Inconsistency of Seams on the Uterus after Cesara Section Problems and Ways of their Solution

Farida Mirzaevna Ayupova[1], Nargiza Mirzaevna Umarova[2], Gulnara Makhsudovna Nigmatova[3], Dilnoza Javdatovna Saidjalilova[4], Feruza Bakhtiyarovna Mirodilova[4]

[1]MD, Professor, Head of the Department of Obstetrics and Gynecology No.1, Tashkent Medical Academy, Farobiy 2, Tashkent city, Uzbekistan, 100109, [2]MD, PhD, Deputy Director of the Republican Perinatal Center, 223 J. Abidova street, Tashkent, Uzbekistan, [3]MD, PhD, Associate Professor of the Department of Obstetrics and Gynecology No.1, Tashkent Medical Academy, Farobiy 2, Tashkent city, Uzbekistan, 100109, [4]MD, PhD, Associate Professor of the Department of Obstetrics and Gynecology No.1, Tashkent Medical Academy, Farobiy 2, Tashkent city, Uzbekistan, 100109, [5]MD, PhD, Assistant of the Department of Dermatovenereology, Tashkent Medical Academy, Farobiy 2, Tashkent city, Uzbekistan, 100109

Abstract

Cesarean section is currently the most common delivery surgery, which leads to the occurrence of diverse in nature changes from the internal genital organs. This primarily concerns the uterus, where a postoperative scar forms in the area of the former incision. This article presents histomorphological and immunohistochemical signs of a uterine scar after cesarean section in the lower uterine segment. The features of tissue regeneration in the area of the incision made on the uterus after cesarean section were revealed. Some method for diagnosing and treating uterine scar failure are also presented.

Keywords: *Cesarean section, uterine scar, ultrasound, hysteroscopy.*

Introduction

In all countries of the world there is a steady increase in the frequency of Caesarean section (CS). Over the past few decades, the frequency of this operation in the world has more than doubled and in Uzbekistan is on average about 24%. According to data published in world literature, one in four women has a cesarean section and a fifth has complications in the early postoperative period and in the long term.

The increase in abdominal delivery is due to the expansion of indications to it from the fetus, an increase in the number of older primiparas[4], with existing comorbidity, and the emergence of new reproductive technologies[25]. At the same time, according to the literature, a small number of criteria or features of the course of the postoperative period in patients with inconsistency of sutures on the uterus after cesarean section were isolated[34].

Currently, issues of repair of the suture on the uterus after CS, which largely determines the favorable course of subsequent pregnancies and childbirths[5], are becoming increasingly urgent[25]. A study of literature data showed a significant variability of information about the causes of the formation of an insolvent scar on the uterus.

Numerous studies have found that the increase in infectious[1] complications in obstetrics is largely due to an increase in the frequency[20] of cesarean section[24]. The increase in abdominal delivery is explained by an increase in severe somatic and obstetric pathology, frequent repeated operations, an increase in the age of primiparas in combination with other factors of obstetric and gynecological history[6]. A little justified tendency to an increase in the frequency of abdominal delivery during pregnancy and childbirth complications or severe somatic pathology poses a risk of secondary or late uterine suture failure[7].

Purulent-inflammatory complications after cesarean section are 8-10 times more common than after spontaneous[24] delivery and occupy one of the first places in the structure of maternal morbidity and mortality[32]. The frequency of peritonitis after CS ranges from 0.2 to 1-1.5% [1, 20, 35].

A large number of factors influence the formation of a suture on the uterus after CS: the presence of somatic and gynecological diseases (especially chronic inflammatory diseases), the course of pregnancy, the duration of labor, the technique of performing CS, the duration of surgery, the amount of blood loss and the adequacy[44] of its replenishment[2], and the course of the postoperative period[8]. It is believed that the main reasons leading to the development of an insolvent uterine scar are postoperative purulent-septic complications[17] in the early postoperative period[17], termination of pregnancy or the presence of any intrauterine interventions during the first year after CS [19]. Many authors consider intrauterine interventions after CS as a possible cause of direct trauma to the area of the postoperative[22] suture and violation of the formation of a scar on the uterus[8],

It is known that the postpartum period, even in the physiological course, and especially in the presence of risk factors, is favorable for the development of infectious complications, with surgical delivery, the postoperative period and associated additional significant risk factors (edema, ischemia, tissue destruction in the seam area, the presence of microhematomas, foreign suture material). Therefore, if the frequency of endometritis after spontaneous labor on average is 5%, then after surgical labor it varies significantly (from 2 to 54.3%), averaging up to 15% [34]. The consequences of severe endometritis are devastating for women's reproductive health: these are chronic endometritis, chronic pelvic pain syndrome, menstrual dysfunction, miscarriage, and secondary infertility [23].

