

International Journal of Medical Science and Dental Research

# Case report of secondary peritonitis due to tuboovarian abscess rupture

İsa KAPLAN<sup>1</sup>, Ömer SERT<sup>2</sup>, Serkan Yücel YILANCI<sup>3</sup>

 <sup>1</sup> Uşak University Faculty of Medicine, Department of Obstetrics and Gynecology Usak/Türkiye
<sup>2</sup> Uşak Training and Research Hospital Anesthesia and Reanimation Clinic Usak/Türkiye

<sup>3</sup> Aydın Maternity and Children's Hospital, Aydın, Türkiye

**Abstract:** The patient is 49 years old. The patient came to the emergency room with severe abdominal pain that started the day before. Abdominal computed tomography (CT) revealed a 6 cm semisolid hypointense cystic mass in the left ovary. Transvaginal ultrasonography (USG) revealed a 5x5x4 cm thick-walled abscess with the dense cystic lesion, bilateral hydrosalpinx, and minimal intra-abdominal fluid in the left adnexal area. There was a rebound and a defense. CRP value was 43 mg/L and there was no fever. The patient with acute abdomen was taken for emergency laparotomy. Intraoperatively, a ruptured abscess was observed around 6x6x5 cm in the left ovary. The entire abdomen was filled with purulent fluid. The patient underwent total abdominal hysterectomy and bilateral salpingo-oophorectomy. During the follow-up, the patient developed adynamic ileus. The ileus picture was followed up medically. The patient had a progressive increase in CRP values (CRP: 324 mg/L) and fever (39.5 °C) in the postoperative period. As a result of intra-abdominal culture, S. Marcescens and P. aeruginosa growth occurred. The final pathology result was a tubo-ovarian abscess (TOA). We wanted to draw attention to the rare case of rupture of TOA and secondary peritonitis in our case.

Keywords - Tubo - Ovarian Abscess, Peritonitis, Rupture of Abscess

## I. INTRODUCTION

Tubo-ovarian abscess (TOA) is an inflammatory disease of the tuba uterine, ovaries and adnexal surrounding tissues. Generally, TOA is a disease of the tuba uterina and ovaries. However, due to its close neighborhood, it also affects the bladder and intestines. It is more common in women of reproductive age. It often occurs following infection of the upper genital tract [1]. Symptoms include pelvic and abdominal pain, nausea, vomiting, high fever and tachycardia. Detailed pelvic examination may not be performed due to diffuse abdominal defense. On bimanual examination, an adnexal mass can usually be palpated. Rarely, when TOA ruptures, it can cause sepsis, which can lead to serious morbidity and mortality. Although there is no current mortality rate, the mortality rate of TOA has been reported to be around 1.7% in different studies [2]. Predisposing factors are multipartner, being in the reproductive period (15-25 years old), having a history of pelvic inflammatory disease (PID), and using an intrauterine device (IUD) as a contraceptive method. TOA is frequently encountered as a polymicrobial infection. Escherichia coli is the most frequently isolated agent in

patients with TOA [3]. Bacteroides species, mycobacterium tuberculosis, neisseria gonorrhea and chlamydia trachomatis can be counted as other factors. The most common complaints in patients are abdominal and groin pain, persistent foul-smelling vaginal discharge, and fever in advanced stages [4]. Apart from pelvic examination, fever, leukocytosis, high C-Reactive Protein (CRP) and sedimentation levels are important findings in the diagnosis. Ultrasonography (USG), on the other hand, is the first imaging method to be applied in TOA because it is easy to apply. Abdominal computed tomography (CT) can be used to evaluate adjacent organs and the clinical course of TOA. In the future, TOA may cause infertility and chronic pelvic pain syndrome due to tubal damage. Although the treatment varies according to the severity of the disease, it is usually medical and/or surgical [5]. In this case, we aimed to draw attention to the secondary peritonitis and adynamic ileus that developed due to TOA rupture.

