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SPONTANEOUS BLADDER RUPTURE IN ASSOCIATION WITH ALCOHOLIC BINGE: A CASE REPORT AND REVIEW OF THE LITERATURE

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□ **Abstract**—Although a few cases of bladder wall rupture have been reported in the literature in association with bladder wall disease, idiopathic rupture of the bladder without injury remains an uncommon phenomenon. We report the case of a patient presenting to the Emergency Department with diffuse abdominal pain from spontaneous bladder rupture in association with an acute alcohol binge. Although cases of spontaneous bladder rupture in association with alcohol use were historically associated with high morbidity and mortality, prompt identification and treatment can lead to favorable outcomes. © 2009 Elsevier Inc.

□ **Keywords**—bladder rupture; alcohol binge; abdominal pain; vomiting; suprapubic tenderness

INTRODUCTION

Cases of bladder wall rupture have been reported in the literature most often in association with ongoing chronic diseases of the bladder wall (1). There are many different causes of bladder rupture. Idiopathic rupture of the bladder without injury remains a relatively uncommon phenomenon. A classification of spontaneous rupture of the bladder into known vs. idiopathic etiologies was developed in response to reports that appeared in the literature in which “spontaneous rupture of the bladder” occurred with no discernible cause (2). The urinary bladder is normally well contained and protected from injury.

When the bladder is full and distended above the pelvic inlet or when the pelvis is fractured, the normal protective influence of the intact pelvic ring is lost (3).

Alcohol intoxication is reported as an etiology within a subset of idiopathic bladder rupture cases. In the Emergency Department (ED) setting, patients presenting with acute alcohol intoxication are notoriously poor historians. Consequently, these cases are often complicated due to the patients lapses in memory of certain events and frequent complaints of non-specific abdominal pain. Physicians searching for the cause of a patient’s abdominal pain typically seek to rule out cases of bleeding ulcer, cholecystitis, pancreatitis, intra-abdominal trauma, bowel obstruction, severe gastritis, or other serious illness. Unless bladder rupture is included in the differential diagnosis, it may not be recognized and serious consequences can result.

A small number of case reports have appeared in the literature involving episodes of spontaneous bladder rupture in association with either acute alcohol intoxication or recent ingestion of significant amounts of alcohol in the absence of associated trauma to the bladder (4–11). When promptly identified, most cases of spontaneous bladder rupture in association with alcohol consumption are treated without complication and have a positive outcome. Historically, delayed diagnosis has led to significant morbidity and mortality (3). Despite the infrequency, the cases in the literature retain a consistency

with regard to features at presentation, course, and appropriate treatment. It is important for clinicians and especially ED health care personnel to be aware of this particular condition due to the diagnostic and subsequent treatment difficulties.

CASE REPORT

A 30-year-old man presented to the ED with a 1-day history of severe abdominal pain. He developed the abdominal pain after an episode of binge drinking with approximately six beers and several shots of alcohol. The patient passed out after the binge and awoke in the early morning with severe vomiting. He then experienced significant abdominal pain. The patient tried to self-medicate with Vicodin and Pepto-Bismol to relieve symptoms, eventually returning to sleep. On the following day, he presented to the ED with persistent acute abdominal pain. The patient was afebrile, and the examination revealed moderate to severe lower abdominal pain, suprapubic abdominal tenderness, and moderate distention. He had no significant past medical or surgical history, no current medications or known drug allergies, and no family history of renal disease. He had a positive history

Figure 1. Acute abdominal X-ray study, initially read as normal. In retrospect, the paucity of gas was likely created by the intra-abdominal fluid.

Figure 2. Ultrasound of the pelvis shows bladder with evidence of free fluid outside of bladder wall (arrow).

of alcohol and tobacco use and denied use of any recreational drugs.

His initial laboratory workup was significant for an elevated white blood cell (WBC) count of $16 \times 10^3/\mu\text{L}$, significantly elevated creatinine of 4.3 mg/dL, a low bicarbonate of 19 mEq/L, and a low calcium of 7.6 mg/dL. His acute abdominal X-ray study was normal (Figure 1). Ultrasound showed evidence of free fluid outside of the bladder wall (Figure 2). Foley catheter output was decreased over several hours duration. As he continued to experience persistent pain, a computed tomography (CT) scan of the abdomen with only oral contrast showed a distended bladder with free fluid (Figure 3). Intravenous contrast could not be used with the CT scan due to the significantly elevated creatinine. Surgical consult was

Figure 3. CT scan of the bladder shows free fluid outside of the bladder wall (arrow).

