CONTINUING MEDICAL EDUCATION

SEXUALLY TRANSMITTED DISEASES AND INFERTILITY

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Infertility, defined as lack of recognized conception after one year of unprotected intercourse, affects 10 to 15% of couples in the reproductive age group. Sexually transmitted disease is a well-recognized cause of infertility.

The main offenders in STD-associated infertility are *Chlamydia trachomatis, Neisseria gonorrhoeae and Mycoplasma hominis.*[1,2] In women STDs affect fertility through salpingitis and pelvic inflammatory diseases (PID). Mardh et al have found that acute salpingitis was associated with chlamydial infection in 58%, gonococcal infection in 8%, and mycoplasma in 12%.[3] However, this may vary in different populations. The most extensive long term follow up after PID is the study from Lund, Sweden by Westrom and colleagues.[6] They found that each new episode of salpingitis approximately doubled the rate of post salpingitis infertility.[4] It is distressing to note that upto one-third of patients suffer from repeated attacks, the second attack usually occurring within one year.[7,8] Women with severe inflammatory changes have five-fold higher rates of subsequent infertility.[5] Other risk factors for tubal infertility are (1) age: women between 15 and 24 years who develop salpingitis have less tubal damage than older women[4] (2) contraceptive choice: women using intrauterine contraceptive devices were found to have a higher risk of tubal factor infertility.[9] Barrier methods provide protection.[10] The association of oral contraceptives with tubal infertility is unclear.[6] In one study OCP use did not protect against tubal infertility.[10] Washington et al suggests that OCP use protects against symptomatic PID, but promotes chlamydial infection of cervix and then causes atypical PID.[11] In contrast the Lund series and certain other studies have reported a protective effect of OCP against PID.[12-14] OCP lowers the risk of ascent of lower genital infections by changes in cervical mucus and decreasing the retrograde flow of menstrual blood. Also OCP were found to have a direct protective effect which prevent growth of chlamydia in the upper genital tract. (3) Smoking in women increases risk of tubal factor infertility by impairing immune defenses. (4) Neither the antibiotic used[4] nor the organism isolated[15] made any discernible impact in subsequent infertility. However some studies contend that gonococci associated salpingitis in younger females (less than 25 years) have a significantly better prognosis.[5] Rosenfeld et al demonstrated that 50% cases with tubal infertility had no history of PID referred to as silent salpingitis.[16]

According to WHO multicenter study in African women tubal infertility due to salpingitis accounted for two-third of the total cases of infertility.[17] In other developing countries the prevalence of tubal occlusion

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among infertile women is 14 to 15% and in developed countries it is 11%.[17] The tubal damage in PID may be in the form of:[18]

1. Endosalpingeal mucosal damage leading to restricted mobility of tubes, paraphimotic ostium, intraluminal adhesions or deciliation all of which can lead to impaired ovum pick up and transport.

2. Tubal occlusion. Either distal or proximal occlusion of distal end of fallopian tubes leads to retort-shaped tubes (sactosalpinx) and hydrosalpinx.

3. Peritubal damage : Includes adhesions and restricted mobility.

***N. gonorrhoeae*:** In the endosalpinx they attach to mucosal epithelial cells penetrate them and cause cell destructiuon. They do not attach to ciliated cells, but produce diminished beating of cilia, by releasing membrane blebs containing Lipid A.[1]

C. trachomatis : They produce self-limited acute infection which resolves into low grade persistent infection in genital tract lasting for years and leading to chronic inflammation and scarring.[1,20] Chlamydial infection leads to release of cytokines (ΓIFN) which delays chlamydial developmental cycle so that reticular bodies persist longer.[19] Lymphokines also lead to fibroblast proliferation and collagen deposition which can cause scarring.[19] Another interesting feature of chlamydia- associated salpingitis is that it produces tubal dysfunction either as deciliation or ciliary dyskinesia.[20] Ciliary dyskinesia can lead to persistent infertility even after re-establishing tubal patency. Also chlamydia can persist in endometrial cavity following therapy for acute salpingitis despite apparent clinical cure leading to relapse or chronic infection.[21] Investigators from 13 different cities have documented that tubal occlusion is strongly associated with the presence of chlamydial antibody (70% of women with tubal infertility had chlamydia antibodies vs 26% of control women).[6]

Mycoplasma : Role of mycoplasma in infertility is controversial. Mycoplasma are considered by some authors to be part of normal genital flora of many sexually active males and females with no clinical disease. Hence they are not considered important in causation of infertility.[1,22] However *Mycoplasma hominis* have been found to cause slowing of mucociliary wave.[23] They may interfere with transmission of spermatozoa and fertilisation of ova directly.[23] Also the inflammation and scarring produced by mycoplasma can interfere with implantation of ova.[23] Moller et al have demonstrated that antibodies to *M.hominis* occured three times more often in women with tubal infertility than in other infertile women (36% vs 11%).[24] The incidence of ureaplasma infection is only significantly higher in those women whose partners have semen abnormalities.

