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Cesarean scar disorder: Management and repair

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ABSTRACT

Cesarean scar disorder (CSD) is an entity recently defined as uterine niche with at least one primary or 2 secondary symptoms. CSDs can be visualized by hysterosalpingography, transvaginal sonography, saline infusion sonohysterography, hysteroscopy, and magnetic resonance imaging, but diagnosis should be performed by exams able to measure the residual myometrial thickness (RMT). Although there is a limited number of studies evaluating fertility and reproductive outcomes after different types of surgery, the following consideration should be kept in mind. Asymptomatic women should not be operated with the hope of improving obstetrical outcomes. It is reasonable to consider hormone therapy for CSDs as a symptomatic treatment in women who no longer wish to conceive and have no contraindications. In case of failure of or contraindications to medical treatment, surgery should be offered according to the severity of symptoms, including infertility, the desire or otherwise to preserve the uterus, the size of the CSD, and RMT measurement. Hysteroscopy is considered to be more of a resection than a repair, so women who desire pregnancy should be excluded from this technique in case of RMT <3 mm. In this instance, repair is essential and can only be achieved by a laparoscopic or vaginal approach. The benefit of laparoscopic approach seems to persist after subsequent CS. Women with CSDs need to be given complete information, including available literature, before any treatment decision is made.

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Introduction

“During the last decade, there has evolved an almost negligible maternal loss from caesarean section (CS) delivery in competent hands. With the emphasis taken off maternal mortality, one would have thought it would have shifted to maternal morbidity or, more precisely to uterine morbidity. There has, in fact, been very little emphasis placed in the uterine scar, as judged by the attention it has been given in the literature. Its safety has been judged by crude and inaccurate deductions such as the patient's convalescence after the operation or the scar's ability to withstand a further pregnancy and labor. There is a need of a more accurate and reliable method of estimating scar safety. Not only has the detection of uterine morbidity but its prevention has enjoyed too little attention” Poidevin L.O.S, University of Adelaide, 1961 South Australia, [1].

Today, these words sound as if we just understood them right now …

Although the 2015 World Health Organization (WHO) recommendations [2] assess that maternal or newborn mortality is not reduced by over 10% with CS, 20% of pregnant women still undergo CS and CS rates continue to grow all around the world [3].

For the first time, already back in 1959, Poidevin described a depression in the uterus at the cesarean incision site [4]. Through the following decades, this depression was differently named as cesarean scar defects [5,6], cesarean scar dehiscence [7–9], isthmocele [10–12], uterine transmural hernia [13], diverticulum [14], pouch formation [15], niche [16,17], and uteroperitoneal fistula (20 > 18) among others.

Now, that said, the question is, “Where are we sixty years after Poidevin's prophecy?” (Fig. 1A, B, 1C, and 1D)

The aim of this review is to collect and present the latest data on management and repair of Cesarean Scar Disorders (CSDs), including guidance on diagnosis, management, and prevention. A PubMed search was conducted using the MeSH terms ‘cesarean scar defect’_ ‘cesarean scar dehiscence’_‘cesarean scar niche’_ ‘cesarean scar isthmocele’_ ‘cesarean scar transmural hernia’_‘cesarean scar diverticulum’_‘cesarean scar pouch’ and ‘cesarean scar uteroperitoneal fistula’. Case reports and oral presentations were excluded, and the review includes articles published in the English language between 1959 and March 2023.

Cesarean scar disorder: a specific entity with symptoms finally recognized!

Sixty years after Poidevin's thoughts, a panel of 31 international experts reached consensus for the constellation of symptoms secondary to a uterine niche and named it CSD [19], defined as a uterine niche in combination with at least 1 primary or 2 secondary symptoms. Defined primary symptoms were postmenstrual spotting, pain during uterine bleeding, technical issues with catheter insertion during embryo transfer, and secondary unexplained infertility combined with intrauterine fluid. Secondary symptoms were dyspareunia, abnormal vaginal discharge, chronic pelvic pain, avoiding sexual intercourse, odor associated with abnormal blood loss, secondary unexplained infertility, secondary infertility despite assisted reproductive technology, negative self-image, and discomfort during participation in leisure activities [19]. Additionally, Gozzi et al. reported more pronounced scar pain and lower abdominal pain when CSD was observed 6 months after CS [20]. Recognizing this iatrogenic condition that might interfere with quality of life is the first step toward consensus of management.

Are there recognized risk factors for developing CSD?

Several risk factors have been identified that must be considered during obstetric monitoring in order to improve the detection of CSDs. First of all, the number of previous CSs is recognized as a risk factor for developing CSD. Incidence of 61%, reported by Osser et al. after one CS, extends to 81% after two and even 100% after three procedures [21]. This was also observed by Antila-Långsjö et al. in women without previous CS with a 35% chance of developing CSD, while the risk was 76% and 88%, respectively, after 2 and 3 CSs [22]. When emergency CS was performed in their series, CSD was significantly more present after long active labor duration. Peripartal infections, elevated body mass index (BMI), and diabetes were recognized as associated risk factors. In their series, 19.7% of patients

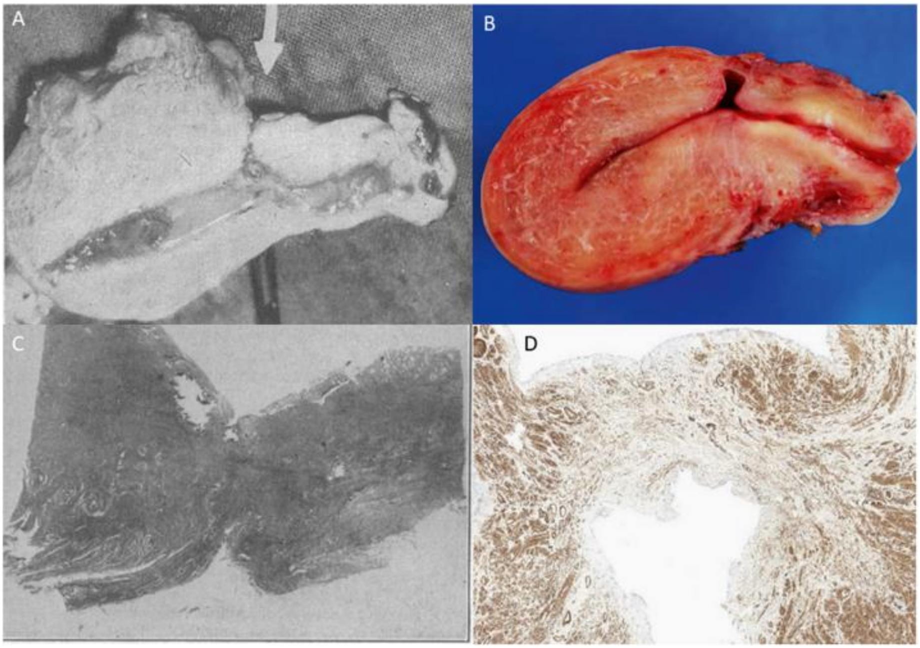

Fig. 1. A. "Where are we sixty years after Poidevin's prophecy?" Sagittal view of a frozen section from a hysterectomy specimen published by Poidevin in 1960 [1]. A depression covered with a thin layer of myometrium (white arrow) can be seen at the level of the supposed site of CS (from POIDEVIN LO. The value of hysterography in the prediction of cesarean section wound defects. Am J Obstet Gynecol. 1961; 81:67–71.). B. "Where are we sixty years after Poidevin's prophecy?" Sagittal view of a frozen section from a hysterectomy specimen published by Donnez [6,18]. A deep anterior defect covered with a thin layer of myometrium (white square) can be seen at the level of the supposed site of CS (from Donnez O. et al., Fertil Steril. 2017 Jan; 107(1):289–296.) and Donnez O. Cesarean scar defects: management of an iatrogenic pathology whose prevalence has dramatically increased. Fertil Steril. 2020 Apr; 113(4):704–716.). C. Microscopic view of the shallow depression from Fig. 1A (from POIDEVIN LO. The value of hysterography in the prediction of cesarean section wound defects. Am J Obstet Gynecol. 1961; 81:67–71.). D. Actin immunostaining in an excised CSD from 1B. The muscular density of myometrium covering the CSD is significantly decreased compared with adjacent healthy myometrium (from Donnez O. et al., Fertil Steril. 2017 Jan; 107(1):289–296. and Donnez O. Cesarean scar defects: management of an iatrogenic pathology whose prevalence has dramatically increased. Fertil Steril. 2020 Apr; 113(4):704–716.).

had residual myometrial thickness (RMT) of <3.0 mm, and peripartal infection and advanced cervical dilatation were risk factors for RMT of <3.0 mm [22].

