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Association of Placental Fetal and Maternal Vascular Malperfusion with Bronchopulmonary Dysplasia and Mortality in Preterm Infants

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Abstract:

Objective: This study aimed to investigate the association of placental histopathological abnormalities with bronchopulmonary dysplasia and mortality in preterm infants.

Methods: This prospective study included preterm infants ≤32 weeks and ≤1500 g who were followed in the NICU of a tertiary university hospital between May 2019 and May 2020. The infants were separated into 2 groups as those with and without bronchopulmonary dysplasia. Placentas were evaluated according to the Amsterdam Placenta Study Group.

Results: In this study, 35 preterm infants were evaluated. Birthweight was determined to be statistically significantly lower in Group 1 (1045±160.63 g) than in Group 2 (1453.95±529.08 g) (P=0.002). According to the median (min-max) gestational week at birth, the age of Group 1 [27.5 (26-29)] was statistically significantly lower than that of Group 2 [30.5 (25-32)] (P=0.001). Nosocomial sepsis was determined at a statistically higher rate in Group 1 [58.3%, n=7) than in Group 2 (13%, n=3) (P=0.015). The median length of stay in hospital was significantly longer in Group 1 [51 days (38-92)] than in Group 2 [21 days (1-74)] (P=0.001). The number of days on non-invasive ventilation was statistically significantly greater in Group 1 [11(2-57)] than in Group 2 [2(0-43)] (P=0.001). Maternal-fetal vascular malperfusion was found to be higher in Group 2. Mortality was observed in Group 2 and not in Group 1.

Conclusion: No statistically significant difference was observed in placental histopathological findings between the groups, with maternal and fetal vascular malperfusion representing the most frequent lesions. Mortality was seen exclusively in the non-BPD group. These findings suggest a potential association between placental vascular pathologies and neonatal mortality; however, larger multicenter prospective studies are required to confirm this relationship.

Keywords: Preterm, Bronchopulmonary Dysplasia, Placental Histopathological Abnormalities, Mortality

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Bronchopulmonary dysplasia (BPD) is a chronic pulmonary disease, which emerges as a result of oxygen toxicity and barotrauma caused by mechanical ventilation in the early period of life. The risk of BPD increases as birthweight and gestational age decrease. Despite advances in care and treatment, BPD remains a serious problem for preterm infants [1]. Preterm birth, low birthweight, the widespread use of reproductive techniques, chorioamnionitis, intubation in the delivery room, surfactant insufficiency (respiratory distress syndrome [RDS]) and maternal factors (smoking during pregnancy, hypertension (HT), and intrauterine growth restriction) have been associated with BPD [1, 2].

In addition, the placenta can be a marker of fetal growth. Chronic inflammatory lesions of the placenta are characterized by infiltration of the organ by lymphocytes, plasma cells, and/or macrophages, and may originate from infections (viral, bacterial, parasitic) or may be of immune origin. There are 3 main lesions in the placenta; villitis, chronic chorioamnionitis, and chronic deciduitis. Villitis of unknown etiology (VUE) is a destructive villous inflammatory lesion, characterized by infiltration of maternal T-cells (CD8+ cytotoxic T-cells) to the chorionic villi. Villitis has been reported to be associated with preterm and term fetal growth restriction, pre-eclampsia, fetal death, and premature birth. Infants with VUE in the placenta are at risk in terms of abnormal neurodevelopmental outcomes and mortality by the age of 2 years [3].

If there are findings of vasculopathy and clotting in the placenta, the risk of necrotizing enterocolitis is increased. Cases that are positive for lesions related to clotting have been determined to be correlated with necrotizing enterocolitis [4]. Moreover, the body mass index of the mother has an effect on the newborn weight. Smoking during pregnancy has been associated with an increase in placenta weight and a decrease in infant weight. In mothers with findings of acute inflammation in the placenta, extended membrane rupture has been determined in 56%, preterm labor in 38%, and pregnancy-induced hypertension in 6% [5, 6].

