Central Asian Journal of Medicine

CURRENT PROBLEMS WITH INTRACANALICULAR FALLOPIAN TUBE OBSTRUCTION (Literature review)

Gulsanam B. Malikova[1], Bakhtiyor M. Abdurahmonov[2]

1 Accosiate Professor, Candidate of Medical Sciences, Multidisciplinary Clinic of the Centre for Development of Professional Skills of Medical Workers, Tashkent, Uzbekistan
E-mail: gulsanam23@mail.ru

2 Multidisciplinary Clinic of the Centre for Development of Professional Skills of Medical Workers, Tashkent, Uzbekistan
E-mail: bakhtiyr.abdurakhmanov@mail.ru

ABSTRACT

The most common cause of tubal obstruction is inflammation. It may be due to sexually transmitted diseases, infectious complications after abortion. According to the literature data, fallopian tubes patency after an infectious process is more often impaired in the ampullary and less often in the interstitial region. This is due to the peculiarities of blood supply in the ampullary sections of the fallopian tubes by the type of venous lacunas and arteriovenous anastomoses, with the predominance of ciliated epithelium, unstable to damaging factors, especially infectious nature. These anatomo-physiological features predetermine the predominant involvement of the ampullary parts of the fallopian tubes, regardless of the type of infection and its route of entry into the tube. In chlamydial, mycoplasma, gonococcal, viral and other ascending infections, adhesions and obliterations develop in the fallopian tubes, resulting in complete or partial tubal occlusion due to epithelial and/or muscular layer lesions, leading to compression of the tube from outside, Pathological curvature of the tubes, impaired contractile function, fimbriae, peritubular and ovarian adhesions, reduced number of endosalpinx cilia, resulting in impaired function of the fallopian tubes in receiving and feeding the egg, sperm and embryo, gamete and embryo transport.

Key words: pregnancy, infertility, fallopian tubes, salpingit, fimbriae, infection, uterus.

INTRODUCTION

Infertility, i.e. the inability to procreate, is a serious condition that impairs a person's social and psychological adjustment and affects their health and quality of life. For this reason infertility in the family remains one of the most important medical and public health problems. According to world statistics, one in 5 married couples suffers from infertility, and so the problem remains acute (1). Numerous

studies in the CIS and non-CIS countries are devoted to this problem. The frequency of infertile marriage, according to some authors, ranges from 10-15% to 18-20%. The cause of infertile marriage in 40-50% of cases is a pathology of the reproductive system in one of the spouses, less often (5-10% of cases) - both. In 60-80% of these marriages the cause of infertility is a woman's health condition and in 30-40% of cases the cause is a disease of the husband. The use of hormonal, endoscopic and ultrasound techniques has made it possible to identify the main factors causing infertility and to determine the structure of infertile marriages. Infertility can be primary if the woman has never had a pregnancy, and secondary if she has had a pregnancy in the past and absolute female infertility (2). Absolute female infertility means that pregnancy is completely impossible (absence of uterus, ovaries and other genital abnormalities). Female infertility is established only after the infertility of the husband (partner) has been excluded. The infertility rate for women is as high as 45%, for men as high as 30% (3).

The main types of tubal abnormalities in infertility:

a) complete or partial permeability;

b) an abrupt disturbance of the tubal function of oocyte retrieval and transfer due to hypothalamic-pituitary-ovarian dysfunction or due to peritubular changes;

c) a combination of anatomical and functional changes.

According to the literature, tubal factor is found in 30-85% of women suffering from infertility, and the peritoneal form of infertility occurs in 9.4-34% of cases (4). This form of infertility is characterised by anatomical and functional impairment of the patency of one or both fallopian tubes and/or pelvic adhesions, usually due to inflammatory diseases, surgical procedures on the pelvic organs, intestines; complicated abortions or deliveries; pelvic peritoneal diseases (external endometriosis). In this form of infertility, there are no violations of hormonal function of the ovaries, the menstrual cycle is not disturbed and has an ovulatory nature, there are no clinical manifestations of endocrinopathies (obesity, hirsutism, virilisation). The incidence of tubal infertility, caused by a mechanical obstruction of sperm-ovine fusion, is 42.5-80.5% in women with primary infertility and 48.2-73.1% in women with secondary infertility [5]. According to WHO summary data, complete fallopian tube occlusion is found in 14.2% of those examined, with post-inflammatory tube changes not resulting in complete occlusion diagnosed in 9.2% of those examined (6). These figures indicate that more than 20% of patients with infertility have marked anatomical changes in the fallopian tubes. Thus, fallopian tube obstruction can be organic (i.e. there are some anatomical changes in the tubes, mechanical obstacles disturbing their patency, adhesions, torsion, sterilisation, etc.) or functional (the structure of the tubes is not altered, no

