Intra-abdominal adhesions are a common complication following any surgical procedure involving the abdominopelvic cavity. The mechanism behind adhesion formation is still not well understood, however, is thought to be caused from inflammatory signaling and mesothelial surface disruption. Adhesion occurrence following a laparotomy is estimated to be as high as 95%, with the other 2 main causes of adhesion formation being chronic inflammation of the bowel and postirradiation therapy. Bladder perforation due to intra-abdominal adhesions has never been reported but may be something to consider when other causes of peritonitis have been excluded. We discuss the case of a male patient who presented with an acute abdomen amid new onset ascites, with surgical exploration revealing a bladder perforation.

CASE PRESENTATION

A 71-year-old male presents to the ED with 2 hours of acute lower abdominal pain after bending over to pull a dish out of the dishwasher. Upon arrival to the ED, he describes his pain as unrelenting, excruciating, and worsening with lumbar flexion. His wife comments that he has appeared “more and more bloated” since the onset of his symptoms. He notes no urinary output since the pain began. He denies any changes in diet, medications, or recent travel history. Review of systems is only remarkable for loose stools, which is normal for the patient at baseline. The patient’s medical history is significant for rectal adenocarcinoma diagnosed 5 years prior with neoadjuvant chemotherapy and radiation, followed by surgical resection. His only complications following cancer treatment were transient peripheral neuropathy and erectile dysfunction. He also underwent an elective laparoscopic cholecystectomy performed 5 weeks prior, with no complications and an uneventful postoperative course.

Upon arrival to the ED, the patient’s vital signs were significant for tachycardia, hypertension, and bradypnea. Upon careful inspection of the abdomen, there were significant adhesions within the pelvic cavity. Upon entering the abdominal cavity copious amounts of fluid was suctioned from the abdomen. Extensive lysis of intra-abdominal adhesions was required. In addition, 1 month prior showed an unusual slightly dumbbell-shaped bladder contour. Physical exam revealed a moderately distended and diffusely tender abdomen with a positive fluid wave and voluntary guarding. At this time, there was no definitive source of the new onset ascites or acute pain that the patient was experiencing. Our differential diagnosis was broad including etiologies stemming from the gastrointestinal tract, hepatic-portal system, urinary system, vascular weakening and injuries, and delayed postoperative complications from his recent laparoscopic procedure. The patient was taken for exploratory laparotomy shortly after presenting to the ED.

OPERATIVE COURSE

Upon entering the abdominal cavity copious amounts of straw-colored ascites fluid was noted. Approximately 3 L of fluid was suctioned from the abdomen. Extensive lysis of intra-abdominal adhesions was required. In addition, there were significant adhesions within the pelvic cavity. The bowel was traced from the ligament of Treitz to the rectum with no perforations appreciated. During this exploration, no undue traction or manipulation of the bowel was done. Upon careful inspection of the pelvic cavity, a 1.5 cm perforation was noted in the dome of the bladder. The location was confirmed by palpating the foley catheter balloon through the perforation. A loop of small bowel was noted in the pelvis near the bladder perforation with a small patch of torn adhesion. This patch clearly corresponded to the size and shape of the bladder perforation. The bladder perforation was repaired with 2 layers of 3-0 polyglactin. The abdomen and pelvic cavity was closed in layers. The patient was taken to the ICU for monitoring and serial labs obtained during his ED course revealed a leukocytosis of 12.3, mild hypokalemia, elevated creatinine, and a normal hemoglobin and hematocrit. He had a normal lactate, bilirubin, liver function tests (LFTs), and lipase. Computed tomography scan of the abdomen with contrast showed a normal appendix, small volume ascites, no free air or bowel obstruction, stable thickening of the ventral wall of the bladder, and a superiorly elongated bladder (Figs. 1 and 2). The operative clips placed during his recent laparoscopic cholecystectomy appeared to be in an appropriate position at the gallbladder fossa. A computed tomography scan of the abdomen done 1 month prior showed an unusual slightly dumbbell-shaped bladder contour. Physical exam revealed a moderately distended and diffusely tender abdomen with a positive fluid wave and voluntary guarding. At this time, there was no definitive source of the new onset ascites or acute pain that the patient was experiencing. Our differential diagnosis was broad including etiologies stemming from the gastrointestinal tract, hepatic-portal system, urinary system, vascular weakening and injuries, and delayed postoperative complications from his recent laparoscopic procedure. The patient was taken for exploratory laparotomy shortly after presenting to the ED.

Financial Disclosures: The authors declare that they have no relevant financial interests.

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Submitted: March 5, 2019, accepted (with revisions): March 16, 2019

https://doi.org/10.1016/j.urology.2019.03.013 0090-4295
cavities were thoroughly irrigated, and a closed suction drain was placed near the repair.

The postoperative course was unremarkable. He was discharged from the hospital 4 days following surgery. The suction drain was removed. A foley catheter was left in place for 7 days following surgery to allow healing of the bladder with plans for a follow-up cystogram.

**DIAGNOSIS AND MANAGEMENT**

Given our patient’s history, significant amount of adhesions, large volume of urine ascites, and the torn adhesion associated with the dome of the bladder, the etiology of the bladder perforation and subsequent urinary peritonitis appears to be rupture of a small bowel adhesion to the bladder. Neoadjuvant radiation therapy to treat his rectal adenocarcinoma was likely a contributing factor to adhesion formation within the pelvis. This therapy could have also played a role in decreasing the tissue integrity of the bladder, predisposing the bladder to injury. Management includes operative repair along with monitoring bladder recovery by the utilization of a cystogram to evaluate for urinary leakage into the pelvic cavity. Close follow-up is warranted in either a general surgery or urology outpatient clinic including monitoring for recurrent peritonitis, urinary obstruction, and urinary tract infection.

**REVIEW OF THE LITERATURE**

We searched the English-language medical literature for reports of patients with bladder perforations secondary to intra-abdominal adhesion rupture and found no reports documented. Bladder perforations are uncommon in general with most cases being associated with an underlying degenerative condition of the bladder wall. This includes radiation, previous surgery, or bladder distention from a decrease in neurological stimulation or outlet obstruction. There are a handful of spontaneous ruptures of the bladder with a nontraumatic history in the literature. The vast majority of bladder perforations, however, are caused from damage during surgical procedures.

**CONCLUSION**

Bladder perforations caused from ruptured intra-abdominal adhesions have not been reported but are something to consider when other causes of bladder injury have been excluded. Findings on surgical exploration including adhesions associated with the bladder perforation should be apparent. Signs and history that should raise the suspicion of bladder involvement in peritonitis include a decrease in urine output, rising creatinine and a history of chronic bowel inflammation or radiation therapy to the pelvis.

**References**