Examination of the placenta in preterm births is important for the determination of the etiology of preterm birth and mortality in infants [3]. Placental lesions (infarctus, chorionic plaque thrombi, and basal perivillous fibrin) reflecting fetal-placental blood flow disorders are seen mostly in the gestational age category of 28-33 weeks [7]. The histopathological findings of placenta obtained from preterm births provide important data in the determination of preterm birth etiology and infant outcomes. It has been determined that the infants of mothers with chorioamnionitis are born preterm, remain longer in hospital, and have a greater requirement for respiratory support [8].

It was thought that the data determined in this study would contribute to decreasing morbidity and mortality rates in preterm infants. The aim of the study was to investigate the relationship between the histopathological findings of the placenta and bronchopulmonary dysplasia in preterm infants.

METHODS

This prospective study included preterm infants ≤32 weeks and ≤1500 g who were followed up in the Neonatal Intensive Care Unit (NICU) of a tertiary-level University hospital between May 2019 and May 2020. Infants were excluded from the study if they had any congenital anomaly, metabolic disease, genetic disease, or syndrome, if the placenta had not been examined, or if they were >32 weeks gestational age and >1500 g weight at birth.

A total of 35 infants were included in the study, as 12 in Group 1 with BPD, and 23 in Group 2 without BPD. All the cases were followed up in NICU. Evaluations were made in respect of birthweight, gestational age at birth, gender, type of birth, APGAR scores, antenatal steroids or MgSO4 administration, cord clamping/cord milking, retinopathy of prematurity (ROP), patent ductus arteriosus (PDA), necrotizing enterocolitis (NEC), resuscitation, infection, number of mechanical ventilations, number of days of non-invasive ventilation, laboratory values, and mortality. A record was also made of maternal age, weight, body mass index (BMI), comorbidities, and placental histopathology results.

All the placentas of the cases in this study were evaluated by a single pathologist according to the international morphological classification designed by the Amsterdam Placenta Working Group (2014) [9, 10]. The placentas were analyzed in 4 main

groups of maternal/trophoblastic (maldevelopment, malperfusion, loss of integrity), fetal stromal-vascular (maldevelopment, malperfusion, loss of integrity), inflammatory (infectious, idiopathic) and other (malformations, distortions, heterotopias, genetic).

This study was conducted in accordance with the Declaration of Helsinki and the principles of Good Clinical Practice and was approved by the Local Ethics Committee of Sivas Cumhuriyet University (Date: 22.05.2019; Decision No: 2019-05/41).

Statistical Analysis

Data obtained in the study were analyzed statistically using SPSS vn. 22 software (IBM, Armonk, NY, USA). Descriptive statistics were stated as arithmetic mean ±standard deviation, or median, minimum, and maximum values for continuous data and as number (n) and percentage (%) for categorical values. Conformity of the data to normal distribution was assessed with the Shapiro-Wilk test. The Independent Samples t-test, the Mann-Whitney U-test, and Fisher's Exact Chi-square test were applied in the analyses. A value of $P<0.05$ was accepted as the level of statistical significance in all tests.

RESULTS

Thirty-five preterm infants were evaluated, of whom 12 (34.2%) developed BPD. The mean birthweight of 1045±160.63 gr in Group 1 with BPD was determined to be statistically significantly lower than the mean 1453.95±529.08 g in Group 2 without BPD (P=0.002). According to the median (min-max) gestational week at birth, the age of Group 1 [27.5 (26-29)] was statistically significantly lower than that of Group 2 [30.5 (25-32)] (P=0.001). In the whole patient group, the infant was delivered by cesarean section in 33 (94.28%) cases and by the vaginal route in 2 (5.72%). No difference was determined in APGAR scores, administration of antenatal steroids or magnesium

