

Acute kidney injury secondary to tubo-ovarian abscess: A case report

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Summary

Acute kidney injury secondary to tubo-ovarian abscess is rare. Here, we present a case of acute kidney injury arising from urethral obstruction secondary to a tubo-ovarian abscess. A 49 year old woman with complaints of persistent fever and abdominal pain was transferred to our hospital. A transvaginal ultrasound revealed two large pelvic masses that were suspected of being bilateral tubo-ovarian abscesses. During broad spectrum parenteral antibiotic treatment, the patient reported oliguria. With no response to fluid resuscitation and renal replacement treatment, we performed a laparoscopic bilateral salpingo-oophorectomy to relieve the urethral obstruction. Surgical removal of the bilateral tubo-ovarian abscess was effective in this patient.

Key words: Acute kidney injury; Oliguria; Pelvic inflammatory disease; Tubo-ovarian abscess; Urethral obstruction.

Introduction

Tubo-ovarian abscess (TOA) is a common complication of pelvic inflammatory disease which is an infection of upper genital tract [1]. It is a potentially life-threatening condition leading to sepsis or rupture of abscesses. Other possible complications include infertility and ectopic pregnancy [2]. Acute kidney injury (AKI) refers to an acute decline in renal excretory function over a period of hours or days leading to the accumulation of waste products, sometimes accompanied by oliguria, hyperkalaemia, hyperphosphataemia, and metabolic acidosis [3]. AKI is a rare, but serious, condition arising secondary to TOA, and few reports have been published concerning this condition. Here, we report a case of acute kidney injury arising from a urethral obstruction secondary to TOA.

Case Report

A 49-year-old woman presented to a local community hospital with abdominal pain, fever, nausea and three incidences of vomiting, all within a 24 hour time period. Her periods were regular and she denied anorexia, recent weight loss, vaginal discharge, dysuria, diarrhea, constipation, and bowel symptoms. She reported that a transvaginal ultrasound revealed bilateral small ovarian cysts three years ago, but these were untreated. Her medical history was unremarkable except having appendectomy for a ruptured appendix at the age of 29. She had no family history of ma-

lignancies. After intravenous antibiotherapy use for 5 days, abdominal pain had improved but not completely resolved. However, she continued to have temperatures of up to 39.5 °C.

The patient was subsequently transferred to our hospital. Vital signs on arrival were a temperature of 39.0 °C, blood pressure of 135/80 mmHg, heart rate of 123 beats per min, respiratory rate of 24 breaths per min, and an oxygen saturation of 99% while breathing ambient air. Her lungs were clear. On physical examination, her abdomen was rigid and she had involuntary guarding with rebound tenderness in the bilateral lower quadrant. A speculum and bimanual examination were refused. Bowel sounds were present. There was no generalized enlargement of lymph nodes. The external genitalia were normal.

The patient had an elevated WBC ($18.5 \times 10^9/L$, normal $3.5-10.5 \times 10^9/L$), an elevated serum level of C-reactive protein (> 200.0 mg/dL, normal 0.00–0.014 mg/dL), and a normal hemoglobin (115 g/L, normal 115–155 g/L). Her urine pregnancy test and urinalysis were both negative as was her blood culture. She had a normal blood urea nitrogen (4.67 mmol/L, normal 2.5–6.1 mmol/L) and creatinine level (105 μ mol/L, normal 58–110 μ mol/L). A transvaginal ultrasound revealed two large pelvic masses (left $9.9 \times 8.6 \times 9.0$ cm, right $10.5 \times 8.4 \times 6.9$ cm) that were suspected of being bilateral TOAs.

The patient was treated empirically with a 0.5 g of lev-

ofloxacin and 1.0 g tinidazole daily for three days for the suspected pelvic infective disease. Over this period, her abdominal pain greatly improved but she still had an intermittent temperature of up to 38.0 °C. Given the intermittent fever and the 10 cm pelvic masses, the patient was scheduled for a laparoscopic exploration.

