

Spontaneous Bladder Rupture: A Diagnostic and Treatment Dilemma Case Studies and Literature Review

Mayank Mohan Agarwal, Shrawan K Singh, Vineet Naja, Ravimohan Mavuduru, Arup K Mandal

Nehru Hospital, Department of Urology, Postgraduate institute of Medical Education and Research, Chandigarh, India.

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ABSTRACT

INTRODUCTION: Spontaneous bladder rupture (SBR) is uncommonly reported and often presents with non-specific clinical features. Therefore, SBR poses a diagnostic and management dilemma for the treating clinician. The authors present 3 cases of SBR with atypical presentation that were promptly diagnosed and successfully managed through conservative treatments.

METHODS: Three males presented to the authors' emergency department. All 3 had features of urosepsis and difficulty voiding, 2 had lower abdominal cellulites, and 1 had uremia, abdominal distention, and tenderness. One patient had untreated urethral stricture negotiable with an 8Fr catheter, and the other 2 had paraplegia with a neurogenic bladder following spinal trauma. Ultrasound revealed a thick-walled diverticulated bladder with bilateral hydroureteronephrosis in all patients. A computed-tomography cystogram was performed because of a high index of suspicion for SBR.

RESULTS: The CT-cystogram showed bladder rupture in all 3 patients (intraperitoneal in 1 and extraperitoneal in 2). All were managed with parenteral antibiotics. Drainage of the urinary bladder and the collection was done through an incision in 2 patients and peritoneal drain in 1. Follow-up CT-cystogram in all patients revealed complete healing of bladder perforation and resolution of the collection. A cystometrogram of 1 patient revealed detrusor overactivity with poor contractility.

CONCLUSION: Physicians must keep a very high index of suspicion for SBR in the presence of diseased bladder associated with overdistension. The CT-cystogram is highly sensitive in diagnosing SBR. Conservative management by indwelling bladder catheter and drainage of extravasated collection is feasible in many cases of extraperitoneal bladder rupture. Conservative treatment may also be considered in carefully selected patients with intraperitoneal bladder rupture, especially if there is intent of temporization. A follow-up functional assessment of the bladder is mandatory to prevent recurrence.

KEYWORDS: Bladder disease, Computed tomography, Neurogenic bladder, Urodynamics, Observation

CORRESPONDENCE: Dr. Shrawan K Singh, Professor, Department of Urology, Level II, B Block, Advanced Urology Center, Nehru Hospital, Postgraduate Institute of Medical Education and Research, Chandigarh, India. PIN-160012.

Email: drmayank_22@rediffmail.com, shrawansingh2002@yahoo.com

INTRODUCTION

Spontaneous bladder rupture (SBR) is an uncommonly reported entity, defined as the presence of rupture without any antecedent trauma [1]. The exact incidence is unknown; however, the reported incidence has been as low as 1:126000 hospital admissions [2]. Huffman et al [3] classified the etiologies of these injuries into lesions: (1) originating in the bladder wall, (2) originating outside the bladder wall, (3) causing overdistention, and (4) originating from idiopathic factors. The term *idiopathic* includes the absence of trauma as well as the above pathologies.

Bladder rupture (BR) commonly presents with hypogastric pain or tenderness, abdominal distension, hematuria, etc [1]. However, the signs and symptoms may be nonspecific and often insidious, leading to a delay in presentation as well as diagnosis [4,5]. Various tests have been employed for diagnosis of bladder rupture, including ultrasonography [6], retrograde cystogram (RC) [1], and computed-tomography (CT) with [1,7] or without [5] cystography. Intraperitoneal BR (IBR) is commonly managed surgically; extraperitoneal (EBR) can be managed conservatively [8]. However, reports of converse forms of management of each group exist in the literature, especially in SBR [9,10].

SBR has been associated with a mortality rate of 25-50% [11]. Many of these deaths can be ascribed to septic complications compounded by delayed presentation and diagnosis. This mortality rate emphasizes the importance of early recognition. The authors present a series of three cases of SBR that were promptly diagnosed and conservatively managed.

