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Tubo Ovarian Abscess: Risk Factors and Clinical Features in Turkish Population

Key Words: Tubo-ovarian abscess, pelvic inflammatory disease, sexually transmitted disease, risk factors.

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Introduction

Tubo-ovarian abscess (TOA) is one of the most common causes of pelvic masses in reproductive-age women (1) and it is a serious sequelae of acute pelvic inflammatory disease (PID) (2).

The incidence of tuboovarian abscess is expected to increase as a result of the current epidemic of sexually transmitted disease and their sequelae (3). As a result of the rising incidence of sexually transmitted disease, such as gonorrhoea, chlamydia and the widespread use of intrauterine contraceptive devices, the problem of both acute and chronic salpingitis (PID) remains a major health concern (3).

These abscesses usually occur in sexually active women between the ages of 20-40 years (1). Risk factors for TOA are similar to those for PID, such as multiple sexual partners, prior history of PID, no contraceptive use, intrauterine device (IUD) use and low socioeconomic status (4).

The parity of these patients is variable with approximately 25% to 50% being nulliparous (3).

Abdominal or pelvic pain is the most frequent presenting complaint in more than 90% of cases of tubo-ovarian abscess reported in the literature (3). Patients with TOA typically present with a history of abdominal or pelvic pain, which may or may not include fever ($>38.5^{\circ}\text{C}$) and chills (1). Laboratory data of patients with TOAs are nonspecific. Despite the surgically confirmed presence of a TOA, 35% of patients were afebrile and 23% of patients had a normal leukocyte count on admission in one study (1). Physical examina-

tion may reveal lower abdominal tenderness, often with peritoneal signs such as guarding and rebound tenderness. Excess cervicovaginal secretions and evidence of cervicitis can be found on pelvic examination (1).

In two years (1994-1995), at gynecology clinic of Dr. Zekai Tahir Burak Women's hospital, twenty-one patients were diagnosed as having tubo ovarian abscess. These cases were evaluated retrospectively for risk factors and clinical diagnosis. The patients ranged from 19 to 41 years of age with a mean of 30.8 ± 5.7 years SD. The parity of these patients were variable. Their parity were between 0-7 with a mean of 3.5 ± 1.7 SD. Three of the twenty-one patients were observed as nulliparous (14%). Abdominal pain was the most frequent complaint, which we have seen in 85% of our cases of TOA. Other complaints were foul smelling vaginal discharge (47%), vaginal bleeding (28.5%). Physical examination revealed lower abdominal tenderness for most of the patients but rebound tenderness were found in five cases of the twenty-one patients. Most of the patients had subfebrile fever, just three of them had fever more than 38.5°C . Ultrasound examination correctly identified a TOA in eighteen of twenty one patients. In our study risk factors for TOAs were colpotomy operation due to prior history of pelvic inflammatory disease (4.7%), septic abortion (4.7%), prior history of laparoscopically tubal ligation (9.5%), IUD usage (33.3%). Fifteen of the twenty-one patients responded to clindamycin, aminoglycoside treatment. Two of the twenty-one patients with medical treatment resistant dysfunctional

uterine bleeding were treated with total abdominal hysterectomy and bilateral or unilateral oophorectomy. Both cases were in late reproductive years without further desire of conception. One of the twenty one patients who did not respond to medical treatment, we performed posterior colpotomy plus abscess drainage following laparotomy.

The pathogenesis of TOA has not been elucidated fully (1). Although TOA are often a sequelae of acute PID, many women have no antecedent history of pelvic infections or sexually transmitted disease (1).

The initiating event in the formation of a TOA is believed to be the invasion of fallopian tube epithelium by a pathogen causing tissue damage and necrosis, thereby providing an ideal environment for anaerobic invasion and growth (1). In our study, history of PID was found in three of the twenty-one patients. A number of studies, however have shown that a prior history of PID is obtained in only one third to one half of patients presenting with tuboovarian abscess (1,3). This may indicate that subclinical infections assessed the sensitivity of ultrasound in the detection of pelvic abscesses to be 93% and the specificity 98.6% (1). Our results were similar to the other series, ultrasound examination was correctly identified a TOA in eighteen of the twenty-one patients (85%).

The management of TOA changed dramatically after the development of broad spectrum antibiotics. Because most women with TOA are in their reproductive years, the usual and accepted approach is conservative; either medical, surgical or a combination of the two, to preserve fertility (1).

The treatment regimens must include antimicrobial agents that have appropriate coverage against organisms commonly found in TOA. Medical therapy also reduces the added morbidity of surgery. In addition to the preservation of fertility, conservative therapy maintains endogenous ovarian steroid production (1). With medical treatment, Landers et al and Sweet et al had excellent clinical cure rates of 90-100% (1,5). Harold et al currently recommend either a combination of clindamycin plus aminoglycoside or cefoxitin plus doxycycline as conservative therapy for TOA (1). In our institution, we used clindamycin plus aminoglycoside with cure rate of 71.4%. Landers et al treated their patients with clindamycin plus aminoglycoside, and their cure rates were 72-73% respectively (5). Our results were similar to these studies. It appears that clindamycin plus aminoglycoside combination is an effective regimen in the conservative treatment of sonographically or laparoscopically confirmed TOA.

There are three main indications for surgical intervention in patients with suspected TOA; 1. Intraabdominal rupture. 2. High index of suspicion of other surgical emergencies, such as appendicitis. 3. Failure to respond antibiotic therapy within 48-72 hours (1).

Surgical therapy is necessary in approximately 25% of patients treated for TOA (1). In our study, we performed surgical therapy in 28.5% of our patients treated for TOA. On the other hand the size of the TOA may influence the decision or timing of the surgery. Harold et al reported that TOA larger than 8 cm were less likely to respond to antimicrobial treatment than smaller abscesses (1).

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