Section: Pathology

Original Research Article

HISTOPATHOLOGICAL ANALYSIS OF PATIENTS WITH ABRUPTIO PLACENTA IN A TERTIARY HEALTH CARE CENTRE

A. Peter Samidoss[1], S.T. Sangeethalakshmi[2], R. Vedamoorthy[1], M.S. Muthu Prabha[1], V. Buvaneswari[1]

[1]Assistant Professor, Department of Pathology, Government Theni Medical College, Theni, Tamilnadu, India.
[2]Assistant Professor, Department of Pathology, Government Chengalpattu Medical College, Chengalpattu, Tamilnadu, India

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Corresponding Author:
Dr. V. Buvaneswari,
Email: drbuvanavinoth@gmail.com

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Abstract

Background: It is the most common pathological cause of late pregnancy bleeding. In humans, it refers to the abnormal separation after 20 weeks of gestation and prior to birth. It occurs on average of 0.5% or 1 in 200 deliveries. The objective is to analyse the incidence of abruptio placenta in a tertiary health care centre. To study the clinical and histopathological features of placenta in abruptio placenta and compare with normal placenta. To study the morphological difference of abruptio placenta of primigravida with that of multigravida patients. **Materials and Methods:** A prospective case-control study included 50 singleton pregnancy with clinical diagnosis of placental abruption (cases) compared to 50 consecutive normal pregnancies (control), attending Coimbatore Medical College Hospital, Tamilnadu, over a period of one and half years from January 2017 to June 2018. Clinical features, gross examination, histopathological analysis was compared between cases and controls, primi and multi gravida. **Result:** The incidence of Abruptio placentae in Coimbatore medical college hospital is 35-40 cases per year. Multiparity, paternal smoking, pregnancy induced hypertension, were the significant risk factors for placental abruption. Median age of occurrence was 27 years. Intervillous haemorrhage, intravillous haemorrhage and increased syncytiotrophoblastic knotting were the significant acute histological features. Decidual vasculopathy was the significant chronic histological feature of placental abruption. Chorioamnionitis with haemorrhage was the significant histopathological feature of placental abruption in primi gravida. Placental infarction, villitis, villous maldevelopment and maternal floor decidual necrosis were the significant histopathological feature of placental abruption in Multi gravida. **Conclusion:** Pathology behind the placental abruption in primi gravida is different from multigravida. Paternal smoking was a significant risk factor for chronic inflammatory processes in placental abruption. Further studies are recommended in primi gravida regarding histopathological, Serological and genetic aspects, to predict the placental abruption in subsequent pregnancies.

INTRODUCTION

Abruptio placenta was first described by Rigby by publishing an essay on Uterine hemorrhage which precedes the delivery of the fetus. Placental abruption is a complication of pregnancy, wherein the placental lining has separated from the uterus of the mother prior to delivery. It is the most common pathological cause of late pregnancy bleeding. In humans, it refers to the abnormal separation after 20 weeks of gestation and prior to birth. It occurs on average in 0.5% or 1 in 200 deliveries.[1] In Coimbatore Medical College, the incidence of patients with placental abruption is about 1 to 2 patients per month and Intra Uterine death is 0-1 per month. Preterm deliveries associated with over half of all pregnancies complicated by placental abruption, and it leads to many adverse maternal and fetal outcomes. The etiology of placental abruption remains hypothetical but is thought to be the consequence of abnormal invasion of trophoblast leading to rupture of spiral arteries and premature separation of the placenta.[2]

In spite of being a unique obstetrical condition, specific diagnostic clinical criteria is not available for placental abruption. Histological evidences supportive to chronic process that often goes with placental abruption has led researchers to hypothesize that the condition is the end result of a

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chronic process which starts very early in pregnancy and perhaps even prolonging to the time of implantation. We have estimated associations between acute and chronic features with placental abruption. This is to determine if clinical and pathological findings from the placenta may perhaps provide some insight as to whether placental abruption is the result of an acute event or a chronic process.

The causes of placental abruption are multifactorial, defective mechanism in early vascularization during placentation, immunologically mediated dysfunction, acute and chronic inflammatory processes might be playing significant roles in the development of placental abruption.[3] Many maternal modifiable risk factors during pregnancy such as pregnancy induced hypertension, polyhydramnios, thrombophilia, preterm premature rupture of membranes, intrauterine infection are associated with increased risk of placental abruption. It has been shown that trophoblast releases a factor that inhibits platelet aggregation and it has been postulated that this factor is needed for normal placental blood flow, when it is decreased, abruption may take place.[4] Toivonen et al., (2004) found that the low activity haplotype C-A (His 113-His139) of the microsomal epoxide hydrolase gene was less frequent in women with abruptio placenta.[5] Tsegaselassie et al., (2013) conducted a study by integrating multiple genomic analytical strategies that provides opportunities for identifying novel biological pathways for exploring the underlying molecular mechanisms for placental abruption.[6]

There are only few studies relating to abruptio placenta in primigravida patients compared to multigravida patients. Present study aims to study the histomorphological features of abruptio placenta of primigravida compared to multigravida patients and to analyze the features more associated with Intra Uterine death incidence. This could enlighten us about the pathway leading to grave prognosis and fatality in such patients, thus enabling us to prevent maternal and fetal deaths in abruptio placenta. If the pathogenesis is unravelled by studying patients with placental abruption, subsequent pregnancies might be given more attention and preventive measures may be undertaken.

