

# Concurrent *Escherichia coli* tubo-ovarian abscess and *Campylobacter jejuni* gastroenteritis: A case report

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## ABSTRACT

Haematogenous or direct spread of bacterial infection causing pelvic inflammatory disease of the upper female reproductive tract is uncommon. We report a diagnostically challenging case of a 41-year-old woman with a background of Stage 4 endometriosis presenting with fever, diarrhoea and abdominal pain with recent history of pyelonephritis. Initially managed for undifferentiated abdominal pain with unclear focus of infection, a broad range of investigations were undertaken. Laboratory samples confirmed the presence of *Campylobacter jejuni* and appropriate treatment for Campylobacteriosis was commenced. Despite treatment, her condition deteriorated and repeat radiological imaging revealed bilateral tubo-ovarian abscess requiring surgical drainage for control of severe sepsis. Sterile surgical samples of the abscess revealed *Escherichia coli*. This case adds to the growing body of evidence of the association between pelvic inflammatory disease, severe endometriosis and development of tubo-ovarian abscess. Sepsis associated with tubo-ovarian abscess has a mortality rate of up to 10%. Hence, we present this case to highlight severe endometriosis as a risk factor for disease and the need for prompt reassessment of the deteriorating woman with sepsis and pelvic pain to direct efforts to minimise morbidity and mortality.

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## 1. Introduction

Endometriosis is a chronic benign inflammatory condition characterised by ectopic endometrial tissue [1]. The pathogenesis is not yet clear, although several causes have been proposed, such as retrograde menstruation [1,2]. It is however, understood to have an underlying genetic component (50%), influenced by various environmental causes such as immunologic, inflammatory and hormonal factors [2,3]. Estimated prevalence is 10%, predominantly affecting pre-menopausal women and commonly implicated in infertility and chronic pelvic pain [2,3]. Disease can be characterised by type of lesions present including: (1) superficial peritoneal endometriosis, (2) ovarian endometriomas or (3) deep infiltrating endometriosis [2].

Pelvic inflammatory disease (PID) is defined by infection of the upper female genital tract involving the uterus, fallopian tubes, ovaries or surrounding structures affected by direct bacterial spread of ascending infection of the lower genital tract [4]. Complications of PID can include chronic pelvic pain, tubal factor infertility, ectopic pregnancy, tubo-ovarian abscess (TOA), perihepatitis (Fitz-Hugh-Curtis syndrome) and peritonitis [4–6]. Actual incidence of PID in Australia is difficult to determine, as many patients have mild disease that is managed conservatively in the outpatient setting and is not a reportable condition [7].

Estimated incidence based on emergency department presentations is around 97 per 100,000 [8], with women of reproductive age more commonly affected than post-menopausal women [6]. Bacteria most commonly implicated are sexually transmitted infections *Chlamydia trachomatis* and *Neisseria gonorrhoea* [4,5], particularly for women aged under 30 years [9]; however, infection can be polymicrobial (30–40% of cases) [10] and even not identified in as many as 60% of cases [9]. Rarely, infection can be secondary to other intra-abdominal pathology such as pyelonephritis or diverticulitis by direct or haematogenous spread [10]. When associated with sepsis, the mortality rate is around 5–10% [10].

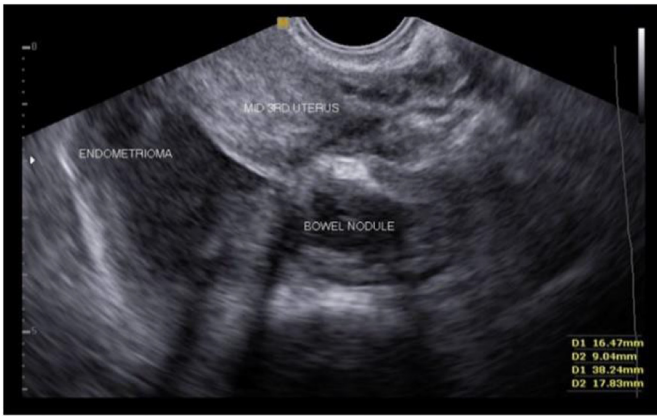
## 2. Case Report

A 41-year-old premenopausal woman re-presented to the metropolitan hospital emergency department with worsening abdominal pain, vomiting, non-bloody diarrhoea and fever 10 days after inpatient treatment for bilateral *Escherichia coli* pyelonephritis. She also reported 3 weeks of intermittent abdominal pain since returning from overseas travel to Indonesia.

