

Placenta and Umbilical Cord Histopathologies in Pathologic Specimens and Relationship Between Pregnancy Outcomes

Patolojik Spesmenlerde Plasenta ve Umbilikal Kord Histopatolojileri ve Gebelik Sonuçları Arasındaki İlişki

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Öz

Amaç: Bu çalışmanın amacı plasenta ve göbek kord spesmenlerinin histopatolojik olarak incelemesi ve gebelik sonuçları arasındaki ilişkiyi saptamaktır.

Hastalar ve Yöntem: Uşak Üniversitesi Eğitim ve Araştırma Hastanesi Patoloji Ana Bilim Dalı arşivinden 112 plasenta materyali retrospektif olarak değerlendirildi. Term plasenta ölçümü, Kaufmann'ın Patolojisine göre; 37-40 hafta, boyut 18 cm, ağırlık 350-750 gr, kalınlık 2-2,5 cm olarak yapıldı. Göbek kordonunun damar sayısı, membran yapısı, koranjyom, infarkt, hematoma, hemoraji kaydedildi. Histopatolojik olarak infarkt, koryoamniyonit, subkoryonik fibrin birikimi, koryonik damarlarda konjesyon, subkoryonik hemoraji (SCH), koranjiozis, koranjyom, kalsifikasyon, perivillöz fibrin birikimi, mekonyum etkisi, fibrinoid nekroz, villit, koryonik damarın ektazisi saptandı. Cinsiyet, doğum şekli, maternal hastalık, doğum ağırlığı, anne yaşı, parite, doğum uzunluğu, baş çevresi ve 0-9 arasında APGAR skoru kaydedildi.

Bulgular: 112 plasenta spesmeni değerlendirildi; 29 plasental infarkt vakasının 4'ünde (% 13,7), 19 koranjiozis vakasının 2'sinde (% 10,5), 17 SCH vakasının 1'inde (% 5,8), 29 koryoamniyonit vakasının 2'sinde (% 6,8), 12 umbilikal kord konjesyonlu vakanın 1'inde (% 8,3) anormal gebelik sonuçları tespit edildi. İstatistiksel olarak anlamlı sonuçlarımız subkoryonik hemoraji ile plasenta boyutu ($p = 0,004$), maternal yaş ile umbilikal kord boyutu ($p = 0,001$) ve doğum ağırlığı ile plasenta ve umbilikal kord boyutları arasında saptandı ($p = 0,000$). Ancak bu parametreler ile gebelik sonuçları arasında bir ilişkiye rastlanmadı. Diyabet, erken doğum, ablatio plasenta ve ölü doğumla sonuçlanan ve seyreden gebeliklerde, bebek ağırlığı ile plasenta ve umbilikal kord boyutu arasında ilişki saptandı. Histopatolojik bulgular ve gebelik sonuçları arasında ise anlamlı bir ilişkiye rastlanmadı.

Sonuç: Çalışmamızdaki temel bulgularımızdan SCH'de görülebilecek abortus, preterm eylem ve intrauterin gelişim geriliği (IUGR) ile kısa kord boyutlarında ileriki yıllarda görülebilecek psikomotor patolojiler açısından düzenli gestasyonel USG takipleri ve iyi bir gebelik kanama anamnezinin bu patolojileri öngörebilme veya önleyebilme de etkin olacağı kanısındayız.

Anahtar Kelimeler: Plasenta, göbek kordonu, histopatoloji, gebelik sonuçları

Abstract

Aim: The objectives of this study are to investigate the placenta and umbilical cord specimens histopathologically, and to determine the relationship between pregnancy outcomes.

Patients and Methods: 112 placenta specimens obtained from the archive of Uşak University Education and Research Hospital of Pathology Department were retrospectively evaluated. The criteria of term placenta measurement were determined as 37-40 weeks, 18 cm size, 350-750g weight, 2-2.5 cm thickness in accordance with the Kaufmann's Pathology. Umbilical cord's number of vessels, membrane structure, chorioangioma, infarct, hematoma, and hemorrhage were recorded. Infarct, chorioamniotitis, sub-chorionic fibrin deposition, congestion of chorionic vessel, sub-chorionic hemorrhage (SCH), chorangioma, calcification, perivillous fibrin deposition, effect of meconium, fibrinoid necrosis, villitis, and ectasia of chorionic vessel were found in histopathological examinations. The gender, mode of delivery, maternal disease, birth weight, maternal age, parity, birth length, head circumference, and APGAR scores between 0-9 were recorded.

