A Growing Concern: Cesarean Scar Defect and Massive Uterine Bleeding

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Short Communication

Cesarean Scar Defect (CSD) is the formation of a diverticulum at the site of the old caesarean incision [1]. With a rising cesarean sections being performed, the complications caused by CSDs has attracted more and more attentions. These include conditions such as scar pregnancy, placenta accreta, placenta previa, and uterine rupture in subsequent pregnancies [2,3]. The CSD is also associated with abnormal bleeding, dysmenorrhea, pelvic pain, adenomyosis, infertility, abscess formation, post-coital bleeding during the non-pregnant state [4-6]. Of note, the most common clinical symptom is abnormal bleeding, and the correlation with CSDs has been well established. The typical presentation is intermenstrual or postmenstrual spotting, lasting for 2 to 12 days. A CSD woman had recurrent life threatening bleeding during her menses only recently being described [7] which was diagnosed CSD and local arterial hemorrhage by hysteroscopy and was managed with laparoscopic correction (Figure 1) of caesarean scar dehiscence?

Herein we also successfully managed a massive hemorrhage CSD woman with laparoscopic repair of the cesarean scar. She was 30 years old, G1P1, underwent an elective lower segment cesarean section 3 year ago. Since then, she had postmenstrual spotting lasting for 11 days, curettage and hormone therapy were ineffective. She was transferred to our hospital because of recurrent massive hemorrhage during her last menses with hemoglobin concentration dropping to 55g/dL. Routine transvaginal ultrasound showed diverticulum hypoechoic image at the site of the previous hysterotomy, pregnancy and arteriovenous fistula was excluded. After homologous transfusion and hemostasis treatment, diagnostic hysteroscopy identified some old blood blot in the cesarean scar defect (Figure 2). The pathology examination revealed endometrial hyperplasia. Massive bleeding reoccurred the next day, emergency uterine artery embolization (UAE) was performed and demonstrated a bleeding point of the distal uterine arteries at the old caesarean scar level; this bleeding site was successfully blocked at that time. However, massive hemorrhage took place again three days thereafter, which suggest failure of UAE treatment. At last, defect repair under laparoscopic has been successfully performed in an attempt to control the bleeding, and the postoperative follow-up was uneventful (Figure 3).

Discussion

Several factors have been reported to be associated with Cesarean section wound healing, including the number of cesarean sections, labor before cesarean section, uterine position, and surgical procedure that leaves a caesarean scar pouch in the anterior lower uterine segment [8-10]. The CSD patients experience abnormal bleeding are not always related to the defect size, presumably the main reason is the lack of coordinated muscular contractions around the caesarean scar allows the defect to collect mucus and menstrual

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product1, which also acts as promoting factors for infection [11]. The other reason was reported by Morris [12] that a congested endometria fold, lymphocytic infiltration and small polyps in the scar area demonstrated by histopathologica study of cesarean section scars specimens may contribute to prolonged menstruation. It is natural to speculate that a woman with CSD will suffer heavy bleeding as long as the local blood vessels are damaged by inflammatory infiltrates, leading to arterial end broken, especially in the menstrual period accompanied by endometrial exfoliation. In addition, the muscular layer in this scar area is less capable of fibromuscular contraction to hemostasis. Hysteroscopy is the most reported approach for the treatment of the defect [13]. However, hysteroscopy can only fulgurated the superficial vessels to prevent bleeding; the entire transected artery can’t completely be coagulated. Once the crust at its broken end falls off, recurrence is still possible, not to mention the complications about the uterine perforation and bladder injury. UAE can successfully diagnose and block the bleeding vessel with gelatin, but the gelatin will be dissolute and absorbed. In addition, local infection and lymphocytic infiltration are not resolved; the uterine arteries may reopen as shown in our study. Perhaps, laproscopic correction of scar defects is the most effective approach for the management this complication of CSDs. However, this approach has not been extensively reported, which mandates further studies to investigate its efficacy and safety for the treatment of CSDs.

References