### II. CASE

Our patient is 49 years old. There is a history of pregnancy that resulted in a total of 9 vaginal deliveries. Our patient has a history of Type 2 diabetes mellitus (DM) and uses oral antidiabetic agents. The operations in our patient's history are cholecystectomy, thyroidectomy, mini laparotomy and bilateral tubal ligation. Our patient had applied to the obstetrics and gynecology outpatient clinic many times with complaints of vaginal discharge and pelvic pain and was given treatment. No pelvic mass or TOA was found in the USG. Probe curettage was performed on the patient with a history of vaginal bleeding twice, and the result was irregular proliferative endometrium in one and atrophic endometrium in the other. The patient last came to the obstetrics and gynecology outpatient clinic 6 months ago, and the ovaries were found to be normal in the transvaginal USG, and no pelvic mass, hydrosalpinx or pelvic inflammation findings were found. Our patient applied to the emergency department of our hospital due to the excruciating abdominal pain that started the day before. In the first examination, the patient had acute abdomen findings, had a CRP value of 43 mg/L and had leukocytosis. A CT scan was performed in the emergency room for diagnosis of acute abdomen. Upon detection of a 6 cm semisolid hypointense cystic mass in the left ovary on CT, an emergency patient was consulted. CT image of the present mass is given in figure 1. In the abdominal examination of the patient, the entire abdomen was tender and rebound and defense were present. In the pelvic examination, there was increased cervical temperature and tenderness. The uterus was palpated at 12 weeks of age. Transvaginal ultrasonography (TV-USG) revealed a 5x5x4 cm dense cystic lesion, which could be a thick-walled abscess, and bilateral hydrosalpinx in the left adnexal area. In addition, minimal intra-abdominal fluid was observed. In the patient's armpit measurements, fever was absent during the first examination. In the patient with acute abdominal findings, an abscess of 6 cm was detected in the left ovary in imaging methods. TOA rupture and secondary peritonitis were considered primarily. The patient was taken to an emergency laparotomy. Under general anesthesia and sterile conditions, the abdomen was entered through a Pfannenstiel incision from the old scar line. It was observed that there was an excessive amount of purulent fluid in the abdomen filling the entire abdomen (Figure 2). A sample was taken from the purulent fluid for microbiology. The uterus was larger than normal and edematous secondary to infection. There was a ruptured 6x6x5 cm thick-walled cystic lesion compatible with TOA in the left ovary. Left tuba uterina was in the form of highly edematous infected hydrosalpenx. The right ovary increased in size and was extremely edematous and infected. The right tuba uterina was highly edematous, infected and hydrosalpenx. The image of the uterus and bilateral adnexa is given in figure 3. The inside of the abdomen was cleaned by aspirating irrigated with 2000 cc of warm sterile saline. Intestines, intestinal meso, omentum and the entire parietal peritoneum were in an extremely edematous state. No abscess formation was found in the liver and spleen on palpation. Appendix was normal. Then, type 1 hysterectomy was performed duly (Figure 3). A hemovac drain was placed in the abdomen. In addition, a hemovac drain was placed in the subcutaneous tissue due to the fact that the patient was obese and was in contact with the purulent fluid. The patient was taken to the ward without complications. The patient was started on ampicillin/sulbactam 2 g IV every 6 hours, gentamicin IV loading dose (2 mg/kg) followed by a maintenance dose (1.5 mg/kg) every 8 hours and clindamycin 900 mg IV every 8 hours. After 24 hours, the patient's CRP value was 157 mg/L, white blood cell value (WBC) was 12110 mm<sup>3</sup>, alanine aminotransferase (ALT) value was 57 IU/L, and aspartate aminotransferase (AST) value was 37 IU/L. In addition, the patient developed adynamic ileus on the first postoperative day. A standing direct abdominal radiography (ADBG) was taken in the patient with a widely distended abdomen without gas and stool output. Diffuse air-fluid levels were present in ADBG (Figure 4). A

nasogastric tube (NG) was inserted in the patient whose oral intake was stopped. In addition, enema treatment and mobilization were performed every 12 hours. Postoperative blood electrolyte levels of the patient were measured. IV potassium treatment was started in the patient with hypokalemia, whose potassium value was 3 mmol/L. The patient was consulted to the general surgeon and did not have any additional recommendations. As a result of the culture sent from the patient intraoperatively, Serratia Marcescens and Pseudomonas Aeruginosa were grown (Table 1). The patient's antibiotherapy was rearranged according to the culture antibiogram result. Ampicillin/sulbactam treatment was stopped in the patient who had ampicillin resistance as a result of the culture antibiogram. Since meropenem is sensitive, meropenem treatment, 500 mg IV every 8 hours, was started. On the second day of follow-up, CRP value was 277 mg/L, AST value was 16 U/L, ALT value was 34 U/L, and potassium value was 3 mmol/L. On the third postoperative day, the patient had a fever of 39 °C and above in the armpit measurement. Blood and urine cultures and control CRP were taken from the patient. Control CRP value came as 324 mg/L. There was no growth in blood and urine cultures. On the third postoperative day of the patient, the intra-abdominal and subcutaneous drains were withdrawn due to the lack of use. Adynamic ileus regressed with medical follow-up and treatment. After meropenem, the patient did not develop high fever again. Control CRP value came as 239 mg/L. No leukocytosis was observed. 48 hours after meropenem treatment, CRP value was 126 mg/L and potassium value was 3.31 mmol/L. Other blood parameters were normal and no fever was observed in the follow-up. In the follow-up, the patient's ileus picture improved. Effective blood glucose monitoring and regulation was performed during the patient's current hospital stay. CRP values measured at 24hour intervals decreased as 64, 31, 20 mg/L. The final pathology result of the patient was found to be TOA in both ovaries and tuba uterina (Table 2). The patient, whose clinically improved ileus picture regressed with medical treatment, did not have high fever, and whose CRP value was negative, was discharged to come to the outpatient clinic. The patient was called for outpatient control on the 3-7th and 15th days after discharge. The patient, who did not experience any problems in the outpatient clinic follow-up, was called for routine obstetrics and gynecology outpatient controls and was excluded from the follow-up.

