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# Evaluation of Risk Factors, Incidence, Perinatal and Maternal Outcome of Placenta Previa Cases with and without Placenta Accreta Spectrum

## Plasenta Akreata Spektrumu Olan ve Olmayan Plasenta Previa Vakalarında Risk Faktörü, İnsidans, Perinatal ve Maternal Sonuçların Değerlendirilmesi

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**ABSTRACT**

**Aim:** Placenta accreta spectrum (PAS) are major risk factor for obstetric hemorrhage, which is a major cause of fetomaternal mortality and morbidity especially in developing countries. It is aimed to investigate the characteristics, incidence, maternal and fetal outcomes of placenta previa cases with and without PAS. Additionally we intended to analyze the clinical features, risk factors of placenta previa cases presenting with PAS.

**Material and Methods:** A retrospective cohort study was conducted to analyze the pregnancies of placenta previa with and without PAS. Written and electronic maternally-linked medical records between January 2005 and December 2014 were reviewed. Placenta previa diagnosis was confirmed ultrasonographically and PAS were defined clinically as placental adherence to the uterus without easy separation

**Results:** A total of 11351 deliveries were analyzed between January 2005 and December 2014. 387 patients were diagnosed to have placenta previa. Multiple pregnancies were detected in 11 of 387 previa cases and those were excluded. The incidence of placenta previa was 3.41‰ in our institution. The number of gravida ≥4 increased the risk of PAS 1.56 folds, ≥2 previous cesarean section (C/S) 9.74 folds, ≥3 abortus 3.83 folds, gestational hypertension (GHT) by 29.72 folds and gestational diabetes (GDM) 2.49 folds. According to logistic regression analysis ≥3 abortus, ≥2 previous C/S, and GTH were statistically significant.

**Conclusion:** Incases of placenta previa, ≥3 abortion, ≥2 previous C/S and GHT were the most important risk factors in terms of developing PAS. We should consider strict evaluation of placenta previa cases with these risk factors for PAS development during pregnancy may have a decreasing effect on maternal-neonatal morbidity and mortality.

**Keywords:** Placenta previa; placenta accreta spectrum; maternal morbidity.

**ÖZ**

**Amaç:** Plasenta akreata spektrumu (PAS), özellikle gelişmekte olan ülkelerde fetomaternal morbidite ve mortalitenin ana sebebi olan obstetrik kanama için önemli bir risk faktörüdür. Bu çalışmada; PAS olan ve olmayan plasenta previa olgularının özellikleri, insidansı, maternal ve fetal sonuçlarının araştırılması amaçlanmıştır. Ek olarak, PAS ile başvuran plasenta previa vakalarının klinik özellikleri ve risk faktörlerinin analiz edilmesi amaçlanmıştır.

**Gereç ve Yöntemler:** Plasenta previa ile birlikte PAS olan ve olmayan gebelikleri analiz etmek amacıyla retrospektif kohort bir çalışma planlandı. Ocak 2005 ile Aralık 2014 arasında yazılı ve elektronik olarak tıbbi kayıtlar gözden geçirildi. Plasenta previa tanısı ultrasonografik olarak, PAS ise klinik olarak plasentanın uterustan ayrılma aşamasında zorluk olarak tanımlandı.

**Bulgular:** Ocak 2005 ile Aralık 2014 arasında toplam 11351 doğumun retrospektif analizi yapıldı. 387 plasenta previa tanısı konulmuş olgu izlendi. Bu olguların 11'inde çoğul gebelik saptandı ve bu vakalar çalışma dışı bırakıldı. Kurumumuzda plasenta previa insidansı ‰3.41 idi. PAS riskini ≥4 gebelik sayısı 1,56 kat, ≥2 geçirilmiş sezeryan sayısı 9,74 kat, ≥3 abort sayısı 3,83 kat ve gestasyonel hipertansiyon varlığı 29,72 kat, gestasyonel diabet varlığı 49 kat arttırmıştır. Risk faktörlerinin lojistik regresyon analizinde ≥3 abort sayısı, ≥2 geçirilmiş sezaryen sayısı ve gestasyonel hipertansiyon varlığı anlamlı olarak değerlendirilmiştir.

**Sonuç:** Plasenta previa vakalarında; ≥3 abort sayısı, geçirilmiş sezaryen sayısının ≥2 ve gestasyonel hipertansiyon varlığı PAS gelişimi açısından en önemli risk faktörleridir. Bu risk faktörlerine sahip plasenta previa olgularının gebeliği boyunca PAS gelişimi açısından sıkı takibi maternal-neonatal morbidite ve mortalite üzerine azalan bir etkiye sahip olabileceğini düşünmekteyiz.

