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Bladder Rupture following Conversion to Enteric Drainage after Pancreatic Transplantation

Vikas Srivastava^a George Passaris^a Rajiv Juneja^a
Mark Siddins^b Jeffrey A.J. Barbara^a

^aRenal Unit, and ^bDepartment of Urology, Flinders Medical Centre and Flinders University, Adelaide, S.A., Australia

Key Words

Pancreatic transplantation · Bladder rupture

Abstract

Complications associated with bladder-drained pancreatic transplant are not uncommon and include urinary tract infections and reflux pancreatitis. Bladder rupture with peritoneal leak is a rare complication after pancreatic transplantation and can present as an acute abdomen with rapidly deteriorating renal function. We describe the first case of a urine leak into the peritoneal cavity occurring after conversion from bladder to enteric drainage. A high index of suspicion is required to diagnose such a complication.

Background

Bladder-drained pancreatic recipients experience a relatively high incidence of metabolic and urologic complications resulting in conversion to enteric drainage in about 15–30% of cases. Spontaneous rupture of the bladder after enteric conversion of a bladder-drained pancreatic transplant has not been previously reported.

Case Report

A 44-year-old man with diabetes mellitus type 1 and end-stage renal failure receiving haemodialysis for four years received a sequential pancreatic transplant after renal transplant in March 2002 and July 2001, respectively. Immunosuppression after transplant included tacrolimus, mycophenolate and prednisolone. The exocrine secretions of the transplanted pancreas drained into the urinary bladder. Due to recurrent urinary tract infection and significant dysuria despite urinary alkalinisation, he underwent pancreatic duodenojejunostomy in June 2003. The following three years were complication free.

In April 2006, he presented to the Emergency Department with generalised abdominal pain associated with non-bilious vomits. He was febrile at 38.6°C, hypotensive and tachycardic with signs of peritonism. The creatinine had increased to 286 µmol/l from 170 µmol/l and the white cell count was $20 \times 10^9/l$. Plain X-ray and CT of the abdomen were non-diagnostic. Empirical therapy with intravenous antibiotics and fluids was administered after an indwelling urinary catheter had been inserted at presentation. He improved rapidly and renal function returned to baseline within 24 h of admission. A very similar presentation occurred two months later. Clinical and biochemical improvement once again occurred quickly and an outpatient colonoscopy and barium swallow returned normal findings.

In December 2006, he presented for a third occasion with abdominal pain and vomiting to the renal outpatients department. The previous evening, he had been lifting a sack of potatoes and experienced some discomfort in his perineum. On examination there were features of peritonism and ascites was detected. The creatinine had risen to 348 µmol/l from his usual baseline and on the following day it was 490 µmol/l. The non-contrast CT of the abdomen confirmed gross ascites. A diagnostic abdominal paracentesis was performed and the creatinine in the ascitic fluid proved to be very similar to serum (501 µmol/l compared to 490 µmol/l). The urinary creatinine at this time was 6.24 mmol/l. A urinary catheter was inserted into the bladder with a rapid improvement in the serum creatinine and a reduction in the volume of ascites. A provisional diagnosis of urinary bladder leak was made and a cystogram ([fig. 1](#)) revealed leakage of contrast into the peritoneal cavity.

In the operating theatre, the leak was confirmed with repeat cystography. An omental patch was mobilised from the transverse mesocolon and placed over the dome of the bladder ([fig. 2](#)). A bladder biopsy showed focal scarring with no dysplasia or malignancy. A cystogram repeated two months later did not reveal any extravasation of contrast into the peritoneal cavity. There have been no complications relating to this procedure. In April 2009, he presented with hyperglycaemia and has become dependent on exogenous insulin.

Discussion

Bladder-drained pancreatic transplants have been associated with complications such as urinary infection and dysuria which can lead to a requirement for enteric drainage [1]. However, bladder rupture after conversion from bladder to enteric drainage has so far not been reported in the literature. This occurred in our case three years after enteric conversion, and the diagnosis was not established until the third acute presentation when a urinary catheter was not initially placed. In our opinion, early urinary catheterisation with the first two presentations allowed the bladder to rest at low volume and resulted in a temporary repair. With the final presentation, the urinary effluent escaped from the bladder directly into the peritoneal cavity resulting in a 'reverse peritoneal dialysis' with recycling or reabsorption of creatinine and a rapid increase in serum creatinine. Metabolic acidosis and hyperkalaemia were also developing quickly. The placement of an indwelling urinary catheter halted this recycling phenomenon.

Urinary bladder rupture is a rare condition but one in which a pathological basis can be identified in most cases [2, 3]. Tarrass et al. [4] identified 23 cases of renal failure following bladder rupture and these were mainly due to intrapelvic gynaecological operations, eosinophilic cystitis, blunt trauma to the abdomen, anatomical outflow obstruction and in association with indwelling catheters [5–10]. The clinical presentation can be variable but often includes oliguria, acute kidney injury and ascites with or without peritonitis. Early diagnosis is important as the condition can be associated with high mortality.

