—Report on Experiments and Clinical Cases—

Clinical Significance of Singleton Pregnancies Complicated by Placental Abruption Associated with Histological Chorioamnionitis

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Abstract

The aim of this study was to determine the perinatal outcomes of placental abruption associated with the presence of histological chorioamnionitis. We reviewed the obstetric records of 96 singleton deliveries complicated by placental abruption after 22 weeks' gestation. Of these 96 cases, 37 cases (39%) were diagnosed as having histological chorioamnionitis in the placenta. The incidence of premature delivery, preterm rupture of the membranes and low birth weight in the cases of placental abruption with chorioamnionitis were higher than in cases without chorioamnionitis. However, there were no significant differences in the incidence of other outcomes, such as fetal demise, low Apgar score, or low umbilical artery pH, between the cases of placental abruption with and without histological chorioamnionitis. Although the incidence of prematurity in the cases of placental abruption with chorioamnionitis was higher than that in cases without chorioamnionitis, there were no significant differences in fetal and neonatal conditions between the abruption cases with and without chorioamnionitis.
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Key words: chorioamnionitis, placental abruption, perinatal outcome

Introduction

Placental abruption or premature separation of the normally implanted placenta is a serious and life-threatening obstetric complication for both mother and fetus. Although the cause of placental abruption remains unclear, the presence of inflammation and infection has been suggested to be the primary cause of placental abruption[1–6]. For example, Nath et al.[5] observed that histologic chorioamnionitis was present in 30.8% of abruption cases in preterm singleton pregnancies and in 34.6% of cases at term singleton pregnancies. Recent evidence has linked neutrophil infiltration into the decidua with preterm placental abruption[5,6]. On the other hand, chorioamnionitis has also long been known to be an independent risk factor for maternal and neonatal morbidity and mortality associated with intrauterine infection leading to premature delivery or neonatal sepsis or both[7,8]. Thus, the perinatal outcomes in cases of placental abruption with chorioamnionitis may be worse than those in cases without chorioamnionitis. To date, however, the perinatal outcomes of placental abruption associated with the presence of chorioamnionitis have not been well

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examined. Therefore, the aim of this study was to determine the perinatal outcomes of placental abruption associated with the presence of histological chorioamnionitis.

Patients and Methods

The protocol for this study was approved by the Ethics Committee of the Japanese Red Cross Katsushika Maternity Hospital. In addition, informed consent concerning analysis from a retrospective database was obtained from all subjects.

We reviewed the obstetric records of 96 singleton deliveries complicated by placental abruption, defined as complete or partial separation of a normally implanted placenta by evidence of retroplacental bleeding after 22 weeks' gestation at Japanese Red Cross Katsushika Maternity Hospital from 2003 through 2009. In this study, we examined as perinatal complications and outcomes the incidence of premature delivery, low neonatal birth weight, hypertensive disorders, such as gestational hypertension and preeclampsia, premature rupture of the membranes (PROM), Cesarean delivery, disseminated intravascular coagulation (DIC), nonreassuring fetal status (NRFS), fetal demise, Apgar score <4 at 1 and 5 minutes and umbilical artery pH <7.

Microscopic histological analysis of the placentas was performed for the diagnosis of chorioamnionitis after delivery according to Blanc's criteria[9]. The severity of chorioamnionitis, i.e., inflammation of the placental surface, was determined via the degree of maternal polymorphonuclear lymphocyte infiltration into either the subchorionic space (intervillositis: stage I), the intervillous space (chorionitis: stage II) or the amniotic cavity (chorioamnionitis in a narrow sense: stage III).

Data are presented as number (%) or mean ± SD. For statistical analysis, the χ^2 test with Yates' correction was used for categorical variables, whereas Student's *t*-test was used for continuous variables. Odds ratios (ORs) and 95% confidence intervals (CIs) were also calculated. Differences with $p<0.05$ were considered significant.

Results

Of the 96 cases of placental abruption, 37 cases (39%) were diagnosed as having histological chorioamnionitis in the placenta. Of the 37 cases of placental abruption with chorioamnionitis, 12 (32%), 16 (44%), and 9 cases (24%) were diagnosed as chorioamnionitis stage I, stage II and stage III, respectively.

The incidence of placental abruption stage ≥II based on a report by Page et al.[10] was 16% (6 of 37 cases; 5 cases of stage II and 1 case of stage III) in the cases with histological chorioamnionitis, which was not significantly different from that in cases without histological chorioamnionitis ($p=0.32$; 27%; 16 of 59 cases; 14 cases of stage II and 2 cases of stage III).