METHODS AND RESULTS

Case 1

A 45-year-old male sustained a wedge fracture of the second lumbar vertebra (L2) following a road-side accident. The accident occurred 8 months before the current evaluation and resulted in paraplegia. The patient was hospitalized elsewhere for 6 weeks and remained on an indwelling perurethral catheter (PUC) during this period. When his paraparesis improved and the PUC was removed, he was discharged. He continued to void in a poor stream with abdominal straining. He had undergone cystolithotomy 10 years ago.

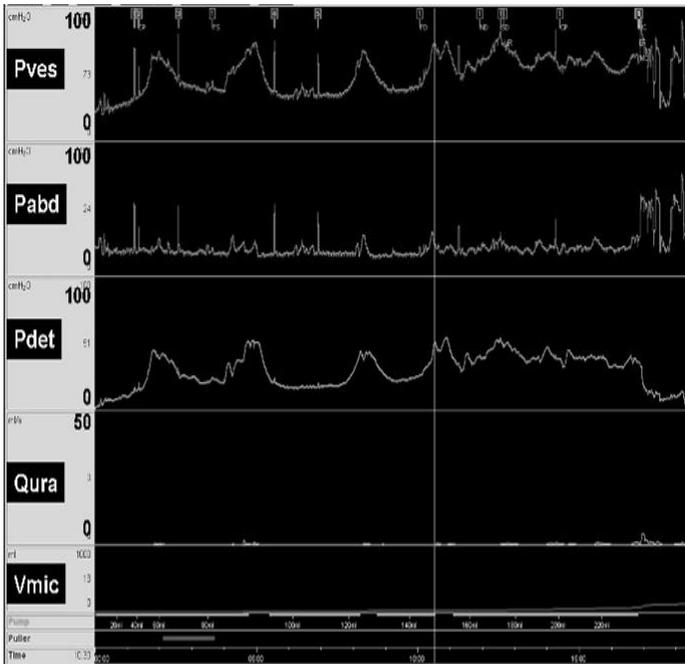
Two-months following discharge, he presented to the authors' emergency department with fever, lower abdominal pain and decreased urine output. Examination revealed a 10x8 cm area of cellulitis with an underlying palpable bladder. His hemoglobin (Hb) was 9.5 g/dL; total leucocyte count (TLC) was 15,300/ μ L (79% polymorphs); and creatinine was 2.2 mg/dl. Ultrasound was suggestive of a thick-walled bladder with bilateral hydronephrosis. Parenteral antibiotics were initiated. Because SBR was highly suspected, CT-cystography was performed. It revealed EBR on the anterior wall with collection (probably from the area of the cystolithotomy scar; see Figure 1a). The indwelling PUC was placed to drain turbid urine, and incision-drainage of the collection was performed. A CT-cystogram was performed at 4 weeks, which confirmed healing of the perforation (Figure 1b).

Figure 1: CT-cystogram of case 1 (a) at presentation, showing a small extraperitoneal perforation on the anterior wall of the bladder communicating with anterior abdominal wall cellulitis, and (b) 4 weeks after the initial presentation, showing healing of the perforation and resolution of cellulitis.

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Figure 2: Cystometry graph of case 1 one month after discharge, showing a poorly compliant bladder with detrusor overactivity associated with urinary leakage. The patient had poorly sustained detrusor contractions during the voiding phase and strain during voiding.
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The patient was discharged free of the catheter with an advice of clean intermittent catheterization (CIC) and anticholinergics. Subsequent cystometrography (CMG) was performed after stopping anticholinergics for 10 days. The CMG showed presence of marked detrusor overactivity and impaired contractility (Figure 2). The patient is doing well 12 months later.

Case 2

A 40-year-old man sustained an 11th-12th dorsal vertebral fracture (D 11-12) following a fall from height 2 years before the current evaluation. The fall resulted in paraplegia and bladder-bowel dysfunction. The patient was managed elsewhere and discharged on indwelling PUC. He returned to the other hospital for follow-up after 18 months. He had retained encrusted obstructed PUC, for which emergency cystolithotomy was performed. The PUC was removed after 4 weeks, and the patient was discharged without any advice about bladder management. He continued to void in poor stream with abdominal straining.