## III. DISCUSSION

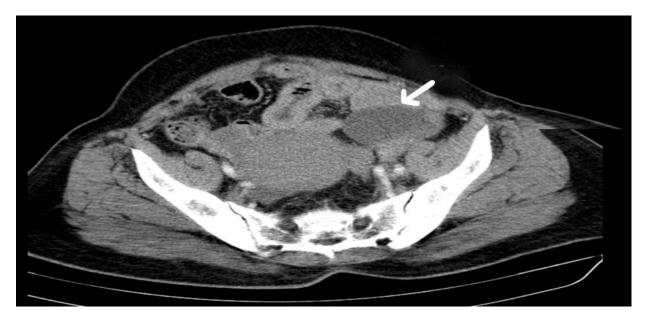
Tubo-ovarian abscess is the most advanced stage of acute pelvic inflammatory disease. In the long term, it causes widespread morbidity and mortality in patients. The diagnosis of TOA is made clinically and laboratory. A good anamnesis, pelvic examination and USG are the most commonly used methods for diagnosis. Abdominal CT, on the other hand, is the method frequently used for differential diagnosis. In the treatment, antibiotic therapy to be given by the IV route in hospital conditions is preferred. 75% of patients respond to IV treatment. However, in some patients who do not respond, surgical treatment is on the agenda. TOA is most common in women of reproductive age, that is, between the ages of 15 and 40, and can occur in any age group. In our case, the patient is 49 years old and the risk factor for PIH and TOA is only type 2 DM. He has no recent history of abdominal surgery. The most common complaints in patients with TOA are inguinal and abdominal pain, vaginal discharge, and fever in advanced stages [6-8]. As in every disease, the first priority is to create a good anamnesis from the patient when diagnosing TOA. Afterwards, TOA can be diagnosed with the help of physical examination, laboratory and imaging methods. In our patient, a preoperative diagnosis of ruptured TOA and secondary peritonitis was made with a good anamnesis, pelvic and physical examination, laboratory and imaging methods. In the diagnosis of TOA, the clinician's first imaging method is USG. With USG, myoma uteri, ovarian cysts, ectopic pregnancy and other genital system pathologies are excluded. Abdominal CT, on the other hand, is an assistive imaging method for the clinician to rule out appendicitis, abscesses that may be associated with inflammatory bowels, and other possible pathologies of the gastrointestinal tract. The formation mechanism of TOA is still not fully explained. It is generally seen after upper genital tract infections, especially following PIH [9-10]. TOA is often a polymicrobial infection. Escherichia coli, aerobic streptococci, Bacteroides fragilis, Prevotella and Peptostreptococci are the most common microorganisms detected in patients with TOA. Serratia Marcescens and Pseudomonas Aeruginosa were grown in our patient. In immunosuppressed and debilitated patients, Mycobacterium tuberculosis may rarely be the cause of TOA [9-10]. TOA may lead to complications such as chronic pelvic pain, ectopic pregnancy and infertility in the term [11]. When TOA ruptures, it causes sepsis and can lead to death. Therefore, patients diagnosed with TOA should be treated medically and/or surgically immediately after diagnosis. Medical treatment can be tried, especially in women