Results: 112 placental specimens were evaluated. As a result of evaluations, 4 of 29 (13.7%) placental infarct cases, 2 of 19 (10.5%) placental chorangioma cases, one of 17 (5.8%) SCH cases, 2 of 29 (6.8%) chorioamniotitis cases, and one of 12 (8.3%) umbilical cord congestion cases were found to have abnormal pregnancy outcomes. Statistically significant relationships were found between sub-chorionic hemorrhage and placenta size ($p = 0.004$), between maternal age and umbilical cord dimension ($p = 0.001$), and between birth weight and placenta and umbilical cord dimensions ($p = 0.000$). However, no association was found between these parameters and pregnancy outcomes. The relationship was determined between birth weight, placenta and umbilical cord size among diabetes, preterm delivery, ablatio placenta, and stillbirths and pregnancies. No significant association was found between histopathological findings and pregnancy outcomes.

Conclusion: Based on the findings obtained in present study, the regular gestational USG follow-up and a good gestational hemorrhage anamnesis may be useful in predicting or preventing these pathologies in terms of abortus that can be seen in SCH, preterm delivery, and the psychomotor pathologies that can be seen in further years in case of intrauterine growth retardation (IUGR) and short cord length.

Key words: Placenta, umbilical cord, histopathology, pregnancy outcome

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INTRODUCTION

Placenta is the only organ responsible for the development of fetus in the body (1-7). The presence of the fetus in utero depends on the placenta. A healthy placenta is necessary for protecting the pregnancy and supporting the delivery of fetus (3, 5-7). Besides the exocrine and endocrine functions, the placenta is also responsible for the oxygen flow, as well as feeding and protecting the fetus (3-7). Placenta pathologies may be related with the systemic diseases or the infant death and stillbirth may accompany (1). The maternal diseases such as diabetes, hypertension, and anemia may cause the pathological disorders in placenta and fetus. For instance, the diffuse placental infarct related with maternal hypertension may cause low APGAR scores (82%) and perinatal death (66.6%) (6). In fact, rather than explaining the death, the pathologies found in placenta are held responsible for the mental problems, which occur after it (1).

Abnormal fetal/placental weight, extensive infarct (>70%), single umbilical artery, infection, retroplacental hemorrhage, chorioangioma, ablatio placenta, maternal hypertension, collagen tissue disease, diabetes, drug addiction, stillbirth, neonatal death, hydrops fetalis, multiple pregnancy, pre-term delivery, post-term delivery, intrauterine growth retardation (IUGR), congenital abnormalities, asphyxia, meconium, and neurological findings such as convulsion are indicators utilized in assessing the placenta (1, 6).

The objective of present study is to compare histological and clinical findings of placenta and umbilical cord in order to confirm the diagnosis.

PATIENTS AND METHODS

112 placenta specimens obtained from pathology archive of Uşak University's Education and Research Hospital were involved in the present study. The placenta size, weight, umbilical cord length, and size were measured macroscopically. The term placenta's measurements performed in accordance with Kaufmann's Pathology (37-40 week) were 18 cm of size, 350- 750 g (mean 400 – 470 g) weight, and 2-2.5 cm thickness. The number of umbilical cord vessels, structures of membrane, chorioangioma, infarct, hematoma, and hemorrhage were recorded. A paraffin block was prepared. The sections were cut into 4-micron thickness and then stained with hematoxylin-eosin (HE). The preparations were evaluated by using a light microscope. The infarct,

chorioamnionitis, sub-chorionic fibrin deposition, congestion of chorionic vessel, SCH, chorangioma, calcification, perivillous fibrin deposition, effect of meconium, fibrinoid necrosis, villitis, and ectasia of chorionic vessels were investigated. The gender, mode of delivery, maternal disease, fetal birth weight, maternal age, parity, length of baby, head circumference, and APGAR score between 0-9 were recorded.

SPSS-22 was employed in all the analyses. In comparing two groups, Mann-Whitney U test was employed. The relationships between variables were examined in correlation analysis. Chi-Square and Fisher's Exact tests were utilized in analyzing the relationship between the categorical variables. The significance level was set to be 0.05, and it was stated that there was a significant difference and a significant relationship when $p < 0.05$, whereas $p > 0.05$ indicates no significant difference or relationship.

