Hypercreatinemia: think beyond acute kidney injury

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Urinary bladder rupture associated with severe hypercreatinemia is a rare clinical presentation. We herein report a 60-year-old interesting patient who was found to have intraperitoneal bladder rupture and pseudo-renal failure. High rate of suspicion and timely diagnosis is the key in management of this condition, which led to complete recovery in our patient.

Key Words: spontaneous bladder rupture, pseudo-renal failure, acute kidney injury, hypercreatinemia, hyperkalemia

Introduction

Urinary bladder rupture and extravasation of urine into peritoneal cavity is a recognized complication of blunt abdominal trauma. However, spontaneous rupture of bladder resulting in pseudo-renal failure is extremely rare with only few reports available in literature.

Case report

A 60-year-old woman was brought to the emergency room by the paramedic team when she was found in an unresponsive state at her home. She lived alone and no additional information was available from her friends. Hospital records showed that she had a dental procedure done 4 weeks back. On initial assessment, she was found to be afebrile and unresponsive to painful stimuli. Vital signs were as follows: heart rate 54/minute, blood pressure 102/60 mmHg, jugular venous pulse flat, and respiratory rate 24/min and oxygen saturation 93% at room air. Respiratory and cardiovascular examination was not contributory. Abdomen examination revealed generalized rigidity. Nervous system exam did not show any localizing signs. There were no rashes or bruises on her body.

Further work up revealed hemoglobin128 g/L, white cell count 14.1 x10^9/L, platelet count 202 x10^9/L, INR 1.3, blood urea 76.7 mmol/L (2.1-8.0), serum creatinine 3222 mmol/L (35-88), sodium 127 mmol/L (136-145), potassium 9.5 mmol/L (3.5-5.1), bicarbonate 5 mmol/L (21-32), random glucose 8.4 mmol/L (3.8-11.0), calcium 2.38 mmol/L (2.12-2.52), phosphorus 4.29 mmol/L (0.81-1.58) and creatine kinase 794 U/L (20-160). Toxic screen was negative. Urine microscopy showed white cells > 100/ high power field (hpf), red cells > 100/hpf and no casts. Urinalysis showed protein 3+ and blood 3+. Urine myoglobin was negative and culture was sterile. Electrocardiogram revealed a sine-wave pattern and arterial blood gas showed severe metabolic acidosis.

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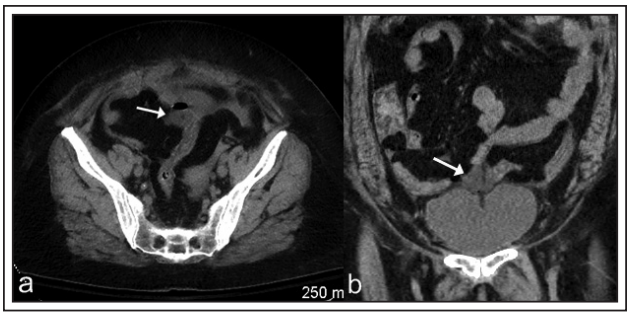

Figure 1. Unenhanced CT axial (a) and coronal (b) images demonstrate bulb of Foley's catheter (white arrow) lying within the peritoneal cavity, outside the urinary bladder. Trace of free fluid is also noted in the pelvis.

She was started on intravenous fluids and a Foley's catheter was inserted. Post-insertion she drained around 700 mL of dark red urine which was sent for analysis. An unenhanced computerized tomogram (CT) scan of abdomen and pelvis, Figure 1, for work up of her hematuria was performed in emergency radiology which showed that the balloon of Foley's catheter was extra-luminal, traversing the bladder dome and was inflated in the peritoneum above the bladder, signifying intraperitoneal bladder rupture. There was no evidence of focal mass or bladder gas on the CT scan. There was mild bilateral hydronephrosis and hydroureter with no perinephric fat stranding. No calculus was identified in the urinary collecting system.