Vikhareva Osser and Valentin [23] reported that the risk for developing CSD dramatically increased if CS is performed after ≥5 h of labor or when cervical dilatation is ≥ 5 cm, considering then that the later in labor CS is performed, the greater the risk of developing larger CSDs. The presence of a retroflected uterus was found to increase the incidence of a CSD when compared to anteverted uterus. Even if advanced labor seems to have an impact on the risk of developing CSD, the position of the uterine incision might also interfere with further uterine healing. Vikhareva et al. reported that the lower the incision, the higher the risk of developing CSD [24]. In their series, low hysterotomy positioning (2 cm below the vesicouterine pouch) in women in advanced labor was associated with a 6-fold higher rate of large CSDs compared to high hysterotomy positioning (2 cm above the vesicouterine pouch) [24].

Do we have to worry about the technique of suture?

The benefits of double-layer closure compared to single-layer remain contradictory. In a systematic review and meta-analysis, Roberge et al. concluded that single-layer and locked first-layer closure were both associated with lower RMT values [25], while double-layer and unlocked first-layer repair were associated with higher RMT values (3.8 ± 1.6 mm vs 6.1 ± 2.2 mm; $p < .001$) and a greater healing ratio than the locked single-layer technique [26]. Bamberg et al. also found RMT to be thicker after double-layer repair at long-term follow-up [27], but CSD incidence was similar when the uterine suture was done via single or double layer. Di Spiezo et al. systematic review [28] reported that the two closure techniques showed similar CSD rates as well as uterine dehiscence and rupture in a subsequent pregnancy (low-to moderate-quality evidence).

Dimassi et al. observed that double-layered purse string uterine suture significantly reduces cesarean scar defect rates, compared to single-layer continuous suture, without increasing the perioperative maternal morbidity [29]. In a randomized clinical trial, Saccone et al. reported that the use of synthetic absorbable monofilament sutures at the time of uterine wall closure was not associated with a reduction in the rate of cesarean scar defect 6 months after delivery compared with the use of synthetic absorbable multifilament sutures [30]. On the other hand, Hosseini et al. observed that Vicryl sutures were associated with a lower risk of CSD formation in comparison with catgut sutures [31].

Moreover, a recent survey illustrated that various uterine closure techniques are performed irrespective of potential adverse sequelae and without consideration for subsequent patient counseling and care, suggesting that further studies should analyze the impact of uterine closure technique on scar healing [32].

Although various data are available in the literature, there is enough evidence to conclude that there is no difference between single- and double-layer suture regarding the risk of developing CSD.

Pathological findings could explain symptoms, and their understanding is decisive for management.

If most cesarean uterine scars remain asymptomatic, they may nevertheless be associated with complications in subsequent pregnancies, such as uterine rupture, an abnormal insertion of the placenta, or scar rupture [33–35].

Morris first reported pathological findings on CSDs originating from hysterectomy specimens [10]. He described distortion and widening of the lower uterine segment, congested endometrium, polyps, lymphocytic infiltration, residual suture material, capillary dilatation, free red blood cells, fragmentation and breakdown of the scar endometrium, and iatrogenic adenomyosis. In a smaller series, Karpathiou et al. reported different pathological features when uterine scar is associated or not with CSD [36]. Most CSD harbor endocervical mucosa, often cystically dilated and/or an atrophic or disorganized endometrial mucosa of lower uterine segment origin. The authors observed frequent regenerative epithelial atypia and fibroblastic stromal reaction, but no granulomatous reaction, important inflammation, or hemorrhage was seen. On the contrary, CS scars without CSD formation do not harbor endocervical mucosa, inclusion cysts, fibroblastic stroma, or regenerative atypia, supporting the idea that CSD might be the site of chronic inflammation.

If most respected specimens only revealed fibrotic tissue, endometriosis was reported in pathological reports from CSD resected specimen analysis. Tanimura et al. [37] and Donnez et al. [5], respectively, found endometriosis rates of 27.2% and 21.1%. When present, endometriotic lesions were interestingly located in the deepest part of the CSD. This key information must be considered when choosing the right surgical approach. Leaving this area in place after excision surgery, or incomplete bottom coagulation, might be in fact responsible for the failure rate. If endometriotic lesions may well explain pain and dysmenorrhea, blood retention might also be related to abnormal bleeding originating from endometriotic foci.

Other mechanisms could explain blood retention inside CSDs. First of all, hysteroscopy frequently described abnormal neovascularization covering the internal part of CSD, suggesting that bleeding can originate from the CSD itself (Fig. 2) [38]. Moreover, the muscular density of the myometrium covering uterine scar differs depending on whether there is a CSD or not (5, 11 > 9, 39). Indeed, when uterine scar is not associated with CSD, the muscular density of the myometrium covering the scar is not significantly different from the surrounding healthy uterine myometrium. When uterine scar was associated with CSD, the muscular density of the myometrium covering the scar was instead dramatically decreased compared to healthy myometrium (Fig. 3A, B, 3C, 3D, 3E, and 3F). This suggests that the myometrium covering CSD might be unable to provide sufficient contractility to expel blood from endometrial shedding during menstruation. Moreover, this poor muscular density should be kept in mind when counseling patients who consider surgical solutions.

Fig. 2. Hysteroscopic view of a CSD. Dendritic blood vessels (red arrows) are present on the surface of the CSD. Old blood retention is observed on the right lateral part of the CSD (white arrow).

According to the literature [39–41], fertility may well be impaired, with the risk of infertility estimated to be between 4% and 19%. Indeed, the presence of blood in the CSD and uterine cavity might prevent penetration of sperm cells and/or embryo implantation [15,39–42]. This toxic environment could then lead to a decline in fertility via several mechanisms, including the flow of bloody fluid or bleeding from the CSD into the vagina and uterine cavity. The cytotoxicity of iron is well known, and an excess of iron after hemoglobin degradation in the uterine cavity [43,44] may be toxic to the embryo and/or impair its implantation through disturbed endometrial receptivity or uterine microbiota [45].

There is obviously a link between large defects detected in nonpregnant women and dehiscence or uterine rupture in subsequent pregnancies (odds ratio [OR], 11.8; 95% confidence interval [CI], 0.7–746), as seen in the study by Vikhareva Osser and Valentin [46]. This risk was even higher in another study in women with large defects (OR, 26.05; 95% CI, 2.36–287.61) [47]. Bujold et al. estimated that a lower uterine segment thickness of 2.3 mm at 35–38 weeks of gestation was associated with a 9.1% risk of complete uterine rupture [34]. However, in a study comparing pregnancy after laparoscopic CSD excision to expectant management, Jordans et al. did not observe any significant difference in uterine rupture between the two groups but more uterine dehiscence in the expectant management group, suggesting that there is actually no evidence to support laparoscopic correction in order to improve obstetrical outcomes [48]. This major information is a critical key point that must be a part of the discussion while giving proper information to asymptomatic patients who may be tempted to undergo surgery in order to avoid the risk of uterine rupture in further pregnancy.

How to diagnose CSDs

Anomalies in the anterior uterine isthmus after CS can be diagnosed by different imaging or endoscopic examen, such as hysterosalpingography, transvaginal sonography (TVS), saline or gel infusion sonohysterography (SIS), hysteroscopy, and magnetic resonance imaging (MRI). If we would be able to recognize CSD during all these examens, everyone would agree that it would provide different information about CSD. As the most useful discriminating factor remains the RMT covering the scar [21,49], this can only be measured by TVS or pelvic MRI.