RESULTS

The placenta and umbilical cord pathologies were observed in 66 out of 112 (58.9%) and 35 out of 112 (31.25%) cases, respectively (Table 1). The mean age and parity of 112 cases were detected 27.04 (16-43) \pm 5.45 and 1.80 (1.00-6.00) \pm 0.86, respectively. The mean birth weight, birth length, and head circumference were measured to be 3293 g (1490-5000) \pm 531.13, 49.91 cm (39-56) \pm 1.93, and 34.72 cm (28-38) \pm 1.31, respectively. The chorioamnionitis

Table 1. Placenta and umbilical cord pathologies

| Placenta pathology | N=112 | % |
|---------------------------------|-------|--------|
| Perivillous fibrin deposit | 112 | 100% |
| Calcification | 68 | 60.7% |
| Infarct | 29 | 25.9 % |
| Chorioamnionitis | 29 | 25.9% |
| Chorangioma | 19 | 17% |
| Subchorionic fibrin deposit | 17 | 15.2% |
| SCH (Subchorionic Hemorrhage) | 17 | 15.2% |
| Subchorionic vessel congestion | 12 | 10.7% |
| Chorionic vessel ectasia | 3 | 2.7% |
| Chorioangioma | 1 | 0.09% |
| Meconium | 1 | 0.09% |
| Fibrinoid necrosis | 1 | 0.09% |
| Villitis | 1 | 0.09% |
| Umbilical cord pathology | | |
| Hematoma | 12 | 10.7% |
| Congestion | 12 | 10.7% |
| Node | 1 | 0.09% |
| Ectasia | 2 | 1.7% |
| Myxoid degeneration | 1 | 0.09% |
| Infarct | 4 | 3.5% |
| Hemorrhage | 3 | 2.7% |

Table 2. Clinicopathologic correlation between placenta and umbilical cord.

| | Placenta size (P) | Umbilical cord size (P) |
|-------------------------|----------------------|----------------------------|
| Maternal age | 0.993 | 0.001 |
| Birth weight | 0.000 | 0.000 |
| Subchorionic hemorrhage | 0.004 | 0.161 |

was found in 29 of 112 (25.9%). All of the placenta specimens were defined to have subclinical chorioamnionitis. No funitis was detected but only one case was defined as villitis. One of 29 cases was stillbirth, another one was preterm delivery, and the resting 27 of 29 cases have normal APGAR scores.

Isolated chorangioma was found in 4 out of 19 cases, infarct in 7 cases, SCH in 3 cases, chorioamnionitis in 4 cases, and maternal uterine anomaly (rudimentary horn) in one case. The umbilical cord insertion was observed at central location in 36 of 112 (32.1%) cases and at peripheral location in 76 of 112 (67.9%) cases. Maternal diseases found were diabetes, hypertension, thyroid, and thrombocyte dysfunction in 17 of 112 (15.2%) cases. 51 of 112 (45.5%) subjects were female and 61 subjects (54.5%) were male. Spontaneous vaginal delivery was reported in 49 of 112 (43.5%) cases and caesarean delivery in 63 of 112 (56.3%) cases. There was a significant difference between the maternal age and the size of umbilical cord ($p = 0.001$), whereas no significant difference was determined between the maternal disease and the placental and umbilical cord pathologies ($p > 0.05$) (Table 2).

No significant difference was found between mode of delivery, parity, placenta, and umbilical cord pathologies ($p > 0.05$). There were significant differences between birth weight, placenta, and

umbilical cord size ($p = 0.000$), whereas no statistically significant difference was found between gender, birth length, head circumferences and placenta, and umbilical cord pathologies ($p > 0.05$). Statistically significant differences were determined between subchorionic hemorrhage and placenta size ($p = 0.004$).

One of 66 cases with placenta pathology was the stillbirth, whereas 6 of 66 cases were hospitalized in NCU and 23 of 66 (34.8%) cases were found to have umbilical cord pathologies such as congestion, hemorrhage, hematoma, ectasia, edema, and infarct. 46 of 112 cases placenta pathologies were normal, whereas 10 of 46 (21.7%) cases were found to have umbilical cord pathologies and 4 of 46 cases were hospitalized in NCU. The hospitalization duration of cases with placenta pathology in NCU was longer than cases with normal pathology and their APGAR scores were lower than the scores of cases with normal pathology (Table 3).

DISCUSSION

SCH may be seen in pregnancy, especially in the first trimester. But, its clinical significance is still controversial. The incidence of SCH is reported to vary between 3% and 48% in the general population in the first trimester (8). The location and size of hematoma, gestational age, duration and vaginal bleeding play important role in pregnancy outcomes (9). Özkaya et al. (10) found that SCH was associated with abortus, preterm labor, and IUGR, and that the incidence of sub-chorionic hemorrhage increased with the increasing placental size. Sub-chorionic hematomas/hemorrhages are believed to result in bleeding in low-pressure venules at the border of the placenta between chorion and myometrium (11). Shayna et al. (12) reported the rate of sub-chorionic

Table 3. Correlation between Neonatal care unit (NCU) and placenta and umbilical cord pathologies.