TABLE 1. The Demographic Characteristics of the Group 1 and Group 2 Cases

	Group 1 (n=12)	Group 2 (n=23)	P-value
Birth weight (g)	1045.25±160.63	1453.95±529.08	**0.002**
Gestational age (weeks)	27.50 (26-29)	30.50 (25-32)	**0.001**
Gender, female	7 (58.3)	7 (30.4)	0.153
Mode of birth, C/S	11 (91.6)	22 (95.6)	0.575
APGAR 1-min score	5 (4-8)	5 (0-8)	0,400
APGAR 5-min score	7 (6-9)	7 (0-9)	0,278
Antenatal steroid	12 (100)	19 (82.6)	0.275
Magnesium sulphate	10 (83.3)	16 (69.5)	0.450
Cord milking/ Delayed cord clamping	10 (83.3)	21 (91.3)	0.594
Maternal age (years)	27.58±3.89	30.52±4.83	0.078
Maternal weight (kg)	76.41±13.31	74.21±15.49	0.679
Maternal height (cm)	160.66±4.24	162.08±5.72	0.456
BMI (kg/m^2)	29.61±5.31	28.40±5.70	0.548
Obesity	7 (58.3)	8 (53.3)	0.329
Pre-eclampsia	3 (25.0)	6 (26.0)	0.639
Chorioamnionitis	2 (16.6)	2 (8.6)	0.594

Data are shown as mean±standard deviation or median (minimum-maximum) or n (%) where appropriate. BMI, body mass index; C/S, caesarean section; APGAR, appearance, pulse, grimace, activity, respiration.

Statistically significant P-values are shown in bold.

TABLE 2. Evaluation of Some Clinical and Laboratory Parameters of the Cases

	Group 1 (n=12)	Group 2 (n=23)	P-value
ROP	2 (16.6)	0 (0.00)	0.111
PDA	2 (16.6)	0 (0.00)	0.111
NEC	0 (0.00)	2 (8.6)	0.536
Resuscitation	1 (8.3)	7 (30.4)	0.216
Nosocomial sepsis	7 (58.3)	3 (13.0)	**0.015**
Total days receiving oxygen	36.50±11.46	9.21±7.27	**0.001**
Number of days in hospital	51(38-92)	21 (1-74)	**0.001**
Number of days of non-invasive ventilation	11 (2-57)	2 (0-43)	**0.001**
Number of days of mechanical ventilation	3 (0-20)	1 (0-36)	0.381
Number of days to full oral feeding	12 (6-43)	10 (0-29)	0.123
Leukocyte count	5910 (2720-15560)	7110 (3940-33540)	0.161
CRP (mg/dL)	2.12 (1-10.50)	1.58 (1-13)	0.327
Neutrophil (count/mm^3)	2950 (480-11580)	3000 (570-7600)	0.771
Neutrophil/lymphocyte ratio	0.93 (0.21-1.68)	1.24 (0.17-7.60)	0.184
Mortality	0 (0.0)	5 (21.7)	0.141

Data are shown as mean±standard deviation or median (minimum-maximum) or n (%) where appropriate. ROP, retinopathy of prematurity; PDA, patent ductus arteriosus; NEC, necrotising enterocolitis; CRP, c-reactive protein.
Statistically significant P-values are shown in bold.

sulfate, cord milking/delayed cord clamping, or maternal characteristics (age, height, weight, BMI, obesity, pre-eclampsia, chorioamnionitis) in respect of the development of BPD (Table 1).

Nosocomial sepsis was determined at a statistically higher rate in Group 1 (70%, n=7) than in Group 2 (30%, n=3) (P=0.015). The median length of stay in hospital was significantly longer in Group 1 [51 days (38-92)] than in Group 2 [21 days (1-74)] (P=0.001). The median number of days on non-invasive ventilation was statistically significantly greater in Group 1 [11(2-57)] than in Group 2 [2 (0-43)] (P=0.001). No statistically significant difference was determined between the two groups in respect of ROP, PDA, NEC, resuscitation, time to full oral feeding, white blood cell (WBC) count, C-reactive protein (CRP), neutrophil count, neutrophil-lymphocyte ratio, and survival (P>0.05) (Table 2).