According to some authors, the structure of complicated forms of purulent inflammation was dominated by complications caused by prolonged wearing of intrauterine contraceptives (44%), and postpartum diseases took the second place (16.9%) [50], while the risk factors for the development of purulent-septic puerperal complications were : "Inflammatory" obstetric and gynecological history (endometritis after childbirth - 35.3%; mastitis - 8.2%; wound infection - 23.5%; endometritis after abortion - 18.2%; cervical erosion - 23.6% acute and chronic salpingoophoritis - 12.7 and 29.1%, respectively); a previous history of infertility (21.8%); wearing an intrauterine contraceptive prior to a real pregnancy (4.1%); previous or existing chronic diseases of the bronchopulmonary system (29%); placenta previa in incision (38.6%); surgery in conditions of sharp thinning of the lower segment (22.7%); the presence of technical errors during the operation (using the Gusakov method - 72.7%; the use of rough manual method for removing the head - 13.6%; the use of a continuous suture for suturing the uterus - 38.6%; the use of reactogenic suture material - 9%; inadequate hemostasis - 9%); the duration of the operation is more than 2 hours (4.5%); the presence of pathological blood loss (9%).[51]

As you know, the optimal technique for conducting COP is still debatable. Most obstetrician-gynecologists are supporters of a "blunt" uterine dissection after a small incision is made with a scalpel according to the Gusakov[47]– Zanchenko method and less commonly, the uterus is dissected using scissors according to Derfler[49]. V.A. Ananyev et al. Found that biopsy specimens during surgery according to Derfler do not affect the most intracellular systems that are most sensitive to damage. Moreover, maintaining the integrity of the cell membranes and the respiratory apparatus of the cell ensures the maintenance of organelles homeostasis and oxidative phosphorylation[16]. At the same time, in the Gusakov – Zanchenko section, deep, mostly irreversible, ultrastructural changes prevail, reflecting the destruction of not only the trophic, but also the contractile apparatus of the myometrium cells [41].

At the same time, there is evidence that the determining factor in the formation of a wealthy scar is the nature of the healing of the wound surface with the predominant formation of muscle or scar tissue. So, if healing proceeds according to the type of primary (with the predominant formation of muscle tissue), then such additional risk factors as placenta localization, the period of time after CS, the production of medical abortions after CS in the final outcome are of unprincipled importance[46]. Gabidullina R.I. et al., based on histochemical studies, noted the complexity of the healing process of the uterus after CS surgery [13]. In later works, it was shown that during the healing of the uterus, two types of regeneration can be observed: substitution, which is defective regeneration, and restitution[45], in which the scar is not clinically detected. Naturally, the replacement of a myometrial defect not with an inert connective[117], but with smooth muscle tissue exhibiting contractile activity is more adequate[146].

Undoubtedly, the main etiological factor in the formation of an inferior uterine scar and generalization of infection is postpartum endomyometritis[15]. Features of the clinical picture of postpartum endometritis are: primary chronic course or short acute phase of

the manifestation of the disease, often hidden behind various "masks" (lochio and hematometer); further erased clinical course, caused, as a rule, by the use of massive antibacterial therapy (on average, two courses - 4–5 antibiotics) and various method of local uterine sanitation: vacuum aspiration, curettage of the uterus, lavage (in 75.5% of puerperas); latent or late (after discharge) manifestation of the inflammatory process with a undulating course of the disease[2] (remission - exacerbation); the predominance of infiltrative and necrotic forms of inflammation in the absence of typical clinical and laboratory signs of infection[3].

All diseases, including postpartum and postoperative, have specific symptoms, reflected in subjective complaints and data from an objective study.

The development of inflammatory and postoperative complications always "goes through" successive stages and is clearly identified when collecting information about the history of the disease, provided that the doctor knows the possible course of the disease and poses directed questions (the presence of risk factors, the initiation of the disease, its development, duration and frequency of hospitalizations, the nature and duration of treatment). Moreover, almost always, even after a certain time, you can identify the cause of the formation of an insolvent scar after CS (more often - endometritis, less often - obvious technical errors of the operation).

It should be noted that information on technical difficulties in performing the operation (difficulties in removing the fetal head, tears in the lower segment, difficulties in hemostasis, additional suturing, etc.) is almost impossible to obtain from extracts from the maternity hospital, while at 40 % of patients with an unsuccessful suture on the uterus after CS, if the extract contains a standard description of the operation, hematomas of parametria and/or hematomas of the postbubble tissue were revealed clinically and by ultrasound, and during the repeated operation[25], multiple unsystematic ad mass ligatures outside the suture line on the uterus (more often in the area of vascular bundles with ligation of one or both uterine arteries) [26].