The patient presented to the authors' emergency department with lower abdominal pain, increased difficulty in voiding, fever, and swelling. He had a discharging wound over the lower abdomen for the last 10 days. Examination revealed suprapubic cellulitis with a discharging wound involving the previous cystolithotomy scar. His Hb was 11.2 g/dl; TLC was 18100/ μ L (81% polymorphs); and creatinine was 2.0 mg/dl. Ultrasound revealed a thick-walled bladder and bilateral hydronephrosis. He was put on parenteral antibiotics. A CT-cystogram was performed because of a high suspicion of SBR. The test showed EBR from the cystolithotomy scar. The patient's management was similar to that used for case 1: incision drainage of the collection, indwelling urethral catheterization, and local wound care. A repeat CT-cystography at 4 weeks showed complete healing of the SBR, and the patient was discharged on CIC regime. He is doing well on CIC and CMG has been planned.

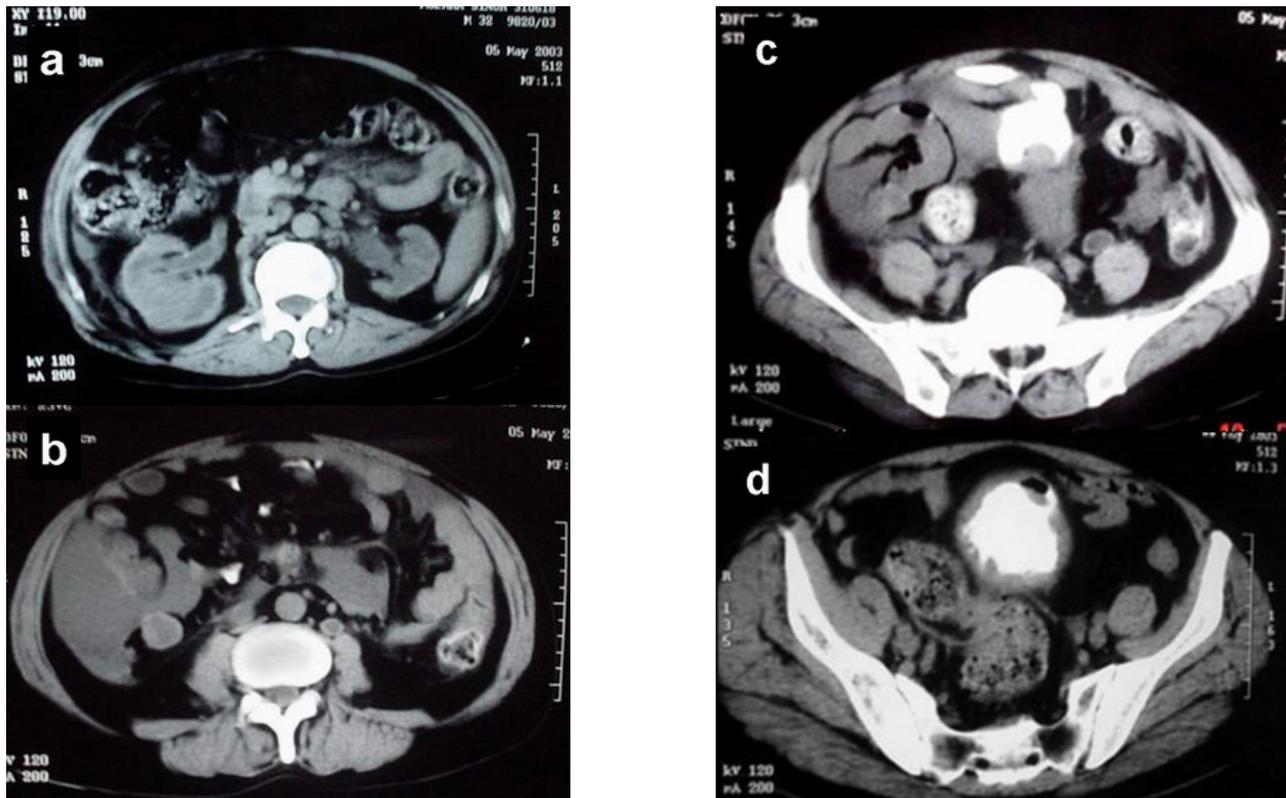
Case 3

A 40-year-old male presented to the authors' emergency department with a history of uremic features, poor urine stream for 6 months, fever, abdominal pain, and near retention for 3 days. He was pale and febrile. Abdominal examination revealed palpable tender bladder and generalized mild abdominal tenderness. His Hb was 8.9g/dL; TLC was 18400/ μ L (polymorphs 85%); and creatinine was 5.7mg/dL. Ultrasound showed bilateral small kidneys with hydroureteronephrosis and a thick-walled distended trabeculated bladder. Parenteral antibiotics were initiated. Urethral catheterization was possible only with an 8Fr infant feeding tube over a floppy-tip PTFE guide wire, due to stricture of the anterior urethra. Purulent urine started draining immediately. Because of the infective setting, a retrograde urethrogram was not performed. After draining approximately 200mL, the catheter became blocked and abdominal distension and tenderness increased. Ultrasound revealed the presence of free fluid in the peritoneal cavity, which was purulent on tapping. The authors performed RC because of suspected IBR; it was noncontributory. A CT-cystogram was performed which revealed a small IBR with ascites (Figures 3a, b, c).

Because of the patient's poor general condition and high risk for anesthesia, the authors placed a 28Fr intraperitoneal drain (Portex Ltd., London, UK) percutaneously for temporarization. It immediately drained 500 mL of purulent fluid. Biochemistry of this fluid was consistent with urine (urea 325 mg/dl; creatinine 22 mg/dl). His general condition started improving rapidly with progressive subsidence of fever and abdominal tenderness. His drain output decreased within 48 hours, with