**Anahtar kelimeler:** Plasenta previa; plasenta akreata spekturum; maternal morbidite.

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## INTRODUCTION

Placenta previa is described as localization of the placenta in the lower uterine segment thus covering the internal os totally or partially. The incidence is 0.3-0.5% (1). Pregnancies complicated with placenta previa are prone for second trimester and postpartum bleeding which increases the risk of adverse perinatal and maternal outcome (2,3). Placenta accreta when the villi penetrate only superficially of the myometrium without invading it, placenta increta when the villi penetrate the myometrium and placenta percreta when the villi penetrate through the uterine serosa (4,5). According to International Federation of Gynecology and Obstetrics (FIGO) 2018 consensus the term "placenta accreta spectrum (PAS)" refers to all three conditions (accreta, increta, percreta) (6). Placenta accreta spectrum (PAS) are commonly together with placenta previa. Uterine damage due to previous surgery (cesarean deliveries, curettage, myomectomy) poor healing allows the placenta to grow through a damaged or absent Nitabuch layer in the myometrium (7,8).

Prolonged hospitalisation, peripartum hysterectomy, massive blood transfusion and intensive care unit admission are potential maternal morbidities of PAS (1).

Prenatal diagnosis of PAS decreases fetal and maternal morbidities and mortalities. Diagnosis of PAS are accomplished sonographically with a sensitivity of 77%-87%, specificity of 96%-98%, a positive predictive value of 65%-93%, and a negative predictive value of 98% (9,10).

Increasing incidence of cesarean section and maternal age will lead to an increase in the number of placenta previa cases and its complications including PAS. These conditions may be diagnosed clinically when there is a failure of the placental detachment or histologically (7,8).

It is aimed to assess the characteristics, incidence, maternal and fetal outcomes of placenta previa cases with and without PAS. Additionally it is intented to analyze the clinical features, risk factors of placenta previa cases presenting with PAS.

## MATERIAL AND METHODS

A retrospective cohort study was conducted to analyze the pregnancies of placenta previa with and without PAS. This study was carried out at Maternity and Women's Diseases Training and Research Hospital, Obstetrics and Gynecology Clinic Istanbul/Turkey. The study was approved by the local Ethics and Research Committee (Approval number: 164/2015).

Written and electronic medical records between January 2005 and December 2014 were evaluated using searches for diagnoses with the terms of "placenta previa", "placenta accreta", "placenta increta", "placenta percreta". Histopathologic studies and laboratory investigations were reviewed via the hospital database. Pregnancies without adequate prenatal surveillance and pregnancies with multiple fetuses were excluded and 376 placenta previa cases were included eventually.

Placenta previa diagnosis was confirmed ultrasonographically. Placenta previa was diagnosed when internal cervical ostium was covered completely or partially. PAS (placenta accreta, increta and percreta) were diagnosed sonographically. Irregularly shaped vascular spaces with turbulent interval flow, loss of retro placental hypoechoic clear zone, thinning of the myometrium over the placenta, absence of a decidual interface with normal placental echogenicity interruption, increased vascularity of uterus serosa-posterior bladder wall interface and apparent protrusion and bulging of the placenta into the bladder are the sonographic features of PAS (11). PAS was defined clinically as firm placental adherence to the uterus. Pathology report was given precedence all over the findings.

Gestational week was calculate by last menstrual period and confirmed with an early ultrasound measurement. Our institution is a tertiary referral center for high risk pregnancies with more than ten thousand deliveries per annum and cesarean deliveries were performed when medically indicated. Cesarean delivery were performed at 37+0 weeks of gestation if placenta previa totalis or partialis is present and delivery electively between 34+0 and 35+0 weeks of gestation is persued if placental invasion anomalies were predicted prenatally.

All women diagnosed with placenta previa accompanying with placenta accreta, placenta increta and placenta percreta were analyzed and evaluated with regard to risk factors. Clinical characteristics including maternal age, gravida, parity, gestational age, obstetric risk factors including previous cesarean deliveries, hypertensive disorders and abortion were evaluated. The evaluation also included additional procedures to control hemorrhage (cesarean hysterectomy, uterine artery ligation, bilateral hypogastric artery ligation, hemostatic sutures, intrauterine balloon tamponade), intensive care unit admission for newborn, neonatal birth weight, Apgar scores at 1st and 5th minute. The study population were categorized into three different groups: placenta previapartialis (PPP) without PAS, placenta previatotalis (PPT) without PAS and placenta previa with PAS. These groups were evaluated regarding the risk factors and perinatal outcome.

### *Statistical Analysis*

All statistical analyses were conducted with the SPSS version 22.0 (SPSS, Illinois, USA). Descriptive statistics were calculated (mean, standard deviation) and One-way ANOVA used for comparison among groups of variables with normal distribution. Tukey HSD test was used to determine the group that caused difference. The Kruskal Wallis test was used to compare the groups for variables with non-normal distribution, and the Bonferroni corrected Mann-Whitney U test was used to determine the group that caused the difference. Pearson's Chi-square test, Continuity Correction (Yates) test, Fisher's Exact test and Fisher Freeman Halton test were used for the comparison of qualitative data. Logistic regression analysis was used to determine the risk factors. Significance was assessed at $p<0.05$ level.

