CASE REPORT

Robotic Repair of Uterine Dehiscence

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ABSTRACT

Background and Objectives: During the past few decades, there has been a significant increase in the number of cesarean deliveries, and thus an increase in the number of complications. A common complication of multiple cesarean deliveries is symptomatic uterine scar dehiscence, for which there are no treatment guidelines available. We report a case of uterine scar dehiscence—the repair of it by robotic surgery—and review the literature on this defect.

Case: The patient was a 39-year-old woman, gravida 4 para 2022, complaining of persistent vaginal spotting for the prior 5 months with a history of a cesarean delivery 3 months before the onset of the symptoms.

Discussion: We report a case of a successful robotic repair of a symptomatic cesarean scar defect.

Conclusion: We propose further studies that include more patients so this technique may become the standard for cesarean scar defect.

Key Words: Cesarean scar defect, Uterine dehiscence, Robotic repair.

INTRODUCTION

During the last few decades, there has been a significant increase in the number of cesarean deliveries, hence, complications including uterine scar dehiscence, whose incidence was previously rare but is now increasing.[1]

Robotic surgery is becoming the repair technique of choice for some types of surgery such as myomectomy. Currently, no guidelines are available for the treatment of symptomatic uterine dehiscence at the cesarean scar. We report a case of uterine scar dehiscence repair by robotic surgery, review the literature, and suggest this entire technique as the possible new gold standard for the repair of this defect.

CASE REPORT

A 39-year-old woman, gravida 4 para 2022, presented to the gynecologic oncology clinic complaining of persistent vaginal spotting for the prior 5 months. She had a cesarean delivery 3 months before the onset of symptoms. Three months before her presentation, the patient resumed normal menses, which was associated with persistent spotting. She denied abdominal or vaginal pain, dyspareunia, fever, nausea, vomiting, or vaginal discharge. During these months, she also complained of progressive fatigue and exercise-induced shortness of breath.

Her obstetric history was significant for 2 cesarean deliveries, the first in 2006 and the last 3 months before the onset of her symptoms. The indication for both surgeries was failure to progress. During the last cesarean delivery, the obstetrician reported that the subcutaneous tissue was very thin but densely adherent to the fascia, with significant scarring. Also, the bladder was densely adhered to the uterus. The uterine incision was low transverse, just above the lower uterine segment. Uterine closure was performed in 3 layers. The postoperative period was uncomplicated. The patient had a laparoscopic ovarian cystectomy in 2008. Her last Pap smear, 6 months earlier, had normal findings.

Her past medical history was positive for iron deficiency anemia and migraine headaches. She reported being allergic to nabumetone. She was taking 325 mg of ferrous sulfate twice per day.

On physical examination, the patient had stable vital signs. Her weight was 63.9 kg, height was 1.7 m, and body

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mass index was 22.1. The pelvic examination revealed normal external female genitalia and some dark blood in the vagina vault, but no visible cervical or vaginal lesions. The uterus was mobile without any masses or nodularity.

As part of the patient's work-up, a magnetic resonance imaging (MRI) scan and an ultrasonogram were obtained and showed dehiscence at the lower uterine segment, with a collection of blood in the area of the dehiscence and hematocolpos **(Figure 1)**. These findings led to the diagnosis of abnormal uterine bleeding secondary to uterine scar dehiscence. Treatment options with associated risks and benefits were discussed with the patient, and she opted for a da Vinci–assisted laparoscopic repair of the lesion.

After general anesthesia, the patient was placed in a low lithotomy position, and an 8-mm skin incision was made in the umbilical area. A pneumoperitoneum was created, and three 5-mm incisions were made—2 pediatric da Vinci ports and 1 assistant port were placed. The uterus was densely adhered to the abdominal wall, so adhesiolysis was performed using sharp dissection and electrocautery. The bladder flap was developed using sharp dissection, mobilizing the bladder from the lower uterine segment. The defect in the lower uterine segment was identified. At this point, hysteroscopy was initiated, and the hysteroscopic view showed an approximately 3-cm defect in the lower uterine segment and a thin scarlike membrane covering it; however, during the hysteroscopy, the laparoscopic view showed a bulging lower uterine segment **(Figure 2)**. The intrauterine cavity appeared to be normal. A uterine incision was made using electrocautery and the blood was removed. The edges of the defect were freshened using unipolar electrocautery. The uterus was closed using 0-Vicryl sutures in 2 layers. The sutures were inspected hysteroscopically. After copious irrigation and evacuating the pneumoperitoneum, the trocars were removed and the skin was closed using 4–0 Caprosyn sutures. Blood loss was <50 mL. The patient was discharged home on postoperative day 1.

Three months later, she had her first follow-up examination and was asymptomatic. She had spotting for about 2 weeks after surgery but has since had no problems. Menses were adequate with no intermenstrual spotting.