Table 3 compares the placental histopathological features of cases in Group 1 (n=12) and Group 2 (n=23). Regarding maternal/trophoblastic findings, maldevelopment was observed in 33.3% of Group 1 and 34.7% of Group 2, with no statistically significant difference between the groups (P=0.618). Malperfusion rates were 58.3% and 65.2%, respectively, and were statistically comparable (P=0.726). Loss of tissue integrity was not observed in Group 1, whereas it was detected in 17.3% of Group 2; however, this difference did not reach statistical significance (P=0.275). When fetal stromal-vascular findings were evaluated, maldevelopment was present in 25.0% of Group 1 and 39.1% of Group 2 (P=0.476), while malperfusion was identified in 41.6% and 60.8% of cases, respectively (P=0.279). Loss of tissue integrity was observed in 16.6% of Group 1 and 43.4% of Group 2, with no statistically significant difference between the groups (P=0.149). With respect to inflammatory findings, infectious inflammation was observed in 50.0% of Group 1 and 21.7% of Group 2; however, this difference was not statistically significant (P=0.130). Idiopathic inflammation was detected only in Group 2 at a rate of 8.6%, with no significant difference between the groups (P=0.536). Other findings (including

TABLE 3. Evaluations of the Placental Histopathological Characteristics of Group 1 and Group 2 Cases

		Group 1 (n=12)	Group 2 (n=23)	P-value
Maternal/trophoblastic, n (%)	Maldevelopment	4 (33.3)	8 (34.7)	0.618
	Malperfusion	7 (58.3)	15 (65.2)	0.726
	Loss of integrity	0 (0.00)	4 (17.3)	0.275
Fetal stromal-vascular, n (%)	Maldevelopment	3 (25.0)	9 (39.1)	0.476
	Malperfusion	5 (41.6)	14 (60.8)	0.279
	Loss of integrity	2 (16.6)	10 (43.4)	0.149
Inflammatory, n (%)	Infectious	6 (50.0)	5 (21.7)	0.130
	Idiopathic	0 (0.00)	2 (8.6)	0.536
Other, n (%)	Malformations, disruptions, heterotopias, genetics	3 (25.0)	6 (26.08)	0.639

malformations, disruptions, heterotopias, and genetic anomalies) were observed in 25.0% of Group 1 and 26.08% of Group 2, with no statistically significant difference between the groups (P=0.639) (Table 3).

DISCUSSION

In this study, comparisons were made of the demographic and clinical characteristics and the placental histopathology of newborn infants in Group 1 and Group 2, and contributory factors were examined.

The importance of gestational age and birthweight in the development of BPD has been well documented in the literature. Bancalari and Claure [11] emphasized that birthweight <1500 g significantly increased the risk of BPD in newborns. Similarly, Walsh *et al*. [12] reported that the risk of BPD was significantly increased with a decrease in gestational age. The current study results support these opinions. The mean birthweight (1045.25±160.63 gr and gestational age (27.5 weeks) of the infants in Group 1 were significantly lower than those of Group 2 (P=0.002, P=0.001, respectively). Consistent with the literature, these findings demonstrate that a low birthweight and early birth week play a critical role in the development of BPD.

No statistically significant difference was determined between the groups with and without BPD with respect to the histopathological findings. However, when all the cases were evaluated, maternal and fetal vascular malperfusion was seen to be the most common histopathological finding. In a study conducted in the Chicago Prentice Women's Hospital between 2005 and 2012, pulmonary hypertension was determined due to BPD associated with maternal vascular malperfusion [13]. As the number of patients in the current study was limited, this may not have been determined statistically.

The most commonly seen placental pathological lesion in preterm births has been determined to be severe chorioamnionitis. However, after the 28th week of pregnancy, a change has been seen in placental pathological lesions, and maternal vascular malperfusion lesions have been found to be predominant in all preterm births [14]. In the current study, although maternal vascular malperfusion was not found to be statistically significant, it was present in most cases. This may have been caused by the premature birth. Nijman *et al*. observed that maternal vascular malperfusion was seen most in both spontaneous and induced multiple and mid/late preterm births. The main finding in spontaneous very preterm births was chorioamnionitis [15]. In the current study, BPD was determined in infants small for gestational age, and maternal and fetal vascular malperfusion in infants large for gestational age, consistent with the literature.

