The Corellation Between the Clinical Diagnosis & Histopathological Findings of Placental Abruption

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ABSTRACT:

BACKGROUND:

The diagnosis of placental abruption is mostly clinical, histopathological diagnosis is poorly sensitive. Acute and chronic inflammatory processes had been proposed to cause placental abruption evidences that supports this remains sparse

OBJECTIVE:

To assess the correlation between the clinical diagnosis and histopathological findings of placental abruption and to examine the profile of chronic and acute histological lesions associated with clinical abruption.

PATIENTS AND METHOD:

The study included fifty singleton pregnant women with a suspected clinical diagnosis of placental abruption compared to fifty consecutive normal pregnancies (control group), attending AL -Yarmouk Teaching Hospital over a period of twelve months, from the first of April 2010 to the end of march 2011 . Examination of the concordance between clinical indicators for placental abruption with those of a histological diagnosis was done. The profile of acute and chronic lesions was also examined histopathologically.

RESULTS:

Among the fifty clinically diagnosed placental abruption cases , thirteen percent (fifteen patients) were confirmed as placental abruption based on gross and histological findings . The most common indication leading to a clinical diagnosis of abruption was evidence of retroplacental clot(s) or bleeding . Acute lesions that were associated with abruption with confirmed pathology included chorioamnionitis , and chorionic villous hemorrhage . Among the chronic lesions, chronic deciduitis, decidul vasculopathy, & dysmaturation were associated with pathologically confirmed placental abruption.

CONCLUSION:

The relation between clinical & histological diagnosis of placental abruption remains weak. Acute and chronic histological lesions were observed more frequently in placentas of pregnancies complicated by placental abruption than the control cases.

KEY WORDS: placental abruption, clinical diagnosis , histopathological lesions.

INTRODUCTION:

Placental abruption (PA) is bleeding after premature separation of the placenta from its implanted site before the birth of the fetus, it's one of the leading causes of fetal and neonatal mortality[1]. The most significant abruption is a retro placental abruption that can compromise fetal oxygenation and perfusion [2]. The reported incidence of PA varies from (0.5% to 2%) and this variation is caused by variation in the diagnosis[3]. There are three grades of PA, these include[1]:

Grade I: are those in which the diagnosis of PA is made retrospectively and the retro placental clot volume of approximately (150 ml), fetuses are usually not at risk and favorable perinatal outcome occurs frequently. Grade II: are those in which ante partum hemorrhage (APH) is accompanied by the classical features of abruption and the fetus is alive, the retro placental clot volume is about (150- 500 ml), (92%) of those patient had abnormal fetal heart rate patterns and perinatal mortality is high especially if the patient delivered vaginally. Grade III: incorporate the features of grade II but fetal demise is confirmed. The classical clinical presentation of placental abruption involving all of these signs-vaginal bleeding, uterine tenderness, hypertonic uterus, fetal demise which

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does not occurs frequently [1] . If blood loss is significant the patient may have signs of shock (tachycardia predominates, blood pressure is poorly correlated with blood volume in this condition). Hypertension may mask true hypovolemia, but increasing abdominal girth or fundal height suggested significant concealed hemorrhage[4]. A common presentation of placental abruption is with mild vaginal bleeding, no uterine tenderness & no coagulopathy , usually occurring in the last 4wk of gestation. The cause of this is peripheral placental separation[1]. The Diagnosis is usually made on clinical grounds. The symptoms and signs are diagnostic in moderate to severe cases .In mild forms , the diagnosis may not be obvious until after delivery ,when retro placental clot is identified. Ultrasonography is not a sensitive method of diagnosing placental abruption, but it is useful in excluding coincident placenta previa, which is present in 10% of cases[4] .The etiology of placental abruption remains speculative, acute and chronic inflammatory processes had been proposed to cause PA by activating cytokines, such as interleukin-1, and tumor necrosis factor, These cytokines up regulate the production and activity of matrix metalloproteinases in the trophoblast. The result is destruction of the extracellular matrices and cell-cell interactions, which may lead to disruption of the normal placental attachment and to premature separation of the placenta[5].