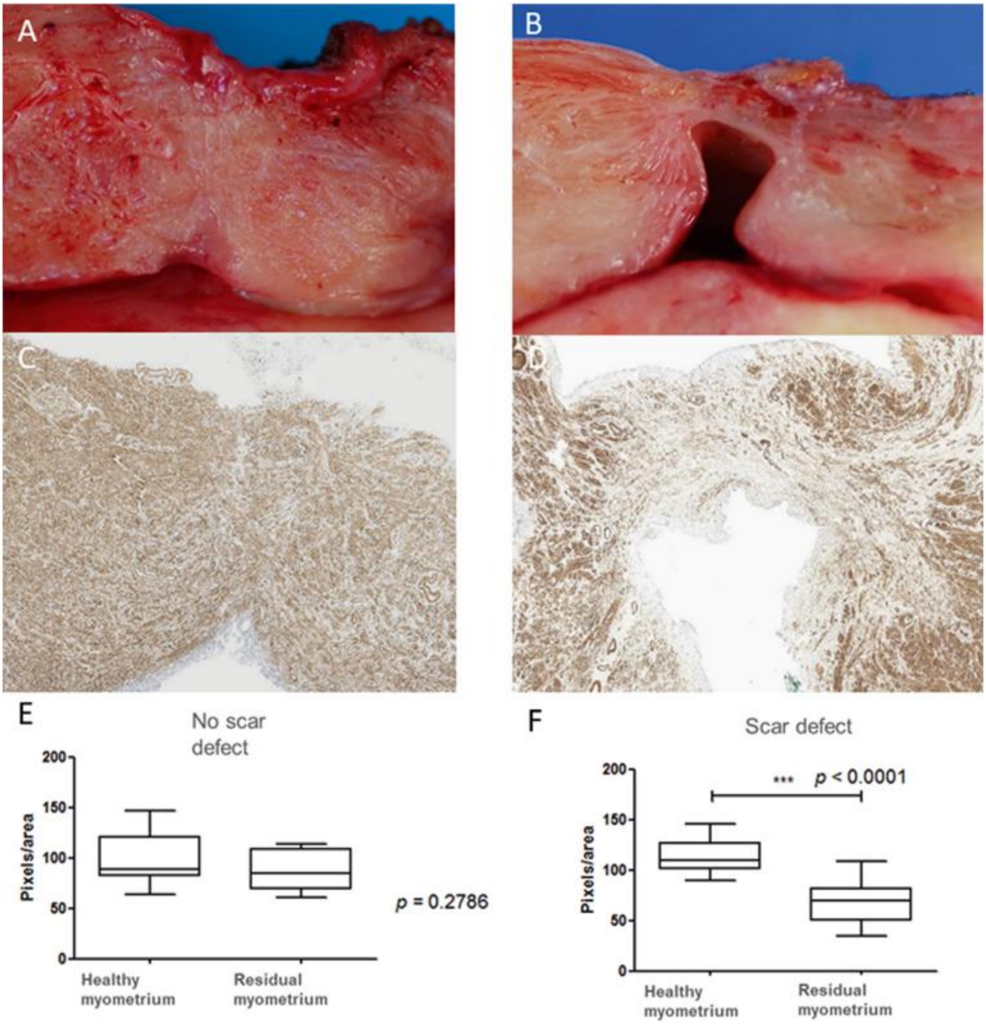

Fig. 3. A. Enlarged view of the shallow depression from a hysterectomy specimen. A shallow depression covered with a thick layer of myometrium can be seen at the level of the supposed site of cesarean section (CS). B. Enlarged view of the deep anterior defect from a frozen section from a hysterectomy specimen. A deep anterior defect covered with a thin layer of myometrium can be seen at the level of the supposed site of CS. C. Actin immunostaining in a hysterectomy specimen. D. Actin immunostaining in an excised Cesarean Scar Disorder (CSD). E. The muscular density of myometrium covering the cesarean scar is similar to adjacent healthy myometrium. F. The muscular density of myometrium covering the CSD is significantly decreased compared with adjacent healthy myometrium. Images in A, B, C, D, E, F reproduced from Donnez et al., Fertil Steril 2017; 107:289–96 and Donnez O. Cesarean scar defects: management of an iatrogenic pathology whose prevalence has dramatically increased. Fertil Steril. 2020 Apr; 113(4):704–716.).

Transvaginal sonography

TVS is an accurate way of diagnosing and measuring CSDs, with or without saline, gel, or even 3D images [17,47], and has high detection rates [22]. Fifteen European experts provided guidance for CSD evaluation by TVS and reached a consensus by means of a modified Delphi procedure [50]. Defects were defined as an indentation with a depth of at least 2 mm at the site of CS. CSDs can be subclassified as follows [1]: simple CSD [2]; simple CSD with one branch [3]; complex CSD (with more than one branch). CSD measurements are based only on myometrial values, and the endometrium should be excluded. Length, depth, RMT, width, adjacent myometrial thickness, distance between the CSD and the vesicovaginal fold, and distance between the CSD and the external os were considered clinically relevant measurements of CSDs. Length, depth, and RMT should be calculated in the sagittal plane, while the transverse plane is used only for the third dimension of these defects (width), not for depth or RMT (Fig. 4A, B, 4C, and 4D). It is useful to vary the pressure with transvaginal probe in order to identify the best plane for measurement. Use of Doppler imaging is not essential but can be helpful to differentiate between CSDs and hematomas, adenomyomas, adenomyosis, or fibrotic tissue. Gel or saline contrast sonography may also be helpful but is not imperative if intrauterine fluid is present.

Magnetic resonance imaging

In a series of 38 patients operated on by laparoscopic CSD resection, we correlated RMT measurement by TVS and MRI to microscopic evaluation of pathological findings, and we did not observe any statistically significant difference [9]. MRI and TVS are therefore appropriate tools to determine RMT, even if MRI use may be disputed, as preoperative RMT values were found to be similar by TVS [9].

However, MRI offers additional benefits, such as reproducibility in interpretation, unlike TVS, which remains dependent on operator training and qualification. If RMT can be easily measured on sagittal (Fig. 5A) and transverse views, the nature of fluid entrapped in the CSD might be defined by MRI (Fig. 5B). Residual blood in the CSD is detected by the presence of hypersignal spots visualized in defects on T1-weighted images with saturation of fatty tissue due to the presence of residual menstrual blood [9]. This blood in the CSD may be retained after menstruation or coming from the CSD itself.

Because it gives information about the whole uterine neighborhood, MRI offers precise mapping of CSDs. Indeed, uterine adhesion with abdominal wall could be seen around the CSD site (Fig. 5C) and are primordial to detect if surgery, especially reconstructive surgery, is planned as the surgeon will have to manage these adhesions before accessing to the vesico uterine fold and the CSD site. In case of attempting vaginal surgery, such adhesion might decrease uterine mobilization and interfere with uterine healing [51]. However, when performed by a trained gynecologist, ultrasound also allows adhesion detection as it is a dynamic exam. In case of an untrained performer, on the contrary, the information is lost.

From my point of view, MRI might be superior to TVS in complex situations when CSD is associated with other gynecologic pathologies such endometriosis, fibroids, or adenomyosis (Fig. 5D). Identifying complete mapping of uterine environment is primordial for proper counseling, leading to appropriate patient information about choosing the right management. In some instances, persistence of symptoms after treatment may be due to ignored associated pathologies and may explain treatment failure.

However, further studies are needed to identify the most accurate means of imaging, taking into consideration the cost-effectiveness of both methods and the undisputable need for specific training for both of them.

Hysteroscopy

CSDs can also be diagnosed by hysteroscopy, even if no information concerning RMT can be obtained unless ultrasound or MRI are performed. In this instance, a cavity is observed on the anterior side of the isthmus, and the presence of neovascularized areas and dendritic vessels (Fig. 2) or polyps [37,38,52] could suggest bleeding from the defect. The presence of old blood (Fig. 2) may also be noted [9,52], suggesting retained menstruation in the CSD or an endometriotic origin [5,37].

From my point of view, gynecologists should be able to recognize CSD when they perform hysteroscopy and, from there, complete an investigation in order to measure RMT and obtain the uterine and peri uterine mapping.

