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EC GYNAECOLOGY
Editorial

Intrauterine Adhesions, an Underreported but Clinical Relevant Condition

Angelo Hooker*

Gynecologist, Zaans Medical Centrum, Zaandam, Netherlands

***Corresponding Author**: Angelo Hooker, Gynecologist, Zaans Medical Centrum, Zaandam, Netherlands.

Received: January 29, 2018; **Published:** February 01, 2018

Intrauterine adhesions (IUAs) were first identified in 1894 by Heinrich Fritsch [1]. In 1948 Joseph Asherman described the etiology and therapeutic aspects of this condition, since then prevalently known as Asherman syndrome [2]. Asherman's original description described post pregnancy intrauterine adhesions. The terms Asherman syndrome and IUAs are both used interchangeably in clinical practice, although in the primary description of Asherman the syndrome required signs and symptoms related to IUAs. It remains unclear if asymptomatic IUAs are of clinical significance. Intentional IUAs, produced from intrauterine treatment such as endometrial ablation are not included in the definition.

IUAs are the major long-term complication of intrauterine surgery. IUAs are formed by the visceral walls of the uterine cavity adhering together in the healing process, causing partial or complete obliteration of the cavity. The prevalence of IUAs is difficult to determine for a number of reasons including geographic location, population studied, and diagnostic method used. IUAs formation is multifactorial with several influencing factors. The underlying mechanism of IUA formation is still unknown; the pathophysiology and mechanism of tissue repair of the endometrium are poorly understood while the process that leads to IUA formation is still obscure. There is often a definable causative event on an unknown predisposing background.

Pregnancy is considered one of the most important predisposing factors. Approximately 91% of cases with IUAs are pregnancy related; adhesions appearing in 67% after miscarriage or abortion curettage and in 22% after postpartum curettage [3]. The enhanced risk of IUAs formation could be that the endometrium of the gravid uterus is in a recovering state; manipulation of the endometrium disturbs the healing process in a way, enhancing IUAs formation. Destruction of the basal layer of the endometrium is considered an essential factor in the process of adhesion formation.

Besides the gravid uterus other determinants play a role in adhesion formation such as infection or inflammation. Infection and inflammation are both known to enhance adhesion formation [4]. Low estrogen state, also known as a hypo-estrogenic condition during the puerperium and lactation are related to an increase risk for developing adhesions [5]. Constitutional characteristics are also considered an important factor.

IUAs may be asymptomatic but often causes menstrual disturbances, infertility and recurrent miscarriages. If pregnancy occurs, it is frequently complicated by (recurrent) miscarriage and ectopic pregnancy. Furthermore, abnormal placentation, fetal growth restriction, fetal anomalies, premature labour and delivery and postpartum haemorrhage also have been described as complications of IUAs [3]. IUAs may have serious consequences. The underlying mechanism for infertility in women with IUAs seems to be related to obstruction of sperm transport into the cervix, impaired embryo migration within the uterine cavity, or failure of embryo implantation due to endometrial insufficiency. The reproductive prognosis of women with minimal IUAs is considered moderately good after adhesiolysis but in women with moderate to severe IUAs, the reported reproductive outcomes are poor [6].

Prevention of IUAs formation is crucial. Clinicians should strictly maintain the indication for surgical intervention during pregnancy or after delivery as the gravid uterus predisposes to adhesion formation. Besides an accurate indication, it is essential to performed surgery in a gentle manner avoiding unnecessary trauma. Matters for the prevention and reduction of IUAs should be considered, but the evidence remains limited. Randomized controlled trails with long follow-up are lacking. Further research on this topic is essential and urgently needed.

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Volume 7 Issue 3 March 2018