Maternal vascular malperfusion has been associated with conditions such as pre-eclampsia,

hypercoagulability conditions, lupus anticoagulant, and sometimes, diabetes [16]. New information related to the basic mechanisms regulating uteroplacental blood flow and the effect on placental health of placental malperfusion has emphasized the importance of the development of diagnostic tests in the early stages of pregnancy for the health of both mother and infant [17].

Fetal vascular malperfusion is characterised by histological changes such as thrombosis, avascular villus, karyorrhexis, and vascular obstruction. This has been associated with various etiologies such as abnormal cord insertion, pre-eclampsia, hypercoagulability, lupus anticoagulant, diabetes, and intrauterine infections. Severe outcomes, including intrauterine growth restriction, poor perinatal outcomes, fetal death, and neurodevelopmental disorders, can result from fetal vascular malperfusion [16]. The current study results showed that while fetal vascular malperfusion was seen less in the BPD group, it was determined in 14 (60.8%) cases in Group 2, and 5 (21.7%) cases in Group 2 developed mortality, but no mortality was observed in Group 1. This suggested that fetal vascular malperfusion can increase mortality during follow-up. However, clinicopathological diagnosis may be helpful in the better management of these cases and resolving subsequent health problems.

In a study by Romero *et al*. [18], placenta samples obtained from 944 women with term births without any complications were examined, and maternal and fetal vascular lesions of malperfusion were determined at the rates of 35.7% and 19.7%, respectively. In the current study, maternal and fetal vascular malperfusion lesions were determined in 15 (65.2%) and 14 (60.8%) cases, respectively, in Group 2.

In another study, vascular malperfusion lesions and chronic inflammation forms were seen at a significantly higher rate in placentas complicated with fetal congenital heart disease, and it was stated that this could contribute to reduced head circumference at birth. It was also emphasized that there is a need for further studies of neuroplacentology to investigate the links between heart defects, placental vascular malperfusion lesions, and fetal brain growth [19]. This shows how valuable the data from our study are and that serious prospective and multidisciplinary studies are needed in the future.

Strengths and Limitations

The main limitation of this study is the small sample size, which limited the statistical power and may have prevented some associations from reaching statistical significance. In addition, as this was a single-center study, the generalizability of the findings is limited.

The prospective study design, evaluation of all placentas by a single pathologist, use of the current and standardized Amsterdam placental classification, integration of clinical and pathological data, and transparent reporting of negative results constitute the main methodological strengths of this study.

CONCLUSION

This study suggests that placental vascular pathologies may have a potential impact on neonatal mortality and morbidity in preterm infants. In particular, maternal and fetal vascular malperfusion findings are thought to be associated with clinical outcomes. However, due to the limited sample size and single-center design, larger-scale, multicenter, prospective studies are needed to further clarify this relationship.

Ethics Approval and Consent to Participate

This study was approved by the Sivas Cumhuriyet University Non-Interventional Clinical Research Ethics Committee (Decision No: 2019-05/41; date: 22.05.2019). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent was obtained from all individual participants (parents) included in the study.

Clinical Trial Registration

Not Available.

Data Availability

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Authors' Contribution

Study Conception: GT, HÖ, GSÜ, SK, EG; Study Design: GT, HÖ, SK; Supervision: GT, HÖ, GSÜ, SK, EG; Funding: HÖ, GSÜ, SK, EG; Materials: GT, HÖ, GSÜ, EG; Data Collection and/or Processing: GT, HÖ, GSÜ, EG; Statistical Analysis and/or Data Interpretation: GT, EG; Literature Review: GT, SK; Manuscript Preparation: GT, HÖ, GSÜ, SK, EG; and Critical Review: GT, HÖ, GSÜ, SK, EG.

Conflict of Interest

The author(s) disclosed no conflict of interest during the preparation or publication of this manuscript.

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The author(s) declare that no artificial intelligence-based tools or applications were used during the preparation process of this manuscript. The all content of the study was produced by the author(s) in accordance with scientific research methods and academic ethical principles.

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