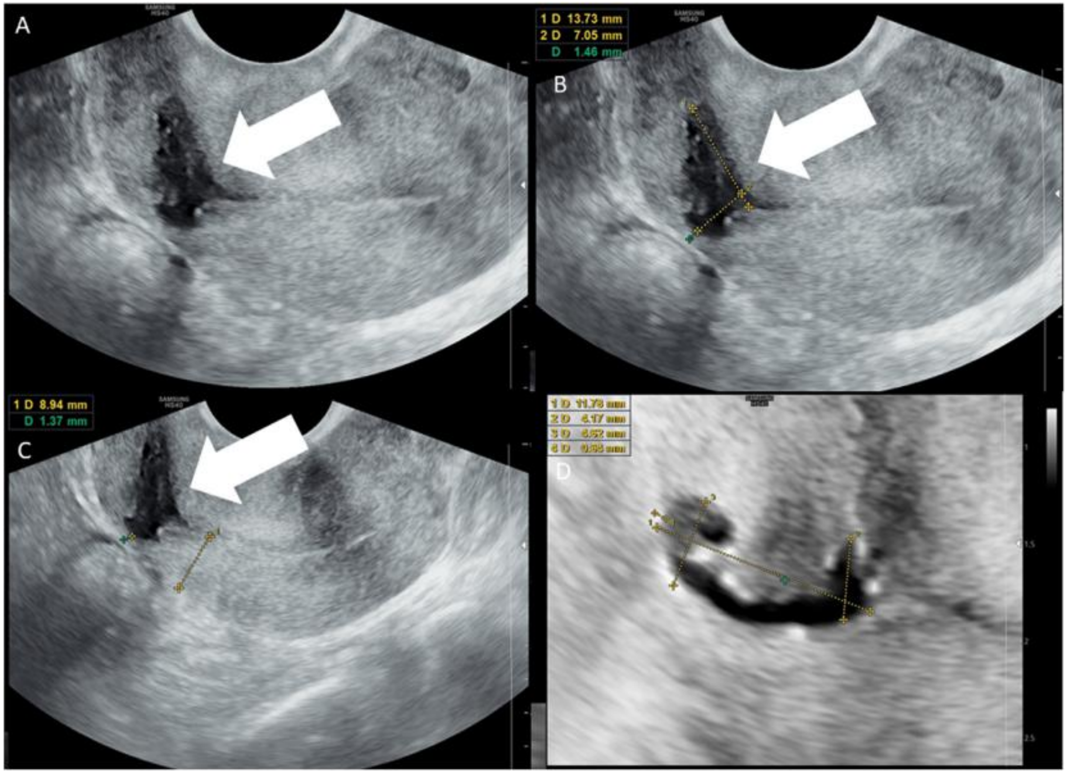

Fig. 4. A. Sagittal transvaginal sonography view of a uterus presenting with a CSD. B. The CSD corresponds to an anechoic area (white arrow) measuring 13.73 mm in length and 7.05 mm in depth. The RMT is 1.46 mm. C. The adjacent myometrium measures 8.94 mm. D. Sagittal transvaginal sonography view of a uterine isthmus presenting with a complex CSD.

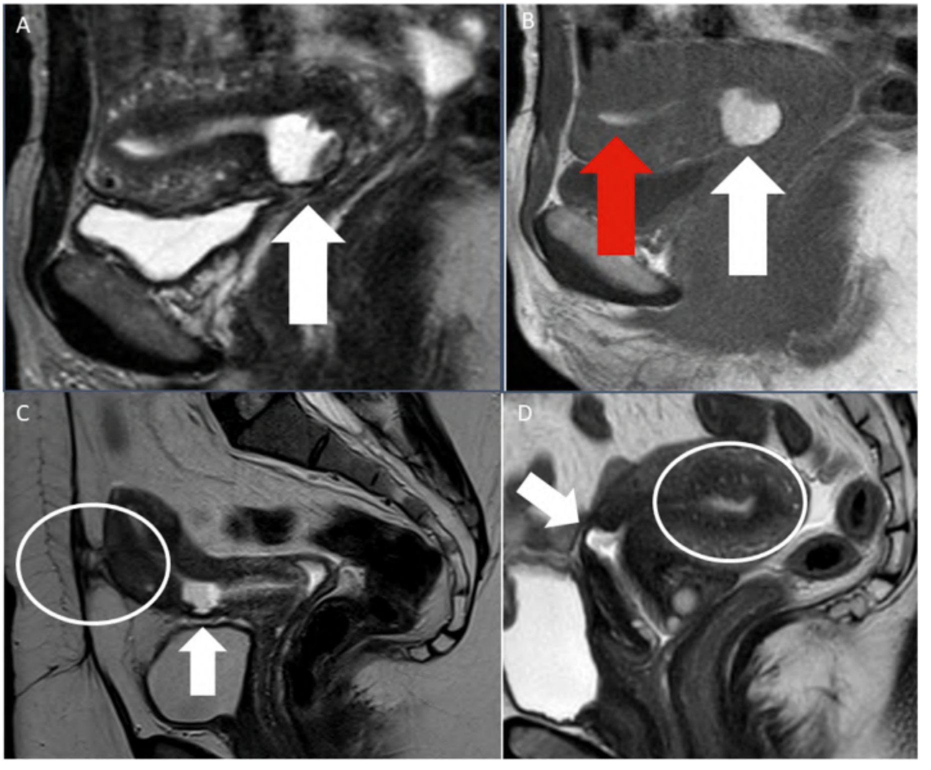

Fig. 5. A. Sagittal view of a T2-weighted image showing a large CSD covered with a thin layer of myometrium (white arrow). B. Sagittal view of a T1-weighted image showing a large CSD with hypersignal (white arrow) from old blood accumulation from endometrial shedding (red arrow). C. Sagittal view of a T2-weighted image showing a large CSD covered with a thin layer of myometrium (white arrow). Uterus reveals a thick junctional zone with hypersignal spots from adenomyosis (white circle). D. Sagittal view of a T2-weighted image showing a large CSD covered with a thin layer of myometrium (white arrow). Dense adhesions (white circle) can be seen between the anterior uterine wall and abdominal wall at some distance from the CSD.

How to manage CSDs

CSD management can be medical or surgical

Medical management

Medical treatment of CSDs has been poorly investigated in small series. According to Tahara et al. [53] and Zhang et al. [54], association of estrogen and progesterone could prevent recurrent bleeding in more than 80% of women in series of, respectively, 11 and 23 patients. Conversely, Chen et al. found the levonorgestrel intrauterine device to be effective in 88.3% of their patients ($n = 5/6$) when treating CSD-related intermenstrual bleeding [55]. This was also observed by Ou et al. in a series of 28 patients with intermenstrual spotting due to CSD. Authors reported intermenstrual bleeding improvement in 22 patients (78.6%) within the first year after levonorgestrel intrauterine device placement without any adverse event, such as uterine perforation or pelvic inflammatory disease [56]. Moreover, in a randomized, open label-controlled trial concerning 208 patients, Zhang et al. reported that the levonorgestrel intrauterine system was superior to hysteroscopic resection in reducing spotting from 9 months onward, as well as the absolute number of spotting days from 6 months onward and pelvic pain from 3 months onward [57].

If medical solutions look interesting, data on a larger population are clearly needed. However, hormonal treatment for CSD might be reasonably proposed to symptomatic patients who no longer wish to conceive and have no contraindications.

Surgical management

In case of failure of or contraindications to medical treatment, surgery should be discussed according to the severity of symptoms, including infertility, the desire or otherwise to preserve the uterus, the size of the CSD and RMT [46].

Hysteroscopic resection

In 1996, Fernandez et al. reported the first conservative treatment by hysteroscopic resection [58], followed later by many other teams. In order to facilitate expulsion of blood, the superior and inferior edges of the CSD were resected (Fig. 6A and B), while the bottom of the defect was fulgurated or electrocoagulated using a rollerball [11,15,37,58–64]. However, a number of authors opted to resect only the inferior edge of the CSD and achieved similar outcomes [65–68], while Shapira et al. proposed also resecting the bottom [69] (Fig. 6B) which seems to be an acceptable option regarding the presence of endometriosis in this area [5]. Most authors were fully aware of the possibility of uterine perforation or bladder injury, but no such complications have ever been described. Indeed, hysteroscopic CSD resection was free of complications in the majority of studies. Vervoort et al. reported pelvic infection in 2% of cases in their series [64].

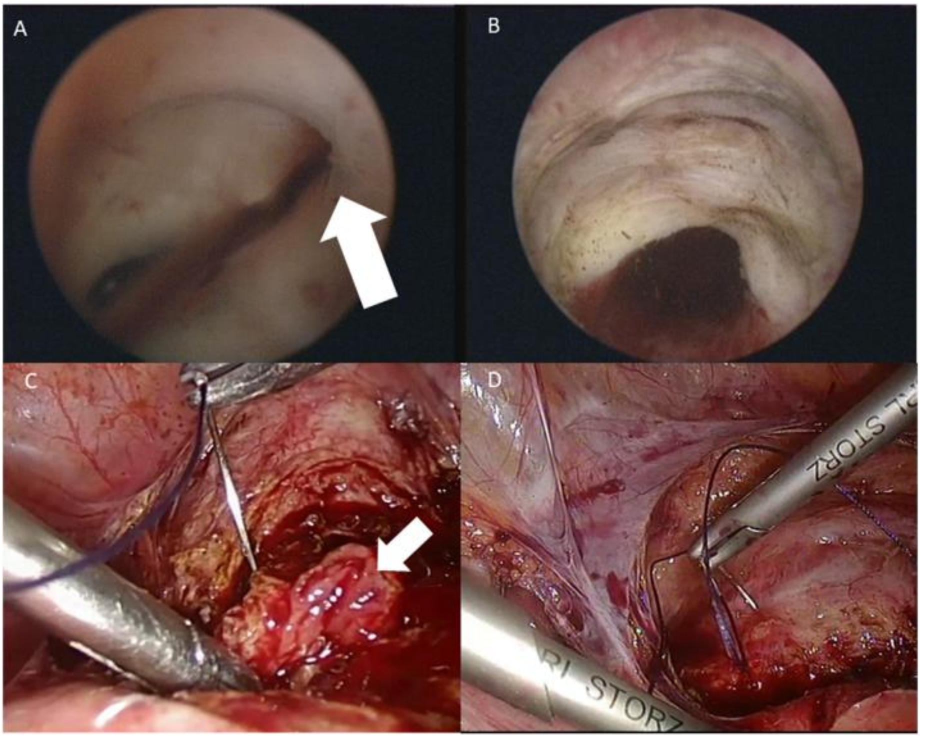

Fig. 6. A. Hysteroscopic view from CSD with old blood retention originating from the left angle (white arrow). B. Final view after hysteroscopic resection of the CSD. The superior and inferior edges of the CSD are resected, as is the bottom of the CSD. C. Laparoscopic view of first-layer suture taking the endocervical mucosa (white arrow). D. Laparoscopic view of second layer suture.