## RESULTS

A total of 11351 deliveries were performed between January 2005 and December 2014. Among these cases, 387 patients were diagnosed to have placenta previa. Multiple pregnancies were detected in 11 of 387 previa cases and those were excluded from the study.

The incidence of placenta previa was 3.41‰ in our institution. PPT was diagnosed in 225 (59.8 %) cases, PPP in 151 (40.2%) cases. Placenta percreta was found in 20 (41.7%), placenta increta was in 6 (12.5%) and placenta accreta was in 22 (45.8%) of the 48 cases with PAS.

Distribution of PPP, PPT and PAS cases among years were shown in Figure 1. In 2005, PPP was observed 27.8%, PPT was 55.6% and PAS was 16.7% of the cases. By 2014, these ratios reached 33.3%, 44.4% and 22.2%, respectively (Figure 1).

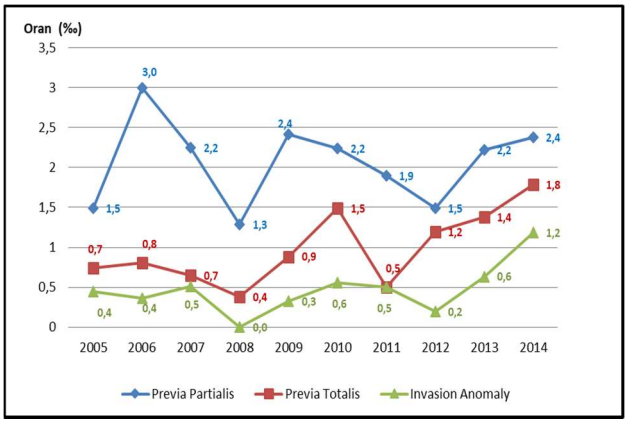


**Figure 1.** Distribution of placental previa and invasion anomaly (placenta accreta spectrum) according to the years.

231 of placenta previa cases were emergency and 145 were elective cesarean delivery between the years of 2005-2014. Patient characteristics and neonatal outcomes were given in Table 1. When the cases in the groups were compared in terms of gravid was found to be higher in the PAS group than in the PPT and PPP groups (p=0.023). The mean gravida of PAS group was significantly higher than PPP group (p=0.006) and PPT group (p=0.002). There was no statistically significant difference between PPP and PPT groups (p=0.237).

The distribution of laboratory and clinical parameters according to groups were shown in Table 2.

In univariate analysis of risk factors, the prevalence of PAS risk was increased 1.56 fold by ≥4 gravida, 3.83 fold by ≥3 abortus, 9.74 fold by ≥2 C/S, 2.49 fold by GDM, 29.72 fold by GHT (Table 3).

**Table 1.** Baseline characteristics and differences in maternal and neonatal outcomes of the groups

| | PPP (n=103) | PPT (n=225) | PAS (n=48) | p |
|---|---|---|---|---|
| | **Mean±Standard Deviation** | | | |
| **Age (year)** | 31.0±5.3 | 30.7±5.6 | 32.1±5.3 | [1]0.285 |
| **BMI (kg/m$^{2)}$** | 28.9±3.1 | 29.2±3.7 | 29.1±3.9 | [1]0.792 |
| **Gestational age (week)** | 35.3±3.5 | 34.8±3.7 | 34.6±2.6 | [1]0.420 |
| **Neonatal weight (kg)** | 2951.1± 669.3 | 2811.2± 656.9 | 2826.3± 660.7 | [1]0.231 |
| | **Median (Minimum-Maximum)** | | | |
| **Gravida** | 2 (1-7) | 3 (1-12) | 3 (1-9) | **[2]0.023** |
| **Apgar score 1$^{st}$ minute** | 8 (2-9) | 7 (1-9) | 7 (4-9) | [2]0.440 |
| **Apgar score 5$^{th}$ minute** | 9 (3-10) | 8 (1-10) | 9 (7-10) | [2]0.352 |
| | **n (%)** | | | |
| **Vaginal Delivery** | | | | |
| 0 | 52 (50.5) | 122 (54.2) | 35 (72.9) | |
| 1 | 26 (25.2) | 39 (17.3) | 6 (12.5) | |
| 2 | 12 (11.7) | 33 (14.7) | 5 (10.4) | [3]0.243 |
| 3 | 8 (7.8) | 19 (8.4) | 2 (4.2) | |
| 4+ | 5 (4.9) | 12 (5.3) | 0 (0.0) | |
| **Previous C/S** | | | | |
| ≤1 | 80 (77.7) | 156 (69.3) | 10 (20.8) | |
| 2 | 19 (18.4) | 41 (18.2) | 22 (45.8) | **[3]0.001** |
| 3 | 2 (1.9) | 22 (9.8) | 12 (25.0) | |
| 4+ | 2 (1.9) | 6 (2.7) | 4 (8.3) | |
| **Previous Abort** | | | | |
| 0 | 18 (30.0) | 58 (44.6) | 1 (4.8) | |
| 1 | 26 (43.3) | 50 (38.5) | 7 (33.3) | |
| 2 | 11 (18.3) | 14 (10.8) | 9 (42.9) | **[3]0.001** |
| 3 | 5 (5.3) | 3 (2.3) | 2 (9.5) | |
| 4+ | 0 (0.0) | 5 (3.8) | 2 (9.5) | |
| **Presentation** | | | | |
| Cephalic | 81 (78.6) | 168 (74.7) | 44 (91.7) | |
| Breech | 14 (13.6) | 43 (19.1) | 3 (6.3) | [4]0.099 |
| Transverse | 8 (7.8) | 14 (6.2) | 1 (2.1) | |
| **NICU administration** | | | | |
| (+) | 21 (20.4) | 56 (24.9) | 11 (22.9) | [4]0.668 |
| (-) | 82 (79.6) | 169 (75.1) | 37 (77.1) | |

