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Asherman's syndrome after an uncomplicated cesarian section

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Abstract

Intrauterine adhesions (IUAs) and their worst form, the Asherman's syndrome, are the result of endometrium basal layer damage, leading to partial or complete obliteration of the uterine cavity by fibrous bridges between the uterine walls. We present the case of a 30 years old woman, gravida 1, para 1 that suffers from secondary amenorrhea and infertility resulted from IUAs formation after an uncomplicated cesarean section, which she underwent 2 years before. Although she never breastfed, she did not start menstruating again after the cesarean section. Clinical and hormonal examinations were in normal ranges and the transvaginal ultrasound revealed an eccentric echogenic endometrium. Hysterosalpingography showed multiple intrauterine filling defects and bilateral tubal occlusion. The operative hysteroscopy revealed multiple intrauterine adhesions originating from the former caesarean incision site and extending to the entire uterine cavity. In spite of hysteroscopic adhesiolysis, intrauterine device insertion and hormonal therapy being provided, the patient never regained fertility. Patients with Asherman's syndrome usually present infertility, recurrent pregnancy loss, amenorrhea or hypomenorrhea. The main cause is known to be the endometrium damage following pregnancy-related curettage. Less common etiologic factors are represented by caesarean section, hysteroscopic procedures, myomectomy and histerotomy. Around 2-2.8% of the patients develop IUAs after cesarian section, more likely as a result of chorioamnionitis, postpartum endometritis, postpartum curettage or uterine compression sutures for postpartum hemorrhage. Severe intrauterine adhesions are a challenging gynecological pathology. The particularity of the case is due to an unusual extensive endometrium damage following uncomplicated caesarian section.
Keywords: *intrauterine adhesions, Asherman's syndrome, infertility*

Introduction

Intrauterine adhesions (IUAs) were first described by Heinrich Fritsch in 1894[1]. Later, the gynecologist Asherman brought more attention to this condition which now bears his name[2]. IUAs are the result of endometrium basal layer damage, leading to partial obliteration of the uterine cavity by fibrous bridges between the uterine walls[3]. The syndrome occurs most often after incomplete abortion (50%), postpartum hemorrhage, and elective abortion[4].Other less common etiologic factors for the Asherman's syndrome are evacuation of a molar pregnancy, myomectomy, hysterotomy, diagnostic curettage, cesarean section, metroplasty, radiation, and tuberculosis. Women who breast-feed may develop IUAs because of the estrogen deficiency[5]. The most common complaint at this syndrome is infertility[6]. Symptoms also include menstrual problems (i.e. amenorrhea, hypomenorrhea, oligomenorrhea,dysmenorrhea), recurrent abortion, intrauterine growth restriction and other complications of pregnancy (i.e. premature rupture of the membranes, abnormal fetal presentation, placenta accrete and placenta praevia)[6].

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Case Report

We present the case of a 30 years old woman, gravida 1, para 1, that suffers from secondary amenorrhea and infertility resulted from IUAs formation after an elective, uncomplicated cesarean section, which she underwent 2 years before. Although she never breastfed, she did not start menstruating again after the caesarean section. Clinical and hormonal examinations were in normal ranges and the transvaginal ultrasound revealed an eccentric echogenic endometrium (Figure 1). Hysterosalpingography showed multiple intrauterine filling defects and bilateral tubal occlusion (Figure 2). The operative hysteroscopy revealed multiple IUAs originating from the former caesarean incision site and extending to the entire uterine cavity (Figure 3). We performed hysteroscopic adhesiolysis. After the surgical procedure in order to prevent the formation of new adhesions, an intrauterine device (IUD) was applied and the patient received hormonal treatment with 1.5 mg estradiol per day. In spite of all these methods, the patient still complained of amenorrhea or hypomenorrhea. The second hysterosalpingography also showed intrauterine filling defects and bilateral tubal occlusion. She restarted the hormonal treatment whit 3 mg estradiol per day, but the menses has not been achieved.

Discussion

Asherman's syndrome occurs due to intrauterine injury causing damage of the endometrium basal layer,

leading to partial or complete obliteration of the uterus by fibrous bridges made between the walls[3,7].

Patients with Asherman's syndrome most of the time present infertility, recurrent pregnancy loss, amenorrhea or hypomenorrhea. The main cause is endometrium damage following pregnancy-related curettage (i.e. incomplete or elective abortion, postpartum hemorrhage). Less common etiologic factor is represented by caesarean section. Around 2-2.8% of the patients develop IUAs after cesarian section[8], more likely as a result of chorioamnionitis, postpartum endometritis, or postpartum curettage.

Asherman's syndrome should be considered in patients with history of cesarean section accompanied by menstrual disturbances, amenorrhea, and infertility, similar to our case.

Hysteroscopy is considered the best method for the diagnosis and should be used as the investigation of choice whenever it is available, but other less invasive modalities such as hysterosalpingogrphy may determine the extent of uterine adhesions and may provide meaningful prognostic information[9].

In our case, the diagnosis of Asherman's syndrome was indicated by hysterosalpingography, which showed multiple intrauterine filling defects and bilateral tubal occlusion and it was confirmed by hysteroscopy, which revealed multiple intrauterine adhesions originating from the former caesarean incision site and extending to the entire uterine cavity. IUAs classification can be useful considered that the prognosis depends on the severity of disease.