Aims and Objectives

To study the incidence, clinical features and histomorphological features of abruptio placenta in a tertiary health care Centre. To compare abruptio placenta with normal placenta and to analyze the differences in histomorphology of abruptio placenta in primi gravida and multi gravida patients.

MATERIALS AND METHODS

Study Population: Women with clinical diagnosis of abruption Placenta attending Coimbatore medical College Hospital.

Study Design: Prospective case control study.

Sample Size: Totally 100 pregnancy cases (50 cases of abruptio placenta and 50 controls)

Prospective case control study was conducted on 100 pregnant women attending the Obstetrics & Gynaecology Department of Government Coimbatore Medical College and Hospital, over a period of one and half years from January 2017 to June 2018. The study was approved by the local Medical Research Ethics Committee, Government Coimbatore Medical College and Hospital. Written, understandable, Informed consent was obtained from all the participants before enrolling in the study. The study included 50 singleton pregnant women with clinical diagnosis of placental abruption compared to 50 consecutive normal pregnancies. All were attending the labor ward. Their gestational age ranged from 24 to 40 weeks calculated from the last menstrual period or early ultrasound.

Inclusion Criteria

All the patients with signs and symptoms of abruptio placenta. Caesarean section and normal delivery included. Booked and immunized cases. Primi gravida and Multi gravida with and without Intra Uterine death. Women with a confirmed or suspected clinical diagnosis of placental abruption were eligible for recruitment as potential cases. Placental abruption cases, or women suspected to have experienced an abruption by the delivering physician, were regarded as true cases if they satisfied at least one of the following 3 specific clinical criteria.[7]

1. Patients presenting with clinical signs of painful vaginal bleeding accompanied by at least one of the following: nonreassuring fetal status, severe abdominal pain, tetanic uterine contractions or uterine hypertonicity.
2. The freshly delivered placenta showing evidence of clinically significant retroplacental bleeding or clots.
3. Placental abruption diagnosed on prenatal ultrasound.[8]

Exclusion Criteria

Cases with type 2 diabetes mellitus/systemic hypertension. Cases with Seropositivity. Cases with TORCH infection. Cases with bronchial asthma/COPD. Cases with history of treatment for primary infertility. Abruptio placenta patients ending in maternal death.

Controls: Women with pregnancies that were not complicated by placental abruption were enlisted as controls and were compared to abruption cases. Control cases were identified in the absence of the following.

1. Any clinical documentation of abruption,
2. Presence of medical illnesses such as Diabetes Mellitus and hypertensive disorders,
3. Presences of PROM,
4. Multiple pregnancies,
5. In cases or controls, women who are diagnosed as placenta previa.

All the placentas (cases & controls) were embedded in 10% neutral buffered formalin and allowed to fix for 24 hours.

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Optimal sampling techniques included 3 placental sections, one with 2 sections of umbilical cord and a roll of extra placental membranes, one section each of fetal and maternal surfaces. When gross lesions were identified, additional sections, up to 3 were made. Then the sections were subjected to tissue processing, section cutting and Haematoxylin & Eosin staining.

Acute histologic lesions included chorioamnionitis, acute deciduitis, funisitis, villous edema, villous stromal haemorrhage and meconium stained membranes associated with amnion necrosis and pigmented macrophages. Chorioamnionitis [Figure 4] was defined by the presence of inflammatory infiltrates of neutrophils at two or more sites on the chorionic plate and extra-placental membranes. It was classified into one of four grades: none, mild, moderate and severe. Mild chorioamnionitis (The presence of few scattered neutrophils (5-10/high power field) in the Subchorionic space and adjacent chorion) [Figure 5d], Moderate chorioamnionitis (Many neutrophils (11–30/high power field) in the lower half of the chorionic plate [Figure 5e] and Severe chorioamnionitis (Dense infiltrates of neutrophils (>30/high power field) throughout the chorionic plate into the amnion [Figure 5f]. Funisitis [Figure 4a] was defined when neutrophils infiltrated the umbilical cord stroma (Wharton's jelly). Villous stromal haemorrhage [Figure 5i,j] was identified if there were erythrocytes within the stroma of chorionic villi.[9,10]

Chronic lesions included chronic deciduitis (lymphocytes with or without plasma cells), maternal floor decidual necrosis [Figure 2b], villitis, decidual vasculopathy (specifically, in the vessels of the extraplacental membrane roll) [Figure 2a], placental infarction [Figure 3d], intervillous thrombosis, villous maldevelopment, and hemosiderin deposition. Villous mal-development included the findings of delayed maturation (increased villous size, increase in stromal cells and decreased syncytial knotting) and accelerated villous maturation (small and slender villi with reduced branching, and increased syncytial knotting). Decidual vasculopathy [Figure 7] comprised of muscular thickening, decidual thrombosis or atherosis occurring within the vessels contained in the extraplacental membrane roll.[5]

RESULTS

In present study, the common age group (50%) of occurrence of placental abruption was 26-30 years [Table 1]. In present study, the mean age of occurrence of placental abruption was 27.2. Median age was 27 years [Figure 1]. The youngest age was 16 years and the oldest age was 37 years. Clinical features among cases and control study, acute onset of abdominal pain, vaginal bleeding and retroplacental haemorrhage [Figure 2c, 3e] were the predominant clinical features (100%) followed by uterine tenderness (92%), and meconium stained membranes (78%) [Table 2].