Whilst routine imaging techniques allow a diagnosis of bladder rupture to be made in the majority of cases, its rare nature demands a high level of clinical suspicion where this constellation of clinical features exists [11]. For a definitive diagnosis, conventional cystography is the gold standard [12] (95% sensitivity) although CT cystograms are considered of equal value [13]. Intravenous urography, duplex sonography, cystoscopy and laparoscopy have also led to the diagnosis in some cases. The management of bladder rupture depends on the site of the rupture. For extraperitoneal rupture, bladder catheterisation alone has been considered safe and effective [14], but for intraperitoneal bladder rupture, open repair with an omental patch is favoured and this is especially the case with recurrent bladder rupture.

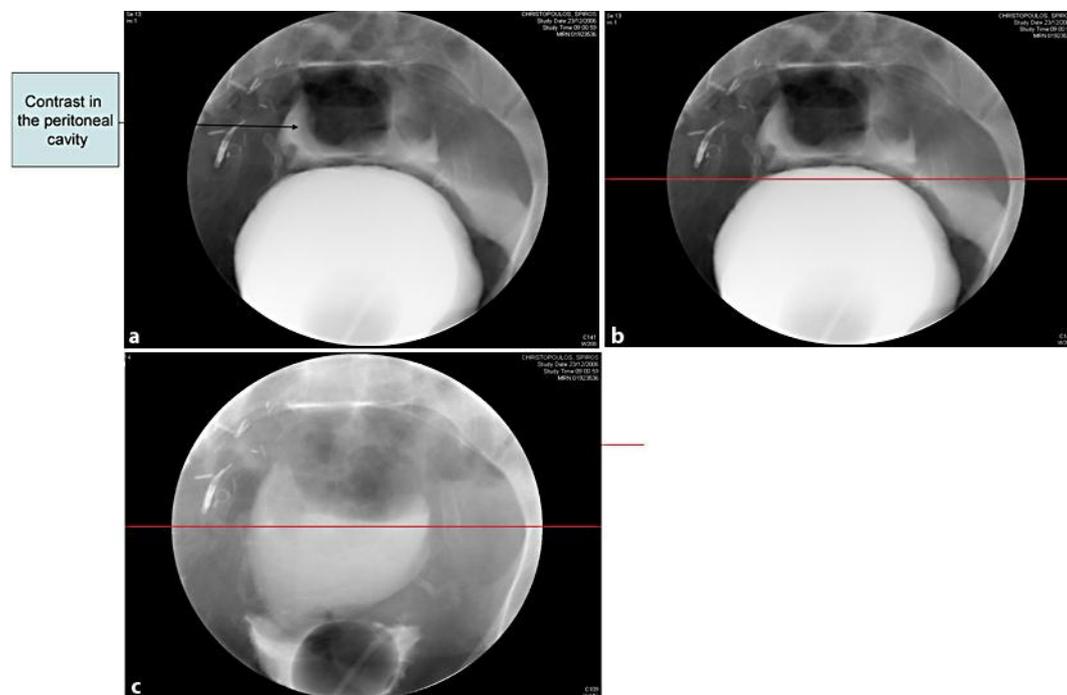


Fig. 1. Cystogram with contrast in the urinary bladder and early escape of contrast superior to the bladder.

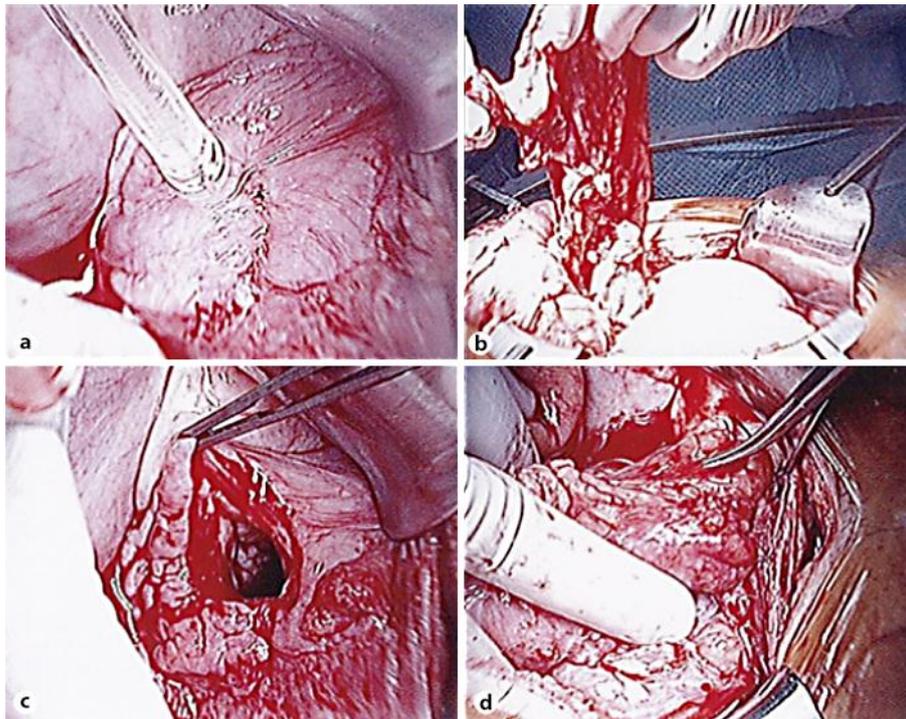


Fig. 2. a Urinary bladder located. b Omentum mobilised. c Rupture on the dome identified. d Omental patch sited.

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