| N | Maternal Disease | Placenta Pathology | APGAR Score | Umbilical cord Pathology | NCU Duration | Diagnosis |
|-----|-------------------------|-------------------------|-------------|--------------------------|--------------|----------------------------|
| 1. | Diabetes | Infarct | 9 | - | 10 day | Maternal diabetes |
| 2. | - | Infarct, corangioma | 7 | - | 8 day | Preterm delivery |
| 3. | - | Infarct | 9 | - | 10 day | Fetal respiratory distress |
| 4. | - | Subchorionic hemorrhage | 9 | - | 8 day | Preterm delivery |
| 5. | - | Chorioamnionitis | 4 | - | 1 month | Preterm delivery |
| 6. | Thrombocyte dysfunction | In fact, corangioma | 3 | - | 1 month | Ablatio placenta |
| 7. | - | Chorioamnionitis | 0 | Node | - | Stillbirth |
| 8. | - | - | 8 | - | 4 day | Tachypnea |
| 9. | - | - | 8 | - | 8 day | Preterm delivery |
| 10. | - | - | 6 | Congestion | 3 day | Fetal respiratory distress |
| 11. | - | - | 9 | - | 5 day | Vomit |

bleeding to be 1.7%. In females cases with sub-chorionic hemorrhage, the increased abruption ($n = 432$, 3.6%) and preterm birth ($n = 6,601$, 15.5%) were reported in 10.5% of cases. The incidence of massive sub-chorionic hemorrhage was reported to be one in every 2000 pregnancies (8). Massive SCH is a rare but serious condition in pregnancy, and large amount of blood accumulate between the uterine wall and chorionic membrane and it may leak into cervical canal. Its etiology has not been completely revealed yet, but large amount of sub-chorionic bleeding with thrombophilia and pseudo-aneurysm were reported in few studies (13). In the present study, there was no correlation between sub-chorionic hemorrhage, low birth weight and IUGR, but the significant correlation was found between sub-chorionic hemorrhage and placenta size, as in some other studies in literature ($p = 0.004$). It was observed that the rate of occlusion of sub-chorionic hemorrhage increased (Figure 1). We believe that this is because of the increased placenta size, vessel diameter, and increased blood flow in parallel with advancing gestational week (11). However, our study was limited from this aspect, because the regular gestational USG findings and bleeding anamnesis during the pregnancy were not available. At the same time, sub-chorionic fibrin

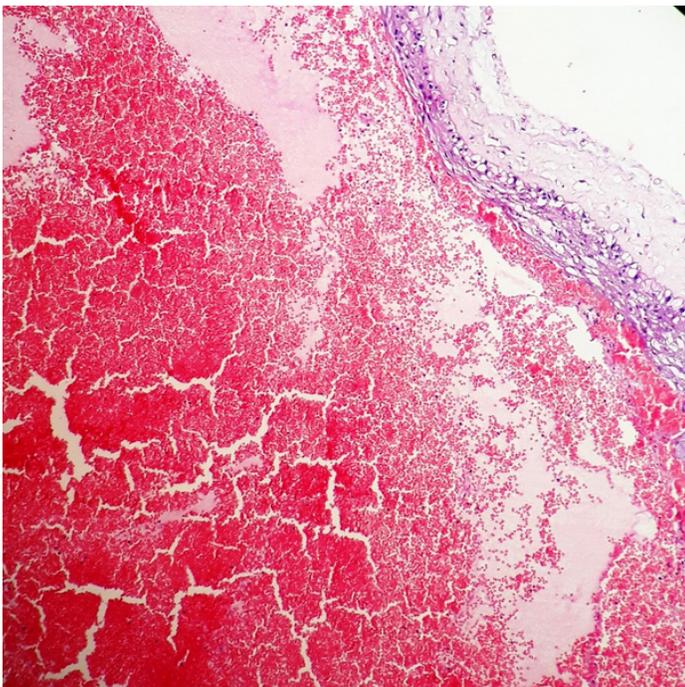


Figure 1. Widespread sub-chorionic hemorrhage area covering the decidual layer is seen at the top of villuses, HE x 40