PATIENTS AND METHOD:

Prospective case – control study was conducted on 100 pregnant women attending the Obstetrics & Gynecology Department of AL-Yarmouk Teaching Hospital, Baghdad, Iraq over a period of 12 months from the first of April 2010 to the end of march 2011 .The study was approved by the local Medical Research Ethics Committee of College of Medicine , Almustansiryia University, Department of Obstetrics & Gynecology. Informed consent was obtained from all participants before enrolling in the study. The study included fifty singleton pregnant women with clinical diagnosis of placental abruption (PA) compared to 50 consecutive normal pregnancies (control group) matched for their age, gestational age & parity. All were attending the labor word either as a labor (cases &control) or ante partum hemorrhage (APH) cases. Their gestational age was ranged from 24 to 40 weeks calculated from the last menstrual period or early ultrasound (U/S) .Inclusion criteria: PA cases, or women suspected to have an abruption by the delivering physician were regarded as true cases if they satisfied at least one of the following three specific clinical criteria:1. patients presenting with the classical signs of painful vaginal bleeding accompanied by at least one of the following: non reassuring fetal status, severe abdominal pain, titanic uterine contractions, or uterine hyper tonicity.2.The freshly delivered placenta showing of clinically significant retro placental bleeding or clot (s) .3.PA diagnosed on prenatal U/S. Control cases were identified in the absence of the following:1. Any clinical documentation of abruption , 2.presence of medical illnesses as DM & hypertensive disorders, 3.presences of PROM, 4. Women with placental abruption in a previous pregnancies, 5.Multiple pregnancies& 6.Women diagnosed as placenta previa among cases or controls. Full history including medical, surgical, social, gynecological &obstetrical histories was taken. Clinical examination general &obstetrical was done for all cases & controls. All the placentas (cases &control) were embedded in formaldehyde 10% & then sent for histopathology, which was done by the same pathologist at the laboratory Department of AL Yarmouk Hospital. The pathologist was blinded to the case -control status. Optimal sampling techniques included three placental blocks: one with two sections of umbilical cord & a roll of extra placental membranes, one block each of fetal & maternal surface. when gross lesions were identified, additional sections, up to three block, were submitted to histological examination. Pathological diagnosis of placental abruption :-Gross examination of the placenta was done looking for retro placental hemorrhage, indentation or hematoma with or without recent or old infarctions. On microscopic examination, the pathologist looked first for histological confirmation of placental abruption; villous infarctions associated with decidual destruction, hemorrhage & adjacent increased syncytiotrophoblast knotting. Placental abruption is the manifestation of clinical event that likely have at least two distinct causative pathways: (i) acute inflammation associated-condition include chorioamnionitis, acute deciduities, funisitis, villous edema, chorionic villous hemorrhage & meconium stained membrane & (ii) chronic processes included chronic deciduitis, maternal floor decidual nicrosis, villitis, decidual vasculopathy, placental

infarction, inter villous thrombosis, villous mal-development & hemosidrine deposition.

Statistical analysis:

Data were analyzed using the computer facility of the available statistical software packages of SPSS-18 (Statistical Packages for Social Sciences-version 18) "PASW Statistics". Data were coded and entered in the computer, then presented in simple measures of frequencies and percentages of acute & chronic lesions and then were compared in two sets of analysis. Pearson Chi-square test was applied for testing the significance of difference using $P<0.05$ as the level of significance. Odds ratio (OR) & 95% confidence interval were determined to evaluate the strength of association in both set of analysis

RESULTS:

The mean maternal age for the patients was 30.1 years ± 6.38 & for the control was 30.6 ± 6.25 years , the mean parity for the patients was 1.7 ± 2.16 & for the control was 1.54 ± 2.03, the gestational age ranged from 24 to 40 weeks , with the mean gestational age for the patients was 33.4 ± 4.88 weeks & for the control was 33.16 ± 4.99 weeks. 10% of patients & 2% of control were smokers, preeclampsia was present only in patients group & it was 14% as shown in table 1. Among the 50 clinically diagnosed placental abruption cases , 30% (n=15) were also confirmed as placental abruption based on gross or histological findings, while the remaining 35 cases pathological examination didn't confirmed PA. The most common indication leading to a clinical diagnosis of abruption was evidence of retro placental clot (s) or bleeding seen in 38 (76%) of abruption cases , followed by vaginal bleeding + non reassuring fetal status in13 (26%) of cases , ultrasound diagnosis in 7 (14%) of cases , and lastly by vaginal bleeding+ uterine hyper tonicity in 5 (10%) of cases as shown in table 2. The distributions of acute histological lesions between cases and controls, as well as between histologically confirmed and unconfirmed PA, are described in table 3. From the 50 clinical cases of PA , chorioamnionities was present in 9 (18%), followed by chorionic villous hemorrhage in 8(16%), then meconium in 5(10%) , funisitis in 3(6%) cases & acute diciduities in1(2%).The distribution of chronic lesions in abruption cases and control & between histiologically confirmed & unconfirmed PA cases are shown in Table 4 .Among the 50 clinically diagnosed PA cases, Chronic deciduitis was present in 47(94%), followed by dysmaturation in 36(72%), then by placental infraction in 9(18%), then decidual vasculopathy in 8 (16%) , then by advanced maturation & villitis in 3(6%) for both, decidual necrosis& inter villous thrombus in 2 (4%) & lastly by hemosiderin deposition in1(2%).