mechanical factors disturbing their patency, etc.).), or functional (the structure of the tubes is not altered, no mechanical factors impede their permeability, nevertheless, the tubes' function is impaired (hypertonicity, hypotonicity, discoordination) and, consequently, the transport of sperm to the egg and of the fertilized egg to the uterus is hampered). The separation of the main groups cannot always explain all causes of infertility, so a group of patients with so-called unexplained infertility, whose cause cannot be determined by the applied research methods. The most common cause of tubal obstruction is an inflammatory process. It can be caused by sexually transmitted diseases, infectious complications after abortion. According to the literature, tubal patency after an infectious process is most often affected in the ampullary and less often in the interstitial region (5). This is due to the peculiarities of the blood supply to the ampullary parts of the fallopian tubes by the type of venous lacunae and arteriovenous anastomoses, with the predominance of ciliated epithelium, unstable to damaging factors, especially of infectious nature. These anatomo-physiological features predetermine the predominant involvement of the ampullary parts of the fallopian tubes, regardless of the type of infection and its route of entry into the tube. In chlamydial, mycoplasma, gonococcal, viral and other ascending infections, adhesions and obliterations develop in the fallopian tubes, resulting in complete or partial tubal occlusion due to epithelial and/or muscular layer lesions, leading to compression of the tube from outside, abnormal tube curvatures, abnormal contractile function, fimbriae structure, peritubular and ovarian adhesions, reduced number of endosalpinx cilia, resulting in abnormal function of the fallopian tubes in receiving and feeding the egg, sperm and embryo, gamete and embryo transport (7). They can also displace the ovaries and fallopian tubes, disrupting their normal anatomy. Various infective agents enter the fallopian tubes through the bloodstream, ascending from the lower genital tract and with spermatozoa. The latter, having reached the fallopian tubes, die after 4-5 hours and the bacteria actively multiply using the sugar contained in the seminal plasma and cause inflammatory disease. Chlamydia is the most common cause of organic obstruction of the fallopian tubes. Unfortunately, in most cases the disease is asymptomatic and the woman may not even be aware of the infection. According to some reports, half of all women with fallopian tubes are found to have chlamydia. For example, according to J. Henry-Suchet et al (1998), chlamydia causes inflammatory changes in the fallopian tubes within the first year of initial infection and can be detected during laparoscopic manipulations. After a single episode of salpingo-phoritis, fallopian tube obstruction is detected in 11-13% of patients, twice in 26-35%, and three times in 54-75% of patients. At the same time, there is evidence that acute salpingitis can

lead to peritubular adhesions without causing marked anatomical and functional disturbances in the fallopian tubes. Thus, according to Westrom (1984) who performed control laparoscopy in patients who had acute salpingitis confirmed by laparoscopy, pelvic adhesions were found in 63% of cases and tubal patency was undisturbed in 40% of these patients (8). Tubal adhesions can develop after pelvic surgery (removal of myoma nodules, removal of the tube after ectopic pregnancy, removal of ovarian cysts, caesarean section). In some cases, tubal obstruction may also develop after abdominal surgery, e.g. after appendicitis. Abortion is one of the frequent causes of subsequent pelvic inflammatory diseases. While after a single abortion these complications occur in 16% of women, after 3 or more abortions they occur in 100%. In 1/3 of women with secondary infertility, an induced abortion has terminated the first pregnancy (9). Tubal adhesions leading to obstruction are particularly common if the operation and the post-operative period have been accompanied by complications. Mynbaev O.A. (1997), conducting a retrospective analysis of the medical records of repeatedly operated women, found a close correlation between the frequency of formation of massive (grade 3-4) postoperative adhesions in the pelvis and the increasing number of repeated operative interventions on abdominal organs. According to A. I. Volobuyev and V. G. Orlova (1985), severe disturbances of the contractile activity of the fallopian tubes are observed in hyperandrogenism, and they are more pronounced in the subclinical form of adrenal hyperandrogenism than in mixed ovarian-adrenal hyperandrogenism. This is evidenced by the fact that 54% of women have tubal pregnancies against a background of various hormonal dysfunctions, with 40% of them having adrenal hyperandrogenism (10). In most women, the cause of occlusive lesions of the TM is an inflammatory process initiated by a genital infection. A mixed infection with chlamydiae, mycoplasmas and gonococci, which form associations with each other as well as with other infections, in particular trichomonads, is now thought to play a major role in the formation of marked anatomical changes of the TM (11). After the first infection, tubal abnormalities occur in 10% of women, after re-infection in 35%, and a triple infection leaves its pathological 'footprint' on the tubes in 75% of women (12). The damage to the fallopian tubes is usually bilateral and, if left untreated, can lead to infertility over time. Pathological changes can affect the fallopian tubes along their entire length. In a primary acute infection of the fallopian tube, it is mainly the mucous membrane that is damaged. The fissures that arise after an initial lesion of the tube usually appear as thin, rather loose membranes that already at this stage disrupt the oocyte retrieval and transport mechanism. After relapses of chronic salpingitis, much denser adhesions appear, deforming the tube and severely impairing its