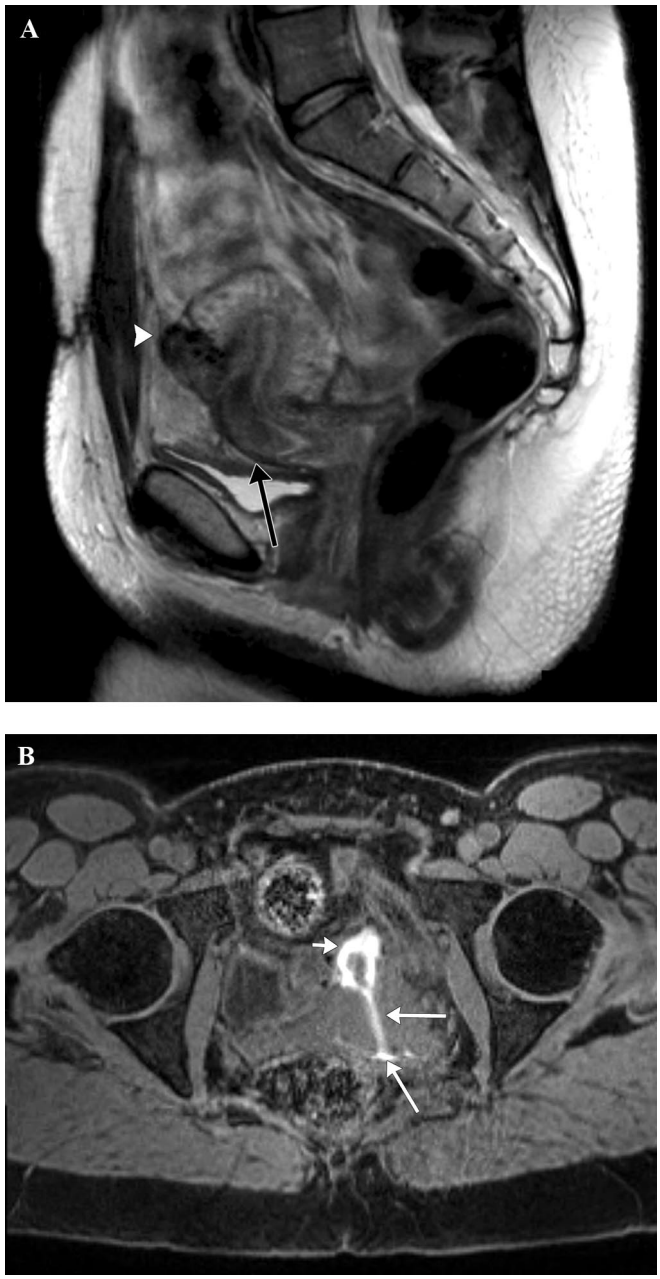

Figure 1. (A) A sagittal T2-weighted scan demonstrates a large clot (*arrow*) at the level of the internal os. Note that no overlying myometrium or fibrous stroma is seen. Dense adhesions involving the anterior uterine wall (*arrowhead*) attach to the abdominal wall on other images (not shown). (B) Fat-suppressed T1-weighted scan demonstrates high signal at cesarean delivery scar site consistent with hematoma (*short arrow*). Long arrows indicate blood in the endocervical canal and vagina.

DISCUSSION

Cesarean delivery is the most common obstetric surgery in the United States, with an increasing incidence of up to 25% of all births.[2,3]

A meta-analysis of birth after a previous cesarean delivery found that the incidence of cesarean scar dehiscence, also

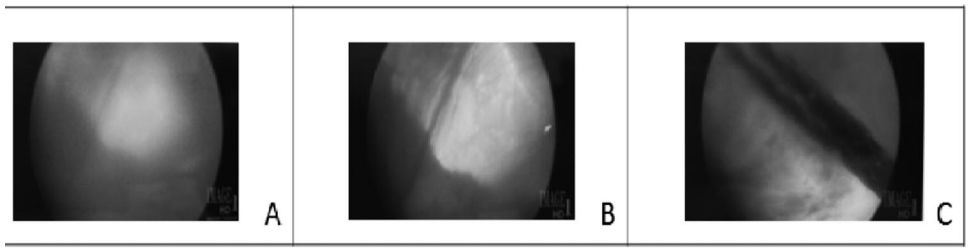

Figure 2. (A and B) Hysteroscopic view of uterine scar dehiscence and (C) the suture post repair of the cesarean scar defect.

known as *cesarean scar defect* (CSD), was 1.9%.[4] Ofili-Yebovi et al found uterine scars in 99.1% of patients with a history of a cesarean delivery, but 19.4% had a defect in their scars; 9.9% of the CSDs were severe, defined as the loss of >50% of myometrial mantle at the scar level.[1] Other studies have reported rates of CSD between 0.6% and 3.8%.[5,6]

Cesarean scar dehiscence is typically defined as discontinuity of the myometrium at the site of a previous cesarean delivery incision or as a subperitoneal separation of the uterine scar in the lower uterine segment, with the chorioamniotic membrane visible through the peritoneum.[3,4]