Histopathological features of Acute abruption in the study, intervillous haemorrhage [Figure 5i,j], intravillous haemorrhage [Figure 5k] and increased syncytiotrophoblastic knotting (Figure 6 p) were the predominant (100%) acute histological features, followed by decidual haemorrhage (88%), and chorioamnionitis (86%) [Figure 4b,c,d,f and Table 2]. Histopathological features of chronic abruption in the study, Decidual vasculopathy [Figure 7] was the predominant chronic histological feature (46%) of placental abruption, followed by villitis (44%), villous maldevelopment (44%), and Maternal floor decidual necrosis [Table 2].

Chorioamnionitis with haemorrhage (65.7%) [Figure 5g] was a significant feature of acute abruption. Acute deciduitis (85%) [Figure 5h] was a prominent feature of acute abruption [Table 3]. The overall picture of placental infarction (100%), villitis (100%), villous maldevelopment (100%), maternal floor decidual necrosis (100%) and decidual vasculopathy (95.7%) were significant features of acute abruption with chronic features cases [Table 3].

Association of Paternal Smoking with cases of acute abruption and cases of acute abruption with chronic features, paternal smoking was present in 4 cases (15.4%) of isolated acute abruption whereas it was present in 22 cases (84.6%) of acute abruption with chronic features. Thus paternal smoking can be considered as a significant independent risk factor associated with acute abruption with chronic features [Table 4].

Gender Distribution of Children in Placental Abruption studies both the cases and controls had 30 male babies and 20 female babies. Thus, there was a significant association between incidence of placental abruption and male babies [Table 5]. Status of Children in Placental Abruption, among the 50 placental abruption cases, 26 male babies (89.7%) had undergone intrauterine death. Thus, there was a significant association between placental abruption and intrauterine death of male babies [Table 5].

Pregnancy Induced Hypertension, 24 placental abruption cases (100%) were associated with pregnancy induced hypertension. Thus, Pregnancy Induced Hypertension has a significant association with cases of placental abruption [Table 7]. Analysis of gravida between cases and control in present study, both the cases and controls had 17 primi gravida and 33 multi gravida. Thus, Multi gravida pregnancies were significantly associated with incidence of placental abruption [Table 7].

Histopathological features of acute abruption in Primi gravida and Multi gravida, chorioamnionitis with haemorrhage [Figure 5g] [Figure 6o] was present in 17 cases (48.6%) of placental abruption in primi gravida. Acute deciduitis [Figure 5h] was present in 17 cases (85%) of placental abruption in primi gravida. Thus, chorioamnionitis with haemorrhage and acute deciduitis were significantly associated with placental abruption in primi gravida

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[Table 8]. Histopathological features of chronic abruption in primi gravida and multi gravida, placental infarction, villitis, villous maldevelopment, and maternal floor decidual necrosis were present in 21 cases (95%) of placental abruption in Multi gravida. Thus, placental infarction, villitis, villous maldevelopment, and maternal floor decidual necrosis [Figure 2b] were significantly associated with placental abruption in multi gravida. These were the features of acute abruption with chronic features. Villous infarction was present in 16 cases (94.1%) of placental abruption in Multi gravida. Thus, villous infarction was a significant feature of placental abruption in multi gravida which was a feature associated with acute abruption with chronic features [Table 8].

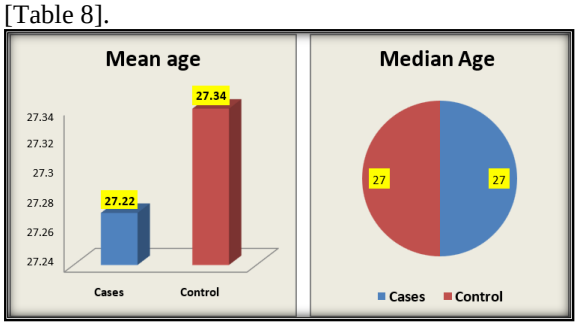

Figure 1: Mean and Median age

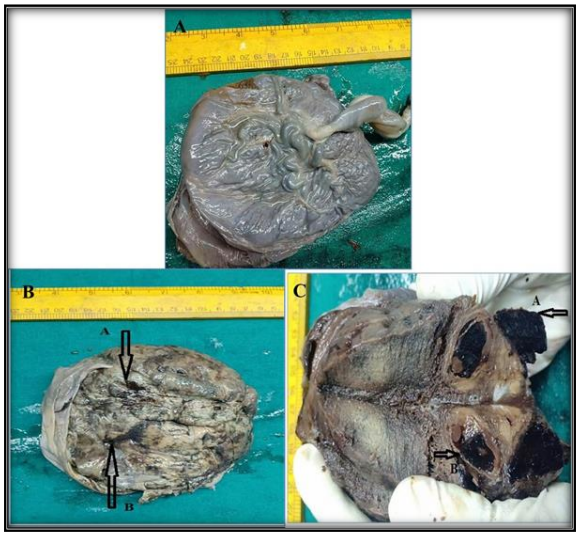

Figure 2: Gross Placental Abruption (a). Dilated Tortuous Vessels Over the Membranes, (b). Maternal Surface- Indentation and Hemorrhagic Areas, (c) Retro placental Blood Clot with Vessel Thrombus

Figure 3: Gross Placental Abruption ((d). Placental Infarction with Congestion and (e) Retro placental Hemorrhage)

