Small Bowel Obstruction Caused by Tubo-Ovarian Abscess Following Chronic Pelvic Inflammatory Disease: A Case Report

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Case report

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Abstract

Background

Although there are reports of small bowel obstruction (SBO) secondary to tubo-ovarian abscess (TOA), there have been no documented cases of unexpected SBO, multiple intestinal ruptures and adhesions in a patient with chronic PID followed by successful surgical treatment of TOA who was successfully treated by surgery after failure by conservative treatment.

Case presentation

A 40-year-old female was admitted with main complaint of abdominal pain and fever for six days. A pelvic mass measuring 6.37x7.85x9.04 cm and ascites at rectovaginal pouch were found despite local treatment with metronidazole and cefazolin. Laboratory tests revealed leukocytosis of 8.9x10^9/L with hyper-neutrocytophilia of 82.8%, C-reactive protein increase at 223 mg/L and Procalcitonin 0.14ng/L. The patient was diagnosed with an acute attack of chronic PID. Tests and body temperature improved after 4 days of IV antibiotics. However, two days later, the patient presented abdominal distension, poor appetite, and difficulty in defecation. Abdominal CT suggested possibility of bowel obstruction. Accordingly, an explorative laparoscopy was performed, revealing 500ml pale yellow ascites within the abdominal cavity. The intestinal tube was clearly dilated with poor peristalsis. Multiple intestinal ruptures and adhesions were found. Dense adhesion existed between the intestinal loop and posterior uterus wall, closing the rectouterine pouch. Pale yellow thick pus could be seen from the end of fallopian tube, and part of the right ovary showed serious pyosis. All the adhesions were split, ruptures were repaired and normal anatomy was restored. Postoperative pathology indicated acute and chronic inflammation of both fallopian tubes with focal abscess formation. The patient was discharged 15 days after operation and followed up at one month without any symptoms.

Conclusion

In such cases, close attention should be paid to changes in the patient's condition and lesion changes. Early laparoscopy is advised when there are significant clinical or CT scan signs of bowel obstruction in TOA patients. Precise predictors or a predictive model for the need of invasive intervention to TOA will require further investigation.

Background

Small bowel obstruction (SBO), a common clinical symptom, is defined as a blockage of gas and stool associated with incoercible vomiting. SBO continues to be a substantial cause of morbidity and mortality, accounting for 12–16% of hospital admissions for the evaluation of acute abdominal pain in the United States [1]. Studies have shown that the most common causes of SBO are intestinal adhesions, tumors, hernias, and inflammatory bowel disease, 10% of which may be further due to infection or inflammation (e.g. appendicitis, diverticulitis, endometriosis, pelvic inflammatory disease (PID), intestinal disease,

abdominal tuberculosis), or chemical peritonitis and irritation caused by foreign bodies [2, 3]. Thirty-three percent of SBO occur in the first year following the triggering factor [2]. Tubo-ovarian abscess (TOA), a complication of PID, can be severe and life-threatening. There is little in the literature about SBO secondary to acute TOA [3, 4]. Herein, we report an educational case of unexpected SBO in a patient with chronic PID followed by TOA in whom multiple intestinal ruptures and adhesions were found that were successfully treated by surgery after conservative treatment failed.

Case Presentation

A 40-year-old female was admitted with main complaint of abdominal pain and fever for six days. A pelvic mass was found five days prior to admission despite local treatment with metronidazole and cefazolin. The G2P1A1 patient had regular menstruation, IUD for contraception, and cesarean Sect. 20 years prior. She suffered intermittent lower abdominal pain for six months without undergoing regular treatment.

On abdominal physical examination, vital signs were normal and there was persistent abdominal distension and tenderness of the lower quadrant, radiating to her waist and iliac fossa. Discomfort followed palpation around the umbilicus. Both Murphy's sign and McBurney's sign were negative. Gynecological examination revealed vaginal swelling, redness, and thick curdy discharge. There was no tenderness on cervix and uterine body. A right adnexal mass could be palpated behind uterus with unclear boundaries, tenderness, adhesion to the uterus and poor movability.