Table 1 shows the perinatal outcomes of the patients who had placental abruption with and without chorioamnionitis. The mean gestational age at delivery and the neonatal birth weight in patients with chorioamnionitis were significantly lower than those in patients without chorioamnionitis. In addition, the incidence of premature delivery (OR: 3.0, 95% CI: 1.2–7.5, $p=0.03$), PROM (OR: 3.6, 95% CI: 1.4–8.9, $p<0.01$), and low neonatal birth weight (OR: 4.5, 95% CI: 1.6–13, $p<0.01$) in patients with chorioamnionitis were significantly higher than those in patients without chorioamnionitis. However, there were no significant differences in the incidence of other perinatal outcomes, such as the incidence of fetal demise, low Apgar score, or low umbilical artery pH, between the 2 groups.

On the basis of the obstetric charts, it was estimated that the rates of placental abruption at <37 weeks' gestation occurred in the hospital were 47% in cases without chorioamnionitis (14 of 30 cases) and 75% in cases with chorioamnionitis ($p=0.05$; 21 of 28 cases).

Table 2 shows the perinatal outcomes of pregnancies complicated by placental abruption associated with the stage of chorioamnionitis. There were no significant differences in these variables in the 3 stages of chorioamnionitis.

Table 1 Perinatal complications and outcomes of pregnancies complicated by placental abruption with or without histological chorioamnionitis

Chorioamnionitis	(−)	(+)	*P* value*
N	59	37	
Maternal age (years)	31.9 ± 4.7	32.2 ± 4.8	0.77
Parity	0.6 ± 0.9	0.7 ± 1.0	0.62
Gestational age at delivery			
Mean (weeks)	36.1 ± 3.5	34.2 ± 3.7	0.02
<32 weeks	6 (10)	7 (19)	0.36
<37 weeks	30 (51)	28 (76)	0.03
Hypertensive disorders	4 (6.8)	4 (11)	0.75
PROM	7 (12)	14 (38)	<0.01
Cesarean delivery	37 (63)	23 (62)	0.87
DIC	5 (8.5)	0 (0)	0.17
Neonatal birth weight			
Average (g)	2,426 ± 684	2,128 ± 698	0.04
<1,500 g	7 (12)	5 (14)	0.94
<2,500 g	27 (49)	28 (76)	<0.01
Fetal demise	5 (8.5)	4 (11)	0.98
Surviving neonates			
n	54	33	
NRFS	44 (82)	25 (76)	0.43
Apgar 1' <4	11 (20)	6 (18)	0.98
Apgar 5' <4	3 (5.6)	2 (6.0)	0.69
Umbilical artery pH<7	5 (9.3)	3 (9.0)	0.75

Values are expressed as n (%) or mean ± SD.
*P values by Student's t-test or χ^2 test with Yates' correction.
PROM, premature rupture of the membranes.
DIC, disseminated intravascular coagulation.
NRFS, non-reassuring fetal status.

Table 2 Perinatal outcomes of pregnancies complicated by placental abruption associated with the stage of histological chorioamnionitis

Chorioamnionitis	Stage I	Stage II	Stage III
N	12	16	9
Geatstional age at delivery			
<32 weeks	0 (0)	4 (25)	3 (33)
<37 weeks	7 (58)	14 (88)	6 (67)
PROM	4 (33)	6 (38)	4 (44)
Neonatal birth weight			
<1,500 g	0 (0)	3 (19)	1 (11)
<2,500 g	9 (75)	12 (75)	6 (67)
Fetal demise	1 (8.3)	2 (13)	1 (11)
Surviving neonates			
n	11	14	8
Apgar 1' <4	1 (9.1)	4 (29)	1 (12)
Apgar 5' <4	0 (0)	2 (14)	0 (0)
Umbilical artery pH<7	1 (9.1)	2 (14)	0 (0)

Values are expressed as n (%).
PROM, premature rupture of the membranes.

Discussion

The major findings of the present study were: (1) the incidence of premature delivery, PROM, and low neonatal birth weight in cases of placental abruption with chorioamnionitis were higher than those in cases without chorioamnionitis, and (2) there were no significant differences in the incidence of other outcomes, such as fetal demise, low Apgar score, or low umbilical artery pH, between the cases of placental abruption with and without histological chorioamnionitis.

In this study, histological chorioamnionitis was present in 39% of abruption cases. This rate was similar to rates reported previously[5,11]. In the present study, unfortunately, we did not examine the incidence of chorioamnionitis in cases without placental abruption; however, some previous studies suggest that the incidence of histological chorioamnionitis in abruption cases is higher than that in normal pregnancies[5,11,12]. In a study by Nath et al.[5], for example, histological chorioamnionitis was present in 12.5% of preterm normal pregnancies and in 20.4% of term normal pregnancies. Thus, the present results also provide further support for the association between histological chorioamnionitis and placental abruption reported previously[1,3–5].

Histological chorioamnionitis, defined as inflammation of the extraplacental membrane, has been consistently linked with prematurity, low neonatal birth weight, and PROM[12]. The relationship between histological chorioamnionitis and infection (positive culture) of the chorioamnion has been reported to be strongest among preterm deliveries and to be less strong in term placentas[13,14]. The present results may support these previous reports[12–14] and suggest the increased risks of prematurity associated with the presence of chorioamnionitis even in cases of placental abruption.

