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METROPLASTY IN REPEATED CESAREAN SECTIONS: DIAGNOSIS AND LONG-TERM SURGICAL OUTCOMES FOR FUTURE REPRODUCTIVE HEALTH **393**

Sodikova D.R., Ermatova H.S.

THE ROLE OF THE VAGINAL MICROBIOME IN THE RISK OF PREMATURE BIRTH **395**

Sultonova J.A.

REHABILITATION AFTER COMPLEX TOOTH EXTRACTION **398**

Aslonova G.B.

IMPACT OF PLACENTAL INVASION (PLACENTA ACCRETA SPECTRUM) ON PREGNANCY AND DELIVERY OUTCOMES IN WOMEN WITH UTERINE SCARS **401**

Kamilova I.A., Shaymurodova M.U.

THE ROLE OF OXIDATIVE STRESS AND CHRONIC INFLAMMATION IN THE PATHOGENESIS OF METABOLIC SYNDROME AND RECURRENT PREGNANCY LOSS **404**

Nabieva R.M., Atakhodjaeva F.A.

ANGIOGENIC AND ANTI-ANGIOGENIC PROFILES IN MILD AND SEVERE PREECLAMPSIA VS GESTATIONAL HYPERTENSION AND HEALTHY PREGNANCIES **406**

Navruzova R.Sadikovna. Sarvarova Maryam Sanjarbekovna.

PSYCHOLOGICAL AND QUALITY-OF-LIFE IMPACT OF ENDOMETRIOSIS IN WOMEN OF REPRODUCTIVE AGE: A COMPREHENSIVE REVIEW **409**

Abdullajonova M.U., Muftaydinova Sh.K., Muminova Z.A.

EFFICACY ASSESSMENT OF PATIENT-CONTROLLED EPIDURAL ANALGESIA IN OPERATIVE GYNECOLOGY FOR PATIENTS WITH HEART FAILURE **411**

Valiev Sh.M, Gaziyev Z.T

COMBINED SPINAL-EPIDURAL ANESTHESIA FOR HYSTERECTOMY IN GYNECOLOGICAL PATIENTS WITH CARDIOVASCULAR PATHOLOGY **413**

Valiev Sh.M, Gaziyev Z.T

TREATMENT OPTIONS FOR UTERINE FIBROIDS: A COMPREHENSIVE REVIEW OF CURRENT AND EMERGING THERAPIES **415**

Mirzaeva D.B., Bekniyazova D.A.

PREVALENCE OF ABNORMAL UTERINE BLEEDING AMONG WOMEN IN UZBEKISTAN: ASSOCIATION BETWEEN SELF-PERCEPTION AND OBJECTIVE PARAMETERS **419**

Mirzaeva D.B., Usmonova D.I.

DIAGNOSTIC ASPECTS OF THE DEVELOPMENT OF FETOPLACENTERAL DISORDERS IN PREGNANT WOMEN WITH COVID-19 **423**

Razzakova N.S., Rakhimjonova H.J.

COMPARATIVE ASSESSMENT OF CERVICAL STUMP AND VAGINAL VAULT PROLAPSE FOLLOWING HYSTERECTOMY **428**

Nigmatova G.M., Begjanova T.U., Sattarova K.A.

DISORDERS OF REPRODUCTIVE FUNCTION IN WOMEN WITH SM PATHOLOGY OF POST-TRAUMATIC GENESIS **430**

Sattarova K.A., Lutfillayeva U.A.

CLINICAL AND LABORATORY ASSESSMENT OF FETAL MEMBRANES IN MISCARRIAGE OF INFECTIOUS ORIGIN **432**

Mirzayeva D.B., Mehmonova I.G.

NEW APPROACHES TO THE TREATMENT OF INFERTILITY OF MIXED GENESIS **434**

Sattarova K.A., Kholmurodova N.N.

THE ROLE OF ENDOVIDEOSURGERY IN THE TREATMENT OF OVARIAN APOPLEXY AND BENIGN OVARIAN CYSTS **436**

Sattarova K.A., Eshpulatova Yu.Sh.