Figure 3: CT-cystogram of case 3 at presentation showing (a) bilateral hydroureteronephrosis, (b) ascites, and (c) a small intraperitoneal perforation at the bladder-dome. A CT cystogram at 2 weeks after the initial presentation shows complete resolution of free fluid and healing of the perforation (d)

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a corresponding increase in perurethral output. At this time, the authors started soft dilatation of the urethra by replacing a PUC of progressively increasing size (over a guide wire) by 2Fr every 3 days, until they placed an 18Fr catheter. The drain was removed on day 8. A CT-cystogram performed at 3 weeks revealed complete healing of the IBR and no collection (Figure 3d). The PUC was then removed, and the patient was discharged with an advice of daily self calibration to check for lumen and post-void residue. He followed up regularly for one year, doing calibration with an 18Fr catheter once per month. He then was lost to follow up.

DISCUSSION

Pressure of more than 300 cm H₂O is required to rupture a normal bladder. Rupture most commonly occurs because of a direct blow to the distended organ. This trauma often leads to perforation in the dome, the thinnest and least supported

part of a distended bladder. In contrast, EBR results from direct laceration usually from a bone-spicule of a fractured pelvis (FP) or, less commonly, from distraction injury associated with pubic diastasis [8]. Bladder-pathologies rarely cause perforation unless associated with overdistention [1]. However, in the presence of overdistention, a formal *blow* or *bone-chip* may not be required for perforation; rather, *spontaneous* rupture may occur. Most of the literature on SBR consists of case reports or case series, and augmentation cystoplasty [12], pelvic radiotherapy [2] and bladder cancer [1] constitute a large majority. Other bladder wall pathologies reported to cause SBR include prolonged cystitis, tuberculosis [1], bladder diverticulum, lipomatosis, scars from previous surgery [13], vaginal delivery [14,15], and atheroembolism affecting the vesical arteries [16]. A neurogenic bladder with or without bladder augmentation is an important cause of SBR. A neurogenic bladder may lead to delayed diagnosis due to loss of sensations, resulting in serious and misleading presentations (eg, peritonitis, sepsis, uremia) [17]. All

patients in the present investigation had an overdistended bladder. In addition, case 1 & 2 (both with a neurogenic bladder) had earlier cystolithotomy done for calculi, and case 3 had severe cystitis with diverticulation. Therefore, overdistention compounded by these weakening factors might have led to the perforation. However, in case 3, minor trauma due to manipulation of floppy-tip guide wire and feeding tube within the bladder cannot be ruled out.

