological Features	Abruptio placentae (Mean ± SD)	Term Placenta (Mean ± SD)	Significant P<0.01

Cytotrophoblastic cells proliferation	16.53 ± 7.58	6.50 ± 4.38	Significant P<0.01
Syncytial Knots	41.80 ± 20.81	6.1 ± 5.76	Significant P<0.001
Vasculosyncytial membranes	2.87 ± 3.71	10.5 ± 3.72	Significant P<0.01
Fibrinoid necrosis	6.0 ± 4.17	1.6 ± 0.84	Significant P<0.001
Basement membrane thickness	7.17 ± 5.35	1.90 ± 0.88	Significant P<0.01
Stromal fibrosis	11.10 ± 8.66	1.6 ± 0.7	Significant P<0.001
Infarction	5.0 ± 4.38	1.6 ± 0.7	Significant P<0.02

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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).

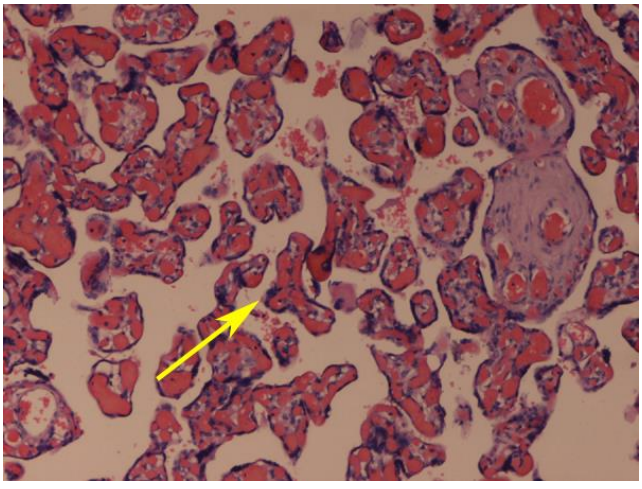

Figure 2: Photomicrograph showing polar cytotrophoblastic proliferation in chorionic villus (H & E stain, 500X).

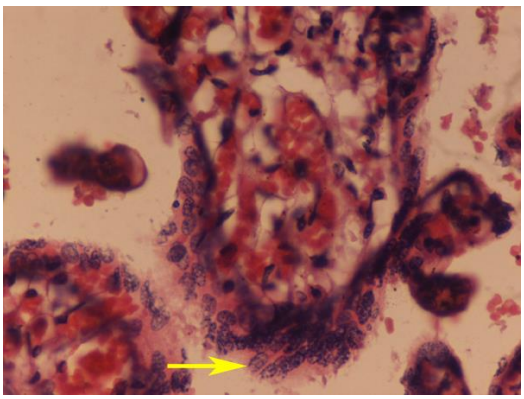

Figure 3: Photomicrograph showing chorionic villi with thickened basement membrane and increased syncytial knots 25% (H & E stain, 500X).

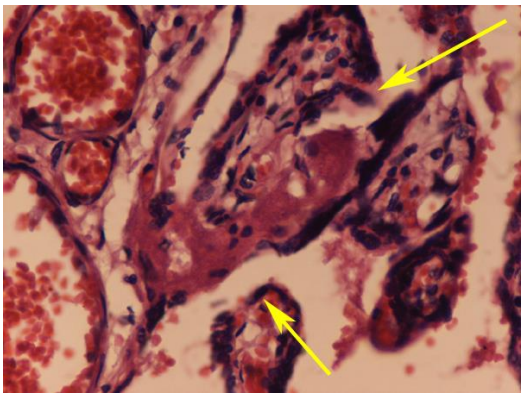

Figure 4: Photomicrograph showing fibrinoid necrosis of chorionic villus as homogenous eosinophilic material (H & E stain 125X).

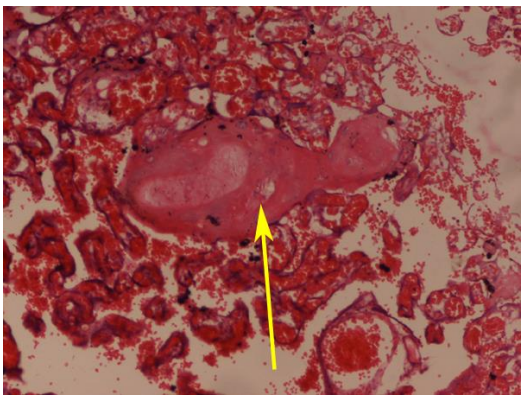

Figure 5: Photomicrograph showing chronic villitis, perivillitis with fibrosis of villi (H & E stain, 125X).

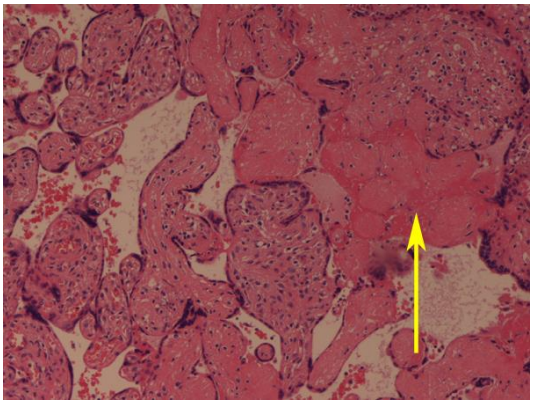

Figure 6: Photomicrograph showing (Retroplacental hemorrhage with hypertension) villi shows fibrosis & intervillus edema (V. G. stain, 125X).