Risk factors for the formation of an insolvent uterine scar after CS[48]:

- The patient has an "inflammatory"[22] obstetric and gynecological history (endometritis, mastitis, wound infection after previous birth, postabortion endometritis; endocervicitis, acute and chronic salpingo-oophoritis, infertility)[47];
- The presence and exacerbation during pregnancy of chronic inflammatory extragenital diseases (especially diseases of the bronchopulmonary system), the presence of anemia;
- Long-term operation, the presence of pathological blood loss;
- Placenta previa in the incision, the operation in conditions of sharp thinning of the lower segment in patients with clinically narrow pelvis ("low" or cervical CS);
- Urgency of the operation, especially the need for rapid delivery due to the development of acute fetal hypoxia, including in patients with premature detachment of the normally located placenta (in this group, the highest perinatal losses are 154 ‰);
- The use of a continuous suture for suturing a wound on the uterus;
- The use of Gusakov's technique, rude manual techniques for removing the head.

Clinical manifestations of a failed seam (scar) on the uterus after CS: Since the main reason for the formation of an unsuitable suture (scar) on the uterus after CS is endometritis, these patients are characterized by a complicated postoperative period and a later discharge from the obstetric hospital (on the 10-11th day).[2] Unfortunately, at present, postpartum endometritis in general and postpartum endometritis after CS[3], in particular, in most patients has a "erased"[9], subclinical course and is characterized by the following,[49]:

- At best, a short "acute" phase of the manifestation of the disease with a further "erased" clinical course of endometritis, often "hiding" under the masks of uterine subinvolution and lochio-hematometers (with a mild or mild inflammatory-intoxication syndrome);
- Late manifestation of the disease, often after discharge from the maternity hospital;
- The appointment in the postoperative period of long or repeated courses of antibiotic therapy;
- Insufficient effect of conservative treatment, which is often supplemented by the use of various instrumental method of sanitation of the uterus: vacuum aspiration, diagnostic curettage, hysteroscopy, lavage (47%);
- The wave-like course of the disease during its progression and episodes of deterioration of the state

of puerperas after the end of antibiotic treatment (short-term effect).

– The predominance of infiltrative, necrotic forms of inflammation in the absence of both typical clinical and laboratory signs of acute inflammatory process. Due to the absence of typical signs of purulent inflammation, necrotic forms of panmetritis were especially difficult for clinical and ultrasound diagnosis and were detected only with hysteroscopy;

– The rapid progression of the disease, despite its "erased" course.

Complaints upon admission of patients do not reflect the actual severity of their condition, therefore, a thorough history and examination are crucial for making a diagnosis. With a vaginal examination, the size of the uterus exceeds the value corresponding to certain days of the postpartum period, its consistency is soft. In most patients, the main pathological changes are observed in the area of the operation zone, i.e., the area of the anterior wall of the uterus, isthmus and neck, while the severity of inflammatory-necrotic changes in the anterior wall of the uterus and isthmus reflects a lag in the formation of the cervix (the neck is free in the form of " sails "hangs in the vagina, freely passes 1-2 fingers). The presence of infiltrates (hematomas) behind the gallbladder is a characteristic unfavorable clinical sign, indicating the development of secondary failure of sutures on the uterus. Even in cases where infiltration is not determined, the uterus is fixed to the tissues of the anterior abdominal wall in the region of its lower segment.

At present, also the features of the course of postpartum infectious diseases are the "worn out" laboratory picture. It is inappropriate to focus on leukocytosis as the main marker of the inflammatory process (it is observed only in 11% of postpartum patients, in the rest the indicators do not exceed the normative). More typical for puerperas with endometritis are an increase in ESR, the presence of moderate anemia (on average, 97.8 g/l), hypo- and dysproteinemia, which is expressed in a decrease in the concentration of albumin and an increase in globulins, as well as an increase in the concentration of C-reactive protein (72, 3%). Leukopenia is a poor prognostic sign indicating a septic course of the process. In severe forms of postpartum infection, systemic disorders prevail, for example, severe hemostasiological disorders (with the presence of uterine and pelvic vein thrombosis), immune disorders with damage to the receptor link (an excessive pathological concentration of antibodies to γ-interferon receptors appears, blocking receptors). Sepsis is a manifestation of a systemic inflammatory response, which is based on a generalized inflammatory reaction[9] - a kind of "malignant intravascular inflammation", leading to endothelial damage and the development of multiple organ failure [42].

Ultrasound diagnosis of insolvent uterine scar: The leading ultrasound method for identifying postpartum endometritis and especially a failed uterine scar[15]. To date, the most informative criteria for the condition of the scar are: the structure of the myometrium, the presence of areas of high acoustic density, vascularization of the uterine segment[38]. The results of echoscopy coincided with intraoperative data in 80.3% of cases. False positive results, when with unsatisfactory echographic parameters, the scar on the uterus was without features, amounted to 12.1%. False negative, when an insolvent uterine scar was an accidental find during surgery, in 7.6% of cases [40].