Mucopurulent cervicitis (MPC) can be caused by either C.trachomatis or N.gonorrhoeae. Although asymptomatic, MPC may lead to endometritis or silent salpingitis. It is stressed that MPC is a treatable condition which when linked with either subclinical or symptomatic salpingitis or endometritis can lead to tubal damage which is much preventable.

In males, infertility occurs as a sequelae of epididymitis and/or prostatitis with or without testicular involvement. Berger et al have found low sperm counts in a high proportion of patients with acute epididymitis.[26] Patients with bilateral epididymitis and bilateral occlusion of vas or epididymis have virtually a very low fertility potential.[1,25] Analogous to silent salpingitis in women, Gartman reported histological evidence of epididymitis in infertile men with no history of epididymitis leading to scarring and decreased fertility.[27] The usual aetiological agents in epididymitis are *Chlamydia trachomatis* and *N. gonorrhoeae*. Ureaplasma induced epididymitis has not

been reported.[1] Predisposing factor is sexually transmitted urethritis. Retrograde passage of infected urine occurs from the urethra along the lumen of vas deferens to epididymis. Ducts of epididymis are distended with PMNL which phagocytose the sperm. Tubular epithelial damage, microabscess formation, mono-nuclear infiltration and formation of sperm granuloma are subsequent events.[25] The inflammation may spread to testis. Vacuolation of sertoli cells is found to occur. After 2-3 years testicular atrophy proportional to degree of inflammation may result.[25] Bilateral occlusion of lumen of epididymis can lead to sterility. In sub Saharan Africa, epididymitis is the leading cause of male infertility.[28] According to Pelouze with only a history of gonococcal urethritis 10.5% of patients had a history of infertility.[29] Ludwig and Haselberger reported on 46 patients with unilateral epididymitis followed for 1 year. They found initially 66% were oligospermic, however only 20% remained so after 1 year. Also, epididymitis may be associated with anti-sperm agglutinating antibodies.[30]

Prostatitis: It is caused mainly by gonococci and chlamydia. Mycoplasma has not been isolated in semen or prostatic fluid. Sperms are infected with genital mycoplasma as they pass through the urethra during ejaculation. Prostatitis may elict an immune response in the form of antisperm antibodies (39% of patients with prostatitis showed spermatozoal antibodies in serum by indirect immunofluorescence). Zinc in the ejaculated seminal plasma is essential for sperm motility and capacitation. This is reduced considerably in prostatitis leading to reduced sperm motility.[31]

Mycoplasma and male infertility: Mycoplasma could reduce fertility in men by decreasing sperm motility, inhibiting spermatogenesis, and forming abnormal sperms.[23,33] However Upandhyaya et al and Talkington et al failed to demonstrate any significant influence of mycoplasma on semen characteristics.[1]

Interestingly, although most authors agree that the presence of serum antichlamydial antibodies or presence of genital mycoplasma do not usually affect semen characteristics, both are suggested as being associated with the presence of sperm agglutinating antibodies in serum. A recent study (Ochsendrof FR, Ozdemir et al, Frankfurt in 1999) has reported that IgA antichlamydial antibodies in seminal plasma appeared to be specific against *C.trachomatis* and were associated with an inflammatory response in male genital tract[34] (higher PMN-elastase level in seminal plasma). The results of another study indicate that in asymptomatic patients the presence of chlamydial antibody IgA or IgG in semen is not associated with reduced semen quality or other determinants of male fertility; however, seminal chlamydial antibodies are significantly related to a tubal infertility factor of female partners.[37] Other organisms in STD associated infertility are:

1. *Trichomonas vaginalis: T. vaginalis* has been isolated from 10% of some series of infertile men.[32] Some in vitro association between spermatozoa and TV have been reported; the significance of which is unknown. TV can immobilise spermatozoa.

2. *C. albicans*: It can immobilize spermatozoa and also causes spermatozoal agglutination.

3. *Herpes simplex virus:* HSV-1 and HSV-2 DNA have been detected in the nuclei of spermatozoa and is found to correlate inversely with sperm count and motility.[35]

4. *Human immunodeficiency virus* (HIV) affects fertility by increasing the mortality and morbidity of reproductive populations.[36]

5. *Treponema pallidum* : Late manifestations of syphilis viz.gumma of testis and tabes dorsalis can lead to impotence.

6. *Bacterial vaginosis* may be risk factor for

development of PID.

7. LGV and granuloma inguinale may distort the architecture of genitalia and contribute to failure of conception. In addition LGV can cause alteration of columnar cells and associated inflammation of urogenital tract in both sexes leading to infertility or ectopic pregnancy in females and infertility and impotence in males.

Management

The diagnosis of STD-associated infertility is restrospective, the time interval between the genital infection and complaint of infertility is often in the magnitude of years and more often than not, it is difficult to identify the causative organism or identify the sequence of events leading to infertility. These factors should be borne in mind before embarking on investigations which may not always be fruitful. In both sexes a detailed history and an astute clinical examination is a must.