Between 59.6% and 100% of patients usually become asymptomatic after surgery, but Wang et al. recorded an 86.6% failure rate in case of retroverted uteri [60] in a series of 57 patients. In a randomized trial of 102 patients comparing hysteroscopic resection and expectant management, Vervoort et al. observed a significant decrease in menstrual blood loss and improved quality of life after resection [64], but no impact on intermenstrual bleeding or dysmenorrhea compared to expectant management [64]. One possible explanation for the incomplete resolution of symptoms after hysteroscopic resection is the absence of resection of the superior edge of the defect in this latest trial. Moreover, electrocoagulation of the deepest part may be insufficient to treat endometriotic lesions frequently encountered in this area [5,37]. This also endorses the technique proposed by Shapira et al., who advocate resecting not only the edges of the CSD but also the bottom [69].

Most authors acknowledge that reduced RMT is a limiting factor for hysteroscopic resection, but inclusion criteria vary greatly between studies. While Chang et al. [65] consider that RMT should exceed 2 mm to avoid uterine rupture, Li et al. [61] added a further criterion, namely desire for pregnancy, in which case RMT should be superior to 3.5 mm. If there is no wish to conceive, RMT should still be over 2.5 mm.

In a series of 8 patients experiencing failure after CSD hysteroscopic resection, Cohen et al. proposed a second hysteroscopic procedure and observed symptom improvement in 6 out of 8 women. They, therefore, concluded that a second hysteroscopic resection is feasible and effective after previous failure. Interestingly, this second surgery had no detrimental effect on further obstetrical outcomes in their series [70]. Nevertheless, hysteroscopic management should be regarded as a resection and not a repair. Lower uterine segment repair can only be achieved by laparoscopy or vaginal access.

Only six studies address the question of RMT after hysteroscopic CSD resection. Tanimura et al. found that RMT values remained unchanged after hysteroscopic resection in a small series [37]. This is also reported by Vervoort et al. in a randomized trial of 102 patients [64]. However, in a series of 24 patients (Li et al.), RMT increased from 4.76 ± 1.55 mm to 5.68 ± 1.69 mm [61], while Tsuji et al. recorded an upturn in RMT values from 2.1 mm to 4.2 mm [63]. Pregnancy rates after hysteroscopic resection for CSD ranged from 6.6% [61] to 100% [69]. Similar results were presented by Casadio et al. [71] who observed a +2 mm significant difference before and after the procedure using channel-like (360°) hysteroscopic resection. Conversely, Zeller et al. reported increased of RMT after hysteroscopic approach even when preoperative RMT was <3 mm and even very thin [72].

However, no author has given a plausible explanation for the increase using a surgical tissue resection technique. This might be questionable regarding the pathological findings of dramatically decreased muscular density in the RMT (Fig. 3D and F).

Laparoscopic repair

The first case of laparoscopic repair of an uteroperitoneal fistula was reported by Jacobson [73]. Our team subsequently described then the laparoscopic repair of wide and deep CSD in 2008 [9], and the first study of 13 patients was published several years later [38], followed by bigger series [5,18].

Laparoscopy allows complete exploration of the pelvis and direct access to CSDs, enabling adhesiolysis if adhesions are present. After bladder dissection, complete resection of any fibrotic tissue is essential to ensure continued healing, followed by double-layer suture (Fig. 6D and E) [5,9,38,54,68,74–76], although some authors opt for single-layer suture [37,61,77–79]. In case of a retroflexed uterus, anteflexion can be performed by shortening the round ligament, as suggested by Vervoort et al. and our team [5,70], or fixing the round ligament to the anterior abdominal aponeurosis through lateral port incisions [5]. Li et al. and Zhang et al. used cut-off values of <5 mm [76] and <6.2 mm [61], respectively. No complications were reported after laparoscopic repair in most series, except bladder dilacerations in 2% of women [74] and hemorrhage on day 10 requiring blood transfusion [80]. Postoperative RMT is measured using either ultrasound or MRI. The mean RMT value obtained after laparoscopic repair by our team and Tanimura et al. was around 10 mm [5,9,37] using double-layer and single-layer suture, respectively. Liu et al. [76] did not encounter any severe CSD recurrence after surgery, while Donnez et al. [9], Nirgianakis et al. [81], Zhang et al. [77], Vervoort et al. [74], and Delaine et al. [80] reported rates of 1.6%, 8.9%, 14%, 12%, and 33% in their respective series. Karampelas et al. used a technique similar to ours and found the same results in terms of postoperative

RMT measures [82]. Very interestingly, the authors found that the benefits seem to persist after the subsequent CS.

The cut-off RMT value for laparoscopic CSD repair is usually <3 mm when data are available and has indeed been proposed by our group [38] based on findings from Bujold et al. These authors estimated that a lower uterine segment of <2.3 mm, also identified as an independent predictor of CSDs, was associated with a higher risk of uterine rupture [34]. However, Jordans et al. did not report any difference in uterine rupture after laparoscopic treatment compared to expectant management, supporting that there is actually no evidence to support laparoscopic treatment to improve obstetrical outcomes [48] even if significantly more uterine dehiscence was observed in the expectant group.

Between 64.1% and 100% of patients were asymptomatic after laparoscopic CSD repair. In our series, we encountered persistence of symptoms in 6.4% of cases (n = 4). Postoperative MRI revealed the RMT to be > 10 mm in 3 of these patients, but with minimal blood retention. As the RMT value was significantly elevated in these patients, we proposed hysteroscopic resection, allowing two of them to conceive. The third patient reported an improvement in symptoms but had no desire for pregnancy. It is important to note one serious failure in a patient with three previous CSs. The achieved RMT was 5 mm, but the CSD was larger, suggesting that the myometrium was unable to heal correctly. This may have been due to excessive fibrotic tissue surrounding the scar or the inability of the surgeon to distinguish healthy tissue after three CSs. The patient asked for total laparoscopic hysterectomy because of worsening symptoms. Patients were advised to wait 3–6 months before attempting pregnancy and to give birth by CS at term in case of subsequent pregnancy. The take-home-baby rate ranged from 21.8% to 75% [77–80].

The use of robotic assistance has also been described, but the benefit of this approach remains unclear when compared to the laparoscopic approach [83].

Vaginal repair

The first vaginal procedure was described in 2005 by Klemm et al. [84]. Some patients in this series were undergoing laparoscopy for bladder dissection, but excision of the CSD was performed vaginally and then followed by single-layer uterine suture and vaginal suture. Although single-layer uterine closure was also reported by Chen et al. [85], most authors use double-layer suture for uterine closure [54,77,86–88]. Low complication rates were observed after vaginal repair: hematoma (2.5%), pelvic infection (2.4%), and bladder dilacerations (2%) [77,89,87].

Blood loss is significantly decreased after vaginal repair (59–89.9%), and three studies recently found a significant increase in RMT from <3 mm to, respectively, 5.6 ± 2.0 mm [87], 6.10 ± 1.43 mm [86], and 8.95 ± 2.51 mm [54]. Other studies did not report any data on RMT measurements. Persistent CSDs were observed in 13% and 31.37% of cases in two further series [54,86]. Concerning obstetrical outcomes, a 47.5% pregnancy rate was achieved by Deng et al. in a series of 183 patients operated on hysteroscopy combined with transvaginal repair [88]. A promising adaptation for vaginal natural orifice transluminal endoscopic surgery (V-NOTES) was recently proposed, but further evaluation is needed [90].

Hysterectomy

Hysterectomy was the first surgical option described [4,10], but it should only be implemented after comprehensive counseling and informed consent from women who no longer wish to conceive. Even if laparoscopic hysterectomy has proven safe in experienced hands [91,92] and can even be performed in an ambulatory setting [93], the principal issue after CS is the possibility of severe adhesions with the bladder or anterior abdominal wall [94]. A history of more than two CSs is a risk factor for bladder injury during vaginal or laparoscopic hysterectomy [95]. Moreover, women undergoing two or more CS deliveries have an adjusted OR for receiving a blood transfusion of 1.93 (95% CI, 1.21–3.07) compared to women with no previous CS [96].