EXACERBATION, CLINIC AND DIAGNOSIS OF RELAPSING CANDIDIASIS VULVOVAGINITIS IN PREGNANT PATIENTS WITH ACQUIRED IMMUNODEFICIENCY VIRUS **438**

Negmadjhanov B.B., Rabbimova G.T., Ochilova U.T.

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IMPACT OF PLACENTAL INVASION (PLACENTA ACCRETA SPECTRUM) ON PREGNANCY AND DELIVERY OUTCOMES IN WOMEN WITH UTERINE SCARS

Kamilova I.A., Shaymurodova M.U.

Tashkent State Medical University, Tashkent Uzbekistan

Abstract: *Placenta accreta spectrum (PAS) is a serious obstetric complication characterized by abnormal adherence of the placenta to the uterine myometrium. This condition has increased markedly among women with a history of prior cesarean section (uterine scar). PAS can lead to postpartum hemorrhage, hysterectomy, bladder injury and even maternal death. Therefore, understanding the etiology and pathogenesis of placenta invasion in women with a uterine scar is of great importance.[1,14,15]. Placenta accreta spectrum is a complex obstetric pathology associated with high maternal morbidity. This relatively recently recognized disorder of placentation results from disruption of the endometrial-myometrial interface of the uterine wall. When first described about 80 years ago, it was mainly associated with manual removal of the placenta, curettage or post-endometritis changes. Superficial damage primarily leads to abnormally adherent placenta and is histologically diagnosed as partial or complete absence of the decidua.[2,13,16] Today, the principal cause of the placenta accreta spectrum is uterine surgery, in particular cesarean section scars that are secondary in nature. If re-epithelialization of the endometrium over the scar does not occur, trophoblast and chorionic tissues may invade the myometrium, including its vascular network, and may extend to the small pelvic organs. The trophoblastic cell changes observed in the placenta accreta spectrum are likely not a primary defect in trophoblast biology that causes excessive myometrial invasion, but rather secondary to an abnormal developing myometrial environment. Pathologists classify the placenta accreta spectrum into three categories: placenta accreta — chorionic villi attach to the myometrium without invading it; placenta increta — chorionic villi invade into the myometrium; and placenta percreta — chorionic villi penetrate through the full thickness of the myometrium. Over the past 35 years several prenatal ultrasound signs of the placenta accreta spectrum have been described, principally loss of the normal retroplacental clear zone (the hypoechoic zone), focal excessive thinning of the underlying myometrium, placental lacunae and placental vascular abnormalities (hypervascularity). The pathophysiological basis of these signs is related to persistent damage of the uterine wall down to the serosa, allowing placental tissue to reach deep uterine vasculature. Adherent and invasive placentation may coexist within the same placental bed and evolve with advancing pregnancy. This may explain why a single ultrasound sign or a fixed combination of signs is not specific enough to differentiate reliably the depth of abnormal placentation or to distinguish adherent from invasive placentation. Correlating pathological and clinical outcomes with prenatal imaging is important to improve screening, diagnosis and management of the placenta accreta spectrum, and standardized protocols should be developed.[3,10,11,12]*

Keywords: *PAS — placenta accreta spectrum , CS— cesarean section (CS) , 3D — 3D ultrasound examination , BPD — biparietal diameter (BPD) , FL— femur length (FL) , UPFBF— uterine-placental-fetal blood flow , DUPFBF — disturbance of uterine-placental-fetal blood flow , CAH — complicated obstetric history , ASH — complicated somatic history , AC — abdominal circumference (AC) , FCI — fetoplacental circulatory insufficiency , CPI — chronic placental insufficiency , BMI — body mass index , MSCT — multislice computed tomography*

Introduction: Placental invasion (placenta accreta) causes massive hemorrhage that threatens the life of both the mother and the fetus. This pathology ranks second among causes of maternal death associated with profuse blood loss. Placental invasion is the most common indication for hysterectomy at delivery and is frequently complicated by coagulopathy, pulmonary injury, infection and acute renal failure, as well as injuries to adjacent organs. The epidemiology of abnormal placental location clearly demonstrates how global trends in obstetrics and changing reproductive attitudes in society are reflected in the structure and frequency of severe pregnancy complications. Over the past 30–40 years worldwide there has been not only a steady increase in cases of abnormal placental attachment but also a significant change in their typical "profile." While placenta previa or accreta were rare in the mid-20th century, today these pathologies are no longer exceptional even among young women and primigravidas [4,8].