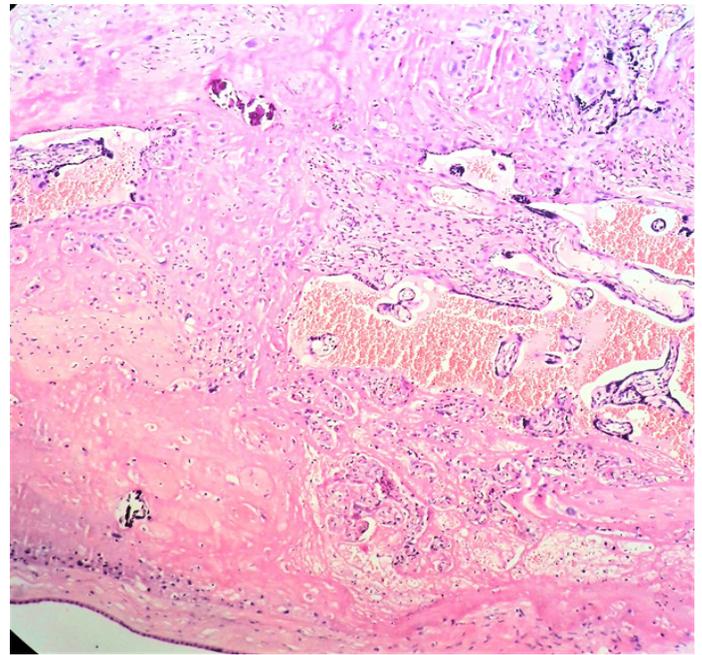


Figure 2. The widespread sub-chorionic fibrin deposition and calcification covering the decidual layer at the top of villuses are seen, HE x 40

accumulation accompanied some of the cases with SCH in our study. The sub-chorionic fibrinoid accumulation is observed under the chorionic plaque and there is an unknown clinical significance (1). In our study, we detected sub-chorionic fibrin deposition in SCH cases (Figure 2).

The umbilical cord and its size are important in fetal development (1, 14). The cord length and the delivery room are measured (19) to be very short or too long along in presence of various pathologies (1). The length of a normal umbilical cord is about 50-60 cm (1, 4). It has been determined that long cord, perinatal mortality, cord entanglement, emergency delivery, fetal thrombotic vasculopathy and neurological pathologies, and short cord may result in IUGR, fetal distress, and fetal mortality (4, 14). It is affected by the maternal factors such as cord length, increasing parity, maternal weight, body mass index, and the fetal factors such as birth weight, and male gender (14). In literature, the excessively long umbilical cords were correlated with the increased maternal age. However, some studies demonstrated no correlation between maternal age and cord length (15-17). In the study carried out by Taner et al. (18), the mean cord length was reported to be 59.44 ± 13.60 cm in normal delivery, 61.28 ± 15.8 cm in cesarean delivery,

and 63.48 ± 10.45 cm in vacuum or forceps delivery, and no significant relationship could be determined between birth length and cord length ($p > 0.05$). In addition, the researchers have suggested that the short cord is seen together with the advanced IQ and psychomotor development abnormalities in the advanced stage of life. However, these cases require long-term follow-up (18). In our study, there was a statistically significant correlation between birth type and cord dimension values, and the cord dimension values were found to be lower in the normal delivery group ($p = 0.0001$). In a study carried out by Carolyn et al. (19) on the male newborns, the cord size was found to be similar to that of our study, but no significant relationship was reported between gender and cord size ($p = 0.263$). In our study, a significant correlation was found between cord size and maternal age ($p < 0.05$) and it was determined that the cord size increased as the maternal age was increased. This result is attributed to the fact that the shortness of the cord measurements of normal births is based on the age factor.

The placental infarct may develop due to maternal uteroplacental vascular insufficiency and it may cause pathologic results in the fetus when the placental volume of 5-10% is exceeded. The multicentric infarct is observed in 25% of pregnancies and the magnitude of the infarct is directly proportional to the severity of maternal illness (1, 2). Particularly those located centrally and larger than 3 cm in diameter are important (1). The maternal hypertension, SLE, and preeclampsia are of significant correlation with the diffuse infarct. The clinical symptom of infarct area depends on the size, location, and severity of maternal disease (1, 2). The incidence of infarct was calculated to be 25.9% in our study, and no statistically significant relationship was found between maternal disease and infarction ($p > 0.05$) (Figure 3).