Conclusion

CSDs should be suspected in women presenting uterine niche in combination with at least 1 primary or 2 secondary symptoms. As prevention is better than cure, risk factors should be identified early in order to ensure appropriate management. The diagnosis must include TVS with adequate measurements. The place of MRI should be discussed, its advantage being that this examination is not operator reliant and allows a whole pelvic exploration. CSD treatment depends on the age of the patient, the severity of symptoms as well as associated infertility, the RMT, and the wish to preserve fertility or at least the uterus. Asymptomatic women presenting with a CSD should not be operated regarding the lack of evidence that surgery improves obstetrical outcomes. An informed consent using the latest scientific data should be thoroughly addressed with the patient. If surgery is indicated, the choice between hysteroscopic resection and laparoscopic or vaginal repair should be guided by RMT. Hysteroscopy is considered to be more of a resection than a repair, so women who desire pregnancy should be excluded from this technique in case of RMT <3 mm. In this instance, repair is essential and can only be achieved by a laparoscopic or vaginal approach. Although both techniques are associated with good anatomical results, laparoscopy allows the surgeon to explore other causes of infertility and pain, enabling simple correction of uterine retroflexion. Further studies are needed to improve knowledge about fertility and obstetrical outcomes after all types of surgeries. Women with CSDs need to be given complete information, including available literature, before any treatment decision is made.

Practice point

- CSDs should be suspected in women presenting uterine niche in combination with at least 1 primary or 2 secondary symptoms.
- Diagnosis should be confirmed by ultrasound or MRI.
- Asymptomatic women should not be operated on with the hope of improving obstetrical outcomes unless future studies prove otherwise.
- Hysteroscopic resection is a valuable surgical option when RMT is > 3 mm. Since it does not repair the anatomy, future studies are needed to evaluate its safety regarding reproductive outcomes.
- RMT restoration can only be obtained by vaginal or laparoscopic repair.
- Laparoscopic repair allows concomitant uterine antefixation in case of retroverted uterus and surgical treatment of other pelvic pathologies.

Research agenda

- Standardize the surgical technique to allow reproducibility.
- Determine the RMT cut-off measurement below which hysteroscopy should not be performed. Analyze the possibility for vaginal delivery after repair.
- Future studies are needed to evaluate fertility and obstetrical outcomes after all surgical procedures.

Declaration of competing interest

The authors have no conflicts of interest.
O.D. has nothing to disclose.

References

[1] Poidevin LO. The value of hysterography in the prediction of cesarean section wound defects. Am J Obstet Gynecol 1961; 81:67–71.
[2] World Health Organization. WHO Statement on caesarean section rates. WHO/RHR/15.02; 2015.
[3] Betrán AP, Ye J, Moller AB, Zhang J, Gülmezoglu AM, Torloni MR. The increasing trend in caesarean section rates: global, regional and national estimates: 1990-2014. PLoS One 2016;11.
[4] Poidevin LO. Caesarean section scar safety. Br Med J 1959;2:1058–61.
[5] Donnez O, Donnez J, Orellana R, Dolmans MM. Gynecological and obstetrical outcomes after laparoscopic repair of a cesarean scar defect in a series of 38 women. Fertil Steril 2017;107:289–96.
[6] Osser OV, Jokubkiene L, Valentin L. Cesarean section scar defects: agreement between transvaginal sonographic findings with and without saline contrast enhancement. Ultrasound Obstet Gynecol 2010;35:75–83.
[7] Van Horenbeeck A, Temmerman M, Dhont M. Cesarean scar dehiscence and irregular uterine bleeding. Obstet Gynecol 2003;102:1137–9.
[8] Klemm P, Koehler C, Mangler M, Schneider U, Schneider A. Laparoscopic and vaginal repair of uterine scar dehiscence following cesarean section as detected by ultrasound. J Perinat Med 2005;33:324–31.
[9] Donnez O, Jadoul P, Squifflet J, Donnez J. Laparoscopic repair of wide and deep uterine scar dehiscence after cesarean section. Fertil Steril 2008;89:974–80.
[10] Morris H. Surgical pathology of the lower uterine segment caesarean section scar: is the scar a source of clinical symptoms? Int J Gynecol Pathol 1995.
[11] Gubbini G, Casadio P, Marra E. Resectoscopic correction of the "isthmocele" in women with postmenstrual abnormal uterine bleeding and secondary infertility. J Minim Invasive Gynecol 2008;15:172–5.
[12] Florio P, Filippeschi M, Moncini I, Marra E, Franchini M, Gubbini G. Hysteroscopic treatment of the cesarean-induced isthmocele in restoring infertility. Curr Opin Obstet Gynecol 2012;24:180–6.
[13] Jeremy B, Bonneau C, Guillo E, Paniel BJ, Le Tohic A, Haddad B, et al. Uterine ishtmique transmural hernia: results of its repair on symptoms and fertility. Gynecol Obstet Fertil 2013;41:588–96.
[14] Luo L, Niu G, Wang Q, Xie HZ, Yao SZ. Vaginal repair of cesarean section scar diverticula. J Minim Invasive Gynecol 2012;19: 454–8.
[15] Fabres C, Aviles G, De La Jara C, Escalona J, Muñoz JF, Mackenna A, et al. The cesarean delivery scar pouch: clinical implications and diagnostic correlation between transvaginal sonography and hysteroscopy. J Ultrasound Med 2003;22: 695–700.
[16] Monteagudo A, Carreno C, Timor-Tritsch IE. Saline infusion sonohysterography in nonpregnant women with previous cesarean delivery: the "niche" in the scar. J Ultrasound Med 2001;20:1105–15.
[17] Bij de Vaate AJ, Brölmann HA, van der Voet LF, van der Slikke JW, Veersema S, Huirne JA. Ultrasound evaluation of the Cesarean scar: relation between a niche and postmenstrual spotting. Ultrasound Obstet Gynecol 2011;37:93–9.
[18] Donnez O. Cesarean scar defects: management of an iatrogenic pathology whose prevalence has dramatically increased. Fertil Steril 2020 Apr;113(4):704–16.
[19] Klein Meuleman SJM, Murji A, van den Bosch T, Donnez O, Grimbizis G, Saridogan E, et al., CSDi Study Group. Definition and criteria for diagnosing cesarean scar disorder. JAMA Netw Open 2023 Mar 1;6(3):e235321.
[20] Gozzi P, Hees KA, Berg C, David M, Wernecke KD, Hellmeyer L, et al. Frequency and associated symptoms of isthmoceles in women 6 months after caesarean section: a prospective cohort study. Arch Gynecol Obstet 2023 Mar;307(3):841–8.
[21] Osser OV, Jokubkiene L, Valentin L. High prevalence of defects in Cesarean section scars at transvaginal ultrasound examination. Ultrasound Obstet Gynecol 2009;34:90–7.
[22] Antila-Långsjö RM, Ju Mäenpää, Huhtala HS, Tomás EI, Staff SM. Cesarean scar defect: a prospective study on risk factors. Am J Obstet Gynecol 2018;219:458.e1–8.
[23] Vikhareva Osser O, Valentin L. Risk factors for incomplete healing of the uterine incision after caesarean section. BJOG 2010;117:1119–26.
[24] Vikhareva O, Rickle GS, Lavesson T, Nedopekina E, Brandell K, Salvesen KÅ. Hysterotomy level at Cesarean section and occurrence of large scar defects: a randomized single-blind trial. Ultrasound Obstet Gynecol 2019;53:438–42.
[25] Roberge S, Demers S, Berghella V, Chaillet N, Moore L, Bujold E. Impact of single- vs double-layer closure on adverse outcomes and uterine scar defect: a systematic review and meta-analysis. Am J Obstet Gynecol 2014;211:453–60.
[26] Roberge S, Demers S, Girard M, Vikhareva O, Markey S, Chaillet N, et al. Impact of uterine closure on residual myometrial thickness after cesarean: a randomized controlled trial. Am J Obstet Gynecol 2016;214:507.e1–6.
[27] Bamberg C, Hinkson L, Dudenhausen JW, Bujak V, Kalache KD, Henrich W. Longitudinal transvaginal ultrasound evaluation of Cesarean scar niche incidence and depth in the first two years after single- or double-layer uterotomy closure: a randomized controlled trial. Acta Obstet Gynecol Scand 2017;96:1484–9.
[28] Di Spiezio Sardo A, Saccone G, McCurdy R, Bujold E, Bifulco G, Berghella V. Risk of Cesarean scar defect following single- vs double-layer uterine closure: systematic review and meta-analysis of randomized controlled trials. Ultrasound Obstet Gynecol 2017;50:578–83.
[29] Dimassi K, Ami O, Merai R, Velemir L, Simon B, Fauck D, et al. Double-layered purse string uterine suture compared with single-layer continuous uterine suture: a randomized Controlled trial. J Gynecol Obstet Hum Reprod 2022 Feb;51(2): 102282.
[30] Saccone G, De Angelis MC, Zizolfi B, Gragnano E, Musone M, Zullo F, et al. Monofilament vs multifilament suture for uterine closure at the time of cesarean delivery: a randomized clinical trial. Am J Obstet Gynecol MFM 2022 May;4(3): 100592.
[31] Hosseini R, Mansoorli S, Pirjani R, Eslamian L, Rabiee M. A comparison of the effects of two suture materials on isthmocele formation: a cohort study. J Gynecol Obstet Hum Reprod 2021 Apr;50(4):101933.
[32] Antoine C, Meyer JA, Silverstein JS, Alexander J, Oh C, Timor-Tritsch IE. The impact of uterine incision closure techniques on post-cesarean delivery niche formation and size: sonohysterographic examination of nonpregnant women. J Ultrasound Med 2022 Jul;41(7):1763–71.