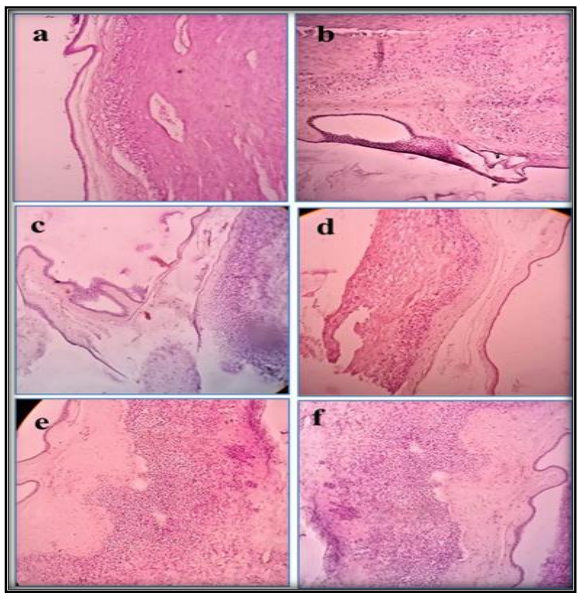

Figure 4: Microscopy Placental Abruption. (a). Funisitis (H&E Stain 400X), (b). Squamous Mataplasia with Moderate Chorioamnionitis (H&E Stain 400 X), (c) Squamous Mataplasia with Moderate Chorioamnionitis (H&E Stain 400 X), (d). Mild Chorioamnionitis (H&E Stain 400 X), (e) Moderate Chorioamnionitis (H&E Stain 400 X) and (f). Severe Chorioamnionitis (H&E Stain 400 X).

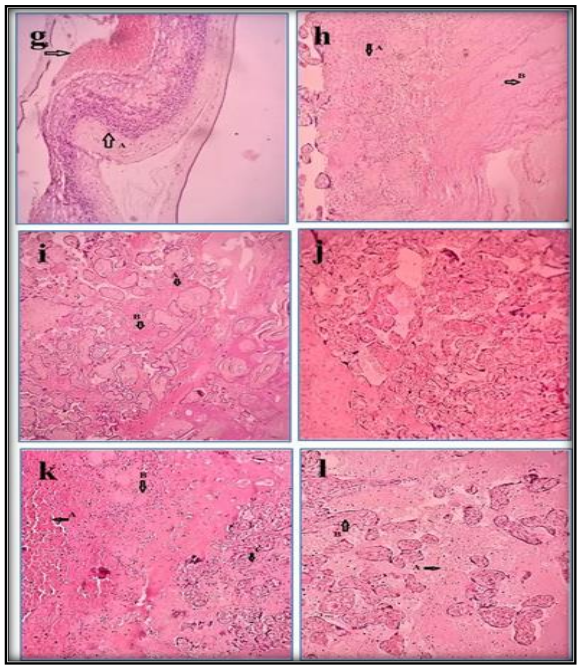

Figure 5: Microscopy Placental Abruption (g). Moderate Chorioamnionitis with Haemorrhage (H&E Stain – 400 X) (A. Moderate Chorioamnionitis and B. Haemorrhage area), (h). Acute Deciduitis with Fibrinoid Necrosis (H&E Stain 400X) (A. Acute Deciduitis and B. Layering of Fibrinoid Necrosis), (i). Intervillous Haemorrhage (H&E Stain 400X), (j). Intervillous and Intravillous Haemorrhage (H&E Stain 400 X), (k). Intravillous Haemorrhage and Adjacent Area of Acute Deciduitis with Haemorrhage (H&E Stain 400 X. A. Haemorrhage, B. Acute Deciduitis and C. Initra Villous Haemorrhage) and (l). Intervillous Haemorrhage and Acute Deciduitis (H&E Stain 400 X. A. Acute Deciduitis and B. Intervillous Haemorrhage).

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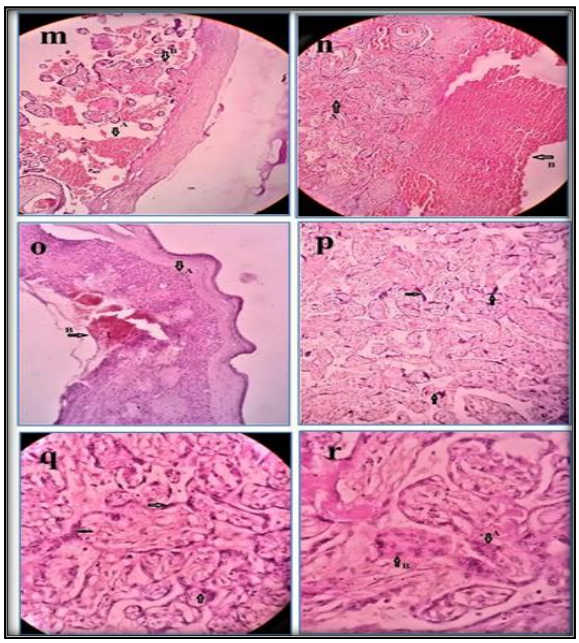

Figure 6: Microscopy Placental Abruption. (m). Intervillous Haemorrhage with Villous Infarction (H&E Stain 400X. A. Intervillous Haemorrhage and B. Villous Infarction), (n). Villous Haemorrhage with Large area of Haemorrhage (H&E Stain 400X. A. Villous Haemorrhage and B. Large area of Haemorrhage), (o). Moderate Chorioamnionitis with Adjacent Area of Hemorrhage (H&E Stain 400X. A. Moderate Chorioamnionitis and B. Hemorrhage Area), (p). Increased Syncytio trophoblastic Knotting (H&E Stain 400X), (q). Increased Syncytio trophoblastic Knotting (H&E Stain 400X) and (r) Increased Syncytiotrophoblastic Knotting with Multinucleated Giant Cell (H&E Stain 400X, A. Increased Syncytiotrophoblastic Knotting and B. Multinucleated Giant Cell)