Perivillous fibrin deposition is seen at frequency varying between 20 and 22% in term placenta specimens, and its clinical significance is controversial. EH and DM are less frequently observed in preterm deliveries and preeclampsia. The fibrin deposition higher than 40% of placenta volume may lead to perinatal morbidity and mortality (1, 2). In our study, we determined perivillous fibrin deposition in all of the placenta specimens, but there was no correlation with clinical findings. It can be examined by making use of diffusiveness of deposition. The calcification is frequently seen in term placenta. It doesn't indicate a fetal maturation and it has no clinical significance

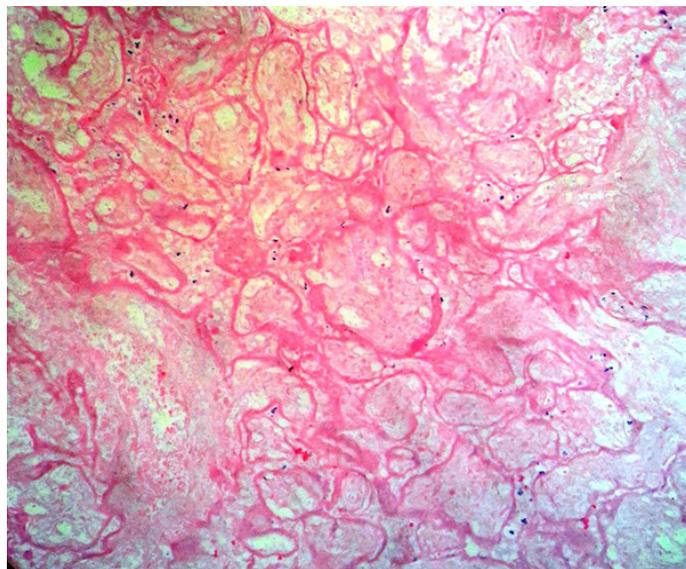


Figure 3. The total infarct areas with pale pink color and villuse silhouette with discredited vividness are observed, HE x 40

(1, 2). The findings obtained in the present study are in corroboration with the literature, we didn't find any correlation between calcification and clinical findings (Figure 4).

The chorioamnionitis is the inflammation of amnion fluid, membrane, and placenta/decidua

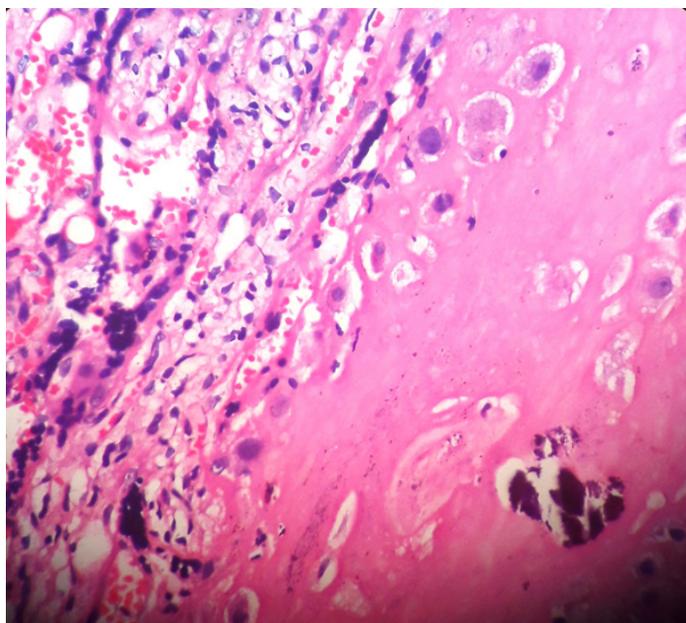


Figure 4. The fibrin deposition and calcification areas in perivillous area are seen, HE x 100

caused by bacteria, virus, fungus or parasitic microorganism. In most cases of chorioamnionitis, the microorganism may reach the membranes transcervically via the ascending pathway and it may also reach at the hematogenous, transabdominal or transfallopian pathways. The microorganisms causing chorioamnionitis increase with the release of prostaglandin. This also results in the premature birth. The most frequent causes are group A and streptococcus, pneumococcus, and HSV, whereas *Listeria*, *E. coli* and staphylococci are observed less frequently (1, 2). The incidence of chorioamnionitis is negatively correlated with gestational age. It is seen in 40-70% of preterm delivery but the incidence decreases down to 2-4% in term deliveries. The prolonged labor and membrane rupture, dilatation of cervix, and short cervix are known as the common etiologic factors. The subclinical or histologic chorioamnionitis is diagnosed by postpartum histologic evaluation of placenta. Low-virulence microorganisms are known as the etiologic factors. The maternal immune system is responsible for chorioamnionitis, whereas the fetal immune system is responsible for the funitis. The cases with funitis are seen in almost all of the chorioamnionitis cases, but the cases with chorioamnionitis constitute 60% of funitis cases (20). If chorioamnionitis is found in more than 20% of placenta specimens, the clinical significance increases. It may lead to preterm delivery, fetal and neonatal infection, respiratory distress, intrauterine hypoxia, and low APGAR score (1, 2, 21). In chorioamnionitis, the chronic villitis plays significant role in fetal infection; more than 30% of villous degeneration leads to perinatal death (1, 2). In our study, the incidence of chorioamnionitis was found to be 25.9%. 29 patients were found to have chorioamnionitis in the placenta; one has died and another one has been hospitalized in intensive care due to premature birth. The remaining 27 patients were observed to have normal values, and there was no intensive care unit admission. There are chronic villitis cases, in which acute and specific forms of villitis bacterial infection play important role in fetal infection (1, 2). In the present study, the rate of villitis was found to be 0.9% and only one case was observed (Figure 5).