Analysis of echograms in postpartum patients with complications after CS revealed a number of common characteristic signs indicating the presence of endomyometritis and impaired repair processes in the suture or scar on the uterus:

– Subinvolution of the uterus, an increase and expansion of its cavity;

– The presence in the uterine cavity of inclusions of various sizes and echogenicity (intracavitary serous fluid, pus); the presence on the walls of the uterus of linear echopositive structures (in the form of an intermittent or continuous contour), reflecting the imposition of fibrin in all patients;

– Heterogeneity of the myometrium in the region of the scar, anterior and posterior walls of the uterus.

The main ultrasound signs of a failed scar on the uterus after CS are:

– of the cavity - the presence of a "niche" conical shape with a depth of 0.5 to 1.0 cm or more, when the top of the niche reaches the outer contour of the anterior wall of the uterus;

– A local change in the structure of the myometrium in the region of the scar or a diffuse change in the myometrium of the anterior wall of the uterus in the form of multiple inclusions of reduced echogenicity with fuzzy contours (microabsection sections);

– Local circulatory disturbance in the scar area - the absence of a diastolic component of blood flow (focal necrosis) or a sharp decrease in blood supply to the entire front wall of the uterus, when it was not possible to visualize the blood flow velocity curves;
– A sharp decrease in blood supply to the anterior wall and an increase in blood flow in the region of the posterior wall.

The following echographic signs are prognostically unfavorable, indicating complete failure of the suture (scar) on the uterus after CS:

– Expansion of the uterine cavity over the entire length of more than 1.5 cm;
– Sharp deformation of the cavity in the scar area: the presence of a cone-shaped niche, the apex of which reaches the outer contour of the anterior wall of the uterus (complete divergence of sutures);
– Diffuse change in the structure of the myometrium of the anterior wall of the uterus in the form of multiple inclusions of reduced echogenicity with fuzzy contours (areas of microabsection);
– A sharp decrease in blood supply to the anterior wall of the uterus (it is not possible to visualize blood flow velocity curves) with an increase in blood flow in the region of the posterior wall (systolodiastolic ratio <2.2 and resistance index> 0.5);
– The presence of echographic signs of hematomas, abscesses or infiltrates in the parameters, pelvis and abdominal cavity.

The development of criteria for the viability of the uterine scar after cesarean section using ultrasound has been the subject of many works by domestic and foreign authors [25, 38,40]. In the study, pay attention to the thickness of the lower uterine segment, its uniformity, the presence or absence of acoustic seals, their heterogeneity, fragmentation. According to the researchers, on the basis of the obtained echograms, scars with a total thickness of less than 4 mm, individual thinning sites (less than 3 mm), heterogeneous scars with a large number of acoustic seals, indicating the growth of connective tissue in the thickness of the anterior wall of the lower uterine segment, are considered insolvent. Almost all authors note the high information content of this method for the study of the uterine scar[2].

Some authors noted that hysteroscopy is indicated for all obstetric patients with endometritis, and the sooner this intervention is undertaken (with a diagnostic or therapeutic diagnostic purpose)[3], the better the outcome of the disease. The only exceptions are patients who have indications for surgical treatment (panmetritis, purulent lesions of the appendages and fiber, the presence of complete failure of the suture (scar) [38]on the uterus according to ultrasound), and hysteroscopy in this case does not make sense[40].

Hysteroscopy allows not only to identify postpartum endometritis and determine its shape (fibrinous, purulent, due to residues of placental tissue, necrotic), determine the condition of the suture on the uterus after CS, but also sanitize the uterine cavity and conduct differential treatment [50].

Morphological and histochemical changes in the uterine scar after CS: The presence of morphofunctional "solvency" of the uterine scar is one of the main criteria that determine[28] the outcome of pregnancy and childbirth[30] in women with a history of cesarean section[32], however, due to the complexity of the object of study, data on factors and mechanisms to date underlying the formation of a full scar on the uterus is not enough. Uterus scar formation should be considered as part of the modern concept of wound healing.