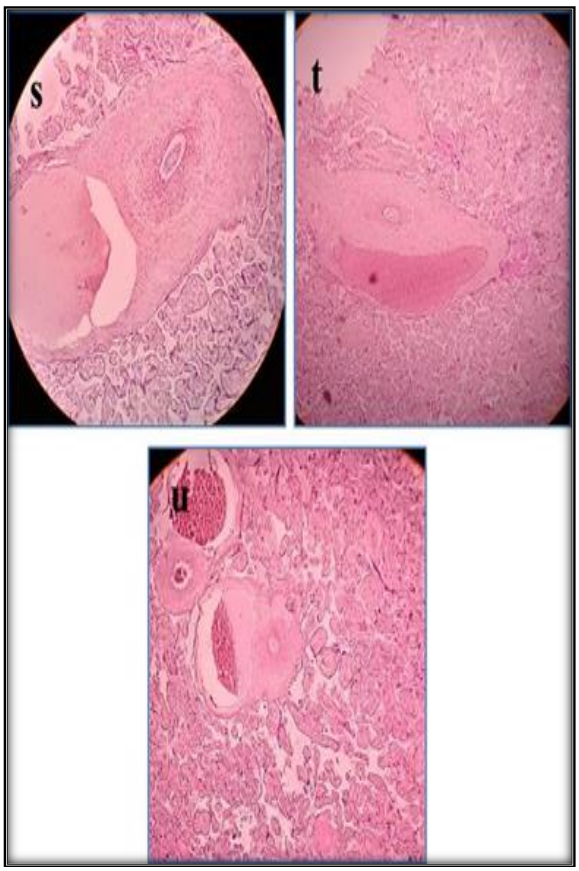

Figure 7: Microscopy Placental Abruption (s). Vasculopathy (Vessels with Marginal Hematoma) (H&E Stain 400X), (t). Vessel with Thick Muscular Cuffing and Adjacent Marginal Hematoma (H&E Stain 400X) and (u). Vessel with Thrombus and Villous Infarction (H&E Stain 400X)

Table 1: Age Distribution among Cases and Controls.

Age	Cases (n=50)	Controls (n= 50)
15- 20 years	6 (54.5%)	5 (45.5%)
21-25 years	13 (48.1%)	14 (51.9%)
26-30 years	17 (50.0%)	17 (50.0%)
31-35 years	12 (50.0%)	12 (50.0%)
36-40 years	2 (50.0%)	2 (50.0%)
Total	50 (100%)	50 (100%)

Table 2: Correlation of Clinical features among cases and control, Histopathological features of Acute abruption and Histopathological features of chronic abruption.

	Cases	Controls
Correlation of Clinical features among cases and control		
Acute onset of abdominal pain	100 (100.0%)	0 (0.0%)
Vaginal bleeding	50 (100.0%)	0 (0.0%)
Uterine tenderness	46 (92.0%)	0 (0.0%)
Retroplacental hemorrhage	50 (100.0%)	0 (0.0%)
Meconium stained membrane	39 (78.0%)	0 (0.0%)
Histopathological features of acute abruption		
Funisitis	20 (40%)	1 (2.0%)
Intervillous haemorrhage	50 (100.0%)	0 (0.0%)
Chorioamnionitis	43 (86.0%)	3 (6.0%)
Chorioamnionitis with haemorrhage	35 (70.0%)	0 (0.0%)
Acute deciduitis	20 (40%)	0 (0.0%)
Decidual hemorrhage	44 (88%)	0 (0.0%)
Increased syncytio trophoblastic knotting	50 (100.0%)	0 (0.0%)
Histopathological features of chronic abruption		
Villitis	22 (44.0%)	0 (0.0%)
Villous infarction	17 (34.0%)	0 (0.0%)
Villous maldevelopment	22 (44.0%)	0 (0.0%)

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Maternal floor decidual necrosis	22 (44.0%)	0 (0.0%)
Decidual vasculopathy	23 (46.0%)	0 (0.0%)

Table 3: Expression and Statistics of Histopathological features of acute abruption in acute abruption cases and in Acute abruption with chronic features cases and Expression and Statistics of Histopathological features of chronic abruption in acute abruption cases and in cases of Acute abruption with chronic features.