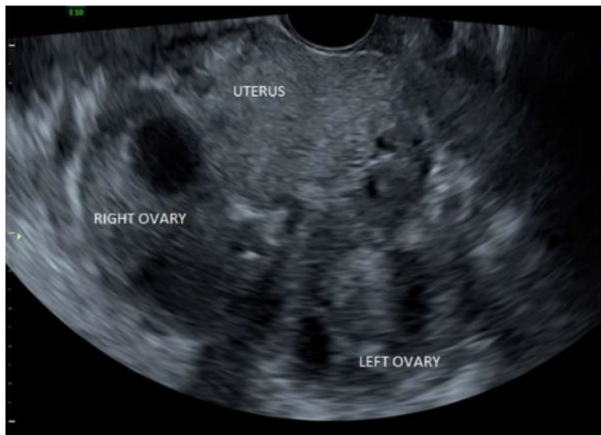
Her background history included Stage 4 (severe) endometriosis (bilateral endometriomas, rectal nodule, obliterated pouch of Douglas and fixed anterior uterus) diagnosed on ultrasound (Figs. 1 and 2), no previous laparoscopies, one previous caesarean section and well controlled asthma.

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**Fig. 1.** Mid sagittal transvaginal ultrasound showing obliterated Pouch of Douglas due to bowel nodule.



**Fig. 2.** Transverse view of pelvis with kissing ovaries.

She denied any recent sexual intercourse, no uterine instrumentation and no history of PID or sexually transmitted infections. At this time, she was not taking any regular medication, was not on any contraceptives and had regular menstrual cycles.

On examination she was tachycardic, hypotensive and febrile with right renal angle tenderness, generalised lower abdominal tenderness with rebound and guarding. There was right adnexal tenderness on bimanual exam though no palpable masses appreciated and no cervical motion tenderness. Speculum examination was unremarkable, normal cervix visualised macroscopically and no abnormal vaginal discharge seen.

Laboratory findings revealed an elevated lactate (3.1 mmol/L) showing the patient was in shock, with elevated inflammatory parameters (leucocytosis of  $16.9 \times 10^9/L$  with 93% neutrophilia, reference range 3.9–11.1  $\times 10^9/L$  and rise in C-reactive protein [CRP] 315 mg/L, reference range <3 mg/L) and acute kidney injury (elevated creatinine 100  $\mu\text{mol/L}$ , reference range 45–90  $\mu\text{mol/L}$  and reduced estimated glomerular filtration rate [eGFR] 60 mL/min/1.73m<sup>2</sup>, reference range  $\geq 90$  mL/min/1.73m<sup>2</sup>). Quantitative beta hCG was negative. Urinalysis, microscopy and culture was negative. Endocervical swabs were negative for *Chlamydia trachomatis* and *Neisseria gonorrhoea*. Blood cultures were also negative. Stool culture was positive for *Campylobacter jejuni*.