The wound and reparative processes are a complex of successive[30] local changes and the numerous common reactions[19] associated with them, which can be combined in three stages regardless of the location of the wound: the stage of inflammation[29], the stage of repair and the stage of remodeling [40]

By remodeling, the authors understand not only the reorganization of the scar, but also (to varying degrees)[28] the morphofunctional[29] restoration of lost tissue[32] (smooth muscle, epithelial, mesothelium, etc.). The wound process, which results in the formation of scar tissue, is accompanied by complex multicomponent interactions of cellular elements[30]: it is carried out mainly with the participation of mast cells, lymphocytes, macrophages and fibroblasts[29]. The interconnection of cells is carried out through cytokines[32] and growth factors: platelet growth factor, transforming β-growth factor, main fibroblast growth factor, epidermal growth factor, vasculoendothelial growth factor, etc.[32] . Studies show that macrophage-fibroblastic and collagen-fibroblastic interactions, through which the feedback between the breakdown[28] and production of collagen is realized, play a key role in the regulation of reparative

growth of connective tissue and sclerogenesis[29]. The optimal course of intercellular interactions underlies physiological neoangiogenesis, since it is the formation of small vessels that is necessary for the full regeneration of the connective tissue and muscle components in the initial stages of wound healing[2]. Hemomicro circulation determines[36] the level of tissue metabolism and oxygenation: the more severe the defeat of the individual components of the microcirculation[46], the more pronounced are violations of the transcapillary exchange and rheological properties of the blood [29].

In a number of works devoted to the study of reparative processes after various operations on the uterus, it has been shown that during the healing of the dissected uterine wall, two types of regeneration can be observed: both substitution (inferior regeneration) and restitution, in which the scar is not clinically detected [36]. Replacing a defect not with an inert connective, but with smooth muscle tissue exhibiting contractile activity is more advantageous. The morphofunctional usefulness of the uterine scar after cesarean section surgery largely depends on the course of the processes of its formation.[5] The various clinical manifestations of anatomical and functional inferiority of the uterus due to inferior wound healing after cesarean section are combined [29]with the concept of "operated uterus disease", the most severe manifestation of which is uterine lacerations in the scar during subsequent pregnancies and childbirths[32].

Features of the repair of uterine tissue determine the formation of the solvency of the scar after CS. Many factors influence the course of repair in the uterus, for example, anemia, preeclampsia, inflammatory processes, immune status, technique of surgical intervention and suture material used, duration of surgery and blood loss, postoperative period, and the time until the next pregnancy [11]. A number of complications (intrauterine interventions, inflammatory diseases of the genitals, intra- and postoperative complications) 1.5 times increase the risk of scar failure [14]. Many researchers have found that the formation of morphofunctional usefulness of the myometrium in patients with CS by a transverse section in the lower segment of the uterus occurs within 1–2 years after surgery [15].

An analysis of the literature on wound repair issues showed that individual, genetically determined features of the course of the wound process, as well as the nature of abdominal delivery, can be considered promising areas in this area. Today, the field of molecular genetic research continues to develop: numerous studies have shown the role of genetic factors, as well as gene polymorphism in the healing process [9]. A certain effect on the nature of wound healing on the uterus can be exerted by the time of the operation of cesarean section - in a planned or emergency manner. The effect of this indicator on the outcome of labor for the fetus is shown.

According to the results of the analysis of significant clinical and statistical material, the most favorable outcome for the fetus is observed with the so-called planned abdominal delivery, when the operation is performed with the beginning of regular labor. This is explained by the influence on the fetus of mechanical and metabolic factors from the mother's body, which lead to a powerful release of stress hormones that provide more optimal conditions for the adaptation of a newborn to extrauterine life [16]. The results of modern and, unfortunately, few studies indicate that the nature of the operation of cesarean section can play an important role in the process of scar formation on the uterus. The potential impact of cesarean section time on wound repair on the uterus can be assumed on the basis of modern ideas about the reparative process described above, taking into account the specific changes occurring in childbirth both at the body level and at the level of the uteroplacental-fetal complex[43]. A change in the reactivity of the immune system during childbirth has been shown to predominate over proinflammatory factors, which leads to structural changes in the cervix, rupture of the membranes and initiation of labor [34].

With the development of regular labor in the myometrium of the lower uterine segment, an increase in the content of leukocytes that produce interleukin (IL) -1β, tumor necrosis factor alpha (TNF-α) and IL-6 is observed [33]. The migration of neutrophils and macrophages into the myometrium during childbirth is due to increased production of chemoattractants for monocytes (MCP-1) and IL-8 [31]. IL-1β and TNF-α stimulate the formation of endogenous prostaglandins due to activation of phospholipid metabolism [43]. IL-6 increases the expression of oxytocin receptors in the myometrium [13]. In addition, IL-1β and TNF-α increase the production of matrix metalloproteinase-9 by myometrial cells, which can play an important role in the process of placenta separation[13]. A study of the concentration of endothelial cell adhesion molecules in the lower uterine segment showed that during urgent delivery there is a significant increase in the level of the intercellular adhesion-1 molecule (ICAM-1),