		Acute abruption (n=28cases)	Acute abruption with chronic features (n=22 cases)	P value
Expression of Histopathological features of acute abruption in acute abruption cases and in Acute abruption with chronic feature cases.				
Intravillous hemorrhage		28 (56.0%)	22 (44.0%)	
Chorioamnionitis		27 (62.8%)	16 (37.2%)	
Chorioamnionitis with haemorrhage		23 (65.7%)	12 (34.3%)	
Acute deciduitis		17 (85.0%)	19 (63.3%)	
Decidual haemorrhage		25 (56.8%)	19 (43.2%)	
Increased syncytiotrophoblastic knotting		28 (56.0%)	22 (44.0%)	
Statistics - Histopathological features of acute abruption.				
Chorioamnionitis with haemorrhage	Yes	23 (65.7%)	12 (34.3%)	.035*
	No	5 (33.3%)	10 (67.7%)	
Acute deciduitis	Yes	17 (85.0%)	3 (15.0%)	.000*
	No	11 (36.7%)	19 (63.3%)	
Decidual Haemorrhage	Yes	25 (56.8%)	19 (43.2%)	.752
	No	3 (50.0%)	3 (50.0%)	
Increased syncytio trophoblastic knotting	Yes	28 (56.0%)	22 (44.0%)	NA
	No	0 (0.0%)	0 (0.0%)	
Expression of Histopathological features of chronic abruption in acute abruption cases and in cases of Acute abruption with chronic features.				
Placental infarction		0 (0%)	22 (100%)	
Villitis		0 (0%)	22 (100%)	
Villous infarction		0 (0%)	17 (100%)	
Villous maldevelopment		0 (0%)	22 (100%)	
Maternal floor decidual necrosis		0 (0%)	22 (100%)	
Decidual vasculopathy		1 (4.3%)	22 (100%)	
Statistics - Histopathological features of chronic abruption.				
Placental infarction	Yes	0 (0.0%)	22 (100.0%)	.000*
	No	28 (100.0%)	0 (0.0%)	
Villitis	Yes	0 (0.0%)	22 (100.0%)	.000*
	No	28 (100.0%)	0 (0.0%)	
Villous Infarction	Yes	0 (0.0%)	17 (100.0%)	.000*
	No	28 (84.8%)	5 (15.2%)	
Villous maldevelopment	Yes	28 (56.0%)	22 (44.0%)	.000*
	No	0 (0.0%)	0 (0.0%)	
Maternal floor Decidual necrosis	Yes	0 (0.0%)	22 (44.0%)	.000*
	No	28 (100.0%)	0 (0.0%)	
Decidual vasculopathy	Yes	1 (4.3%)	0 (0.0%)	.000*
	No	27 (100.0%)	22 (95.7%)	

*Statistically significant ($p<0.05$)

Table 4: Association of Paternal Smoking with cases of acute abruption and cases of acute abruption with chronic features.

Paternal smoking	Acute abruption	Acute abruption with chronic features	P value
Present	4 (15.4%)	22 (84.6%)	.000*
Absent	24 (100.0%)	0 (0.0%)	

*-Statistically significant ($p<0.05$)

Table 5: Gender Distribution of Children in Placental Abruption and Status of Children in Placental Abruption.

	Male	Female	P value
Gender Distribution of Children in Placental Abruption.			
Abruption cases (Total=50)	30(60.0%)	20(40.0%)	.000*
Control (Total=50)	30(60.0%)	20(40.0%)	
Status of Children in Placental Abruption.			
Alive	4(19.0%)	17(81.0%)	.000*
Intra uterine death	26(89.7%)	3(10.3%)	

*Statistically significant ($p<0.05$)

Table 6: Correlation between chorioamnionitis and preterm deliveries among placental abruption.

Chorioamnionitis Vs Preterm deliveries		Preterm deliveries		P value
		Yes	No	
Chorioamnionitis	Yes	42 (97.7%)	1 (2.3%)	.134
	No	6 (85.7%)	1 (14.3%)	

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Table 7: Pregnancy Induced Hypertension and Analysis of gravida between cases and control.

Pregnancy Induced Hypertension	Cases (Total=50)	Controls (Total=50)	P value
Present	24 (100.0%)	0(0.0%)	.000*
Absent	26 (34.2%)	50(65.8%)	
Analysis of gravida between cases and control	Primi gravida	Multi gravida	P value
Cases	17(34%)	33(66%)	.000*
Control	17(34%)	33(66%)	

*-Statistically significant (p<0.05)

Table 8: Histopathological features of acute abruption and chronic abruption in Primi gravida and Multi gravida.

Histopathological features	Multi gravida (33 cases)	Primi gravida (17 cases)	P value
Histopathological features of acute abruption in Primi gravida and Multi gravida.			
Funisitis	12 (60.0%)	8 (40.0%)	.465
Intravillous haemorrhage	33 (66.0%)	17 (34.0%)	NA
Chorioamnionitis	26 (60.5%)	17 (39.5%)	.041
Chorioamnionitis with haemorrhage	18 (51.4%)	17 (48.6%)	.001*
Acute deciduitis	3 (15.0%)	17 (85.0%)	.000*
Decidual haemorrhage	27 (61.4%)	17 (38.6%)	.061
Increased syncytiotrophoblastic knotting	33 (66.0%)	17 (34.0%)	NA
Histopathological features of chronic abruption in primi gravida and multi gravida.			
Placental infarction	21 (95.0%)	1 (4.5%)	.000*
Villitis	21 (95.0%)	1 (4.5%)	.000*
Villous infarction	16 (94.1%)	1 (5.9%)	.003*
Villous maldevelopment	21 (95.0%)	1 (4.5%)	.000*
Maternal floor decidual necrosis	21 (95.0%)	1 (4.5%)	.000*
Decidual vasculopathy	22 (95.7%)	1 (4.3%)	.000*

*Statistically significant (p<0.05)

DISCUSSION

The incidence of Abruptio placenta in Coimbatore Medical College hospital is 40 cases per year. In this study, we have collected placentas from 50 consecutive clinically diagnosed cases of placental abruption attending Coimbatore Medical College Hospital for the period of one and half years (from January 2017 to June 2018). In the same period, we have collected placentas from 50 consecutive normal deliveries as Control. Being a tertiary health care centre, many of the placental abruption cases are referred from Primary Health Centres and Taluk Government Hospitals around Coimbatore including Nilgiris, Pollachi, Valparai, Dharapuram, Erode and Namakkal district. Among the 50 placental abruption cases, 43 cases were referred from Primary Health Centres and Taluk Government Hospitals, 7 cases were attending the Government Coimbatore medical college itself. In present study, clinical and histopathological features of placental abruption were studied and compared with the normal placenta. In addition to, the clinical and histopathological features of placental abruption in primi gravida and multi gravida were compared.