function (13). In parallel, there is an increase in fibroblast activity in the serous membrane of the tube and peritubal adhesions often develop. Uterine tubal patency is most often impaired in the ampullary, less often in the interstitial, and most rarely in the isthmic region as a result of the infection process. This phenomenon is associated with the peculiarities of blood supply to the ampullary parts of the fallopian tubes by the type of venous lacunas and arteriovenous anastomoses as well as with the predominance of ciliated epithelium unstable to damaging factors, especially infectious nature. These anatomo-physiological features predetermine the predominant involvement of the ampullary parts of the fallopian tubes, regardless of the type of infection and its route of entry into the tube. Inflammatory process in the ampullary parts of the fallopian tubes is often accompanied by fimbriae adhesion at the sites of ciliated epithelium death and gross anatomical changes of the fallopian tube as hydrosalpinxes. N.I. Kondrikov (1969) found morphofunctional changes in all layers of the fallopian tubes in chronic salpingitis. As the chronic inflammatory process progresses, collagen fibres proliferate in the stroma of the mucosal folds, the muscular wall of the fallopian tubes and under the serosal cover. Blood vessels gradually undergo obliteration and acidic mucopolysaccharides accumulate around them. Functional changes develop as well, with decreased levels of RNA and glycogen and reduced glycoprotein content in the tubal secretion. All these changes can impair oocyte transport or cause oocyte death (14). However, there is evidence that acute salpingitis can lead to peritubular adhesions without causing marked anatomical and functional abnormalities in the fallopian tubes. The most common cause of secondary tubal infertility is abortion. Tumours of the uterus and ovaries can lead to tubal infertility. The tubes are either mechanically compressed or their function is impaired. Tubal permeability is affected by foci of endometriosis, the development of which is associated with endometrial implantation in the tubes due to antiperistaltic menstrual blood flow or intrauterine manipulations (mucosal curettage, blowing, hysterography, etc.). Endometrioid heterotopias in the tubes, which have been increasing in frequency in recent years, may cause infertility (complete occlusion of the tube) or the development of a tubal pregnancy. Tubal obstruction due to endometriosis occurs in 10-15% of cases. The intensity of the adhesion process is determined by the classification of S. Hulka et al (1978). This classification is based on a visual analysis of the adhesions. According to this classification, all adhesions fall into four categories.

Grade I: adhesions are minimal, the tubes are passable and most of the ovary is visible ovary.

Grade II: more than 50% of the ovarian surface is free, ampullary occlusion with retained folds.

Grade III: Less than 50% of the ovary is free, ampullary occlusion with destruction of the folds. destruction of the folds.

Grade IV: no ovarian surface visible, bilateral hydrosalpinx.

Grade I and II adhesions are found to be filmy, easily ruptured adhesions around the fallopian tubes and ovaries. In grade III and IV pelvic adhesions, the adhesions are more often dense, vascular, and difficult to separate; the uterus, intestines, and omentum are involved in the pathological process. Almost all types of adhesions result in a disturbance of the functional and anatomical contact between the tubal and ovarian fibrils. With minor adhesions, the tubes are passable, but even so, one has to be very careful about infections. In severe adhesions (stage III and IV), the tubes are completely closed and the ovaries are separated from the outside world (15). Uterine tube lesions do not only appear as tubal occlusions. Less profound disturbances may not be accompanied by occlusion of the tubal lumen, but there is a disorder of the coordinated mechanisms of ciliary, secretory and muscular activity, resulting in impaired sperm promotion, oocyte capture and post-fertilisation transport to the uterus. This kind of disturbance can cause an increased risk of ectopic implantation. The latter explains the fact that the treatment of tubal diseases increases the risk of ectopic pregnancy, since some surgical or therapeutic interventions do not always normalise tubal function after restoring tubal anatomical patency. Researchers have found that tubal infection results in a threefold increase in the number of ectopic pregnancies. Thus the study of the causes of infertility has made some progress, the factors that cause its development have been established and sufficiently effective methods of diagnosis and surgical treatment have been developed. However, the problem of further improving the methods of surgical treatment and rehabilitation of the reproductive function of women suffering from intracanalicular obstruction needs to be solved and remains topical.

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