Pelvic inflammatory disease with obstructive complications: two cases and a literature review

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Dear Sir,

Pelvic inflammatory disease (PID) is an ascending infection of the lower genital tract that results in endometritis, salpingitis and/or pelvic peritonitis. The infection leads to suppurative damage to the epithelium of the fallopian tube, resulting in a favourable anaerobic environment for the formation of tubo-ovarian abscesses (TOAs).\(^{(1,2)}\) TOAs may involve adjacent pelvic organs such as the bowel, omentum and bladder.\(^{(3)}\) As infection progresses, tissue planes are lost with the destruction of normal pelvic anatomy,\(^{(2)}\) with subsequent adhesion formation and scarring.\(^{(1)}\) TOAs complicate the clinical course of up to 10%–15% of women hospitalised with PID,\(^{(4)}\) which can result in severe sepsis and is potentially life-threatening.

PID and TOA resulting in obstructive complications such as intestinal obstruction (IO) or hydronephrosis have rarely been described in the literature. During our review of articles published in PubMed (MEDLINE) (restricted to English language, full-text-only manuscripts before April 2020), we identified only six cases of complications of bowel obstruction and eight cases of hydronephrosis, secondary to PID. Herein, we describe two cases of PID with TOA, one resulting in adhesions leading to small bowel obstruction (SBO) and one with complications of hydronephrosis due to extrinsic compression by the mass. This highlights the importance of considering infective causes arising from gynaecological organs as a possible differential in women presenting with obstructive complications.

Case 1 was a healthy 29-year-old woman with a body mass index (BMI) of 16 kg/m\(^2\) who had no previous history of PID or sexually transmitted infection (STIs), and her husband was her sole sexual partner. She presented with a one-week history of lower abdominal pain and fever. On examination, her vital parameters were stable, apart from a low-grade fever of 37.9°C, and physical examination was significant for bilateral adnexal tenderness. Laboratory investigations did not indicate any systemic infection. Pelvic ultrasonography revealed bilateral
TOA (5.0 cm on the right and 5.3 cm on the left) and computed tomography imaging of the abdomen and pelvis (CTAP) was consistent with PID with bilateral TOA. An endocervical swab confirmed *Chlamydia trachomatis* infection, and screening for other STDs showed negative results. The patient was administered intravenous antibiotics; she showed a good clinical response to intravenous ceftriaxone, metronidazole and oral doxycycline, and was discharged after 48 hours with two weeks of oral antibiotics.

She subsequently re-attended the Emergency Department five days later with new symptoms of obstipation, vomiting and abdominal pain. She was afebrile with stable parameters, but examination revealed a distended abdomen, with generalised tenderness and guarding. There was significant cervical excitation and adnexal tenderness. Her inflammatory markers remained unremarkable, and there was no hyperlactaemia or evidence of metabolic acidosis. A repeat CTAP showed diffuse inflammatory changes in the pelvis, with reduction in size of the TOA, and interval high-grade SBO with a transition point.

The patient was admitted to general surgery for SBO and restarted on intravenous antibiotics. She was kept on bowel rest and a nasogastric tube was inserted for gastric decompression. However, as her pain did not improve with conservative management, she was counselled for diagnostic laparoscopy, which was carried out within 24 hours of admission. Intraoperatively, dilated bowel loops, evidence of Fitz-Hugh-Curtis perihepatitis and extensive filmy adhesions in the pelvis were observed (Fig. 1a). Two transition points were found, the first 20 cm proximal to the terminal ileum, adherent to a pus-filled right fallopian tube (Fig. 1b), and the second 30 cm distal to the duodenal-jejunal flexure, caused by another adhesion band. The laparoscopic approach was abandoned in favour of open adhesiolysis to allow a more thorough evaluation of the bowel. The right fallopian tube was milked to drain the pus, with good success.
Postoperatively, the patient recovered well in the general ward. Intravenous antibiotics were continued, and she was discharged on postoperative day 6 with a two-week course of oral doxycycline. She subsequently defaulted all follow-up appointments and was uncontactable despite all efforts.