Urology was consulted at this point and patient was started on hemodialysis via a temporary non-tunnelled femoral catheter in view of life threatening hyperkalemia and azotemia. The blood and dialysate flows were adjusted to avoid dialysis disequilibrium. She was transferred to intensive care unit for further care and was subsequently taken to operating room where she underwent repair of her bladder rupture. Intraoperatively, a significant laceration to the bladder approximately 3 cm in length, was detected which was closed and a Jackson-Pratt (JP) drain was left behind. A suprapubic catheter was inserted as well as a Foley's catheter remained in place once the bladder was repaired. Postoperatively, her sensorium improved and hematuria slowly cleared with renal function returning back to normal over a period of 4 days. Further questioning did not reveal any history of trauma, abdominal pain, alcohol abuse, or any chronic illness. She denied any history of suggestive of chronic cystitis or repeated urinary tract infections.

Histopathology report of the bladder wall showed mucosal ulceration with fibrosis and chronic inflammation of bladder mucosa at the site of rupture. The mucosa adjacent to the site of ulceration had reactive papillary hyperplasia and cystitis cystica. There was no evidence of urothelial dysplasia or malignancy or diverticular disease. She subsequently recovered well and was discharged a week later.

Discussion

Urinary bladder rupture is an uncommon event with occurrence in the range of 1 in 126000 hospital admissions.[1] Most often, it is associated with blunt trauma to abdomen, pelvic radiotherapy, outflow obstructions either anatomical or functional or a diseased bladder wall state.[2] In rare circumstances, bladder spontaneously ruptures spontaneously after an alcohol binge[3] or in immediate postpartum period.[4] The usual site of rupture is the dome, the weakest point in the bladder wall. The diagnosis of spontaneous bladder rupture is often missed in absence of history of trauma or a preexisting bladder disorder.[5]

Lower abdominal pain, supra-pubic tenderness, inability to pass urine, oligo-anuria, hematuria, and rapidly increasing ascites should prompt the suspicion of an intraperitoneal bladder rupture. Delay in diagnosis could result in frank peritonitis.[6] CT cystogram is diagnostic investigation of choice in such cases.

Our patient denied any of the symptoms suggestive of bladder rupture when she gained consciousness. The etiology of spontaneous bladder rupture appears to be multi-factorial in our case. She had a very thin, high volume bladder intraoperatively suggesting long term retention or chronic bladder distention. Secondly, her histology showed cystitis cystica, a common chronic reactive inflammatory disorder occurring in the setting of chronic irritation. Metaplasia of the urothelium is incited by irritants such as infection, calculi, outlet obstruction, or even tumor. Though, we did not have any confirmatory evidence for any of those irritants, we postulate that a low grade chronic transmural infectious process might have weakened the bladder and caused it to rupture. Historically, there were no clues to point towards either of these processes.

The abnormally high levels of creatinine and potassium in our patient have never been reported. Hyponatremia, hyperkalemia and hypercreatinemia occurs due to reverse auto-dialysis across the peritoneal membrane.[7] The nitrogenous waste products from the peritoneal cavity get re-absorbed along the concentration gradient which results in azotemia mimicking acute kidney injury. We did not perform peritoneal fluid analysis or retrograde cystography, as the diagnosis was confirmed on the CT scan report.

The striking feature of normalization of biochemical parameters after urinary drainage discriminates true acute kidney injury from pseudo-acute kidney injury from dialysis of urine across the peritoneum.[8] Our patient had a good recovery clinically and her biochemical parameters were normalized within a period of 4 days as expected in pseudo-kidney injury.

Conclusion

Spontaneous rupture of urinary bladder is a serious complication resulting in urinary ascites (uroperitoneum) and subsequent pseudo-acute kidney injury. A high index of suspicion is needed to identify this condition. Management involves urinary catheterization, prompt surgical repair and work up of any underlying cause, if any. This condition should be included in differential diagnosis for acute kidney injury so that emergency physicians, nephrologists and urologists are aware of this rare but completely curable condition. □

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