PPP: Placenta previapartialis, PPT: Placenta previatotalis, PAS: Placenta acreta spectrum, C/S: Caesarean Section, NICU: Neonatal intensive care unite [1]Oneway ANOVA, [2]Kruskal Wallis, [3]Fisher Freeman Halton, [4]Pearson Chi-square test

**Table 2.** Evaluation of clinical parameters according to the groups

| | PPP (n=103) | PPT (n=225) | PAS (n=48) | p |
|---|---|---|---|---|
| | **Mean±Standard Deviation** | | | |
| **Prepartum Hb** | 11.5±1.3 | 11.2±1.2 | 11.2±1.1 | [1]0.280 |
| **Postpartum Hb** | 9.7±1.6 | 9.3±1.3 | 7.8±1.6 | **[1]0.001** |
| | **n (%)** | | | |
| Maternal bleeding ≥1000cc | 70 (68.0) | 149 (66.2) | 43 (89.6) | **[2]0.005** |
| TAH | 0 (0.0) | 3 (1.3) | 45 (93.8) | **[2]0.001** |
| Ballontamponade (Bacri®) | 7 (6.8) | 13 (5.8) | 1 (2.1) | [2]0.492 |
| Hemostatic suturation | 7 (6.8) | 17 (7.6) | 6 (12.5) | [2]0.452 |
| BHAL | 1 (1.0) | 4 (1.8) | 17 (35.4) | **[2]0.001** |
| Uterin artery ligation | 0 (0.0) | 6 (2.7) | 0 (0.0) | [3]0.206 |
| Preeclampsia | 6 (5.8) | 19 (8.4) | 5 (10.4) | [2]0.575 |
| GDM | 10 (9.7) | 11 (4.9) | 7 (14.6) | **[2]0.040** |
| Chronic hypertansion | 0 (0.0) | 3 (1.3) | 2 (4.2) | [3]0.127 |
| GHT | 0 (0.0) | 1 (0.4) | 4 (8.3) | **[3]0.002** |
| Oligohydramnios | 11 (10.7) | 18 (8.0) | 4 (8.3) | [2]0.724 |
| Polihydramnios | 3 (2.9) | 8 (3.6) | 2 (4.2) | [3]0.847 |
| PPROM | 7 (6.8) | 13 (5.8) | 1 (2.1) | [2]0.492 |
| Preterm Labour | 8 (7.8) | 9 (4.0) | 5 (10.4) | [2]0.142 |
| IUMF | 2 (1.9) | 6 (2.7) | 0 (0.0) | [3]0.877 |
| IUGR | 3 (2.9) | 10 (4.4) | 3 (6.3) | [3]0.573 |
| ART pregnancy | 1 (1.0) | 10 (4.4) | 0 (0.0) | [3]0.145 |
| Transfusion ≥4IU | 5 (4.9) | 17 (7.6) | 15 (31.3) | **[2]0.001** |
| Uterine Anomaly | 1 (1.0) | 4 (1.8) | 0 (0.0) | [3]1.000 |

PPP: Placenta previapartialis, PPT: Placenta previatotalis, PAS: Placenta acreta spectrum, Hb: Hemoglobin, TAH: Total Abdominal Hysterectomy, BHAL: bilateral hypogastic artery ligation, GDM: Gestational Diabetes Mellitus, GHT: Gestational Hypertansion, IUMF: Intrauterine mort fetalis, IUGR: Intrauterine growth retardation, ART: Assisted reproductive technology, [1]Oneway ANOVA, [2]Pearson Chi-square, [3]Fisher Freeman Halton test