with TOA who have a mass less than 9 cm in size and who have no suspicion of rupture on examination [9]. Another treatment modality in patients with TOA is percutaneous abscess drainage under the guidance of CT or USG. However, despite 48-72 hours of IV antibiotic treatment, the patient's fever persists, and emergency surgery is recommended for patients with no reduction in abdominal pain, an abscess of 9 cm or more, and a clinical suspicion of rupture [9]. Although the size of the mass was 6 cm in our patient, medical treatment was not considered, and direct surgical treatment was planned for the patient, since the examination findings and USG findings were compatible with ruptured TOA and secondary peritonitis. Intraoperatively, TAH+BSO was applied to the patient considering the intense infection of both ovaries, tuba uterina and uterus, the contact of the entire abdomen with the abscess contents, and the age of the patient. Conventional treatment in patients with TOA is TAH+BSO and surgical removal of all infected tissue. There are studies showing that performing unilateral salpingo-opherectomy (USO) is sufficient in the treatment [12]. The method of operation to be performed in patients with TOA varies depending on the age of the patient and whether there is a desire for fertilization. TAH+BSO was performed in our patient, considering his comorbid conditions and age. In our case, the fever did not occur during the first examination, but came out later. In addition, high CRP is not at a stimulating level for sepsis or peritonitis. This situation may have been caused by the fact that the patient had type 2 DM for many years. Emergency surgery in the patient prevented possible sepsis and related morbidity and mortality. Effective diagnosis and treatment and good patient management provide a significant reduction in morbidity and mortality in these patients.

#### IV. FIGURES AND TABLES

Table 1. Culture result	
Bacteria: Pseudomonas spp	Number of Colonies: cfu mL
Antibiotic Name	Result
Meropenem	Sensitive
Seftazidim	Sensitive
Amikasin	Sensitive
Ciprofloksasin	Sensitive
Gentamisin	Sensitive
Aztreonam	Resistant
Piperasilin Tazobaktam	Sensitive
Bacteria: Serratia spp	Number of Colonies: cfu mL
Antibiotic Name	Result
Piperasilin Tazobaktam	Sensitive
Sefuroksim oral	Resistant
Trimetoprim Sulfametoksazol	Resistant
Amoksosisilin Klavulonik Asit	Resistant
Ampisilin	Resistant
Sefotaksim	Sensitive
Ertapenem	Sensitive
Gentamisin	Resistant
Siprofloksasin	Sensitive
Sefriakson	Sensitive
Seftazidim	Sensitive

Table 2. Pathology Result	
Diagnosis	
Squamous metaplasia nabolthi cysts	Uterine cervix
Irregular proliferative endometrium	Endometrium
Tubo-ovarian suppurative inflammation/abscess	Right ovary and tuba
corpus albicans	
Tubo-ovarian suppurative inflammation/abscess	Left ovary and tuba
corpus albicans	



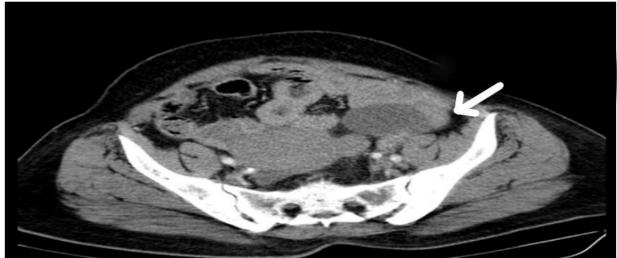


Figure 1. Abscess and intra-abdominal fluid are shown with arrow .