Case 2 was a healthy 28-year-old foreign domestic helper with a BMI of 24 kg/m², who had an intrauterine contraceptive device (IUCD) inserted more than a year ago. She had no previous history of PID or STIs and her husband residing overseas was her sole sexual partner. She presented with worsening lower abdominal pain for three weeks, associated with fever and inability to pass urine for 12 hours. Her temperature was 38.1°C and she was tachycardic, with a pulse rate of 111. On examination, she appeared toxic-looking, with lower abdominal tenderness but no peritonism. On speculum examination, the IUCD stem was observed at the cervical os and removed. The patient had significant cervical excitation, with bilateral adnexal tenderness. An indwelling catheter (IDC) was inserted, which drained 400 mL of urine. Her inflammatory markers were raised, but renal function was normal. She was diagnosed with severe sepsis secondary to PID and admitted to a high dependency unit. Intravenous ceftriaxone, metronidazole and oral doxycycline were commenced. Her endocervical swab was positive for *Chlamydia trachomatis*, but blood, urine and IUCD cultures were negative for any pathogens. Her STD screen was unremarkable. She responded to antibiotics, with resolution of her fever and tachycardia within 24 hours. A CTAP revealed a 10.2-cm TOA in the pouch of Douglas (Fig. 2a), with anterior displacement of the urinary bladder and moderate right hydroureteronephrosis with a transition point at the right distal ureter secondary to compressive effect from the TOA (Fig. 2b). Pelvic ultrasonography confirmed bilateral TOA with an interconnecting 10.8-cm mass in the pouch of Douglas.

After receiving intravenous antibiotics for 72 hours, the patient managed to void successfully upon removal of the IDC. She also underwent CT-guided drainage of her pelvic
collection, which drained 250 mL of pus (Fig. 2c). Cultures from the pelvic collection confirmed *Chlamydia trachomatis*. The drain was removed after three days when the output was minimal, and the patient was subsequently discharged with oral azithromycin owing to intolerance to doxycycline. At the follow-up outpatient review two weeks later, she was asymptomatic and her inflammatory markers were normal. She was lost to follow-up thereafter; however, ideally, interval renal imaging should be repeated to ensure resolution of hydronephrosis. Resolution of the hydronephrosis would help to establish causality between her PID resulting in obstructive nephropathy as well as exclude other causes of hydronephrosis.

Intestinal obstruction and hydronephrosis are commonly encountered clinical conditions, of which PID/TOA is an uncommon aetiology. Herein, we report rare obstructive complications of SBO and hydronephrosis in two patients with TOA. We hypothesise that in Case 2, it was not a true episode of acute urinary retention; rather, the inability of the patient to pass urine was due to a combination of pain, compression from the TOA and low urine output secondary to sepsis. *Chlamydia* was the organism implicated in both cases, and both patients underwent invasive interventions to relieve the source of obstruction. These cases demonstrate that when infection from PID progresses to TOA, adjacent organs can get involved.\(^5\) With reactive inflammation, oedema and scarring from infection, this can result in functional or mechanical obstructive sequelae of the gastrointestinal and renal tract.\(^5\) These patients will require multidisciplinary team care by gynaecologists, general surgeons, urologists and/or interventional radiologists.