Table 1 : Demographic characteristics of clinically diagnosed placental abruption cases and controls.

Maternal characteristics	Abruption cases (n=50)	Controls (n=50)
Mean maternal age(years)	30.1 ±6.38	30.6 ±6.25
Mean parity	1.7±2.16	1. 54.±2.03
Mean gestational age(weeks)	33.4±4.88	33.2±4.9 9

Table 2: Clinical Criteria for the diagnosis of placental abruption

Indicators for a clinical diagnosis Of placental abruption	Clinically diagnosed placental abruption cases			
	All clinical cases (n=5o)	Pathology confirmed (n=15)	Pathology Un confirmed (n=35)	P value
Retro placental clot(s) /hemorrhage	38 (76%)	12 (80%)	26 (74.3%)	0.665
Vaginal bleeding + non reassuring feta l status	13 (26%)	2 (13.3%)	11 (31.4%)	0.181
Ultrasound diagnosis	7 (14%)	3 (20%)	4 (11.4%)	0.426
Vaginal bleeding + uterine hypertonicity	5 (10%)	2 (13.3%)	3 (8.6%)	0.607

*Significant difference in percentage using Pearson Chi-square test at 0.05 level of significance.

Table 3: The association between acute histological lesions and placental abruption cases and controls, and between histologically confirmed & unconfirmed abruption cases.

Acute histological lesions	Clinically diagnosed placental abruption cases			Controls (n=50)	Adjusted OR (95%CI)	
	All clinical cases (n=50)	Pathologically confirmed+ (n=15)	Pathologically unconfirmed++ (n=35)		All clinical Vs Control	Pathologically confirmed Vs Unconfirmed
Chorioamnionitis	9(18.0%)	7(46.7%)	2(5.7%)	4(8.0%)	2.52 (0.72-8.82)	14.44 (2.51-83.17)*
Funisitis	3(6.0%)	3(20.0%)	-	1(2.0%)	3.13 (0.31-31.1)	-
Acute Deciduities	1(2.0%)	1(6.7%)	-	1(2.0%)	-	-
Meconium	5(10.0%)	1(6.7%)	4(11.4%)	3(6.0%)	1.74 (0.39-7.71)	0.55 (0.06-5.41)
Chorionic villous hemorrhage	8(16.0%)	5(33.3%)	3(8.6%)	1(2.0%)	9.33 (1.12-77.7)*	5.33 (1.08-26.36)*
At least one lesion	48(96.0%)	14(93.3%)	34(97.1%)	33(66.0%)	12.36 (2.68-57.1)*	0.41 (0.02-7.05)

+clinically diagnosed placental abruption cases also confirmed on placental pathology
++ Clinically diagnosed placental abruption cases not confirmed as placental abruption on placental pathology.

Table 4: The association between chronic histological lesions and placental abruption cases and controls, and between histologically confirmed & unconfirmed abruption cases.