which provides adhesion of neutrophils, monocytes and lymphocytes to activated vascular endothelium with subsequent extravasation and migration to the lesion inflammation. ICAM-1 is also involved in cell contact interactions in immune responses: a T-lymphocyte with a monocyte, a cytotoxic T-lymphocyte with a target cell [13]. In a study on the mechanical properties of the lower uterine segment during full-term pregnancy and urgent delivery, it was shown that the viscoelastic properties of the myometrium change during childbirth, while the lower uterine segment becomes denser [27]. The time taken to perform a caesarean section affects the thickness of the lower uterine segment in subsequent pregnancies: with abdominal delivery during pregnancy, the thickness of the lower uterine segment, according to ultrasound, is 2.4 mm (interquartile range from 2 to 3.2 mm), while as with the operation in the active phase of labor, 3.1 mm (interquartile range from 2.5 to 3.9 mm)[37]. It has been suggested that the migration of leukocytes into the uterine wall during childbirth and their production of proinflammatory[26] cytokines play an important role not only in initiating and maintaining normal labor[14], but also in the process of repairing the wound surface in the placental area in the postpartum period, however, detailed studies in this direction[2] have not been carried out[9].

Therefore, it is necessary to further study the process of wound repair on the uterus using modern molecular genetic[9] and immunological method[15], which will expand the existing understanding of the course of the wound process with the subsequent development[29] of new diagnostic method, as well as optimizing the prevention of "insolvency" of the uterine scar after cesarean section[14] .

In their observations, the authors noted the expression of vascular endothelial growth factor VEGF and epidermal growth factor receptor EGFR, which is also common for transforming growth factor[29]. It is these growth factors that provide mitogenic activity of endothelial cells and fibroblasts, cell migration and differentiation, stimulate angiogenesis and the development of granulation tissue, which occupied a significant amount of the forming scar[31]. It should be noted that the presence of extensive areas of granulation tissue in the period of 6 months[14] or more after cesarean section indicates a slow healing process[27]. This fact may be associated with massive areas of necrosis[19]. A large volume of necrotic tissue slows down the process of granulation formation and their maturation into connective tissue, the organization of the extracellular matrix, scar formation and, moreover, its remodeling. The formation of foci of disorganization in fibrous and smooth muscle tissue lacking cellular components, collagen, fibrin, blood vessels, acid mucopolysaccharides and glycoproteins, proteinases, with perifocal expression of the macrophage marker CD68 may be associated with the outcome of persistent granulomatous inflammation[39].

Hence, the main reason for the formation of an insolvent uterine scar after CS is the primary severe inflammatory or traumatic necrotic damage to the endo- and myometrium with delayed and inferior maturation of connective tissue[29].

Given the high frequency of postpartum complications in women after cesarean section, as well as the absence of a tendency to reduce it[31], there is a need to determine preclinical markers of complications[19], in our case, uterine suture failure, to develop an algorithm for managing them[39], depending on the degree of risk of complications and new highly effective method of surgical treatment that can preserve the reproductive function of women.

The significance of immunological and biochemical changes in the development of uterine suture failure after cesarean section in their functional relationship, as markers of preclinical diagnosis, remains virtually unexplored, which determines the urgency of the problem and the need for targeted research in this direction.

The diagnosis of uterine scar (suture) on the uterus and the possibility of reconstructive surgery remain poorly understood.

Discussion

Diagnosis of a failed suture (scar) on the uterus after CS is always difficult, therefore, the diagnosis should be verified during a consultative examination after a sequential comprehensive examination including a clinical examination (special attention is paid to a thorough medical history, study of the features of the course of a previous pregnancy, surgery and postoperative period, analysis of discharge from obstetric and other hospitals), ultrasound and endoscopy. Even if it is possible to use the most modern research method, the main diagnostic method that determines the professional qualifications, the level of clinical thinking of a doctor and the subsequent direction of diagnostic measures is clinical.

Currently, laparoscopic metroplasty is known only at the stage of pregnancy planning with localization of the postoperative scar within the lower segment of the uterine[27] body, while hysteroresection with "smoothing" of the edges of the niche and ablation of the endometrium in the defect zone in patients not planning pregnancy[16], which allows to effectively save woman from complaints and significantly improve the quality of her life [40].

Conclusion

In view of the increase in the frequency of CS and its complications, it becomes important to search for prognostic criteria for the usefulness of the uterine scar in the postpartum period. However, the currently accepted[38] method for assessing the usefulness of the scar are not sufficiently informative and are often invasive, which limits their application. Currently, the issues of diagnosis and reconstructive surgery for an inconsistent uterine scar after a cesarean section in the postpartum period, as well as the volume and advisability of surgical intervention in patients with this pathology, have not been fully studied.

Ethical Clearance: No ethical approval is needed.