A number of classification systems has been proposed for Asherman's syndrome[10].

We classified our case as severe Asherman'ssyndrome according to the modified classification of IUAs based on The European Society of Gynaecological Endoscopy[11].

The treatment of Asherman's syndrome should be considered only if it is symptomatic with pain, menstrual dysfunction, including hematometra, infertility, or recurrent pregnancy loss[10,12]. The management of Asherman's syndrome has a first objective: to restore the volume of the uterine cavity to normal conditions. Standard criterion for achieving this objective is the hysteroscopic adhesiolysis[10,12]. Second objectives include treating associated symptoms including infertility, and preventing recurrence of adhesions. Several methods can be used in order to prevent the adhesion reformation[12]: an IUD or a Foley catheter acts as mechanical barrier between the uterine walls, keeping them apart and thus preventing the contact between the two faces of the uterus and the formation of new adhesions. Intrauterine application of hyaluronic acid gel also could prevent adhesion formation[13]. Hormone therapy with estrogen showed to promotes endometrial proliferation and re-epithelialization of the scarred surfaces[3,10,12].

In our case, in spite of using hysteroscopic adhesiolysis, IUD insertion and hormonal therapy, we failed in restoring the patient with normal menses.

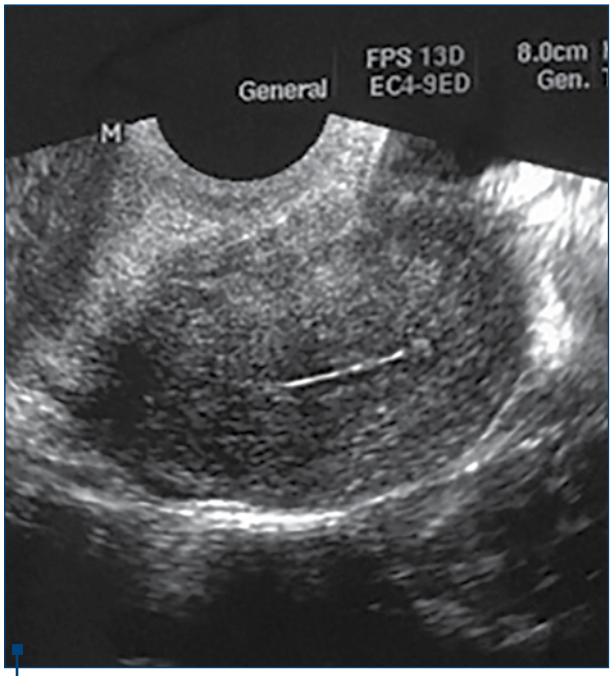

Figure 1. Transvaginal ultrasonography image

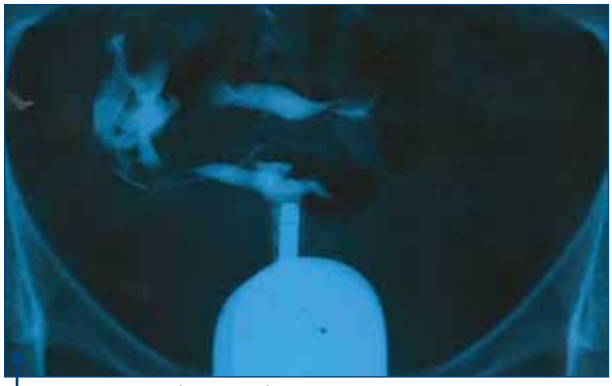

Figure 2. Hysterosalpingography image

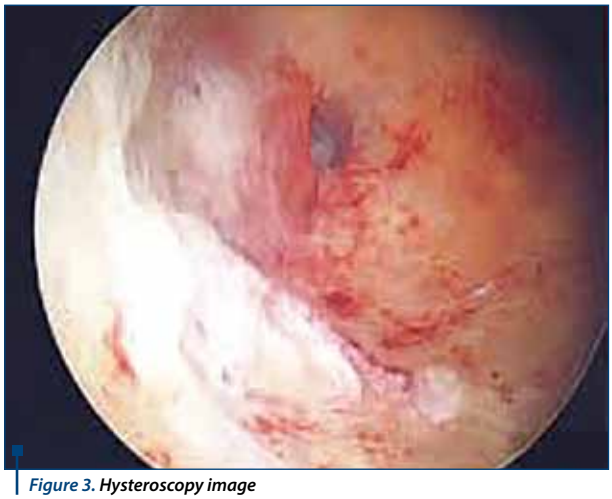

Figure 3. Hysteroscopy image

A new promising therapeutic option for patients with refractory Asherman's syndrome showed to be the cell therapy with cluster of differentiation (CD133+). In a prospective study, CD133+ cells were isolated through peripheral blood apheresis and subsequently delivered into the spiral arterioles of the patient by a minimal invasive technique[14].

Conclusions

Severe IUAs are a challenging gynecological pathology. The peculiarity of this case is represented by the occurrence of uterine synechiae after uncomplicated caesarian section, even in the absence of chorioamniotitis, postpartum endometritis, or postpartum curettage. ■

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