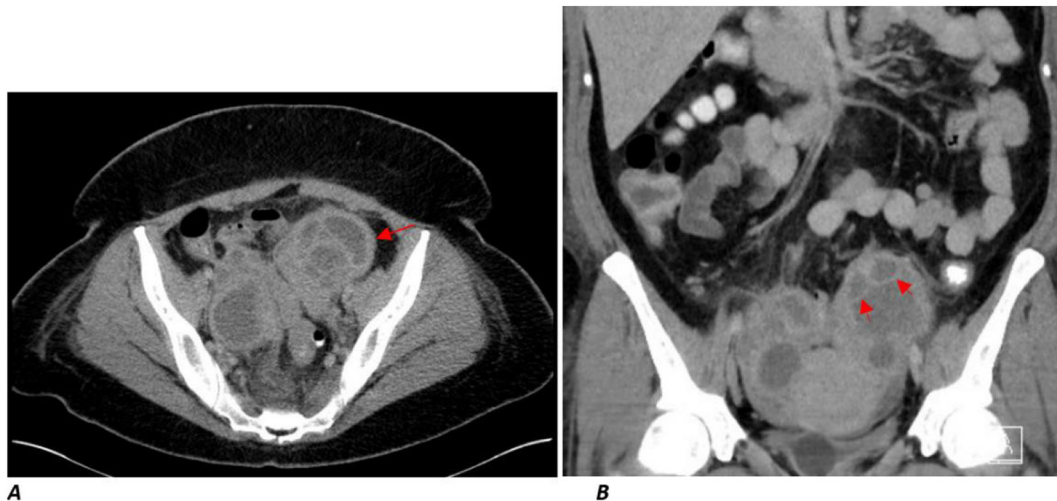
CT abdomen showed bilateral enlarged ovaries with septated cysts and a low-density mass in the presacral space with fat stranding. Transvaginal deep infiltrating endometriosis ultrasound demonstrated multiple endometriomas, a right ovarian cyst 54 mm  $\times$  31 mm  $\times$  32 mm and three left ovarian cysts, the largest being 39 mm  $\times$  33 mm  $\times$  38 mm. The ovaries were fixed in the pelvis and demonstrated the “kissing ovaries” sign (Fig. 2). The sliding sign was negative with the bowel adherent to the posterior wall of the uterus. Two bowel nodules were noted.

Differential diagnoses included colitis, urosepsis, appendicitis, and tubo-ovarian abscesses. Tubo-ovarian abscess was less likely given the stable appearance compared with previous imaging since diagnosis four years earlier.

Initially, she was managed empirically for sepsis as per the adult sepsis pathway [11], including fluid resuscitation and broad-spectrum intravenous antibiotics (ceftriaxone and metronidazole).

Her condition deteriorated over the next 24–36 h with ongoing fever, tachycardia and hypotension. Despite resuscitative treatment, she required transfer to the intensive care unit for inotropic support. She was changed to intravenous amoxicillin with clavulanic acid and additionally commenced on azithromycin for *Campylobacteriosis*.

On day 5 of her hospital admission, having ongoing pain and infective signs, she underwent repeat CT abdomen pelvis demonstrating interval increase in the left multiloculated adnexal mass from 5.8 cm  $\times$  5.7 cm  $\times$  7.2 cm to 7.6 cm  $\times$  7.6 cm  $\times$  9.3 cm as well as increased surrounding fat stranding and free fluid in the pelvis (Fig. 3).



**Fig. 3.** Tubo-ovarian abscess in 41-year-old women with abdominal pain and fevers. A: Contrast enhanced CT abdomen-pelvis axial view demonstrates bilateral adnexal thick-walled (arrowhead) multiloculated collections. B: Contrast enhanced CT abdomen-pelvis coronal view showing multiple enhancing septae within abscesses (arrowheads).

There was re-demonstration of bowel wall thickening of the sigmoid, descending and transverse colon, possibly inflammatory or infective in nature given the clinical history (Fig. 4). A decision was made for exploratory laparoscopy.

She underwent urgent laparoscopic washout and drainage of the tubo-ovarian abscesses for source control of infection. Sterile abscess culture fluid was positive for *Escherichia coli* and the right adnexal tissue histopathology showed evidence of abscess formation with no evidence of endometriosis. She was managed with intravenous antibiotics, intravenous fluids and her condition greatly improved. She was discharged from hospital several days later with plan for outpatient follow-up with repeat pelvic scan to assess for resolution.