Chronic histological lesions	Clinically diagnosed placental abruption cases			Controls =	Adjusted OR (95%CI)	
	All clinical cases (n=50)	Pathologically Cc	Pathologically unconfirmed ++	(No.50)		
		confirmed (no.15)	Unconfirmed (no.35)		All clinical Vs Control	Pathologically confirmed Vs Unconfirmed
Chronic deciduitis	47(94.0%)	14(93.3%)	33(94.3%)	40(80.0%)	3.92 (1.0115.22)*	0.85 (0.07-10.14)
Decidual necrosis	2(4.0%)	1(6.7%)	1(2.9%)	2(4.0%)	-	2.43 (0.14-41.60)
Decidual vasculopathy	8(16.0%)	6(40.0%)	2(5.7%)	7(14.0%)	1.17 (0.39-3.52)	11.0 (1.89-64.06)*
Placental infarction	9(18.0%)	9(60.0%)	-	4(8.0%)	2.52 (0.72-8.82)	-
Advanced maturation	3(6.0%)	2(13.3%)	1(2.9%)	3(6.0%)	-	5.23 (0.44-62.72)
Dysmaturation	36(72.0%)	14(93.3%)	22(62.9%)	23(46.0%)	3.02 (1.32-6.93)*	8.27 (0.97-70.42)
Hemosiderin deposition	1(2.0%)	-	1(2.9%)	1(2.0%)	-	-
Inter villous thrombus	2(4.0%)	-	2(5.7%)	9(18.0%)	0.19 (0.04-0.93)*	-
Villitis	3(6.0%)	-	3(8.6%)	4(8.0%)	0.73 (0.16-3.46)	-
At least one lesion	48(96.0%)	14(93.3%)	34(97.1%)	48(96.0%)	-	0.41 (0.02-7.05)

+clinically diagnosed placental abruption cases also confirmed on placental pathology
++ clinically diagnosed placental abruption cases not confirmed as placental abruption on placental pathology.
*Significant difference in percentage using Pearson Chi-square test at 0.05 level of significance.

DISCUSSION:

There is a growing body of evidence to suggest that placental abruption is a pathologic condition, chiefly associated with longstanding chronic vascular lesions, and to a lesser extent, acute

inflammatory processes. Evidence from previous studies collectively suggests that placental abruption is the manifestation of clinical events that likely have at least 2 distinct causative pathways: 1) acute inflammation–associated conditions, and 2) chronic processes(vascular dysfunction and chronic inflammation). If true ,then clues to these pathways may be found in associated clinical risk factors identified throughout pregnancy[6] .

The etiology of placental abruption remains speculative, perhaps, to a large extent the lack of appropriate diagnostic criteria. The diagnosis of placental abruption should be based on clinical criteria, since examination of the placenta for conformational purposes is not sensitive [7].The current study showed that the most common indication for clinical diagnosis of PA was retro placental clot or bleeding . Retro placental clot was the indicator most consistent with a positive diagnosis of PA upon histological examination , followed by, vaginal bleeding +non reassuring fetal status, U/S diagnosis & lastly by vaginal bleeding uterine hyper tonicity .

U/S diagnosis was present in(14%) from the clinical diagnosis of PA in the current study ,a finding which is consistent with the following investigators Glantz et al in 2002[8] & Tikkanen et al in1987 [9] studies ; 17 (11%) of 149 had sonographic evidence of abruption in Glantz et al study[8] & Retro placental clot in 15% of 75 of cases with abruption by Tikkanen et al study [9] . U/S demonstration of hematoma is clinically important since these pregnancies may have a worse prognosis than placental abruptions that do not show a hematoma .Fetal demise and premature labor have been correlated with the size of the hematoma in several clinical studies[9]. During the histological examination of the placenta, cord, & membrane, chorioamnionitis was the most significant acute histological finding between histologically confirmed & unconfirmed PA pathology. This is agreed with Nathen et al in 2006[6] who stated that Severe histological chorioamnionitis is associated with abruption in both preterm and term gestations, implicating inflammation as a potential contributor causal pathway . The result of Darby et al in 1989[10] showed that histological chorioamnionitis and funisitis were present significantly more often in patients with abruption than in control patients (41% versus 4%) a significant association exists between preterm placental abruption and histological chorioamnionitis . Lockwood et al in 2005[11] explained this as neutrophillus are a rich source of proteases that can degrade extracellular matrix.

All placental infarction cases were histologically confirmed as PA. Infarctions, when present, are likely to cause under perfusion to the utero placental bed [12], which, over an extended period may result in increased placental resistance. This in turn is likely to lead to destruction of blood vessels and extend to feto-maternal bleeding [13]. Katzman and Genest in 2001[14] stated that maternal floor infarction is a poorly understood placental lesion reportedly associated with intrauterine growth restriction (IUGR) and tend to recur [14].The chronic lesions significantly involved in the pathophysiologic process of abruption in the current study included; chronic deciduitis, decidual vasculopathy, inter villous thrombus & villitis . Ananth et al in 2006[12] stated that increased risk associated with placental lesions, especially chronic inflammatory lesions even in the absence of early vaginal bleeding, suggests that prolonged inflammation may be implicated in placental abruption[15] . Ananth in 2006[6] stated that among women with placental abruption, conditions associated with acute inflammation are more prevalent at preterm than term gestations, whereas chronic processes are present throughout gestation [6].