**Table 3.** Univariate analysis of risk factors for invasion abnormalities

| | Previa (n=328) | Invasion (n=48) | p | Odds Ratio (%95 CI) |
|---|---|---|---|---|
| **Abortus** | | | | |
| ≥3 | 38 (11.6) | 13 (27.1) | **[1]0.007** | 3.835 (1.379-5.828) |
| < 3 | 290 (88.4) | 35 (72.9) | | |
| **C/S** | | | | |
| ≥2 | 92 (28) | 38 (79.2) | **[1]0.001** | 9.748 (4.664-20.371) |
| < 2 | 236 (72) | 10 (20.8) | | |
| **Gravida** | | | | |
| ≥4 | 103 (31.4) | 20 (41.7) | [2]0.157 | 1.560 (0.840-2.899) |
| < 4 | 225 (68.6) | 28 (58.3) | | |
| **GDM** | | | | |
| (+) | 21 (6.4) | 7 (14.6) | [3]0.069 | 2.496 (0.999-6.234) |
| (-) | 307 (93.6) | 41 (85.4) | | |
| **GHT** | | | | |
| (+) | 1 (0.3) | 4 (8.3) | **[3]0.001** | 29.727 (3.249-272.015) |
| (-) | 327 (99.7) | 44 (91.7) | | |

CI: Confidence Interval, GDM: Gestational Diabetes mellitus, GHT: Gestational Hypertansion, [1]Continuity Correction (Yates) test, [2]Pearson Chi-square test, [3]Fisher's Exact test

When we evaluate the effects of number of abortion, C/S and GHT parameters on invasion anomalies by Enter logistic regression analysis; the model was found to be significant (p=0.001). Negelkerke R square value was found to be 0.284 and the explanatory coefficient of the model was found to be good (87.2%). The effects ≥3 abortion (p=0.005), ≥2 C/S number (p=0.001) and GHT (p=0.016) were found to be statistically significant (Table 4).

In the analysis of postpartum maternal morbidities, the risk of TAH was 1625 times, the risk of BHAL was 35.426 times, the risk of ≥4 IU transfusion was 6.322 times, the risk of bleeding ≥1000 cc was 4.280 times higher when comparing patients with PAS than previa without PAS (Table 5).

There was a statistically significant difference between the groups according to postpartum hemoglobin (PP Hb) measurements (p<0.01). As the result of the Post-Hoc Tukey HSD test to determine the group from which the difference originated, the mean PP Hb of the PAS group was significantly lower than the PPP group (p=0.001) and PPT group (p=0.001). There was no statistically significant difference between PPP and PPT groups (p>0.05).

**Table 4.** Logistic regression analysis of risk factors for invasion anomalies

| | B | S.E. | p | Exp(B) |
|---|---|---|---|---|
| Constant | -3.448 | 0.352 | **0.001** | 0.032 |
| Abort ≥3 | 1.185 | 0.422 | **0.005** | 3.272 |
| C/S ≥2 | 2.269 | 0.388 | **0.001** | 9.666 |
| GHT | 3.120 | 1.292 | **0.016** | 22.654 |

B: Beta, S.E: Standard Error, Exp (B): Exponential (ODDS ratio)

**Table 5.** Univariate analysis of the postpartum complications

| | Previa (n=328) | PAS (n=48) | p | Odds Ratio (%95 CI) |
|---|---|---|---|---|
| **TAH** | | | | |
| (+) | 3 (6.3) | 45 (93.8) | [1]**0.001** | 1625.0 (318.26-8296.99) |
| (-) | 325 (99.1) | 3 (0.9) | | |
| **BHAL** | | | | |
| (+) | 5 (22.7) | 17 (77.3) | [2]**0.001** | 35.426 (12.236-102.564) |
| (-) | 323 (91.2) | 31 (8.8) | | |
| **Transfusion** | | | | |
| ≥4IU | 22 (59.5) | 15 (40.5) | [2]**0.001** | 6.322 (2.992-13.359) |
| <4IU | 306 (90.3) | 33 (9.7) | | |
| **Bleeding** | | | | |
| ≥1000cc | 219 (83.6) | 43 (16.4) | [1]**0.002** | 4.280 (1.649-11.114) |
| <1000cc | 109 (95.6) | 5 (4.4) | | |

CI: Confidence Interval, [1]Continuity Correction (Yates) test, [2]Fisher's Exact test

## DISCUSSION

The aim of the present study was to evaluate neonatal and maternal outcomes in cases with placenta previa with and without PAS over ten years period in a tertiary referral center.

The results of the present study showed that the presence of ≥3 abortus, ≥2 previous C/S and GHT in placenta previa cases were important risk factors for PAS development. The risk of TAH was 1625 fold, the risk of BHAL was 35.426 fold, the risk of ≥4 IU transfusion was 6.322 fold, the risk of bleeding ≥1000 cc was 4.280 fold higher than that of non-PAS related previa.

Due to an unknown reason, the frequency of pregnancies complicated by placenta previa has increased in the last decade. As a matter of fact, when we was examined the placenta prevalence data of our clinic for 2005-2014, the PAS cases were observed in 16.7% in 2005, and reached to 22.2% in 2014.