On the third hospital day, the patient reported anuria (a urine output of 0.21 mL/kg/h; normal > 0.5 mL/kg/h for 6–12 hours). The patient's vital signs were stable except she was febrile. A full blood count showed an elevated serum creatine level of 454 μ mol/L. Renal ultrasound showed bilateral moderate hydronephrosis. With no response to fluid resuscitation, the Department of Nephrology was consulted and an urgent renal replacement was performed.

The following day, the patient's anuria did not resolve. Her urine output was 6 mL in 24 hours and her serum creatine level was greatly elevated (598 μ mol/L). Given both sepsis and renal failure, an exploratory laparoscopy was performed. Intraoperative findings included a 10 cm left TOA adhering to the left pelvic side wall and anterior abdominal wall, and a 9 cm right TOA adhering to the right pelvic side wall. Adhesions between the right TOA, bowel, omentum, and uterus were noted. After adhesions were lysed, approximately 500 mL of yellow pus was aspirated from TOAs. Considering the patient's age and condition, the medical team decided to perform a bilateral salpingo-oophorectomy. When the bilateral TOAs were removed, clear urine was drained by an indwelled urethral catheter.

Bilateral TOAs resulting in acute renal failure, obstructive uropathy, and reproductive tract infection were diagnosed. The abscess fluid was sent for microbiological culture which subsequently indicated an *E. coli* infection. The abdomen was irrigated with 1500 mL of sterile water. A drain was packed gently into the pelvis. A few hours following the surgery, the patient's anuria had improved and her other symptoms improved after administration of combined antibiotics for a week as the sepsis resolved. The drain was removed on the third post-operative day.

Discussion

Tubo-ovarian abscess is a common complication of pelvic inflammatory disease. Progression of the TOA can affect any abdominal organ leading to other possible near and long-term complications including sepsis, rupture of abscesses, infertility, and ectopic pregnancy. AKI is rare life-threatening consequence secondary to TOA. We present this report to alert gynecological surgeons to this rare potential complication of TOA.

Two reasons may cause an association between TOA and AKI. The first is that severe infection results in sepsis-induced AKI. Severe sepsis, the systemic inflammatory response syndrome caused by an infection in the presence of at least one organ failure, is a common and often fatal condition. Acute kidney injury is common in patients with

sepsis [4]. Although the patient was hemodynamically stable during her inpatient clinical course, it was reasonable to hypothesize that the acute serum creatinine elevation and oliguria were attributable to sepsis-induced AKI. However, AKI stemming from an inflammatory response resulting in a redistribution of renal blood flow failed to explain deteriorating renal function after renal replacement treatment. Based on the size of the abscesses and the proximity of the female urinary and genital tract, we hypothesized a second reason for AKI was that compression of the TOAs resulted in urethral obstruction and renal failure. As the patient's serum creatinine level returned to normal quickly after salpingo-oophorectomy, the urethral obstruction resulting from the TOAs is thought to be the primary cause of the deteriorating renal function. In retrospect, bilateral moderate hydronephrosis should have raised the possibility of diagnosis of postrenal AKI.

Available literature supports surgical management for hemodynamically unstable patients who have signs of a TOA rupture (acute abdomen, sepsis), or a TOA that does not respond to appropriate antibiotics [5-7]. Surgery is effective for TOA, and has a high healing rate despite being burdened by a high morbidity rate in critical ill patients. Given the severity of consequences resulting from TOA, early surgical intervention, although many view this as an aggressive approach, should be an ideal modality for prompt treatment for TOA. Alternately, minimally invasive techniques such as TOA drainage are a conservative treatment approach [8].

In conclusion, AKI is a rare complication resulting from TOA. Close monitoring of hemodynamic status in the perioperative period may discover early evidence of AKI. If fluid resuscitation and renal replacement therapy fail to stabilize the patient promptly, immediate surgical exploration is necessary. Prompt diagnosis and immediate treatment of this rare complication are important to prevent kidney injury early in the course of illness.

Conflict of Interest

The authors declare no competing interests.

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