Laboratory tests revealed leukocytosis of 8.9x10^9/L with hyper-neutrocytophilia of 82.8%, C-reactive protein increase at 223 mg/L (normal values: 0.0–8.0 mg/l) and Procalcitonin (PCT) 0.14ng/L. Serum biochemistry and human chorionic gonadotropin were normal. Leucorrhea DNA was positive for candida and negative for trichomonas and Gardnerella. Gynecological ultrasonography suggested an inflammatory right pelvic mixed mass measuring 6.37x7.85x9.04 cm and ascites at rectovaginal pouch measuring 4.87x4.25x1.77cm. The patient was diagnosed with an acute attack of chronic PID.

The patient received a vaginal clotrimazole tablet and IV administration of cefamandole and morinidazole for four days. Laboratory review revealed the following WBC count: 6.7x10^9/L, neutrophil percentage: 75.8%; Procalcitonin: 0.09ng/ml; C-reactive protein: 107.0mg/L. As tests and body temperature of patient improved, she continued to receive conservative treatment. Six days after initial treatment, the patient presented abdominal distension, poor appetite, and difficulty in defecation. Physical examination showed abdominal distension, lower abdominal tenderness, but normal borborygmus. Blood routine reexamination revealed WBC count: 9.6x10^9/L, neutrophil percentage: 83.0%; Procalcitonin: 0.06ng/ml; C-reactive protein: 35.8mg/L.

Reexamination of gynecological ultrasonography indicated a liquid tubular mass with thick wall measuring 6.30x3.84x1.69cm posterior to uterus. Abdominal CT suggested possibility of bowel obstruction (Fig. 1a). In light of the symptoms and imaging findings, an explorative laparoscopy was performed, revealing 500ml pale yellow ascites within the abdominal cavity. The intestinal tube was

clearly dilated with poor peristalsis. Partial intestinal wall was congested with dark color (Fig. 1b). The uterus retained a normal size and smooth anterior wall, but showed dense adhesion between intestinal loop and posterior wall, closing the rectouterine pouch and largely preventing exposure of bilateral adnexa (Fig. 1c). During dissection, part of the intestinal tube was found to be attached to the posterior wall of the uterus and bilateral adnexa in the form of "W" loop adhesion, resulting in intestinal segment stenosis at the adhesion site and above intestinal obstruction. Pale yellow thick pus could be seen from the end of the tube, while fallopian tubes and part of the right ovary showed serious pyosis. Left ovary retained a normal appearance. Multiple intestinal ruptures were found after separation (Fig. 1d).

Subsequent exploration of the bowel was performed with a total revision from Treitz to rectum under laparotomy. The intestinal tube was observed to be black and weak at 1.3m from Treitz, with "W" shape adhesion at 1.7m, a rupture of 1cm in diameter at 2m and 2.1m adhesions, pelvic adhesions at 2.8m, and a hole of 2mm visible at 3.2m. No rupture was found in the appendix, sigmoid colon or rectum. All the adhesions were split, ruptures were repaired and normal anatomy was restored. Flushing out the contents of the small intestine into the colon reduced pressure in the small intestine. Abdominal cavity was fully flushed, and two "double cannula" drains were placed from the left and right lower abdomen, respectively. A mushroom-head drainage tube was placed in the anal canal.

Postoperative pathology indicated acute and chronic inflammation of both fallopian tubes with focal abscess formation. The patient rapidly regained bowel movement and was given food and antibiotics (cefoperazone sodium sulbactam sodium 3g Q8h and morinidazole 0.5 g BID) for 14 days. Bacteriology analysis of ascites found E. coli. The patient was satisfied and discharged 15 days after operation and followed up at one month without any symptoms.