Chorangiomas are a lesion of terminal villus but its etiology is not clear. It is thought to have genetic origins (1, 22-24). The low oxygen pressure, cytokines, diabetes, preeclampsia hypertension, Rh immunization, air pollution, smoking, and major

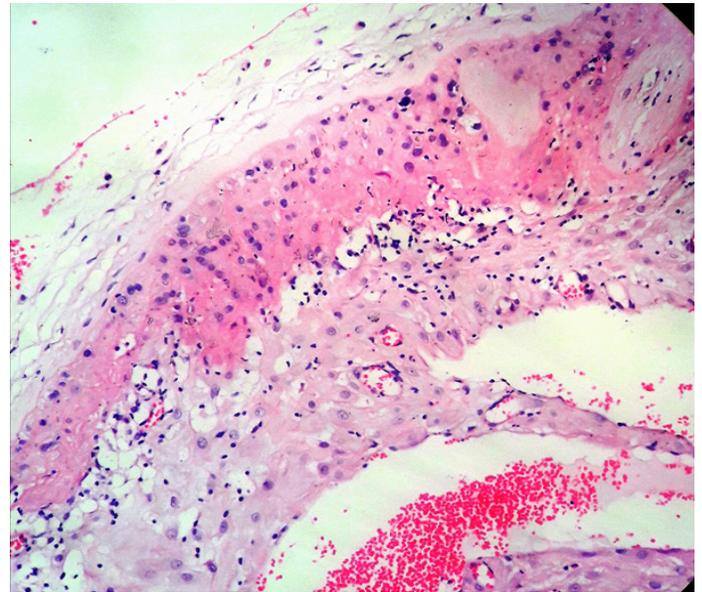


Figure 5. The chorioamnionitis area incorporating mixed inflammatory cell infiltration that is rich in terms of leucocyte with polymorph-core adhering to the amnion and upper chorion membrane can be seen, HE x 40

congenital anomalies such as Beckwith Wiedemann syndrome are related with the chorangiomas (1, 22-24). Similarly, Shariska S. et al. (22) reported that the reduction in oxygen transportation from mother to baby within the intervillous range leads to remodeling of vessels, which is caused by chorangiomas. In

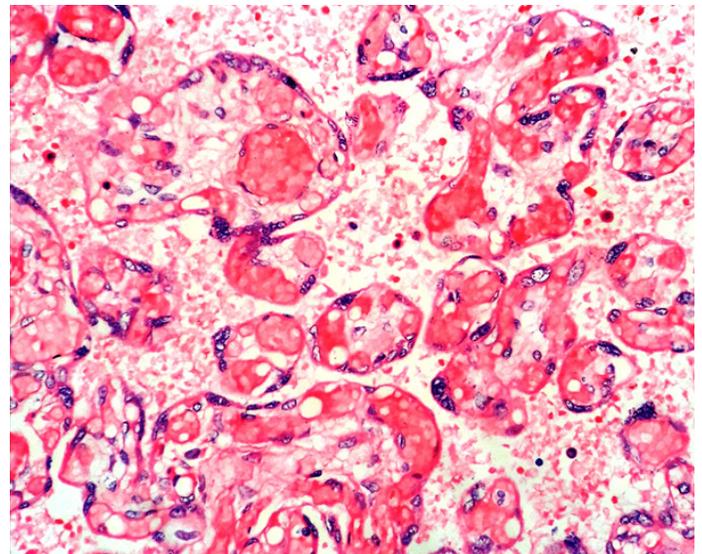


Figure 6. The chorangiomas area representing the vascular hyperplasia seen in terminal chorionic villus structures are seen, HE x 100

our study, we didn't find any correlation between chorangioma and clinical findings. However, 36.8% of the chorangioma specimens were seen to have infarct and one of the subjects was found to have uterine anomaly (rudimentary horn). Thus, it may be related with hypoxia or genetic factors (Figure 6).