Surgery of placental penetration abnormalities are a real challenge and serious complications including massive intrapartum hemorrhage, maternal mortality, morbidity exist (12,13). This study revealed that neonatal and maternal morbidity is increased significantly in the case of placenta previa (PP) is complicated with PAS. PP and PAS are associated with adverse neonatal and maternal outcome. Therefore, prevention and detection of risk factors is crucial.

The incidence of PAS during the study period was %0.043. This finding was reported previously with an incidence of 0.17 per 1000 pregnancies for placenta accreta (14). In the study of Palova et al. (15) placenta accreta was verified 3% among the women with PP. Miller et al. (4) showed that placenta accreta occurred in 9.3% women with PP. In our study; placenta accreta occurred in 5.8% women with PP during the years of study.

Increasing number of cesarean delivery and PP have noted to be major risk factors for PAS (14,16,17). Kassem (18) published retrospective cohort study 122 PP patients 25 of them was with placenta accreta. 96% had a history of previous caesarean section.

As the number of previous cesarean deliveries increases, the risk of PP and placenta accreta coexistence increases. This rate is even higher in women with classical cesarean history (5,19,20). In a multicenter cohort study, women with PP were assessed with cesarean delivery birth and the risk of placenta accreta after birth was reported as 3%, 11% and 40% after first, second and third cesarean section. It has been reported that these risks are independent of other maternal characteristics such as body mass index, parity, tobacco use, and concomitant diabetes or hypertension (7,3,21). We found that ≥2 previous C/S surgery is statistically significant risk factor for development of PAS and if the number of past cesarean sections is 2 or more, the risk of placenta accreta spectrum increased 9.74 folds.

It is a debate why some women with prior cesarean and placenta previa develop accreta while others do not. Advanced maternal age, smoking, hypertensive disorders were associated with PAS in some studies (14,16,17). In these studies; maternal age was evaluated as a known risk factor owing to PP and increasing numbers of cesareans. Smoking was reported to have negative effects on wound healing and hypertension thought to lead to accreta by vascular endothelial trauma (16). In the large prospective cohort study of Bowman and colleagues' (3), multiple cesarean section history was reported to be only risk factor for the association of PP and placenta accrete. No other risk factors including maternal age, smoking, parity, hypertension, diabetes were significant when controlling other variables. In Lebanon studies, hypertensive diseases were shown to be associated with placenta accreta. It has been suggested that hypertension may cause placenta accreta as a result of vascular endothelial injury, or that placenta accreta may cause hypertension as a result of abnormal trophoblast invasion (16). We did not find any relation between maternal age/ parity with placenta previa and accompanying PAS. However, the relationship between gravida and ≥3 abortion PAS was significant. In our cohort ≥3 abortion increased the PAS risk by 3.83 fold and in multivariate analysis showed that ≥3 abortion had significantly effect on the risk of PAS development.

It has been reported that in a single-centered large series of Eshkoli et al. (17), placenta accreta risk was increased 2.12 fold in cases with ≥2 consecutive abortus. However, in the same study, it was seen that recurrent abortus lost statistical significance as an independent risk factor after multiple logistic regression analysis. In our study, the number of ≥3 abortus increased the incidence of accreata by 3.83 fold. Multivariate logistic regression analysis also showed a statistically significance for ≥3 abortus.

We also found that gestational diabetes mellitus (GDM) and gestational hypertension (GHT) were more frequent in placenta previa cases with PAS than placenta previa without PAS. In Lebanon studies hypertensive diseases have been associated with

accreta, it has been suggested that hypertension may cause accreta as a result of vascular endothelial injury, or that PAS may cause hypertension as a result of abnormal trophoblast invasion (16). We did not find maternal age and parity with placenta previa and PAS in our study. However, the relationship between gravida and PAS was significant. We also found that GDM and GHT were more frequent in placenta previa cases with PAS than placenta previa without PAS. In the univariate analysis; while GHT increased the PAS risk by 29.72 fold and GDM 2.49 fold. In multivariate analysis showed that GHT had significantly effect on abnormal placental penetration.

In placenta previa cases, especially in the case of placenta previa totalis, engagement of fetal head can be prevented and this may lead to fetal malpresentation. Senkoro and colleagues (22) reported an increased rate of fetal malpresentation, low apgar score, low birth weight, admission to neonatal intensive care unit, stillbirth and early neonatal death. In our study; PPP, PPT and PAS were not significantly different in terms of fetal presentation, birth weight, apgar scores and stillbirth. However, Senkoro was compared the cases with and without placenta previa. In our study; all of our cases consisted of placenta previa cases. Sekiguchi et al. (23) and Omokanye et al. (24) reported a significant relationship between placenta previa types and the APGAR score in their studies. In addition, Omokanye and colleagues (24) reported that there was a significant relationship between placenta previa types and gestational week of birth, birth weight of newborn and intraoperative blood loss.