Risk factors for CSD are a history of multiple prior cesarean deliveries, a retroflexed uterus, and the inability to visualize the entire cesarean scars by ultrasonography.[4]

Uterine healing is a complex process that depends on several biochemical variables, such as transforming growth factor–β family, tumor necrosis factor–α, platelet-derived growth factor, fibroblast growth factor, and vascular endothelial growth factor. All of these factors control the production of collagen in the healing area.[7,8] Pollio et al[9] reported higher collagen content in the areas of CSD compared with the well-healed cesarean scars. This finding was attributed to lower-than-normal transforming growth factor–β3 and connective tissue growth factor. These defects showed increased expression of fibroblast growth factor, vascular endothelial growth factor, platelet-derived growth factor, and tumor necrosis factor–α.[9]

One of the principal concerns with uterine dehiscences is their possible association with uterine rupture. Several studies have found that no relationship exists between these 2 variables. However, one study reported a positive association between uterine dehiscence and uterine rupture, but it was not statistically significant, probably because of the low power of the study, given the rareness of the condition.[10] Thus, there is no consensus about screening nonpregnant women for uterine scar defects.

The width and depth of the defect is proportional to the number of previous cesarean deliveries. The width is also greater in patients who have a retroflexed uterus, maybe a result of the reduced vascular perfusion. The width of the defect is greater in patients who present with vaginal spotting, dyspareunia, and chronic pelvic pain.[6]

Cesarean scar dehiscence is asymptomatic in most patients but can cause symptoms, such as dysmenorrhea[11] and intermenstrual bleeding.[12,13] In a 3-year study,[14] Wang et al found that among the 293 patients diagnosed with CSD by transvaginal sonography, the most common symptom was intermenstrual spotting (64%), followed by dysmenorrhea (53%), chronic pelvic pain (40%), and dyspareunia (18%).

Some mechanisms that could explain these symptoms have been proposed; for example, vaginal bleeding could be the result of the presence of congested endometrial folds (61% of the cases) or the presence of a polyp in the scar recess (16% of the cases). A lack of coordinated muscular contractions around the CSD can also contribute to the collection of debris and therefore cause intermenstrual spotting. Chronic pelvic pain and dyspareunia can be caused by a lymphocytic infiltration (65% of the cases) or a distortion of the lower uterine segment (75%). Finally, the presence of dysmenorrhea in these patients could be produced by iatrogenic adenomyosis.[1]

In addition, the cesarean scar dehiscence can also be the site of implantation in an ectopic pregnancy,[15] increasing the prevalence of this disease during the last decade of life.[16,17]

In an ultrasonography study,[18] it was reported that the risk of a defective scar is directly related to the degree of thinning of the lower uterine segment at 37 weeks of pregnancy. The sensitivity and negative predictive value of ultrasonography for the diagnosis of scar dehiscence in pregnant patients was 88.9% and 96.2%, respectively. Retention of blood in the scar dehiscence was also found, likely accounting for intermenstrual spotting.

MRI has been shown to be the most definitive modality to evaluate uterine incision healing after cesarean deliveries.[19,20] It has the advantage of superior contrast resolution, enabling detailed visualization of tissue planes. Fur-

thermore, MRI is not impeded by body habitus or bowel gas. The size of the defect can be better estimated by using MRI. In a case of posterior wall dehiscence from laparoscopic myomectomy, MRI was able to clearly depict the defect, whereas the initial ultrasonographic evaluation did not.[21] Hemorrhage or hematomas have a characteristic signal on MRI and therefore can be readily distinguished from other fluid collections or masses. One study suggested that the presence of a bladder flap hematoma >5 cm should prompt a careful search for dehiscence.[22]

In expectant management of uterine dehiscence during pregnancy, MRI can be used to confirm the diagnosis, and then the lesion can be followed by ultrasonography if it is adequately visualized by the latter modality.[23]

There are no guidelines for the treatment of intermenstrual bleeding caused by CSD. Nevertheless, multiple techniques had been proposed for the repair of uterine scar dehiscence—for example, hysteroscopic resection of the fibrotic tissue[5] or the technique proposed by Donnez et al,[24] who reported 3 cases of satisfactory laparoscopic repair of uterine scar dehiscence.

Regarding the type of suture used, Greenberg et al[25] conducted a small pilot study in ewes and concluded that the absorbable knotless barbed suture was equivalent to knotted smooth sutures for closing of the uterus.[19]

CONCLUSION

To our knowledge, this is the first reported case of a CSD repair using a pediatric robot that uses 5-mm trocars. The surgical result was excellent. Postoperatively, the patient's symptoms resolved and she is asymptomatic. This technique has been used for treatment of several other diseases, but never previously for CSD. We believe that this technique can become, after further studies, the gold standard surgery for this specific defect.

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