In this study, although the incidence of prematurity in the cases of placental abruption with chorioamnionitis was higher than in cases without chorioamnionitis, there were no significant differences in the other perinatal outcomes, such as the incidence of fetal demise, low Apgar score, or low umbilical artery pH, between the 2 groups. Although mothers with histological chorioamnionitis usually have no clinical evidence of infection, their infants have been reported to have increased rates of sepsis and death[7,8]. Thus, the present results may conflict with previous reports[7,8]. One reason for the present results may be the small sample size. In this study, for example, there were only 9 cases of chorioamnionitis stage III. Another reason may be differences in perinatal management before the onset of placental abruption. The timing of the start of perinatal management for cases with chorioamnionitis might be earlier than that for cases without chorioamnionitis, because chorioamnionitis is associated with an increased incidence of preterm labor or preterm PROM or both before the onset of placental abruption. In this study, the rate of preterm placental abruption in the cases with chorioamnionitis seemed to be higher than that in the cases without chorioamnionitis (p=0.05). In cases with chorioamnionitis, the prematurely delivered placentas often have an acute marginal hemorrhage that undermines the edge of the placenta and originates from deciduitis[6]. This hemorrhagic process can cause premature labor or preterm PROM or both and has been reported to differ from the typical placental abruption due to other causes, such as preeclampsia[2]. Vintzileos et al.[3] have suggested that true placental abruption following the presence of chorioamnionitis usually occurs after PROM. Thus, the earlier admission for perinatal management in the cases with chorioamnionitis may have prevented the adverse outcome of placental abruption compared with the cases without chorioamnionitis. In addition, the influence of placental abruption on fetal and neonatal condition may also be larger than that of the presence of chorioamnionitis. In the present study, the severity of placental abruption was not changed irrespective of the presence of chorioamnionitis, and then the fetal and neonatal outcomes may not be attributed to the presence of chorioamnionitis.

In conclusion, although the incidence of prematurity in the cases of placental abruption with chorioamnionitis was higher than that in cases without chorioamnionitis, there were no significant

differences in fetal or neonatal condition between the abruption cases with and without chorioamnionitis.

References

1. Harris BA Jr, Gore H, Flowers CE Jr: Peripheral placental separation: a possible relationship to premature labor. Obstet Gynecol 1985; 66: 774–778.
2. Harris BA Jr: Peripheral placental separation: A review. Obstet Gynecol Surv 1988; 43: 577–581.
3. Vintzileous AM, Campbell WA, Nochimson DJ, Weinbaum PJ: Preterm premature rupture of the membranes: A risk factor for the development of abruptio placentae. Am J Obstet Gynecol 1987; 156: 1235–1238.
4. Ananth CV, Getahun D, Peltier MR, Smulian JC: Plaental abruption in term and preterm gestations. Obstet Gynecol 2006; 107: 785–792.
5. Nath CA, Ananth CV, Smulian JC, Shen-Schwarz S, Kaminsky L: Histologic evidence of inflammation and risk of placental abruption. Am J Obstet Gynecol 2007; 197: 319.e1–319.e6.
6. Benirschke K, Kaufmann P, Baergen RN: Chorioamnionitis. In Pathology of the Human Placenta (Benirschke K, Kaufmann P, Baergen RN, eds), 5th ed. 2006; pp 657–694, Springer, New York.
7. Russel P: Inflammatory lesions of the human placenta. I. Clinical significance of acute chorioamnionitis. Am J Diagn Gynecol Obstet 1979; 1: 127–137.
8. Bracci R, Buonocore G: Chorioamnionitis: A risk factor for fetal and neonatal morbidity. Biol Neonate 2003; 83: 85–96.
9. Blanc WA: Amniotic infection syndrome: pathogenesis morphology, and significance in circumnatal mortality. Clin Obstet Gynecol 1959; 2: 705–734.
10. Page EW, King EB, Merill JA: Abruptio placentae: Danger of delivery. Obstet Gynecol 1954; 3: 385–393.
11. Rana A, Sawhney H, Gopalan S, Penigrahi D, Nijhawan R: Abruptio placentae and chorioamnionitis-microbial and histological correlation. Acta Obstet Gynecol Scand 1999; 78: 363–366.
12. Gibbs RS, Romero R, Hiller SL, Eschenbach DA, Sweet RL: A review of premature birth and subclinical infection. Am J Obstet Gynecol 1992; 166: 1515–1528.
13. Hillier SL, Martius J, Krohn M, Kiviat N, Holmes KK, Eschenbach DA: A case-control study of chorioamnionic infection and histologic chorioamnionitis in prematurity. N Engl J Med 1988; 319: 912–918.
14. Dong Y, St Clair PJ, Ramzy I, Kagen-Hallet KS, Gibbs RS: A microbiologic and clinical study of placental inflammation at term. Obstet Gynecol 1987; 70: 175–182.

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