As placenta previa can lead to previa-associated bleeding, hypoxia, intrauterine growth retardation and prematurity, it is natural that the results of Senkoro were different from ours (22). The progress of ultrasound technology increases the probability of diagnosing PAS cases in the prenatal period and early diagnosis provides a chance to plan the right time for cesarean delivery. Accordingly, that the perinatal morbidity and mortality of the newborn decrease.

Data related to the depth of villus invasion are limited and cesarean hysterectomy is the most common management approach for PAS when the diagnosis is prenatal. Most cases of conservative treatment require secondary hysterectomy (25). In our case, no case of secondary hysterectomy was needed. Hysterectomy rate was given between 5-19% in patients with placenta previa (26-28). Hysterectomy was necessary in 1.3% of our placenta previa cases without placental penetration abnormalities and 93.8% placenta previa cases with PAS. In our study; hysterectomy was not needed in any of the cases of placenta previa partialis without PAS.

Miller et al. (29) reported placenta accreta to be the most common indication for an emergency peripartum hysterectomy. Palova et al. (15) found placenta accreta to be the second most common indication for an emergency peripartum hysterectomy. In our study, 231 of placenta previa cases were emergency and 145 were elective cesarean delivery between the years of 2005-2014. Peripartum hysterectomy was performed in 45 of 48 patients with placenta previa cases with PAS.

Wright et al. (30) reported a median blood loss of 3000 ml and a median red blood cell transfusion of five units in patients undergoing hysterectomy for placenta accreta. Kasem et al. (18) reported a median blood loss of 2000 ml as a result of placenta accrete. In our study, intraoperative hemorrhage was detected in 89.6% of PAS cases and ≥4 IU blood transfusions were performed in 31.3% of the cases. The most intraoperative bleeding rate and the highest blood transfusion frequency was detected in cases of PAS group.

Some centers perform elective surgery at 34-35 weeks for placenta accreta. They advocates that this practice is not associated with increased neonatal morbidity (31). Kassem et al. (18) reported that waiting from 34 weeks+5 days until 36 weeks+1.4 days resulted in reduction in neonatal intensive care unit admissions and an increase in mean neonatal weight. They also expressed that the obstetrician must weight the risks of the benefits of a planned delivery against neonatal prematurity. In our institution; cesarean delivery was performed at 37+0 weeks of gestation if PPT or PPP is present and delivery between 34+0 and 35+0 weeks of gestation is persued if PAS were predicted prenatally.

In conclusion, history of ≥2 cesarean delivery, ≥3 abortus, GHT were major important risk factors for development of PAS in placenta previa cases. We state that antenatal screening of these patients of PAS will have a decreasing effect on maternal-neonatal mortality and morbidity. The prenatal diagnosis of PAS events will allow us to make clinical decisions in terms of timing, place of birth and precautions to be taken at birth and reduce complications.

**REFERENCES**


1. Oyelese Y, Smulian JC, Placenta previa, placenta accreta, and vasa previa. Obstet Gynecol. 2006;107(4):927-41.
2. Kollman M, Gaulhofer J, Lang U, Klaritsch P. Placenta praevia: incidence, risk factors and outcome. J Matern Fetal Neonatal Med. 2016;29(9):1395-8.
3. Bowman Z, Eller A, Bardsley T, Greene T, Varner M, Silver R. Risk factors for Placenta Accreta: A Large Prospective Cohort. Am J Perinatol. 2014;31(9):799-804.
4. Jauniaux E, Jurkovic D. Placenta accreta: pathogenesis of a 20th century iatrogenic uterine disease. Placenta 2012;33(4): 244-51.
5. Fox H, Sebire NJ. Pathology of the placenta. 3rd ed. Philadelphia: Saunders- Elsevier; 2007.
6. Jauniaux E, Chantraine F, Silver RM, Langhoff-Roos J; FIGO Placenta Accreta Diagnosis and Management Expert Consensus Panel. FIGO consensus guidelines on placenta accreta spectrum disorders: Epidemiology. Int J Gynaecol Obstet. 2018;140(3):265-73.
7. Wu S, Kocherginsky M, Hibbard JU. Abnormal placentation: twenty-year analysis. Am J Obstet Gynecol. 2005;192(5):1458-61.
8. Ananth CV, Savitz DA, Luther ER. Maternal cigarette smoking as a risk factor for placental abruption, placenta previa, and uterine bleeding in pregnancy. Am J Epidemiol. 1996;144(9):881-9.
9. Warshak CR, Eskander R, Hull AD, Scioscia AL, Mattrey RF, Benirschke K et al. Accuracy of ultrasonography and magnetic resonance imaging in the diagnosis of placenta accreta. Obstet Gynecol. 2006;108(3 Pt 1):573-81.
10. Comstock CH, Love JJ Jr, Bronsteen RA, Lee W, Vettraino IM, Huang RR et al. Sonographic detection of placenta accreta in the second and third trimesters of pregnancy. Am J Obstet Gynecol. 2004;190(4):1135-40.
11. Asıcıoglu O, Şahbaz A, Güngördük K, Yildirim G, Asıcıoglu BB, Ülker V. Maternal and perinatal outcomes in women with placenta praevia and accreta in teaching hospitals in Western Turkey. J Obstet Gynaecol. 2014;34(6):462-6.
12. Chou M. Prenatal diagnosis and perinatal management of placenta previa accreta: past, present and future. Taiwanese Journal of Obstetrics and Gynecology. 2004;43:64-71.
13. Heller DS. Placenta accreta and percreta. Surg Pathol.2013;6(1):181-97.
14. Fitzpatrick KE, Sellers S, Spark P, Kurinczuk JJ, Brocklehurst P, Knight M. Incidence and risk factors for placenta accreta/increta/percreta in the UK: a national case-control study. PLoS ONE. 2012;7(12):52893.
15. Palova E, Redecha M, Malova A, Hammerova L, Kosibova Z. Placenta accreta as a cause of peripartum hysterectomy. BratislLekListy. 2016;117(4):212-6.
16. Usta IM, Hobeika EM, Musa AA, Gabriel GE, Nassar AH. Placenta previa- accreta: risk factors and complications. Am J Obstet Gynecol 2005;193(3Pt 2) :1045-9.
17. Eshkoli T, Weintraub AY, Sergienko R, Sheiner E. Placenta accreta: Risk factors, perinatal outcomes, and consequences for subsequent births. Am J Obstet Gynecol. 2013;208 (3):219.e1-7.