### 3. Discussion

Females of reproductive age presenting with low pelvic pain can often pose a challenge to the physician, yielding broad differentials and warranting investigation of several body systems. PID itself can be difficult to diagnose given the wide range of presenting signs and symptoms which are often vague and can be mild [12]. In the first instance, women of reproductive age importantly should have investigations to rule pregnancy in or out.

The above case demonstrates the diagnostic challenge in having several factors on history, examination and investigations pointing to several possible diagnoses for the infective source of her sepsis. This was due to her recently having had treatment for pyelonephritis, convincing evidence for colitis secondary to *Campylobacter* gastroenteritis and recent overseas travel. Her history of endometriosis perhaps was the only indicator that TOA was implicated as part of the diagnosis, however, as she had no other risk factors present.

TOA is a relatively uncommon complication of PID affecting 15–35% of women diagnosed with PID [10]. Women with endometriomas are more likely to have co-existent PID with TOA [10]. *Escherichia coli* has been found to be the primary culprit bacteria in cases of ruptured TOA; however, this did not occur in our case [12]. A retrospective study of 3215 women by Chen et al. found, with statistical difference, that women with advanced stage endometriosis (Stage III and IV) were more likely to develop TOA than those without endometriosis [13]. Also, there was an association with TOA for women with any type of endometriosis if there was also a history of pelvic surgery [13]. Similarly, Elizur et al. found that women with severe endometriosis who were hospitalised with PID or TOA were much more likely to fail antibiotic therapy and require surgical intervention than women without endometriosis (48% vs 6%) [14]. Rarely infection can be caused by bacterial spread to the female upper genital tract by either direct contact

with inflamed bowel or haematogenous route particularly where there is bacterial diarrhoeal illness [15,16]. As demonstrated in this case, we consider haematological or direct spread to be implicated in this patient's development of severe sepsis subsequent to TOA. Given the known diagnosis of *Campylobacteriosis* and previous *Escherichia coli* pyelonephritis, this case represents the uncommon instance where TOA develops from other intraabdominal infection via direct or haematogenous spread aided by the immune-inflammatory milieu created by the patient's severe Stage 4 endometriosis [10,13].

The Clinical Excellence Committee (CEC) route cause analysis committee demonstrates the most frequent cause for mortality in sepsis is inadequate treatment [11]. This case also acts as an important reminder for early recognition of the deteriorating septic patient. Deterioration may be due to inadequate treatment and should prompt clinicians to revisit the history, examination and investigations to ensure treatment is appropriate. In the above case the patient was identified as not improving despite resuscitation and intensive care support, prompting repeat pelvic imaging, which demonstrated interval increase of pelvic mass size, leading to subsequent surgical management for the source control of infection.

### 4. Conclusion

Given the severe sequelae of untreated or inadequately treated PID there should be prompt initiation of appropriate antibiotics when the diagnosis is suspected [10]. Women with severe endometriosis should be considered at increased risk of severe pelvic infection and TOA. Those failing to improve with conservative management may warrant surgical management, though additional risk factors, appropriate timing and the best approach in surgical intervention remain unclear [10].

Multidisciplinary management is important as demonstrated in this case with input from surgical, gynaecological, infectious diseases and intensive care teams, with senior clinician involvement from the outset, to ensure appropriate and timely treatment to optimise patient outcomes.

### Contributors

Alison Laura King acquired data, performed the literature review and drafted the case report.

Nicole Stamatopoulos revised the draft manuscript.

Both authors were responsible for the conceptualisation of the case report, and both saw and approved the final version of the paper and take full responsibility for the work.

### Conflict of interest

Both authors declare that they have no conflict of interest regarding the publication of this case report.

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### Patient consent

Obtained.

### Provenance and peer review

This case report was peer reviewed.

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**Fig. 4.** Magnetic resonance imaging pelvis T1 weighted fat saturated axial image demonstrating mixed cystic and solid appearance of adnexal lesions with post-contrast enhancement.

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