Age and Obstetric History

In present study, the most common age of occurrence was 26-30 years accounting to 32% (16 cases). The age group between 21- 35 years comprising 84% (42 cases) of placental abruption cases. Two patients were more than 35 years, 6 patients were ≤20 years. One patient was 16 years old. Mean age of occurrence of placental abruption was 27.2 years. Among the 50 placental abruption patients, 17 patients were primi gravida, 33 patients were multi gravida. Among the multi gravida, 6 patients were grand multi gravida (≥4 deliveries). This signifies the multiparity being the common risk factor for placental abruption.

Paternal Smoking: In present study, there was no history of maternal smoking among the cases. But 26 cases had history of paternal smoking which was accounting for 52% of placental abruption. In acute abruption, paternal smoking was present in 4 cases (15.4%), whereas in acute abruption with chronic features, 22 cases had history of paternal smoking (84.6%) which is statistically significant (P<0.05). Thus, it signifies that paternal smoking is a significant risk factor for acute abruption with chronic features and in multi gravida.

Anaemia: In present study, 41 cases were associated with clinical findings of anaemia which was accounting to 82% of placental abruption.

Pregnancy Induced Hypertension: In present study, 24 cases are associated with the pregnancy induced hypertension which was contributing to 48% of placental abruption cases which is statistically significant (P<0.05). Placental abruption and pre-eclampsia might be sharing the same common aetiology with failed placentation in early pregnancy. This could lead to the placental dysfunction and further increases the risk of abruption of placenta in women with pregnancy induced hypertension.

Gestational Diabetes Mellitus: In this study, one patient had gestational diabetes which was accounting to 2% of the placental abruption.

Hydramnios: In present study, one patient had hydramnios which was accounting to 2% of the placental abruption cases.

Premature Rupture of Membrane: In present study, 2 patients were associated with premature rupture of membranes which was accounting to 4% of placental abruption. Usually PROM precedes abruption, and sometimes abruption may cause

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PROM. Placental abruption is usually associated with dense infiltration of neutrophils in decidua which secretes more amount of proteases that could destroy the cellular matrix, ultimately leads to PROM60. It is very difficult to fix whether decidual neutrophil infiltration is secondary to disruption of vessels or whether it is the primary reason of abruption.

Clinical Features: In Present study, retroplacental haemorrhage, vaginal bleeding, uterine tenderness and acute onset of abdominal pain were found in all the 50 cases of placental abruption which was accounting to 100% of placental abruption. Placental abruption cases are graded by the amount of retroplacental haemorrhage, status of the child and coagulopathy. Among the 50 patients, 5 patients were under grade 1 category which was accounting to 10% of placental abruption, 15 patients were under grade 2 which was accounting to 30% of placental abruption, 28 patients were under grade 3a which was accounting to 56% of placental abruption, 2 patients were under grade 3b which was accounting to 4 % of placental abruption.

Disseminated Intravascular Coagulation: In present study, 2 patients were developed disseminated intravascular coagulation which was accounting to 4% of placental abruption.

Mode of Delivery: In present study, 4 patients (8%) had the vaginal delivery whereas 46 patients (92%) underwent emergency Caesarean section. Out of 50 placental abruption patients, 15 patients were found with the history of previous caesarean delivery which was accounting to 30% of placental abruption.

Intrauterine Death: In present study, 29 patients out of 50 had intrauterine death of fetus which was accounting to 58% of placental abruption that suggests poor perinatal outcome. 21 patients (42%) had alive babies. Among the 29 intrauterine death, 26 patients had male baby (89.7%) which was statistically significant (P<0.05), and 4 patients had female baby (13.8%). Among the 21 alive babies, 17 babies were female (81%) and 4 babies were male (19%). Acute placental separation cut off the oxygen supply and nourishment to the fetus, so that the fetus frequently dies. The significance of male babies more prone to intrauterine death is yet to be deciphered.

Preterm Delivery: In present study, 49 patients delivered preterm babies which was accounting to 98% of placental abruption. The risk to the fetus depends on the severity of placental abruption and the gestational age at which the placental abruption occurs.

Second Trimester Fetal Loss: In present study, one patient of placental abruption had a history of previous intrauterine death which occurred in third trimester. This signifies that the previous second and third trimester fetal loss has been identified as a major risk factor for subsequent stillbirth.

Histopathological Features

Acute Abruption: In present study, funisitis was found in 20 patients (40%), intervillous and intra villous haemorrhage were found in all the 50 patients (100%), chorioamnionitis with haemorrhage was found in 35 patients (70%), acute deciduitis was found in 20 patients (40%), decidual haemorrhage was found in 44 patients (88%), increased syncytiotrophoblastic knotting was found in all the 50 patients (100%).