18. Kassem GA, Alzahrani AK. Maternal and neonatal outcomes of placenta previa and placenta accreta: three years of experience with a two-consultant approach. Int J Womens Health. 2013;28(5):803-10.
19. Thurn L, Lindqvist PG, Jakobsson M, Colmorn LB, Klungsoyr K, Bjarnadóttir RI, et al. Abnormally invasive placenta-prevalence, risk factors and antenatal suspicion: results from a large population-based pregnancy cohort study in the Nordic countries. BJOG. 2016;123(8):1348-55.
20. Gyamfi-Bannerman C, Gilbert S, Landon MB, Spong CY, Rouse DJ, Varner MW, et al. Risk of uterine rupture and placenta accreta with prior uterine surgery outside of the lower segment. Obstet Gynecol. 2012;120(6):1332-7.
21. Silver RM, Landon MB, Rouse DJ, Leveno KJ, Spong CY, Thom EA, et al. Maternal morbidity associated with multiple repeat cesarean deliveries Obstet Gynecol. 2006;107(6):1226-32.
22. Senkoro EE, Mwanamsangu AH, Chuwa FS, Msuya SE, Mnali OP, Brown BG, et al. Frequency, Risk Factors, and Adverse Fetomaternal Outcomes of Placenta Previa in Northern Tanzania. J Pregnancy. 2017;2017: 5936309.
23. Sekiguchi A, Nakai A, Kawabata I, Hayashi M, Takeshita T. Type and location of placenta previa affect preterm delivery risk related to antepartum hemorrhage. Int J Med Sci. 2013:24;10(12):1683-8.
24. Omokanye LO, Olatinwo AWO, Salaudeen AG, Ajiboye AD, Durowade KA.A 5- year review of pattern of placenta previa in Ilorin, Nigeria. Int J Health Sci. 2017;11(2):35-40.
25. Jauniaux E, Bhide A. Prenatal ultrasound diagnosis and outcome of placenta previa accreta after cesarean delivery: a systematic review and meta-analysis. Am J Obstet Gynecol. 2017;217(1):27-36.
26. Daskalakis G, Simou M, Zacharakis D, Detorakis S, Akrivos N, Papantoniou N et al. Impact of placenta previa on obstetric outcome. Int J Gynaecol Obstet. 2011;114(3):238-41.
27. Rosenberg T, Pariente G, Sergienko R, Wiznitzer A, Sheiner E. Critical analysis of risk factors and outcome of placenta previa. Arch Gynecol Obstet. 2011;284(1):47-51.
28. Crane JM, Van den Hof MC, Dodds L, Liston R. Maternal complications with placenta previa. Am J Perinatol. 2000;17(2):101-5.
29. Miller DA, Chollet JA, Goodwin TM. Clinical risk factors for placenta previa-placenta accreta. Am J Obstet Gynecol. 1997;177(1):210-4.
30. Wright JD, Pri-Paz S, Herzog TJ, Shah M, Bonanno C, Lewin SN et al. Predictors of massive blood loss in women with placenta accreta. Am J Obstet Gynecol 2011;205(1):38.e1-6.
31. Warshak CR, Ramos GA, Eskander R, Benirschke K, Saenz CC, Kelly TF et al. Effect of predelivery diagnosis in 99 consecutive cases of placenta accreta. Obstet Gynecol. 2010;115(1):65-9.