Ninety five to 100% of patients with traumatic bladder rupture present with abdominal pain and gross or microscopic hematuria [1,8], often with bruising at the impact-site. More than 80-90% of traumatic bladder injuries may be diagnosed without significant delay upon presentation [5]. However, when IBR is not associated with FP, the patient may not feel the urge to void. This may delay the presentation by few hours or more. These patients may present with abdominal distention and *oliguric acute renal failure* secondary to reabsorption of urinary constituents from the peritoneum [8]. The ability to void does not rule out BR, because the bladder may still act as a reservoir in cases of small IBRs and many EBRs, leading to further delay in presentation [18]. Moreover, in patients with SBR, the diagnosis is often delayed for various reasons. First, absence of a history of trauma may prevent the physician from considering SBR. Second, because of diminished sensations and altered motor function in the abdomen and bladder, there is an absence of abdominal pain and signs which may significantly delay presentation as well as diagnosis [17]. Third, the perforation may be small and the collection often contained or loculated due to adhesions of previous surgery, radiotherapy, or advanced malignancy. Therefore, these patients retain the ability to void and are often treated by physicians not specializing in urology as urinary tract infection (UTI), pelvic inflammatory disease, pyelonephritis, or intestinal obstruction. The patient may present with peritonitis, abscesses, sepsis, and uremia before they are referred to urological services [3,6]. Many of these bladders are rendered weak by diverticulation and operative interventions (eg, augmentation cystoplasty, orthotopic neobladder, cystolithotomy). In the presence of inefficient voiding or CIC, UTI, and ischemia, perforation may occur in up to 5-8% of cases [12,19]. Therefore, in the appropriate clinical setting as discussed in the present article and classified by Huffman et al [3], the physician must keep a high index of suspicion to prevent misdiagnosis and associated complications. The authors followed these

guidelines and diagnosed all 3 perforations promptly.

Various radiological modalities have been employed to diagnose BR. Because ultrasonography has overall poor sensitivity [6], its role is limited. Static or fluoroscopic retrograde cystography (RC) is highly accurate (85-100%) in diagnosing traumatic BR [1,6]. However, Carrol and McAnnich [20] emphasized that any deviation from the protocols would deteriorate the accuracy from 100% to 79%. RC has also been used to diagnose SBR [6,21]; however, sensitivity in delayed presentations has been reported to be low [2,6]. RC may also lead to underestimation of the extent of perforation. The authors performed RC in case 3 and missed the SBR diagnosis, possibly due to a small perforation and free fluid in the abdomen. CT-cystography with retrograde instillation of contrast (1-4%) has proven to be highly sensitive and specific (~100%) for diagnosis of EBR as well as IBR in trauma cases [1,7,8]. CT-cystography is replacing conventional RC wherever facilities are available. It has the obvious advantage of imaging the whole abdomen, which is particularly important in delayed and atypical presentations. However, a contrast-enhanced CT with delayed films without a formal RC is inaccurate (60.6%) and not recommended to rule out BR [5]. There are few reports on the use of the CT cystogram in the diagnosis of SBR. The largest report on SBR in patients on augmentation cystoplasty (n=43) did not specify the diagnostic investigation(s) [12]. In a series of 107 bladder augmentations, Defoor et al [19] found CT cystography to be 60% sensitive in 5 patients of SBR diagnosed on laparotomy.

The authors could diagnose SBR in all cases with CT cystography. The authors believe that the difference in sensitivity in various case series may be due to variability in technique and resealing of perforation by the time of the investigation. Apart from the investigations discussed in the present article, there have been reports of proceeding with operative or conservative treatment based solely on peritoneal fluid biochemistry (urea and creatinine) [4,10]. In addition, exploratory laparotomies have been performed for acute abdomen with peritonitis and BR detected intraoperatively [17].