This dynamic is directly linked to radical changes in delivery practice: the increasing prevalence of cesarean section, active adoption of new fertilization technologies and the expansion of indications for various uterine surgical interventions have led to a continual "rejuvenation" and expansion of the risk group. In some developed countries the cesarean section rate already exceeds 30–40% of all deliveries and the number of women with more than one uterine scar is steadily growing. As a result, abnormal placental location is increasingly encountered even in patients without clear traditional risk factors such as grand multiparity, advanced maternal age or multiparity [5].

Geographic analysis confirms that the highest incidence rates are observed in countries and regions with high surgical activity in obstetrics and reproductive medicine. For example, multicenter studies report that the frequency of placenta previa ranges from 0.3% to 0.8% of all pregnancies, while the incidence of placenta accreta varies from about 1 in 500 to 1 in 2500 births depending on population characteristics and diagnostic criteria. On the other hand, data from developing countries show substantial variability, where limited access to modern diagnostics and lack of systematic registration may lead to underestimation of the true prevalence of the pathology [6,9].

The influence of social factors must also be considered: trends toward postponing first pregnancy, an increase in the number of women of older reproductive age, and higher use of assisted reproductive technologies — all create additional conditions for formation of new risk groups. Whereas abnormal placental location was previously mainly associated with "older" pregnant women or patients with an adverse obstetric history, today complications are increasingly recorded in young women, as confirmed by both domestic and foreign authors [7,14].

Relevance: The clinical significance of the placenta accreta spectrum (PAS) is underscored by its increasing incidence and the severe complications it engenders. Epidemiological data indicate that the incidence of PAS has risen from approximately 1 in 2,500–4,000 deliveries in the 1980s to roughly 1 in 500–1,000 deliveries at present. This trend is associated with the growing rate of cesarean sections, advances in assisted reproductive technologies, and increasing maternal age. PAS not only poses

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a life-threatening risk to the mother but also has detrimental consequences for future reproductive health.

Material and methods: The study population comprised pregnant women who were followed during pregnancy and delivery at the Obstetrics and Gynecology Complex of the 2nd Clinic and at the Women's Health Center of the 2nd Clinic, Tashkent State Medical University; deliveries were managed in the 1st and 2nd obstetric units of these clinics.

We retrospectively and prospectively reviewed the medical records of 30 women with a uterine scar and sonographically diagnosed placental invasion during 2024–2025. A total of 30 deliveries were analyzed. For comparative analysis the patients were assigned to two groups: Group 1 included 15 women with a uterine scar but without placental invasion; Group 2 included 15 women with a uterine scar and confirmed placental invasion. The inclusion criterion was sonographic evidence of placental invasion during pregnancy in women with a uterine scar.

Study methods comprised retrospective and prospective analysis of the course of pregnancy in the context of placental invasion on a uterine scar (history taking), obstetric and gynecologic examination, general clinical assessment, ultrasound examination, biochemical tests and pathoanatomic (histopathological) investigations.

Recorded characteristics of the pregnant women included age, body mass index (BMI), parity, complicated obstetric history, complicated somatic history, and presence of placental invasion on a uterine scar. In our study we evaluated the course of pregnancy in women with scar-associated placental invasion, the presence of extragenital comorbidities, gestational age at delivery and indications for delivery (spontaneous vaginal delivery or cesarean section). Neonatal outcomes were assessed by birth weight, Apgar score, requirement for neonatal intensive care, and the need for transfer to second-level rehabilitation.

For the retrospective component we examined medical histories of patients aged 18 to 42 years with uterine scars and placental invasion. The mean maternal age was 27.4 ± 6.1 years. The largest age group comprised women aged 21–30 years (see Table)

AGE GROUPS	18-20	21-25	25-30	31-35	36-40	41-42
ABS	2	8	9	4	3	2
%	6,7%	26,7%	30%	13,3%	10%	6.7%

By parity, the pregnant women were distributed as follows: women with one uterine scar constituted 46% of the cohort, while women with two or more uterine scars constituted 54%.