Discussion And Conclusions

PID comprises a spectrum of inflammatory disorders of the upper female genital tract, including any combination of endometritis, salpingitis, TOA, and pelvic peritonitis [5]. PID is difficult to diagnose because of the wide variation in symptoms and signs associated with this condition. Many women with PID have subtle or nonspecific symptoms or are asymptomatic. Pelvic inflammation involves the intestinal canal and appendix, leading to local edema, followed by fibrinous purulent exudate, and in turn, adhesion with surrounding organs. From a pathophysiological standpoint, it is well accepted that adhesions occur within seven days following the peritoneal injury, becoming progressively organized with fibrotic tissues. On average, patients must be readmitted twice over the subsequent 10 years due to such adhesions [2, 6]. Acute PID primarily cause paralytic ileus, while recurrent chronic pelvic viscera adhesion mainly result in mechanical ileus [4, 7].

Ultrasound scanning may be useful to confirm a pelvic abscess while computed tomography (CT) or magnetic resonance imaging (MRI) can help rule out other causes of peritonitis. The potential utility of MRI scanning of the pelvis in excluding differential diagnoses has been well established [8].

The choice and timing of surgery in PID is still controversial. Conservative management of PID with antibiotics alone remains first-line, with success rates of about 70% [5, 8, 9]. A five-year retrospective study aimed at investigating whether early surgical management improves outcomes in patients presenting with a TOA suggested that early surgery may be beneficial in the management of TOAs. Nineteen (38.0%) patients were treated with antibiotics (medical group) and thirty-one (62.0%) were treated surgically on admission (early surgical group). The early surgical group was associated with a high success rate of 96.8% and the lowest risk of readmission within 12 months (16.1%) [10]. A systematic review indicates that better outcomes in the management of TOA were achieved via a minimally invasive approach compared to conservative treatment with antibiotics only [11]. But how to predict the necessity for invasive intervention in TOA? Ribak et al. [12] evaluated 48 of 94 patients (51.1%) hospitalized with complicated PID who were sonographically diagnosed with TOA. They found that CRP was a sensitive, specific inflammatory marker for predicting TOA in patients with complicated PID, while levels > 49.3 mg/L suggested the presence of TOA. They concluded that increasing CRP levels may be used as a predictor for invasive intervention. Levin et al. [13] found that CA-125 level was the only independent factor associated with failure of conservative parenteral antibiotic therapy for TOA (OR; 95% confidence interval [CI], 1.27,1.08–1.48, p = 0.03). Moreover, abscesses larger than 10 clinepagem have greater than 60% chance of surgery in addition to antibiotics [11]. The size of TOA may be another predictor for the need of invasive intervention.

In summary, PID is a rare cause of SBO. Close attention should be paid to changes in the patient's condition and lesion changes. Early laparoscopy is advised when there are significant clinical or CT scan signs of bowel obstruction in PID patients. Precise predictors or a predictive model for the need of invasive intervention to TOA will require further investigation.

Abbreviations

SBO: Small bowel obstruction

PID: pelvic inflammatory disease

TOA: tubo-ovarian abscess

PCT: procalcitonin

Declarations

Ethics approval and consent to participate

Ethics approval was not required and the patient gave her written consent form to participate. In the form, the patient has given her consent for her images and other clinical information to be reported. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

Consent to publish

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

Availability of data and materials

The datasets used during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors have no financial or non-financial competing interests to declare.

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Authors' contributions

Z.Z., Y.Z., Y.X.Z. and L.M. managed the patient and participated in the surgery. Literature search and data analysis were performed by Y.L.. A first draft of the manuscript was written by X.Z. and was critically reviewed and edited by Y.L., and Y.D.. X.H. supervised the work. All authors read and approved the final manuscript.