Acute Abruption with Chronic Features: In present study, we didn't get isolated chronic placental abruption cases, instead we got cases of acute placental abruption with chronic features. In 50 cases, 28 cases were acute placental abruption, 22 cases were acute placental abruption with chronic features. When these two categories were compared, Intervillous haemorrhage was found in 28 cases (56%) of acute placental abruption, whereas it was found in 12 cases (44%) of acute abruption with chronic features. Intravillous haemorrhage was found in 28 cases (44%) of placental abruption, whereas it was found in 22 cases of acute placental abruption with chronic features. Chorioamnionitis was present in 27 cases (62.8%) of acute placental abruption, whereas it was found in 16 cases (37.2%) of acute placental abruption with chronic features. Chorioamnionitis with haemorrhage was found in 23 cases (65.7%) of acute placental abruption which is statistically significant (P<0.05), whereas it was found in 12 cases (34.3%) of acute abruption with chronic features. Acute deciduitis was found in 17 cases (85%) of acute placental abruption which is statistically significant (P<0.05), whereas it was found in 3 cases (63.3%) of acute abruption with chronic features. Decidual haemorrhage was found in 25 cases (56.8%) of placental abruption, whereas it was found in 19 cases (43.2%) of acute abruption with chronic features. Increased syncytiotrophoblastic knotting was found in 28 cases (56%) of placental abruption, whereas it was found in 22 cases (44%) of acute abruption with chronic features.

In present study, 48 placental abruption cases associated with preterm deliveries, among that 42 cases are found with the features of chorioamnionitis (P<0.05). Placental infarction was not found in 28 cases of acute placental abruption (0%), whereas it was found in all the 22 cases (100%) of acute abruption with chronic features (P<0.05). Villitis was not found in 28 cases of acute placental abruption (0%), whereas it was found in all the 22 cases (100%) of acute abruption with chronic features (P<0.05). Villous infarction was not found in 28 cases of acute placental abruption (0%), whereas it was found in 17 cases (77.3%) of acute abruption with chronic features (P<0.05). Villous maldevelopment was not found in 28 cases of acute placental abruption (0%), whereas it was found in all the 22 cases (100%) of acute abruption with chronic features (P<0.05). Maternal floor decidual necrosis was not found in 28 cases of acute placental abruption (0%), whereas it was found in all the 22 cases (100%) of acute abruption with chronic features (P<0.05). Decidual vasculopathy was found in one case (4.3%) of acute placental abruption, whereas it was found in all the

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22 cases (100%) of acute abruption with chronic features (P<0.05).

Bleeding at the decidual-placental interface causes abruption. Acute vasospasm of the small blood vessels might be the event that precedes the separation of placenta. Lack of adequate invasion of trophoblasts might be the cause of early separation 11. A prospective study has found that at 20-24 weeks of gestation, notching changes in the Doppler waveform of the uterine artery which is an important marker of impaired uteroplacental circulation, had a strong association with placental abruption.[12] Thus uteroplacental insufficiency might be playing a critical role in the pathology of abruption. The acute and chronic inflammatory processes are playing a major role in causing placental abruption by triggering the activation of cytokines like Interleukin-1 and Tumor necrosis factor- α. These cytokines are upregulating the production and activity of trophoblast matrix metalloproteinases. This leads to extracellular matrix destruction and loss of cell-cell interactions, which might lead to interruption in the attachment of normal placenta which leads to premature separation of placenta.[13] The cases of placental abruption, acute inflammatory conditions are more common at preterm than term pregnancies whereas chronic inflammatory processes are present throughout the gestation.[14]

Furthermore, in this study, we have compared the histopathological features of placental abruption between primi and multi gravida. This comparative study showed that the feature of acute placental abruption was significantly seen in primi gravida when compare to multi gravida, even though intervillous haemorrhage, intravilloushaemorrhage and increased syncytiotrophoblastic knotting is seen in both primi and multi gravida. In addition to that, the features of chronic placental abruption were significantly seen in multi gravida.

CONCLUSION

This study is the first type of it, which is carried out in Government Coimbatore Medical College Hospital, Tamilnadu. The incidence of abruptio placenta in Government Coimbatore Medical College Hospital, Tamilnadu, is 3 per month. Out of 50 placental abruption cases, 29 cases had intrauterine death and these were significantly associated with male babies. Multiparity and Grand multiparity are strongly associated with placental abruption, so that, the proper health education regarding the complications of multiparity should be given to the mothers through the Primary health centres and Taluk Government hospitals. Modifiable risk factors like anaemia, pregnancy induced hypertension, and multiparity in placental abruption should be early detected and managed appropriately to reduce the incidence of placental abruption. Acute inflammatory process was playing a major role in placental abruption of primi gravida, whereas in multi gravida in addition to the acute events, chronic inflammatory process was also taking part in placental abruption. Infection and inflammation causing tissue damage mediated by IL-1 and TNF-α. Unfortunately, either accurate prediction or prevention of placental abruption is not possible at present time. Further studies regarding acute inflammatory process in the placenta of primi gravida are recommended to predict the placental abruption in subsequent pregnancies. Serological markers to detect acute placental infarction in primi gravida may be tried in future. Paternal smoking is a major risk factor for chronic inflammatory processes in placental abruption. The hypoxic changes developed by nicotine and carbon monoxide could cause placental infarcts, signifying that capillary fragility is increased and it might result in arterial rupture, leading to placental abruption. In this study, chronic vascular changes are predominantly present in multi gravida. Being a preventable risk factor, cessation of smoking reduces adverse pregnancy outcomes including placental abruption. Further studies regarding the vascular changes in the placenta of primi gravida would be useful in predicting the placental abruption in subsequent pregnancies.

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