Figure 4. CT cystogram shows contrast leaking outside of bladder wall (arrow).

obtained and the patient was admitted to the hospital. Diagnostic paracentesis was performed for laboratory analysis and evaluation. Due to suspicions of bladder injury, specific fluid analysis revealed a creatinine of 19 mg/dL. The patient's urinalysis was significant for an elevated creatinine of 42.9 mg/dL, red blood cells 600/high-power field, and a protein level of 500 g/dL. A urologic consult recommended a CT cystogram. After administration of 150 cc of contrast, an intraperitoneal rupture at the left posterior dome of the bladder was demonstrated (Figure 4). Laparoscopic repair confirmed the diagnosis and revealed a 5-cm defect in the wall. The patient tolerated the procedure with minimal blood loss and the recovery was unremarkable. Before discharge, the patient's biochemistry returned to normal levels and he was able to void normally.

DISCUSSION

The majority of spontaneous bladder rupture cases reported in the literature have been associated with bladder wall disease (1,2). The rare reports of spontaneous bladder rupture in the past 20 years often seem to occur in association with alcohol intoxication (4–6,9). Bastable et al. in 1959 reported a series of cases associated with alcohol intoxication that had a mortality rate of 50% (4). Subsequent case studies and meta-analyses have differentiated between those cases associated with alcohol or substance abuse and spontaneous rupture of the bladder secondary to urinary retention or other disease processes (5,6,12,13). The reported mortality rate associated with the complications from bladder rupture has declined due

to better management of hyperkalemia, renal failure, and sepsis (7).

It is important to consider the possibility of bladder rupture in patients who present with difficult-to-discern symptoms associated with acute alcohol intoxication. Several case reports of bladder rupture have reported that these cases mimicked acute renal failure and myocardial infarction (12–14). In addition to the non-specific complaints of abdominal pain and vomiting, there have been reports of electrocardiogram changes, including wide QRS complexes and ST segment elevations (14). These changes are likely due to the metabolic derangements, including an increased potassium load in the peritoneum after bladder rupture. Clearly, the presentation of cases of spontaneous bladder rupture in the absence of other associated symptoms or a history of bladder disease has led to significant delay in diagnoses and treatment. In early stages after bladder rupture, symptoms can still be minimal, vague, and diffuse. Although many of these patients will have signs of ascites, the presence of red blood cells in the urine may not be visible for 24 to 36 h after the initial rupture (10). Over 24 to 48 h after the symptoms have become more severe, the risk of mortality increases dramatically (3,10).

Idiopathic bladder rupture is a diagnostic consideration in patients with no known history of bladder wall disease or injury complaining of an inability to void, hematuria, diffuse abdominal pain, suprapubic tenderness, and abdominal distention. With alcohol intoxication, the bladder begins to fill and becomes severely distended. The dome or fundus is the weakest area of the bladder and therefore virtually all spontaneous bladder rupture cases occur there. The decreased sensation associated with increasing levels of intoxication, along with the diuretic effects of the alcohol, places the patient at greater risk.

Cases of acute alcohol intoxication are rising across the United States. Studies conducted with college students in 1974 and replicated again in 1994 found almost the same percentage of students drinking (79.4% and 78.6%, respectively) but found a significant increase in binge drinking in 1994 (15). It has typically been thought that men have a greater tendency to drink alcohol, to drink more heavily and more frequently in comparison to women. However, those differences seem to be disappearing over time (15,16). Thus, as the drinking patterns of the general population change, especially in light of the inherent dangers of binge drinking, physicians and other emergency health care professionals need to be alerted to the potential for bladder rupture and associated symptoms. Although no data are available, it seems likely that an increase in binge drinking in both men and women, as well as drinking in younger age groups,

would increase rates of idiopathic bladder rupture in association with alcohol intoxication (17).

Retrograde cystography or CT cystogram is the procedures of choice to diagnose a ruptured bladder. CT cystography is equivalent to retrograde cystography for detecting bladder rupture (18). In our experience, CT cystography is preferred because it is less invasive. Paracentesis can be used for further confirmation of intra-peritoneal bladder rupture by demonstrating biochemical abnormalities resulting in elevated creatinine levels in the ascitic fluid. Once the diagnosis is established, surgical intervention is indicated for intra-peritoneal rupture, whereas extra-peritoneal rupture usually can be managed conservatively (19).

CONCLUSION

This case is presented to increase awareness of the key features of spontaneous bladder rupture in association with alcohol intoxication or substance abuse (4,7). With the documented increases in binge drinking noted in the literature, it is possible that more cases of spontaneous bladder rupture will be presenting across the country (15). The appropriate diagnostic protocol will ensure that these conditions no longer result in delayed or ineffective treatment and the associated morbidity and mortality inherent in missed diagnoses.

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