Acute Renal Failure Following Ruptured Bladder After Electroresection of a Polyp

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Urinary bladder perforation due to electroresection of vesical polyps or cancers is a rare iatrogenic complication. Bladder rupture might cause acute renal failure because of leakage of urine into the peritoneal cavity. We report a case of unexplained renal failure as a consequence of urinary bladder perforation, following the electroresection of a bladder dome polyp. Urinary drainage resulted in rapid resolution of the biochemical abnormalities. Literature on renal failure after urinary bladder rupture is reviewed. [Hong Kong J Nephrol 2006;8(1):36–9]

Key words: bladder rupture, polyp resection, renal failure, urinary drainage

膀胱穿孔乃膀胱息肉或惡性腫瘤的電切除中罕見的醫源性?發症。基於尿液漏出至腹腔中,膀胱的破裂可能會導致急性腎衰竭。以下是一宗在膀胱半球形息肉的電切除術後,膀胱穿孔所致的原因不明性腎衰竭個案,其生化異常於尿液引流後迅速緩解。本文亦回顧了膀胱破裂後腎衰竭的相關文獻。

Introduction

Urinary bladder perforation should be in the differential diagnosis of acute oliguric renal failure soon after bladder surgery [1,2]. We present a case of unexplained acute renal failure after electroresection of a bladder polyp. Biochemical features of uremia occurred as a result of intraperitoneal extravasation of urine.

CASE REPORT

An 81-year-old man was referred to our hospital because of deteriorating renal function shortly after undergoing electroresection of a bladder dome polyp, performed through a transurethral route. Over the ensuing 2 days after the operation, the patient developed oliguria with increasing painless abdominal distension, whilst his serum creatinine rose to 640 µmol/L. The patient's past medical history was marked by type 2 diabetes mellitus and hypertension diagnosed 5 years previously. There was no known history of diabetic ketoacidosis, retinopathy or polyneuropathy.

On physical examination, blood pressure was 140/90 mmHg, with a heart rate of 72 beats/min. His

respiratory rate was 20 breaths/min, and he was afebrile. Cardiothoracic examination was unremarkable. Abdominal examination revealed mild distension, with shifting dullness and a large fluid wave. There was no tenderness to deep palpation or organomegaly. Urogenital examination was unremarkable. Digital rectal examination demonstrated a nontender prostate of normal size and contour. Extremities were without edema.

Laboratory investigation revealed an acute rise in his serum creatinine to 640 µmol/L 2 days after surgery, from a baseline value of 120 µmol/L (normal range, 30–115 µmol/L), corresponding to an estimated glomerular filtration rate below 10 mL/min/1.73 m² by the abbreviated Modification of Diet in Renal Disease (MDRD) study equation. Blood urea was 19 mmol/L, serum sodium was 125 mmol/L, potassium was 4.1 mmol/L, and bicarbonate was 16 mmol/L. Complete blood cell count and coagulation studies were essentially normal. All viral serologies, including hepatitis, were nonreactive. Liver function tests were also within normal limits, including proteins of 71 g/L and serum albumin of 48 g/L.

The patient was admitted to the nephrology service for further diagnostic evaluation and management of

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his acute renal failure. Abdominal ultrasound showed normal-sized kidneys without hydronephrosis, with a large volume of ascitic fluid. Paracentesis yielded approximately 2.6 L of transudative peritoneal fluid, with the following concentrations: glucose, 9 mmol/L; protein, 4 g/L; creatinine, 1,782 µmol/L; urea, 41 mmol/L. Cultures and cytologic examinations were negative. Evaluation of the anesthesia and operation notes did not identify causes for his clinical picture of acute renal failure. In particular, there was no history of profound hypotensive episodes, and use of nephrotoxic medications or intravenous contrast medium.

Despite the absence of hydronephrosis on renal ultrasound, a Foley catheter was placed, yielding 1.5 L of urine output within the first 30 minutes. Although hemodialysis was required initially to correct the patient's azotemia and electrolyte imbalance, his subsequent clinical course was unremarkable, and he was discharged with normal renal function and a serum creatinine level of 100 µmol/L on the third day. Conservative management of the iatrogenic perforation, which consisted of percutaneous drainage of ascites and prolonged Foley catheter drainage of the bladder, was conducted. The patient was discharged from hospital with the Foley catheter *in situ*. Follow-up was

performed through weekly cystographic examination. The Foley catheter was removed on the 20th day, after a normal urethrocystography.

DISCUSSION

This case demonstrates that after bladder surgery, a high degree of suspicion of urinary bladder rupture is warranted in patients with unexplained renal failure and subsequent ascites. The bladder is an extraperitoneal pelvic organ, but has close proximity to the peritoneal cavity, especially over the dome and anterior wall when the bladder is full. Urinary leakage might, therefore, occur intraperitoneally after bladder rupture [2]. In patients with ruptured bladder, renal failure is caused by fluids and electrolytes equilibrating across the peritoneal surface, as occurs in peritoneal dialysis. Serum values of creatinine, urea and potassium will be high, and sodium and chloride concentrations will be low [3–5]. Water and sodium diffuse into the peritoneal cavity to maintain osmolar equilibrium, which emphasizes ascites [4,5].

A review of the medical literature further identified 23 cases of renal failure following bladder rupture (Table) [1,2,4,6–22]. The key diagnostic findings in

Table. Reports in the literature of renal failure secondary to urinary bladder rupture

Author	Year	Age (yr)	Gender	Predisposing condition	Clinical manifestation	Diagnosis and treatment	Timing of resolution
Sullivan et al [6]	1972	1	M	Spontaneous rupture of urinary bladder diverticulum	Anuria with ascites, disproportionate blood urea to creatinine ratio	Cystogram, excision of ruptured diverticulum and cystostomy	24 hr
Sullivan et al [6]	1972	87	F	Necrotizing cystitis	Acute abdomen, disproportionate blood urea to creatinine ratio	Cystogram	No details
Shinotoh et al [7]	1985	38	M	Alcoholic intoxication and traumatic injury	Hyperkalemic paralysis and gross hematuria	Cystogram, surgical repair	No details
Ratliff & Scoble [8]	1987	67	M	Bladder transitional cell carcinoma with repeated cystodiathermy	Disproportionate rise in serum creatinine	Micturating cystogram and laparotomy bladder repair	No details
Davenport & Goldsmith [9]	1989	35	M	Blunt trauma	Anuria after trauma, lower abdominal and left iliac fossa pain, hyperkalemia and hyponatremia	Cystogram, laparotomy and bladder repair	24 hr
Dees et al [4]	1990	47	F	Bicycle accident	Painful micturation followed by anuria and distended abdomen	Hemodialysis, bladder catheterization and urine drainage	4 d
Dees et al [4]	1990	50	F	Bruch operation for urinary incontinence	Abdominal ileus and disproportionate rise in serum creatinine	Excretory urography	No details
Dees et al [4]	1990	64	M	Transurethral resection of bladder polyp	Nausea, lower abdominal pain, and ascites with acute rise in serum creatinine	Retrograde cystography	No details

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