Table I summarises the 14 patients with obstructive complications of PID described in the literature. There are several possible theories regarding the mechanism of obstruction in PID. Obstruction can be either mechanical or functional. Firstly, TOA can be large enough to cause mass effect and extrinsic compression on the bowel\(^6\) or ureter.\(^7\) This was demonstrated in Case 2, supported by radiological findings of a large TOA compressing the right ureter, with
a transition point at the level of the TOA. In Case 1, the bowel obstruction was due to adhesion bands induced by the PID/TOA, creating a transition point that was visible on imaging. There have also been case reports of patients presenting with SBO without signs of acute pelvic infection. In patients with a history of past PID, fibrotic adhesions from previous episodes of PID have also been shown to cause mechanical obstruction.\(^8\) Abul-Khoudoud et al have described a rare case of SBO from perihepatic adhesions from Fitz-Hugh-Curtis syndrome due to internal herniation of the bowel above the liver.\(^9\) Obstruction can also be functional. Inflammatory changes result in failure of peristalsis of the bowel, as observed in conditions such as severe peritonitis or gastroenteritis.\(^10\) Reactive inflammation of the bowel or ureter due to the PID/TOA itself can result in small or large bowel ileus,\(^10\) or ureteric obstruction due to inflammatory changes involving the ureter.\(^11\)

It is important to understand the mechanisms of obstruction induced by PID in order to guide management decisions. In the literature, most patients with PID complicated by SBO underwent surgical management, especially those with a virgin abdomen. Delays in timely operative intervention significantly increase the risk of morbidity and mortality.\(^12\) Clinical red flags that warrant urgent surgery include peritonitis, haemodynamic instability or biochemical markers suggestive of bowel ischaemia or radiological signs of closed loop obstruction.\(^13\) However, in a select population of patients who are clinically stable with no signs of bowel ischemia, there may be an option for a trial of conservative management.\(^14\) Harel et al\(^10\) described a case of a 19-year-old African-American woman with a history of recurrent PID admitted for SBO. CTAP demonstrated partial high-grade small bowel obstruction at the duodenum but no transition point or TOA. She was successfully treated with intravenous antibiotics and remained symptom-free during the six-month follow-up period.

In patients with complications of hydronephrosis secondary to PID, the first-line management would be intravenous antibiotics to treat the underlying infection.\(^15\) Renal
function should be closely monitored to assess response to treatment. Patients with complications of acute kidney injury (AKI) may benefit from a percutaneous nephrostomy (PCN) insertion to relieve the obstruction and preserve renal function.\(^{(7)}\) Surgical intervention to drain the TOA should be considered in case of clinical non-response to antibiotic therapy after 24–48 hours or signs of worsening AKI. Čizmarević et al described a case of a 40-year-old woman admitted for bilateral pyosalpinx complicated by bilateral hydronephrosis that was worse on the right side. She underwent a right PCN insertion to resolve the obstruction of the upper urinary tract. However, repeat imaging after the procedure revealed SBO, and therefore, she underwent a laparoscopic right adnexectomy, left pyosalpinx drainage and adhesiolysis.\(^{(7)}\) Galal et al emphasised the need for prompt intervention to avoid permanent dilatation of the renal tract and renal impairment.\(^{(16)}\) Interval imaging should be carried out to ensure resolution of the hydronephrosis and exclude other aetiologies of ureteric obstruction.

Obstructive symptoms have been the presenting complaint for majority of the case reports in the literature. Out of the 14 cases, 12 had presented with symptoms of obstruction (such as symptoms of IO or voiding difficulty and loin pain) at admission and were diagnosed in the same setting. In the remaining two cases,\(^{(8,17)}\) the time course for the development of obstructive complications was subacute, in part owing to failure to recognise the symptoms of obstruction. The case report by Harel et al\(^{(10)}\) described recurrent presentations for PID with a delayed diagnosis of SBO only on the third admission of the patient. This was possibly because IO was not considered as a differential until much later during her disease course, when her vomiting became persistent and refractory to anti-emetics. Christodoulidou et al\(^{(17)}\) described a case of a 39-year-old woman admitted three times over a period of four weeks with abdominal pain, loin pain and fever. She was initially treated for appendicitis and then for pyelonephritis, and only on her last admission was she diagnosed with PID complicated by right hydronephrosis. The failure to recognise and treat PID at the initial episode resulted in her PID
progressing into a large TOA, resulting in obstructive nephropathy. This case highlights the importance of suspecting the diagnosis of PID in women of a reproductive age who present with abdominal pain, and early treatment with appropriate antibiotics could prevent re-admissions and development of obstructive sequelae.