In women smears should be taken form the cervix, endometrium, tubes, cul de sac and duct of Bartholin's gland, for gram staining and culture. The rate of recovery of gonococci in cases of chronic PID may be unrewarding. However, chlamydia has been isolated by culture (which has 100% specificity) in upto 30% cases of tubal infertility.[38,39] Antigen detection can be done by PCR or ELISA or DFA testing. A clean catch mid stream sample of urine should be obtained for microscopy and culture. DNA hybridisation techniques provide additional information (Chlamdia DNA detected in 49% of infertile patients with tubal infertility).[40]

Serological tests : There are several serological tests available. Enzyme immuno assay using purified pili antigen of N.gonorrhoea helps in detecting gonococcal antibodies.[2] Enzyme immuno assay (EIA) helps in demonstrating mycoplasmal antibodies.

Serological test for C.trachomatis: The direct immunoperoxidase assay is both specific and sensitive for the detection of chlamydial IgM antibodies.[41] Other tests include indirect fluorescent antibody assay, rELISA (based on a recombinant lipopolysaccharide antigen which detects chlamydia genus specific antibodies) and peptide EIA (which detects chlamydia genus specific antibodies and is based on a peptide derived from major outer membrane protein)[42]

Evaluation of tubal patency and peritoneal factors:

1. Hysterosalpingography (HSG)-Intraluminal adhesions are observed as patchy filling defects - Leopard skin appearance

2. Laparoscopy is the gold standard for diagnosis of PID

3. Others include color doppler flow USG, trans-cervical salpingoscopy, and fimbrioscopy.

Men are referred to infertility clinics because of oligospermia and are unaware of any degree of genital infection. Examination of urethral smear, prostatic massage and two glass urine tests are done. In men in the absence of evidence of infection or inflammation (Gram strain of urethral discharge >5WBC/OIF or urine microscopy >10WBC/HPF) routine cultures of genital tract are not indicated.[43] A semen analysis is imperative and this includes sperm count, motility, morphology and detection of potential markers of infection. Antigen and antibody detection should be carried out in semen. Antibody detection in serum should also be done.

Other investigations done are scrotal USG, seminal vesiculography, vasography and testicular biopsy.[43]

Prevention: Among the many causes of infertility those related to STD are possible to prevent but difficult to treat.

Primary prevention involves STD control measures and blocking acquisition of infection. Secondary prevention blocks the progression of low genital infections to upper genital tract by institution of prompt and

appropriate antibiotic therapy. Tertiary prevention blocks the progression of upper genital tract infection to tubal obstruction and eventual infertility. Tertiary prevention is disappointing as the type of antibiotic given for PID has only a minimal influence on fertility outcome. The currently recommended regimes will cure acute PID,[44] but have limited effect on existing tubal damage or they may not produce actual eradication of chlamydial infections.[45]

Treatment: Women with bilaterally obstructed fallopian tubes are sterile and will not conceive unless the tubes are either surgically re-opened or by passed as in invitro fertilisation (IVF)[46] and embryo transfer (ET).

Surgery - Microsurgical techniques and operative laparoscopy includes salpingo-ovariolysis, fimbrioplasty and salpingostomy.[47] Surgery is contra-indicated in case of frozen pelvis, leopard skin appearance on HSG and bipolar tubal occlusion.[47] The overall pregnancy rates after surgery is 23% within two years of surgery.[46]

For those whom surgery is contraindicated assisted reproductive techniques (ART) have to be resorted to and includes Invitro fertilisation-Embryo transfer (IVF-ET), Gamete intra fallopian transfer (GIFT) and Zygote intra fallopian transfer (ZIFT). The pregnancy rates with IVF is 9-10 percent per cycle.[48]

Lunenfeld et al have demonstrated that the take home baby rate in IVF-ET programmes was higher in patients who are chlamydia seronegative.[49] Hence many authors propose a course of doxycycline prior to IVF to optimise pregnancy rates.[50]

There is conflicting data on the use of doxycycline in infertile couples to eradicate ureaplasma. Toth et al using doxycycline successfully eradicated ureaplasma from 129 infertile couples.[51] They found that the 3-year conception rate was 60% compared with only 5% for 32% of infertile couples in whom ureaplasma could not be eliminated.[51] In contrast Upandhyaya et al reported that pregnancy rates were uninfluenced by treatment with doxycycline.[52]

In men, if there is evidence of genital tract infection or inflammation a combined anti-microbial, anti-inflammatory treatment is required.[43-53] Surgical treatment in males is feasible only if vas deferens is uninvolved and includes laterolateral vaso epididymostomy and epididymolysis.[43]

Antisperm antibodies are the cause of immunologic infertility.[54] Chlamydia and mycoplasma are but two of the many causes of antisperm antibodies. They pose a therapeutic challenge and may respond to high dose corticosteroids, danazol or insemination with washed sperms.[54,55]

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