Figure 2. Content of intra-abdominal abscess

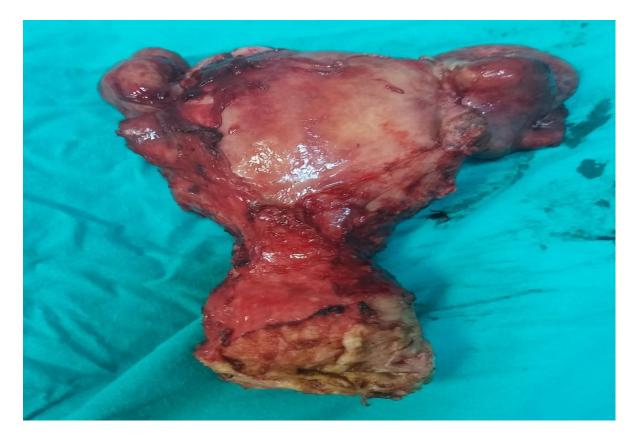


Figure 3. Hysterectomy Material

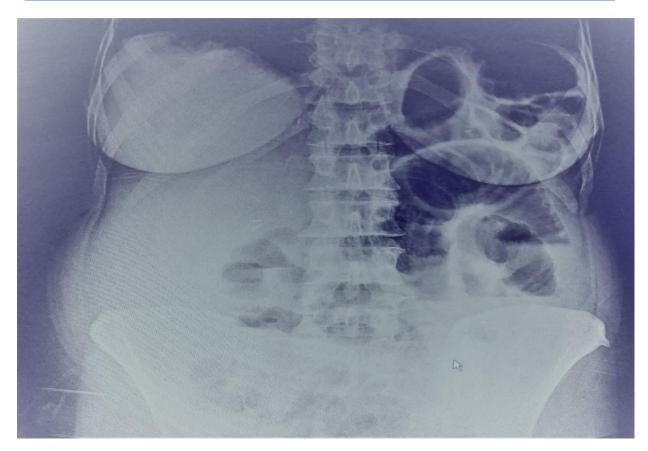


Figure 4. ADBG image of air-fluid levels

## Acknowledgements

## **Conflict of Interest**

On behalf of all authors, I accept and declare that as the responsible author, we do not have a partnership or membership relationship with any financial or non-financial institution or business regarding the products mentioned in this article.

## **Support Resources**

No financial support was used by authors during this study.

## Notification

This study was not presented as an oral presentation/poster in any congress/symposium or meeting.

## **Ethical Declaration**

Informed consent was obtained from the participant and Helsinki Declaration rules were followed to conduct this study.

## Thanks / Thanks

I would like to thank the nurses and midwives of Iğdır State Hospital and also the staff for their hard work

## REFERENCES

- Granberg S, Gjelland K, Ekerhovd E. The management of pelvic abscess. Best Pract Res Clin Obstet Gynaecol. 2009 Oct;23(5):667-78. https://doi.org/10.1016/j.bpobgyn.2009.01.010. Epub 2009 Feb 20. PMID: 19230781.
- [2] Pedowitz P, Bloomfield RD. Ruptured adnexal abscess (tuboovarian) with generalized peritonitis. Am J Obstet Gynecol. 1964 Mar 15;88:721-9. https://doi.org/10.1016/0002-9378(64)90604-0. PMID: 14130334.
- [3] Landers DV, Sweet RL. Tubo-ovarian abscess: contemporary approach to management. Rev Infect Dis. 1983 Sep-Oct;5(5):876-84. https://doi.org/10.1093/clinids/5.5.876. PMID: 6635426.
- [4] Lareau SM, Beigi RH. Pelvic inflammatory disease and tubo-ovarian abscess. Infect Dis Clin North Am. 2008 Dec;22(4):693-708. https://doi.org/10.1016/j.idc.2008.05.008. PMID: 18954759.
- [5] İngeç M, Kumtepe Y, Börekçi B, Kadanalı S. Tuboovarian Abse: 24 vakanın analizi. Jinekoloji ve Obstetrik Dergisi 2004; 18(5): 162-165. https://app.trdizin.gov.tr/makale/TkRRNE56VTE/tuboovarian-abse-24olgunun-analizi
- [6] Chao AS, Chang SY, Soong YK. Postmenopausal tuboovarian abscess. Changgeng Yi Xue Za Zhi. 1992 Sep;15(3):128-33. PMID: 1468035.
- [7] Lipscomb GH, Ling FW. Tubo-ovarian abscess in postmenopausal patients. South Med J. 1992 Jul;85(7):696-9. https://doi.org/10.1097/00007611-199207000-00006. PMID: 1631680.
- [8] Cohen CR, Sinei S, Reilly M, Bukusi E, Eschenbach D, Holmes KK et al Effect of human immunodeficiency virus type 1 infection upon acute salpingitis: a laparoscopic study. J Infect Dis. 1998 Nov;178(5):1352-8. https://doi.org/10.1086/314465. PMID: 9780255.
- [9] Richard H Beigi. www.uptodate.com, 2012; June, 2012.
- [10] Ilmer M, Bergauer F, Friese K, Mylonas I. Genital tuberculosis as the cause of tuboovarian abscess in an immunosuppressed patient. Infect Dis Obstet Gynecol. 2009;2009:745060. https://doi.org/10.1155/2009/745060. Epub 2010 Mar 8. PMID: 20224814; PMCID: PMC2834956.
- [11] Soper DE. Pelvic inflammatory disease. Obstet Gynecol. 2010 Aug;116(2 Pt 1):419-428. https://doi.org/10.1097/AOG.0b013e3181e92c54. PMID: 20664404.
- [12] Sweet RL. Soft tissue infection and pelvic abscess. In: Infectious diseases of the female genital tract, 5th ed, Sweet RL, Gibbs RS (Eds), Lippincott Williams and Wilkins, Philadelphia 2009.