In conclusion, obstructive complications of PID/TOA are uncommon but must be considered as a possibility, especially in sexually active women. It is essential to have a high index of suspicion for PID in such patients to facilitate early diagnosis and initiate appropriate treatment, thereby avoiding repeated admissions due to missed diagnosis. In patients with non-resolving intestinal obstruction or hydronephrosis, early surgical intervention and drainage of TOA should be considered for source control and to resolve the obstruction. Timely intervention will ensure good clinical outcomes in these cases.

Yours sincerely,

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REFERENCES


FIGURES

**Fig. 1** Intraoperative photographs show (a) multiple filmy adhesions from the sigmoid colon to the uterus, adnexa and pelvic side wall; and (b) a transition point 20 cm from the terminal ileum adherent to the right fallopian tube and tubo-ovarian abscess.

**Fig. 2** (a) Coronal CT image of the abdomen and pelvis shows a 10-cm complex multiloculated cystic mass suggestive of tubo-ovarian abscess (TOA). (b) Axial CT images show right hydroureteronephrosis, and delayed enhancement and excretion of contrast by the right kidney and (c) CT-guided drainage of the TOA, with a pigtail drain left *in situ* for passive drainage.
Table I. Summary of case reports of obstructive complications due to pelvic inflammatory disease.

<table>
<thead>
<tr>
<th>Author</th>
<th>Age (yr)</th>
<th>Presentation</th>
<th>Causative organism</th>
<th>Cause of obstruction</th>
<th>Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cases of intestinal obstructions secondary to PID</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Abul-Khoudoud et al(9)</td>
<td>51</td>
<td>Abdominal pain and vomiting</td>
<td><em>Chlamydia trachomatis</em></td>
<td>SBO secondary to adhesions and Fitz-Hugh-Curtis</td>
<td>Diagnostic laparoscopy and adhesiolysis</td>
<td>Discharged on POD 1</td>
</tr>
<tr>
<td>Al-Ghassab et al(8)</td>
<td>32</td>
<td>Abdominal pain, vomiting and constipation</td>
<td>None isolated</td>
<td>SBO secondary to adhesions, Fitz-Hugh-Curtis and left hydrosalpinx</td>
<td>Diagnostic laparoscopy and adhesiolysis</td>
<td>Discharged on POD 5, remained symptom-free after 2 month</td>
</tr>
<tr>
<td>Haumann et al(13)</td>
<td>27</td>
<td>Abdominal pain and vomiting</td>
<td><em>Chlamydia trachomatis</em></td>
<td>SBO secondary to inflammatory changes in the pelvis</td>
<td>Exploratory laparotomy and adhesiolysis. Received antibiotics for 3 weeks</td>
<td>Discharged on POD 3, Remained symptom-free after 1 month</td>
</tr>
<tr>
<td>Harel at al(10)</td>
<td>18</td>
<td>Abdominal pain and vomiting</td>
<td><em>Chlamydia trachomatis</em></td>
<td>SBO with transition point at level of terminal ileum</td>
<td>Conservatively treated with gastric decompression and bowel rest. Received 11 days of IV antibiotics followed by 10 days of oral antibiotics</td>
<td>Discharged on Day 11 of hospitalisation, remained symptom-free after 6 months</td>
</tr>
<tr>
<td>Weledji et al(14)</td>
<td>22</td>
<td>Abdominal pain, vomiting, constipation 1 week after dilatation and curettage for surgical abortion</td>
<td>None isolated</td>
<td>SBO secondary to adhesions and strictureing due to a right 6-cm TOA</td>
