Conservative treatment of an intraperitoneal bladder perforation

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KEY WORDS
urinary ascites » conservative treatment » bladder perforation

ABSTRACT
Introduction. The management of bladder rupture depends on its anatomical location.
Material and methods. Case report and review of the pertinent English language literature.
Results. A 56-year-old man with history of an anterior rectum resection with partial cystectomy presented with signs of acute renal failure, and later with a tender, distended abdomen. Work-up including serum and ascites biochemistry, cystoscopy, and CT cystography diagnosed urinary ascites. The small intraperitoneal bladder rupture was treated conservatively via continued urinary drainage under urinary antibiotic prophylaxis until closure. Conclusion. A conservative treatment of a small intraperitoneal bladder perforation is possible under certain conditions.

INTRODUCTION
The management of bladder rupture depends on its anatomical location. Extraperitoneal lesions are managed conservatively with prolonged bladder drainage, while intraperitoneal ruptures are usually closed surgically. A conservative approach may be effective and successful even for intraperitoneal lesions, but patients must be warned beforehand that failure of conservative treatment is a real possibility.

Table 1. Blood analysis showing acute kidney insufficiency, hyponatriemia, and increased inflammatory parameters on admission, with complete remission of his kidney insufficiency and hyponatriemia after aspiration of ascites fluid.

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|---|---|---|---|---|
| | On admission | Three days after aspiration ascites fluid | Unit | Reference values |
| URM | 41 | 17 | mg/dL | 15-40 |
| CREA | 1.86 | 0.93 | mg/dL | 0.50-1.50 |
| EGFR | 38 | >60 | mL/min/1.73m² | >60 |
| NA | 132 | 145 | mEq/L | 137-145 |
| K | 4.3 | 4.7 | mEq/L | 3.6-5.0 |
| CL | 94 | 107 | mEq/L | 101-111 |
| BIC | 22 | 31 | mEq/L | 22-31 |
| CRP | 39.5 | 18.4 | mg/L | <5 |
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insufficiency were ruled out using ultrasonography; urinary and coprocultures were negative.

Three months later the patient was readmitted with abdominal pain, which started abruptly during forceful micturition after a night of heavy drinking. The onset was intense, non-crampoid, and initially located in the lower abdomen. On physical examination the abdomen was distended. CT imaging showed recurrence of ascites fluid (Fig. 1). Aspiration with analysis of the peritoneal fluid was performed. The presence of elevated creatinine levels (56 mg/dL compared to a normal serum level of 1.41 mg/dL) and urea levels (545 mg/dL compared to a normal serum level of 25 mg/dL) was suggestive of urinary ascites. The presence of a small bladder rupture was confirmed by cystoscopy (Fig. 2). A conservative approach with indwelling catheter and prophylactic urinary antiseptics (nitrofurantoin 100 mg OD) was initiated. After a few days, the patient was able to leave the hospital. CT cystography with 3-dimensional reconstruction three weeks later showed no more leakage and complete closure of the bladder perforation (Fig. 3).

Urodynamic evaluation showed bladder instability during the filling phase with an obstructive voiding pattern characterized by high pressures intravesically and a weak urinary flow. After starting the patient on a combined alpha-blocker (tamsulosin 0.4 mg) and anticholinergic (solfenacin 5 mg) therapy [1], voiding was good with no post-void residual.

DISCUSSION

Urinary ascites is an unusual clinical finding associated with intraperitoneal urinary tract injury. Traumatic and non-traumatic causes have been reported in medical literature. Traumatic bladder perforations are known complications from gastrointestinal and urogynecological interventions, trauma (blunt or penetrating) to the lower abdominal wall, pelvic irradiation [2-6], and delivery-related interventions such as forceful forceps [7], vacuum device [8], and cesarean section [9]. But also long-term catheterization in the presence of urinary tract infections can lead to chronic irritation with weakening of the bladder wall integrity and spontaneous rupture as a result. Non-traumatic causes are mostly the result of cancerous invasion from surrounding structures such as rectosigmoid and cervical cancers. In our case, the bladder perforation was probably caused by the concurrence of two events. First of all, the laparoscopic closure of the partial cystectomy one year before led to a zone of smaller resistance. Secondly, the presence of obstructive voiding worsened by obsessive alcohol consumption led to high intravesical pressure in an overdistended bladder. The combination led to a rupture at the “fragile” bladder wall, resulting in urinary ascites.

Patients with urinary ascites, as in our case report, present with a diffusely tender, distended abdomen without evidence of sepsis [3]. Their urological symptoms may range from normal micturition to dysuria and hematuria [3, 7, 10]. In the absence of a traumatic event, medical and surgical histories are important clues in the diagnosis. It is important to educate patients who have had bladder repair of the possibility of sutures releasing with bladder perforation as a consequence.

However, the presentation may be more dramatic. Patients can present with biochemical signs of acute renal failure [11]. In our patient, no ionogram was performed on the ascites when he first presented. His acute renal failure was blamed on dehydration. The signs of acute renal failure cleared rapidly after aspirating the ascites fluid. Possibly the acute renal failure was a misdiagnosed case of “reverse self-dialysis” of the peritoneal membrane [2, 12-13]. Instead of diffusion of metabolic waste products into a dialysis solution, the peritoneum re-absorbs urea and creatinine from the leaked urine with a subsequent rise in serum levels. Moreover, in extreme conditions there may exist a physiological attempt to dilute the hypertonic uroperitoneum at the expense of extracellular fluid [14]. In this situation, serum values of urea, creatinine, and potassium will be high, while sodium and chloride concentrations will be low [15, 16]. In order to maintain osmolar...
equilibrium, water and sodium diffuse into the peritoneal cavity leading to an increase in ascites fluid. The level of natremia is unspecific for diagnosis of acute renal insufficiency.

Work-up of urinary ascites includes a combination of serum and ascites biochemistry, cystoscopy, conventional cystography, or CT cystography to pinpoint the diagnosis [11]. Urine cytology or biopsies taken at the rupture site can be used to exclude malignant perforation. Cystography is the most sensitive “gold standard” technique. In earlier times, Carroll and McAninch diagnosed 100% of cases, while Sandler et al. diagnosed 19 of 21 intraperitoneal bladder perforations with a conventional cystogram [17, 18]. Today some trauma centers rely exclusively on CT cystograms to evaluate suspected bladder rupture with equally good results [19].

The management of bladder perforations has been a controversial issue. In the beginning, all bladder perforations used to be treated by open repair. Publications by Hayes and Corriere [20, 21] supported a conservative management with temporary bladder drainage in extraperitoneal perforations. Intraperitoneal ruptures on the other hand, are usually still managed primarily by open repair, mostly because of concern about communication between the environment and peritoneal cavity [17]. Our case illustrates that even a conservative approach in the treatment of small intraperitoneal ruptures may be considered if a number of conditions are maintained: urinary antibiotic prophylaxis and continued urinary drainage through an indwelling catheter for a minimum of two weeks until closure of the perforation is evident on CT cystogram. Patients should be warned however that an unsuccessful result with persistent communication to the peritoneal cavity is possible and more typical.

CONCLUSION

Urinary ascites should be considered in the differential diagnosis of ascites in patients who have had bladder surgery in their past surgical history. Serum and ascites biochemistry in conjunction with CT cystography are key to diagnosis. Our case illustrates that a conservative approach is possible, even in cases with intraperitoneal rupture. Nevertheless, we recommend urinary prophylaxis until urinary drainage via an indwelling catheter has allowed the lesion to heal.

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