Placental chorioangioma is the most common benign tumor found in the placenta. The incidence of chorioangioma was found to be 1% in all of the pregnancies (25). It may cause fetal morbidity in relation to tumor size (26-29). From histopathological aspect, they are the lesions having anastomosis with each other, relatively regular edges, showing lobular and island-like growth pattern, consisting of capillary vessels and cavernous vascular structures, and covered with endothelial cells (26, 28, 30). In literature, chorioangioma has been found to be associated with maternal age, hypertension, DM, prematurity, newborn girl genders, and multiple gestations (27). A retrospective study carried out on 22,000 cases by Kuhnel et al. (31), 138 cases of chorioangioma were reported and the incidence was calculated to be 0.6%. In the present study, we did not find any correlation between chorioangioma and clinical findings, and only one of the patients had chorioangioma (0.9%) (Figure 7).

The placental growth retardation may develop

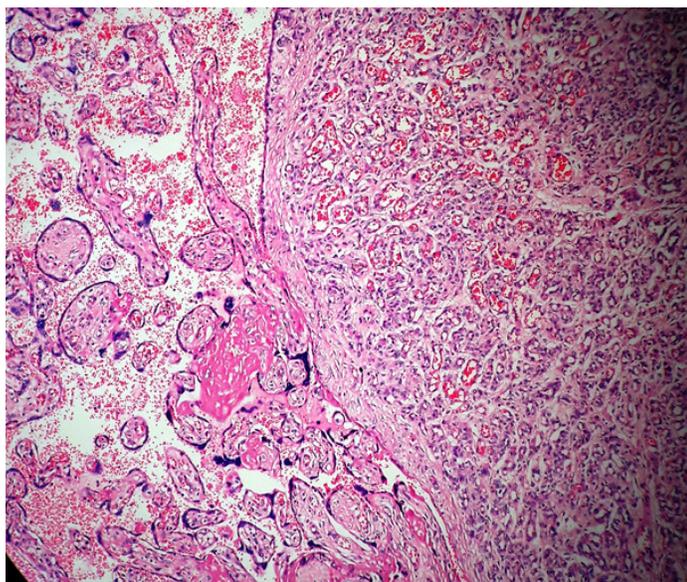


Figure 7. The lesion of chorangioma separated from the adjacent tissue with relatively regular edges, forming several island and lobule structures with different sizes, some of which anastomose with each other, and consisting of capillary-type vascular structures can be observed, HE x 20

because of factors such as Diabetes Mellitus (DM), immune and non-immune hydrops, maternal anemia, and syphilis, but there generally is no accompanying pathology. There are publications on the generalization that may be associated with villous maturation, fetal hydrops, and growth retardation (1, 2, 6, 7). Kayaselçuk et al. (2) also found a significant relationship between the villous maturation tension and the intrauterine growth retardation. In the present study, the maternal pathologies such as DM, Multi Nodular Goiter (MNG), hypertension, smoking, hepatitis B infection were among the maternal diseases observed. The infarction, chorioamnionitis, chorioamnionitis – chorangioma, and chorangioma - infarction were observed in some cases with DM, MNG, Hepatitis, and using cigarette, respectively. However, there was no statistically significant correlation between these pathologic findings and histopathological findings.

The reasons of this study's limitation are the limited number of placenta specimens and the lack of patient records and monitoring. These specimens were evaluated in terms of existence in the pathologic lesion but from the aspects of size, diffusiveness, and location. According to our opinion, besides the fact that the number of cases is insufficient, the placenta and umbilical cord examination, which should be started in the birth room, is not performed sufficiently and the standardization of pathology reports cannot be ensured. We believe that placental and umbilical cord specimens taken from pregnant women undergone regular obstetric checks and follow-ups to be performed in large series should be subjected to adequate examination in the delivery room, and a standard report template should be established for the pathology reports. We think that clinical efficacy of placenta and umbilical cord pathologies will be better understood.

Placenta and umbilical cord pathologies are correlated with clinical findings. Therefore, the size, location, and number of lesions should be particularly defined while performing a macroscopic evaluation. The duration, gestational week at detection, and vaginal bleeding also significantly affect the clinical findings. SCH increased in parallel with the placenta size. Therefore, it should be taken into consideration while carrying out a clinical and ultrasonographic evaluation. We believe that complications such as abortus, preterm labor, and IUGR that may develop during SCH are predictable or preventable by making use of regular gestational USG follow-up and good

history of pregnancy bleeding. The length of umbilical cord increases in parallel with the maternal age. In case of an abnormality of obstetrics examination during labor or delivery, the short or long umbilical cord length should be thought from the aspect of maternal age. The shortness of cord size was related to low IQ and psychomotor development in previous studies carried out on normal births and on those with a small maternal age. Therefore, we believe that evaluating the cord size together with the other anomalies may be useful in predicting the neurological pathologies that may develop in future.

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