[33] Bujold E, Jastrow N, Simoneau J, Brunet S, Gauthier RJ. Prediction of complete uterine rupture by sonographic evaluation of the lower uterine segment. Am J Obstet Gynecol 2009;201:320.e1–6.
[34] Diaz SD, Jones JE, Seryakov M, Mann WJ. Uterine rupture and dehiscence: ten-year review and case-control study. South Med J 2002;95:431–5.
[35] Naji O, Wynants L, Smith A, Abdallah Y, Stalder C, Sayasneh A, et al. Predicting successful vaginal birth after Cesarean section using a model based on Cesarean scar features examined by transvaginal sonography. Ultrasound Obstet Gynecol 2013;41:672–8.
[36] Karpathiou G, Chauleur C, Dridi M, Baillard P, Corsini T, Dumollard JM, et al. Histologic findings of uterine niches. Am J Clin Pathol 2020 Oct 13;154(5):645–55.
[37] Tanimura S, Funamoto H, Hosono T, Shitano Y, Nakashima M, Ametani Y, et al. New diagnostic criteria and operative strategy for cesarean scar syndrome: endoscopic repair for secondary infertility caused by cesarean scar defect. J Obstet Gynaecol Res 2015;41:1363–9.
[38] Marotta ML, Donnez J, Squifflet J, Jadoul P, Darii N, Donnez O. Laparoscopic repair of post-cesarean section uterine scar defects diagnosed in nonpregnant women. J Minim Invasive Gynecol 2013;20:386–91.
[39] Gurol-Urganci I, Bou-Antoun S, Lim CP, Cromwell DA, Mahmood TA, Templeton A, et al. Impact of Caesarean section on subsequent fertility: a systematic review and meta-analysis. Hum Reprod 2013;28:1943–52.
[40] Gurol-Urganci I, Cromwell DA, Mahmood TA, van der Meulen JH, Templeton A. A population-based cohort study of the effect of Caesarean section on subsequent fertility. Hum Reprod 2014;29:1320–6.
[41] CORONIS Collaborative Group, Abalos E, Addo V, Brocklehurst P, El Sheikh M, Farrell B, Gray S, et al. Caesarean section surgical techniques (CORONIS): a fractional, factorial, unmasked, randomised controlled trial. Lancet 2013;382:234–48.
[42] Bij de Vaate AJ, van der Voet LF, Naji O, Witmer M, Veersema S, Brölmann HA, et al. Prevalence, potential risk factors for development and symptoms related to the presence of uterine niches following Cesarean section: systematic review. Ultrasound Obstet Gynecol 2014;43:372–82.
[43] Defrere S, Lousse JC, Gonzalez-Ramos R, Colette S, Donnez J, Van Langendonckt A. Potential involvement of iron in the pathogenesis of peritoneal endometriosis. Mol Hum Reprod 2008;14:377–85.
[44] Van Langendonckt A, Casanas-Roux F, Donnez J. Iron overload in the peritoneal cavity of women with pelvic endometriosis. Fertil Steril 2002;78:712–8.
[45] Moreno I, Simon C. Relevance of assessing the uterine microbiota in infertility. Fertil Steril 2018;110:337–43.
[46] Vikhareva Osser O, Valentin L. Clinical importance of appearance of cesarean hysterotomy scar at transvaginal ultrasonography in nonpregnant women. Obstet Gynecol 2011;117:525–32.
[47] Baron J, Weintraub AY, Eshkoli T, Hershkovitz R, Sheiner E. The consequences of previous uterine scar dehiscence and cesarean delivery on subsequent births. Int J Gynaecol Obstet 2014;126:120–2.
[48] Jordans IPM, Vissers J, de Leeuw RA, Hehenkamp WJK, Twisk JWR, de Groot CJM, et al. Change of the residual myometrial thickness during pregnancy in women who underwent laparoscopic niche resection compared with controls without niche surgery: a prospective comparative cohort study. Am J Obstet Gynecol 2022 Dec;227(6). 901.e1-901901.
[49] Ludwin A, Martins WP, Ludwin I. Evaluation of uterine niche by three-dimensional sonohysterography and volumetric quantification: techniques and scoring classification system. Ultrasound Obstet Gynecol 2019;53:139–43.
[50] Jordans IPM, de Leeuw RA, Stegwee SI, Amso NN, Barri-Soldevila PN, van den Bosch T, et al. Sonographic examination of uterine niche in non-pregnant women: a modified Delphi procedure. Ultrasound Obstet Gynecol 2019;53:107–15.
[51] Vissers J, Hehenkamp W, Lambalk CB, Huirne JA. Post-Caesarean section niche-related impaired fertility: hypothetical mechanisms. Hum Reprod 2020 Jul 1;35(7):1484–94.
[52] Chen YY, Tsai CC, Kung FT, Lan KC, Ou YC. Association between hysteroscopic findings of previous cesarean delivery scar defects and abnormal uterine bleeding. Taiwan J Obstet Gynecol 2019;58:541–4.
[53] Tahara M, Shimizu T, Shimoura H. Preliminary report of treatment with oral contraceptive pills for intermenstrual vaginal bleeding secondary to a cesarean section scar. Fertil Steril 2006;86:477–9.
[54] Zhang X, Yang M, Wang Q, Chen J, Ding J, Hua K. Prospective evaluation of five methods used to treat cesarean scar defects. Int J Gynaecol Obstet 2016;134:336–9.
[55] Chen YY, Tsai CC, Lan KC, Ou YC. Preliminary report on the use of a levonorgestrel intrauterine system for the treatment of intermenstrual bleeding due to previous cesarean delivery scar defect. J Obstet Gynaecol Res 2019;45:2015–20.
[56] Ou YC, Chen YY, Lan KC, Tsai CC, Chu LC, Cheng LY. Levonorgestrel intrauterine system for the treatment of intermenstrual spotting in patients with previous cesarean delivery scar defect. J Obstet Gynaecol Res 2022 Jan;48(1):155–60.
[57] Zhang J, Zhu C, Yan L, Wang Y, Zhu Q, He C, et al. Comparing levonorgestrel intrauterine system with hysteroscopic niche resection in women with postmenstrual spotting related to a niche in the uterine cesarean scar: a randomized, open-label, controlled trial. S0002-S9378 Am J Obstet Gynecol 2023 Mar 17;(23):167–9.
[58] Fernandez E, Fernandez C, Fabres C, Alam VV. Hysteroscopic correction of cesarean section scars in women with abnormal uterine bleeding. J Am Assoc Gynecol Laparosc 1996;3:S13.
[59] Gubbini G, Centini G, Nascetti D, Marra E, Moncini I, Bruni L, et al. Surgical hysteroscopic treatment of cesarean-induced isthmocele in restoring fertility: prospective study. J Minim Invasive Gynecol 2011;18:234–7.
[60] Wang CJ, Huang HJ, Chao A, Lin YP, Pan YJ, Horng SG. Challenges in the transvaginal management of abnormal uterine bleeding secondary to cesarean section scar defect. Eur J Obstet Gynecol Reprod Biol 2011;154:218–22.
[61] Li C, Guo Y, Liu Y, Cheng J, Zhang W. Hysteroscopic and laparoscopic management of uterine defects on previous cesarean delivery scars. J Perinat Med 2014;42:363–70.
[62] Muzii L, Domenici L, Lecce F, Di Feliciantonio M, Frantellizzi R, Marchetti C, et al. Clinical outcomes after resectoscopic treatment of cesarean-induced isthmocele: a prospective case-control study. Eur Rev Med Pharmacol Sci 2017;21:3341–6.
[63] Tsuji S, Kimura F, Yamanaka A, Hanada T, Hirata K, Takebayashi A, et al. Impact of hysteroscopic surgery for isthmocele associated with cesarean scar syndrome. J Obstet Gynaecol Res 2018;44:43–8.
[64] Vervoort A, van der Voet LF, Hehenkamp W, Thurkow AL, van Kesteren P, Quartero H, et al. Hysteroscopic resection of a uterine caesarean scar defect (niche) in women with postmenstrual spotting: a randomised controlled trial. BJOG 2018;125:326–34.