To assess the condition of the fetoplacental system and to identify placental dysfunction in pregnant women with a uterine scar and suspected placental invasion, ultrasound examination including color Doppler assessment of the maternal–placental–fetal circulation was performed in the second and third trimesters. Examinations were conducted using Sonoscape S-22 and Mindray DC-30 ultrasound systems with a 3.5 MHz transducer.

Fetal biometric parameters recorded included biparietal diameter (BPD), abdominal circumference (AC), femur length (FL), placental location and thickness, fetal growth restriction, and the amniotic fluid index.

Scanning protocol: Initially, a longitudinal scan was performed to determine fetal presentation and position. The transducer was then oriented strictly perpendicular to the frontal axis of the fetal head and a transverse scan was obtained to visualize midline brain structures (M-echo). BPD was measured only when the M-echo demonstrated symmetric positioning of the two parietal bones. BPD measurement was taken from the outer surface of the proximal parietal bone to the inner surface of the distal parietal bone. For BPD, the largest biparietal diameter was selected from several measurements. Femur length (FL) was identified in the proximal portion of the lower limb and measured along the long axis of the visualized femoral diaphysis.

Color Doppler mapping was produced to evaluate blood flow in the uterine arteries, the umbilical arteries, and the middle cerebral artery. Doppler indices were then compared with the course and outcome of pregnancy as well as with fetal status. Doppler assessment of uteroplacental–fetal blood flow was performed with the pregnant woman in the supine position, since placing the patient on her left side may reduce the sensitivity and specificity of Doppler for detecting fetoplacental blood-flow disturbances and for prognosticating perinatal outcomes.

Hemodynamic disturbances within the mother–placenta–fetus functional unit are considered a principal pathophysiological mechanism linking a variety of pregnancy complications with impaired fetal condition and development.

To improve diagnostic quality and the predictive accuracy of Doppler studies for circulatory disturbances in the mother–placenta–fetus system, we used the following classification [68,89], which defines three degrees of circulatory impairment: Grade 1a — hemodynamic disturbance confined to the maternal–placental circulation;1b — disturbance confined to the fetal–placental circulation. Grade 2 — disturbances are present in both circulations but do not reach clinically significant values. Grade 3 — severe disturbances of the fetoplacental circulation with fetal life at risk.

Results: Dopplerography of the maternal–placental–fetal circulation revealed abnormalities in both study groups. Women without circulatory disturbances comprised 35% of the cohort. Circulatory disturbances classified as grade 1a and 1b occurred with approximately equal frequency. Grade 2 disturbances were less frequent. Although grade 3 disturbances were rare, they were observed; in these cases severe adverse outcomes were recorded, including antenatal fetal death. (The original abbreviation "XOQS" appears in the source text and was not expanded; it is retained here as presented.)

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Results: The frequency of placental invasion identified at delivery increased 5.9-fold over the past 10 years ($p < 0.001$); this steady rise correlated with an increase in the rate of operative deliveries. The degree and depth of placental invasion at the time of delivery significantly affected the volume of blood loss ($p < 0.001$) and increased the risk of hysterectomy ($p < 0.001$). The postoperative course in Group 1 (women with a uterine scar but no placental invasion) was milder compared with Group 2 (women with a uterine scar and placental invasion).

Conclusion: Early diagnosis of placental invasion is feasible only after 16 weeks' gestation. The volume of blood loss during reconstructive surgical management at delivery is directly related to the depth and extent of placental invasion into the uterus. A system of measures to improve pregnancy and delivery outcomes in women with placenta invasion on a uterine scar should include: identification of the high-risk contingent at the first antenatal registration; prompt referral to a specialized center for diagnostic confirmation after high risk has been identified, together with patient counselling about potential complications and their prevention; intensified surveillance of high-risk pregnancies; timely hospitalization (not delayed before 34 weeks when indicated); and an individualized approach to the timing and mode of delivery to minimize the risk of complications.

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