Source of Funding: Self

Conflict of Interest: Nil

References

1. Abramchenko V.V. KDF, KED. Purulent-septic infection in obstetrics and gynecology. St. Petersburg. Special Literature. 2005.
2. Ailamazyan E. K. KTU. Features of myometrium repair after cesarean section//Obstetrics and gynecology. 2008; 1: p. 34-36.
3. Ed. E.K. Aylamazyan VIKVERGMSM. Obstetrics: National leadership. GEOTAR-Media. 2011; 1200.
4. KVI. Alternative delivery of pregnant women with an operated uterus. Journal of Obstetrics and Women's Diseases. 2003; 1(1): p. 20-25.
5. A AV.. Spontaneous birth in pregnant women with a scar on the uterus. Russian Bulletin of the obstetrician-gynecologist. 2002; 2(4): p. 30-31.
6. Ananyev V. A. PNM, CEA. Complications and morbidity after cesarean section in the postpartum and late periods. Obstetrics and Gynecology. 2005; 2: p. 52-54.
7. OV. B. Modern possibilities of assessing the condition of the scar on the uterus after cesarean section. Obstetrics and gynecology. 2009; 5: p. 7-9.
8. Buyanova SN PN. nsufficient uterine scar after cesarean section: diagnosis, management tactics, reproductive prognosis. Russian Bulletin of the Obstetrician-Gynecologist. 2011; 4: p. 36-8.
9. Buyanova SN SNCMMMTYPNBR. Modern method for diagnosing insolvency of sutures or scar on the uterus after cesarean section. Russian Bulletin of the Obstetrician-Gynecologist. 2013; 1: p. 73-7.
10. Buyanova S.N. YNV. Puchkova N.V. and other Modern possibilities for diagnosing the solubility of the uterine scar after organ-saving operations. Russian Bulletin of the obstetrician-gynecologist. 2014; 2: p. 94-100.
11. Gabidullina RI FIKAADRP. Clinical and morphological parallels in assessing the condition of the uterine scar after cesarean section. Kazan medical journal. 2002; 6: p. 424-9.
12. Ed. IN AND. Kulakova IBMGMS. Gynecology: National leadership. M: GEOTAR-Media. 2007.
13. Gurtovoi B.L. VLV, KNM. Clinical and immunological features of puerperas with postpartum endometritis. obstetrics and Gynecology. 2006; 1: p. 30-34.
14. Gustovarova TA IAKS. Management of pregnancy and childbirth in patients with a scar on the uterus after cesarean section. russian Bulletin of the Obstetrician-Gynecologist. 2007; 4: p. 45-9.
15. al.] GAV[. Diagnosis of the condition of the scar on the uterus in pregnant women undergoing a cesarean section. Obstetrics and gynecology. 2008; 1: p. 4-44.
16. Dobrokhotova Yu.E. MLM, NNIao. The state of the reproductive system of patients who underwent reconstructive plastic surgery on the uterus. Obstetrics and gynecology. 2017; 8: p. 42-48.
17. AB. F. A mathematical model for predicting the state of the uterine scar after cesarean section in practical medicine. Young scientist. 2011; 2(3): p. 167-72.
18. TI. K. Hereditary collagenopathies (clinic, diagnosis, treatment and medical examination). SPb.: Nevsky dialect. 2000; 272.
19. Krasnopolsky VI LdBvS. Insufficient scar on the uterus after cesarean section: causes of formation

and therapeutic tactics. Obstetrics and gynecology. 2013; 12: p. 28-33.