The relation between chronic inflammatory lesions & PA was weak in the current study, this may be explained by the small sample size & the short duration. While acute inflammatory lesions were significantly associated with PA between cases & control odd ratio C/I was 12.36(2.68-57.1).This may be explained by the higher percentage of preterm labor 58% of the sample size in comparison to term labor which was 34%.

CONCLUSION:

The relation between the clinical & histological diagnosis of placental abruption remains weak. Clinical diagnosis for abruption should include one or more of the followings: retroplacental bleeding or clot(s), sonographic visualization of abruption, or painful vaginal bleeding accompanied by non reassuring fetal status or uterine hypertonicity. Acute and chronic histological lesions were observed more frequently in placentas of pregnancies complicated by placental abruption than in control cases .This may support the hypothesis that PA is the result of an acute event or a chronic inflammation & vascular dysfunction.

REFERENCES:

1. Arias F. Bleeding during pregnancy . Practical Guide to High –Risk pregnancy & delivery3rd edition New Delhi . Elsevier; 2008;13:299-328.
2. Kay H H, Placenta previa and Abruption . Scott J. R., Gibbs R. Eds . Danforth Obstetrics& gynecology. Lippincott Williams & Wilkins ,2003;20:365-79.
3. Baker P N. Disorder of placentation . Obstetrics by ten Teacher18th edition. London . Edward Arnold; 2006;13:168-70.
4. Konje JC, Taylor D J. Bleeding In late Pregnancy . James. D.K, Steer P. J Weiner. CP, Gonik B. Eds. High Risk Pregnancy Management 3rd edition . Elsevier; 2006;59:1266-71.
5. Carl A. Nath, Cande V,Ananth,John C, Smulian et al. Histological evidence of inflammation and risk of placental abruption Am J Obstet Gynecol 2007;197:319.
6. Cande V. Ananth, Darios Getahun, Morgan R. Peltier et al. Placental Abruption in Term and Preterm Gestations. The American College of Obstetricians and Gynecologists 2006;107:785-92.
7. Denise A, Elsasser, Cande V, Ananth,Vinay Prasad et al. Diagnosis of placental abruption, Relationship Between Clinical and Histopathological Findings .European Journal of Obstetrics & Gynecology and Reproductive Biology 2009;148:125–30
8. Chris Glantz, Leslie Purnell, Clinical Utility of Sonography in the Diagnosis and Treatment of Placental Abruption. J Ultrasound Med 2002; 21:837-840 ,4278-97.
9. David A. Nyberg, Dale R. Cyr, Laurence A Mack et al. Sonographic Spectrum of Placental Abruption AJR 1987;148:161-64.
10. Darby MJ, Caritis SN , Shen-Schwarz S. et al. Placental abruption in the preterm gestation: an association with chorioamnionitis. Obstet Gynecol 1989;74:88-92.
11. Charles J. Lockwood, Paolo Toti , Michael Paidas et al . Mechanisms of Abruption-Induced Premature Rupture of the Fetal Membranes. American Journal of Pathology 2005;167:1443-49.
12. Salafia CM, López-Zeno JA,Shere DM et al . Histologic evidence of old intrauterine bleeding is more frequent in prematurity. Am J Obstet Gynecol 1995;173:1065-70.
13. Salafia CM. Placental pathology of fetal growth restriction. Clinical Obstetrics& Gynecology 1997;40:740-49.
14. Piliph J. Katzman, David R. Genest. Maternal Floor Infarction and Massive Perivillous Fibrin Deposition: Histological Definitions, Association with Intrauterine Fetal Growth Restriction, and Risk of Recurrence . Pediatric And Developmental Pathology 2001;5:159-64.
15. Ananth CV, Oyelese Y, Prasad V et al. Evidence of placental abruption as a chronic process: associations with vaginal bleeding early in pregnancy and placental lesions . Eur J Obstet Gynecol Reprod Biol 2006;128:15-21.