<td>Exploratory laparotomy, SO, bowel resection with anastomosis. Received 1 week of IV antibiotics, then 1 week of oral antibiotics</td>
<td>Discharged on POD 9</td>
</tr>
<tr>
<td>Pines et al(6)</td>
<td>35</td>
<td>Abdominal pain, vomiting, obstipation</td>
<td>None isolated</td>
<td>SBO secondary to pelvic abscess, right pyosalpinx entrapping small bowel</td>
<td>Diagnostic laparoscopy converted to open adhesiolysis, left salpingectomy and right salpingotomy</td>
<td>Discharged on POD 8 and remained symptom-free after 2 months</td>
</tr>
<tr>
<td><strong>Cases of hydronephrosis secondary to PID</strong></td>
<td></td>
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<tr>
<td>Yonemura et al(11)</td>
<td>62</td>
<td>Left flank pain and fever, IUCD in situ</td>
<td>None isolated</td>
<td>Left hydronephrosis with stricture in the lower third of the left ureter</td>
<td>Left PCN inserted, subsequently THBSO and left nephroureterectomy</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Case No</td>
<td>Symptoms</td>
<td>Organisms Found</td>
<td>Complications</td>
<td>Treatment</td>
<td>Outcome</td>
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<tr>
<td>Čizmarević et al(7)</td>
<td>40</td>
<td>Left flank pain</td>
<td>None isolated</td>
<td>Right hydronephrosis with bilateral pyosalpinx</td>
<td>Laparoscopic right adnexectomy, left-pyosalpinx drainage, adhesiolysis.</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Nasu et al(18)</td>
<td>63</td>
<td>Abdominal pain and fever, IUCD in situ</td>
<td>None isolated</td>
<td>Right hydronephrosis secondary to adhesions</td>
<td>Benzyl penicillin for 4 weeks, THBSO and adhesiolysis</td>
<td>Remained symptom-free after 1 year</td>
</tr>
<tr>
<td>Christodoulidou et al(47)</td>
<td>39</td>
<td>Abdominal pain and fever, IUCD in situ</td>
<td>Bacteriodes</td>
<td>Right hydronephrosis secondary to right TOA</td>
<td>THBSO, right retrograde ureteric stent insertion</td>
<td>Remained symptom-free after 1 month</td>
</tr>
<tr>
<td>Lee et al(19)</td>
<td>42</td>
<td>Pelvic discomfort and constipation</td>
<td>Actinomycosis</td>
<td>Left hydronephrosis and colonic stricture due to pelvic mass with adhesions</td>
<td>En-bloc excision of pelvic mass with THBSO, low anterior resection and</td>
<td>Remained symptom free after 1 year</td>
</tr>
<tr>
<td>Fite et al(15)</td>
<td>27</td>
<td>Abdominal pain and fever</td>
<td>None isolated</td>
<td>Bilateral hydronephrosis secondary to bilateral TOA</td>
<td>IV Meropenem</td>
<td>Not documented</td>
</tr>
<tr>
<td>Cox et al(20)</td>
<td>29</td>
<td>Abdominal pain and dysuria</td>
<td>None isolated</td>
<td>Right hydronephrosis secondary to right TOA</td>
<td>Laparotomy, right adnexectomy, received IV antibiotics</td>
<td>Remained symptom-free after 3 months</td>
</tr>
<tr>
<td>Galel et al(16)</td>
<td>30</td>
<td>Abdominal pain, dysuria, urinary frequency</td>
<td>None isolated</td>
<td>Bilateral hydronephrosis secondary to mass effect from TOA and adhesions</td>
<td>Laparotomy, left salpingo-oophorectomy, right salpingectomy, uterolysis</td>
<td>Defaulted follow-up</td>
</tr>
</tbody>
</table>

**IUCD**: intrauterine contraceptive device; **IV**: intravenous; **PCN**: percutaneous nephrostomy; **POD**: postoperative day; **SBO**: small bowel obstruction; **SO**: salpingo-oophorectomy; **THBSO**: total hysterectomy bilateral salpingo-oophorectomy; **TOA**: tubo-ovarian abscess