20. Krasnopolsky VI BSSN. Purulent-septic complications in obstetrics and gynecology: features of the course at the present stage. Tactics of reference. Russian Bulletin of the Obstetrician-Gynecologist. 2013; 4: p. 82-5.
21. Krasnopolskaya K.V. PAA, CMA. Pregravid metroplasty for insolvent uterine scar after cesarean section. effect on natural fertility and IVF results// Problems of reproduction. 2015; 21(3): p. 56-62.
22. Kramarsky VA MLDV. The main risk factors for the development of uterine scar inferiority after cesarean section.. Russian Bulletin of the Obstetrician-Gynecologist. 2003; 2: p. 37-9.
23. Krasnopolsky VI LLBS.. Reproductive problems of the operated uterus.. M.: Miklos. 2006; 157.
24. Krasnopolsky VI BSSN.. Obstetric sepsis as a reproductive problem. Obstetrics and gynecology. 2007; 3: p. 38-42.
25. Kulakov VI CEKL. Cesarean section.. M.: Triad-X. 204; 320.
26. section. LdDcfusac. Russian Bulletin of the Obstetrician-Gynecologist. 2003; 59: p. 64.
27. M MZ. Assessment of the condition of the scar on the uterus in the first three months after cesarean section.. Medical aspects of women's health. 2010; 3: p. 57-9.
28. Manukhin I.B. PAD, PYN, LTSae. Organ-preserving operations in case of failure of sutures on the uterus and obstetric peritonitis after cesarean section. XII All-Russian Scientific Forum "Mother and Child". 2011;: p. 131-132.
29. al.]AED[.Morphologicalandimmunohistochemical features of the condition of the uterine scar after cesarean section. Russian Bulletin of the Obstetrician-Gynecologist. 2007; 7(4): p. 7-13.
30. scar Mafeotlusateoppaipwwa. Ailamazyan E.K. [et al.]. Journal of Obstetrics and Women's Diseases. 2006; 4: p. 11-18.
31. al.] NP[. Morphological and functional assessment of reparative processes in the lower segment of the operated uterus at the end of pregnancy. Journal of Obstetrics and Women's Diseases. 2007; 7: p. 50-51.
32. Ordzhonikidze NV FTDlS.. Endometritis and wound infection in puerperas. Problems and solutions. Obstetrics and gynecology. 2004; 5: p. 3-5.
33. Pavlova TU AVFR. Clinical and morphological aspects of the uterine scar after cesarean section. Siberian Medical Journal (Tomsk). 2007; 22: p. 10-2.
34. Savelyeva G. M. KEY, KMA, KY. Yu., Panina O. B., Trofimova O. A. Kesareva section in modern obstetrics. Obstetrics and gynecology. 207; 2: p. 3-8.
35. V.N. S. Obstetric pathology and systemic inflammatory response syndrome.. Russian Medical Journal. 2004; 12(13): p. 741-742.
36. Dry GT KMKNKEDTMABOONAE.. Pregnancy, childbirth and the postpartum period in women after operations on the uterus with undifferentiated forms of connective tissue dysplasia. Obstetrics and gynecology. 2010; 2: p. 26-9.
37. Dry GT KEKMDTDAMATDKISMKNKPONAE. Morphological and molecular genetic features of neoangiogenesis in uterine scar tissue in patients with undifferentiated connective tissue dysplasia.. Obstetrics and gynecology. 2010; 6: p. 23-7.
38. Dry GT KMDAKNBOKEDTKIAESMADKBONTD.. The role of estrogen receptor alpha gene polymorphism (ESR1) in the formation of an inferior uterine scar after cesarean section. Obstetrics and gynecology. 2011; 4: p. 40-4.
39. Titchenko Yu.P. LLS, KIV. Modern aspects of the ultrasound diagnosis of complications in the postpartum period. Russian Bulletin of the Obstetrician-Gynecologist. 2006; 3: p. 22-5.
40. Telegin IV NIPRAVDG.. Features of wound repair on the uterus after cesarean section. Medical Bulletin of the North Caucasus. 2013; 8: p. 89-92.
41. section Tatusbac. Azanova D. B. [et al.]. Actual issues of obstetrics and gynecology. 2001; 1(1): p. 118-119.
42. Cahill AG MG.. Vaginal birth after cesarean delivery: evidence-based practice.. Clin Obstet Gynecol. 2007; 50(2): p. 518-25.
43. Cauwe B. VdSPE, OG.. The biochemical, biological, and pathological kaleidoscope of cell surface substrates processed by matrix metalloproteinases. Crit. Rev. Biochem. Mol. Biol. 2007; 42(3): p. 113-85.

44. Chen H. Y. WL, CYW, CWC, TFJ, TCH. Estrogen receptor beta gene haplotype is associated with pelvic organ prolapse. Eur. J. Obstet. Gynecol. Reprod. Biol. 2008; 138(1): p. 105-9.
45. Chilaka V.N. CMY, HOX, KJC. Risk of uterine rupture following induction of labor in women with a previous caesarean section in a large UK teaching hospital. J Obstet Gynaecol. 2004; 24-3: p. 264-265.
46. Berghella V BJCS. Evidence-based surgery for cesarean delivery.. Am J Obstet Gynecol. 2005; 193(5): p. 1607-17.
47. Guise JM HJOP. Evidence-based vaginal birth after Caesarean section.. Best Pract Res Clin Obstet Gynaecol. 2005; 19(1): p. 117-30.
48. Kumar V AAFNAJ.. Tissue renewal, regeneration and repair. In: Robbins SL, Kumar V, Cotran RS, editors. Robbins and Cotran pathologic basis of disease.. 8th ed. Philadelphia: Saunders/Elsevier. 2010;: p. 79-110.
49. Olsen M. A. BAM, WDM, DP, GGA, FVJ. Risk factors for surgical site infection after low transverse cesarean section.. Infect. Control Hosp. Epidemiol. 2008; 29: p. 477-84.
50. Olsen M. A. BAM, WDM, GGA, DtP, FVJ.. Risk factors for endometritis after low transverse cesarean delivery. Infect.. Control Hosp. Epidemiol. 2010; 31: p. 69-7.
51. V. PE. Clinical significance of office hysteroscopy in examination algorithm of partly faulty uterus scar after cesarean section on the stage of pregnancy planning.. J. Minim. Invasive Gynecol. 2015; 22: p. 190.