[65] Chang Y, Tsai EM, Long CY, Lee CL, Kay N. Resectoscopic treatment combined with sonohysterographic evaluation of women with postmenstrual bleeding as a result of previous cesarean delivery scar defects. Am J Obstet Gynecol 2009;200: 370.e1–4.
[66] Feng YL, Li MX, Liang XQ, Li XM. Hysteroscopic treatment of postcesarean scar defect. J Minim Invasive Gynecol 2012;19: 498–502.
[67] Raimondo G, Grifone G, Raimondo D, Seracchioli R, Scambia G, Masciullo V. Hysteroscopic treatment of symptomatic cesarean-induced isthmocele: a prospective study. J Minim Invasive Gynecol 2015;22:297–301.
[68] Lv B, Xie X, Liu C, Lin Y. Laparoscopic combined with hysteroscopic repair or operative hysteroscopy in the treatment of symptomatic cesarean-induced diverticulum. Med Sci 2018;34:47–51.
[69] Shapira M, Mashiach R, Meller N, Watad H, Baron A, Bouaziz J, et al. Clinical success rate of extensive hysteroscopic cesarean scar defect excision and correlation to histologic findings. J Minim Invasive Gynecol 2019 8;(19):30118–9. https://doi.org/10.1016/j.jmig.2019.03.001. pii: S1553-4650.
[70] Cohen SB, Mashiach R, Baron A, Goldenberg M, Schiff E, Orvieto R, et al. Feasibility and efficacy of repeated hysteroscopic cesarean niche resection. Eur J Obstet Gynecol Reprod Biol 2017 Oct;217:12–7.
[71] Casadio P, Raffone A, Alletto A, Filipponi F, Raimondo D, Arena A, et al. Postoperative morphologic changes of the isthmocele and clinical impact in patients treated by channel-like (360°) hysteroscopic technique. Int J Gynaecol Obstet 2023 Jan;160(1):326–33.
[72] Zeller A, Villette C, Fernandez H, Capmas P. Is hysteroscopy a good option to manage severe cesarean scar defect? J Minim Invasive Gynecol 2021 Jul;28(7):1397–402.
[73] Jacobson MT, Osias J, Velasco A, Charles R, Nezhat C. Laparoscopic repair of a uteroperitoneal fistula. J Soc Laparoendosc Surg 2003;7:367–9.
[74] Vervoort A, Vissers J, Hehenkamp W, Brölmann H, Huirne J. The effect of laparoscopic resection of large niches in the uterine caesarean scar on symptoms, ultrasound findings and quality of life: a prospective cohort study. BJOG 2018;125: 317–25.
[75] Yalcinkaya TM, Akar ME, Kammire LD, Johnston-MacAnanny EB, Mertz HL. Robotic-assisted laparoscopic repair of symptomatic cesarean scar defect: a report of two cases. J Reprod Med 2011;56:265–70.
[76] Liu SJ, Lv W, Li W. Laparoscopic repair with hysteroscopy of cesarean scar diverticulum. J Obstet Gynaecol Res 2016;42: 1719–23.
[77] Zhang Y. A comparative study of transvaginal repair and laparoscopic repair in the management of patients with previous cesarean scar defect. J Minim Invasive Gynecol 2016 May-;23:535–41.
[78] Li C, Tang S, Gao X, Lin W, Han D, Zhai J, et al. Efficacy of combined laparoscopic and hysteroscopic repair of post-cesarean section uterine diverticulum: a retrospective analysis. BioMed Res Int 2016;2016:1765624. https://doi.org/10.1155/2016/1765624. Epub 2016.
[79] Dosedla E, Calda P. Outcomes of laparoscopic treatment in women with cesarean scar syndrome. Med Sci Mon Int Med J Exp Clin Res 2017;23:4061–6.
[80] Delaine M, Lecointre L, Akladios CY, Hummel M, Host A, Garbin O. Laparoscopic treatment of cesarean scar pouch - a case series study of 9 patients. Gynecol Obstet Fertil Senol 2017;45:262–8.
[81] Nirgianakis K, Oehler R, Mueller M. The Rendez-vous technique for treatment of caesarean scar defects: a novel combined endoscopic approach. Surg Endosc 2016;30:770–1.
[82] Karampelas S, Salem Wehbe G, de Landsheere L, Badr DA, Tebache L, Nisolle M. Laparoscopic isthmocele repair: efficacy and benefits before and after subsequent cesarean section. J Clin Med 2021 Dec 10;10(24):5785.
[83] Gkegkes ID, Psomiadou V, Minis E, Iavazzo C. Robot-assisted laparoscopic repair of cesarean scar defect: a systematic review of clinical evidence. J Robot Surg 2022 Nov 27. https://doi.org/10.1007/s11701-022-01502-w.
[84] Klemm P, Koehler C, Mangler M, Schneider U, Schneider A. Laparoscopic and vaginal repair of uterine scar dehiscence following cesarean section as detected by ultrasound. J Perinat Med 2005;33:324–31.
[85] Chen Y, Chang Y, Yao S. Transvaginal management of cesarean scar section diverticulum: a novel surgical treatment. Med Sci Mon Int Med J Exp Clin Res 2014 8;20:1395–9.
[86] Zhou X, Yang X, Chen H, Fang X, Wang X. Obstetrical outcomes after vaginal repair of caesarean scar diverticula in reproductive-aged women. BMC Pregnancy Childbirth 2018 19;18:407.
[87] Chen H, Wang H, Zhou J, Xiong Y, Wang X. Vaginal repair of cesarean section scar diverticula diagnosed in non-pregnant women. J Minim Invasive Gynecol 2019;26:526–34.
[88] Deng K, Liu W, Chen Y, Lin S, Huang X, Wu C, et al. Obstetric and gynecologic outcomes after the transvaginal repair of cesarean scar defect in a series of 183 women. J Minim Invasive Gynecol 2021 May;28(5):1051–9.
[89] Luo L, Niu G, Wang Q, Xie HZ, Yao SZ. Vaginal repair of cesarean section scar diverticula. J Minim Invasive Gynecol 2012;19: 454–8. https://doi.org/10.1016/j.jmig.2012.03.012.
[90] Baekelandt JF, Vnotes Kapurubandara S. A novel approach using vaginal natural orifice transluminal endoscopic surgery to repair a symptomatic uterine isthmocele. Fertil Steril 2023 Feb;119(2):328–30.
[91] Donnez O, Jadoul P, Squifflet J, Donnez J. A series of 3190 laparoscopic hysterectomies for benign disease from 1990 to 2006: evaluation of complications compared with vaginal and abdominal procedures. BJOG 2009;116:492–500.
[92] Donnez O, Donnez J. A series of 400 laparoscopic hysterectomies for benign disease: a single centre, single surgeon prospective study of complications confirming previous retrospective study. BJOG 2010;117:752–5.
[93] Donnez O, Donnez J, Dolmans MM, Dethy A, Baeyens M, Mitchell J. Low pain score after total laparoscopic hysterectomy and same-day discharge within less than 5 hours: results of a prospective observational study. J Minim Invasive Gynecol 2015;22:1293–9.
[94] Hesselman S, Högberg U, Jonsson M. Effect of remote cesarean delivery on complications during hysterectomy: a cohort study. Am J Obstet Gynecol 2017;217:564.e1–8.
[95] Bogani G, Cromi A, Serati M, Di Naro E, Casarin J, Marconi N, et al. Hysterectomy in patients with previous cesarean section: comparison between laparoscopic and vaginal approaches. Eur J Obstet Gynecol Reprod Biol 2015;184:53–7.
[96] Lindquist SAI, Shah N, Overgaard C, Torp-Pedersen C, Glavind K, Larsen T, et al. Association of previous cesarean delivery with surgical complications after a hysterectomy later in life. JAMA Surg 2017;152:1148–55.