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Figures

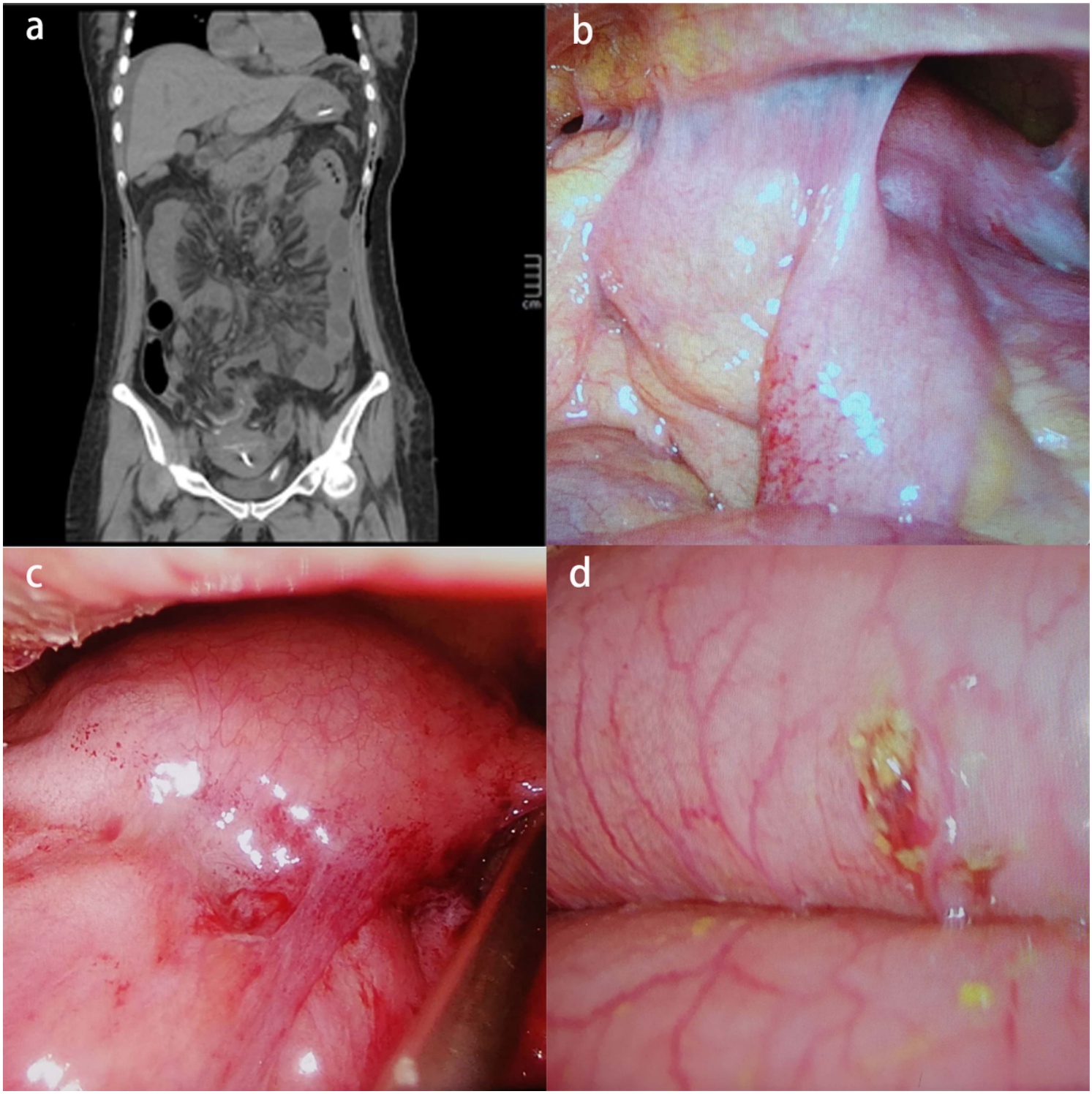

Figure 1

a Computed tomography (frontal section) showing edema and diffuse in exudation mesentery, intestinal tube and abdominal wall, multiple gas shadows and increased density in the ascending colon of the right lower abdomen. b: Obvious intestinal distension, poor peristalsis, partial intestinal wall congestion, and multiple adhesions could be seen. c: The posterior wall of the uterus adhered tightly to the intestinal loop, sealing the pelvic cavity behind the uterus. Bilateral attachments cannot be exposed. d: A ruptured bowel.

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