Intraperitoneal bladder rupture is usually managed surgically because of the high probability of associated injuries and possibility of herniation of bowel-loops through the tear, which causes adhesions [5,8]. Extraperitoneal bladder rupture can be managed conservatively [8]. Osman et al [10] managed 4 children presenting with traumatic IBR with a percutaneously placed peritoneal drain and indwelling catheter. These methods worked well when compared with 4 children who were managed surgically. Other physicians have utilized a nonsurgical approach with favorable outcome [6]. Conversely, Kotkin and Koch [9] advocated surgical

treatment of EBR in view of a 26% failure rate of conservative management. However, these authors did not identify risk factors for failure of the latter management. McAnich and Santucci [8] have discussed the indications of exploration in such cases. Similar types of controversies exist in SBR and no definite recommendations are available. In two of the largest series of patients with augmentation cystoplasty, Metcalfe et al [12] (n=43) and DrFoor et al [19] (n=5) managed all of the patients surgically with favorable outcomes. In a similar group, Fontaine et al [6] (n=10) favored a surgical approach in patients with immediate diagnosis (7/10); patients with delayed diagnosis without evidence of generalized peritonitis and hemodynamic instability (3/10) were managed with drainage of a pouch and percutaneous peritoneal drainage. The outcome was successful in 2 patients. Garfinkle et al [17] surgically managed two patients with SBR due to neurogenic bladder with diverticulae; both patients presented with peritonitis and sepsis. Conversely, there have been reports of successful conservative management in various groups of SBR [6,22]. In the present study, the authors could plan a conservative management because of accurate diagnosis using CT-cystography and careful case selection. All of the patients presented with sepsis; both patients with EBR had localized infected collection, and the patient with IBR had infected free fluid with peritonism. In the latter case, a percutaneous peritoneal drain was placed for temporization; however, because the patient's general condition improved dramatically, the authors elected to continue with the same management and ultimately succeeded. The authors suggest primary conservative management in well-selected patients with SBR. Conservative management should also be considered for temporization in severely septic patients with IBR, if a delay in surgery is contemplated. Accurate diagnosis is mandatory before conservative management. In the presence of discrepant investigative results, the physician's clinical judgment should be used to decide the overall management because of the potential fatal nature of the disease.

Identifying risk factors for SBR is important because the recurrent nature of perforation has been reported by many authors [6,12,17,19,22]. Some important factors in the etiology of SBR and their recurrences are: weakening of the bladder wall with enterocystoplasty; radiation injury; and surgical scar of cystolithotomy or diverticulum. These factors are compounded by decreased compliance, overdistension, increased bladder outlet resistance and severe cystitis. Because the *weakening factor* may not be modifiable, it is pertinent to meticulously manage the *compounding factors* to prevent occurrence of SBR. In neurogenic/non-neurogenic voiding

dysfunction, CMG provides important guidelines for evidence-based management and has shown to decrease the incidence of perforation [19]. The authors performed UDS in case 1 and have kept him on anticholinergics and CIC regime.

CONCLUSION

A well-informed clinician must keep a very high index of suspicion for SBR in the presence of a suggestive clinical history, risk factors (weakening and compounding factors) and an abnormal physical examination. A CT-cystogram is highly sensitive in diagnosing SBR and has an additional advantage of imaging the whole abdomen. This imaging increases the clinician's confidence, especially if a conservative approach is contemplated. In light of the current body of evidence and success of the patient management in the present investigation, a conservative approach can be considered standard for patients with EBR; however, such an approach may only be considered in carefully selected IBR patients, mostly as temporization. Good drainage of bladder and the extravasated collection is mandatory under all circumstances. A follow-up UDS/PFS is warranted in patients with known neurogenic bladder or voiding symptoms in the absence of mechanical obstruction. This follow-up provides an evidence-based management of the bladder with the aim of preservation of upper tracts and decreases the probability of rupture.

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