

# Spontaneous intraperitoneal rupture of a neurogenic bladder; the importance of ascitic fluid urea and electrolytes in diagnosis

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**Summary:** Spontaneous intraperitoneal bladder rupture in a 38 year old man with a spastic paraparesis since infancy is described. Delayed diagnosis resulted in peritoneal autodialysis so providing an opportunity for documentation of biochemical abnormalities. Surgery resulted in a successful outcome. The literature on this rare condition is outlined and difficulties in diagnosis are discussed. The diagnostic value of urea and electrolyte levels in ascitic fluid is emphasized.

## Introduction

Spontaneous rupture of neurogenic bladders is rare, a recent review finding only 14 published cases in adults.<sup>1</sup> The earliest reports described twelve cases associated with either tabes dorsalis or paraplegia.<sup>2,3</sup> More recent reports have included rupture of a diabetic neuropathic bladder<sup>4</sup> and bladder rupture in a man with post-traumatic spastic paraparesis.<sup>1</sup> A similar entity has been described in neonates and recently the third case of urinary ascites in a new born with a myelomeningocele was reported.<sup>5</sup> In previous reports of ruptured neurogenic bladder in adults, difficulties in diagnosis have not been particularly highlighted, unlike reports of traumatic or spontaneous intraperitoneal bladder rupture that is not associated with neurological disease. We describe a case of spontaneous intraperitoneal bladder rupture associated with a long-standing paraplegia of undetermined aetiology and highlight the diagnostic usefulness of urea and electrolyte levels in urinary ascites due to bladder rupture.

## Case report

A previously well 38 year old man who had bilateral lower limb weakness since childhood was admitted with a history of sudden onset of severe epigastric and periumbilical pain, vomiting and diarrhoea of 5 days duration. On the day before the onset of his symptoms he drank seven pints of beer but denied any abdominal trauma.

On admission the pulse was 120 per minute and blood pressure was 120/80 mm Hg. There was diffuse

abdominal tenderness without rigidity or guarding. Neurological examination revealed a spastic paraparesis without sensory deficit. On presentation the serum amylase was 350 somogyi units/100 ml, sodium 135 mmol/l, potassium 6.0 mmol/l, chloride 102 mmol/l, urea 31 mmol/l, blood sugar 9 mmol/l, calcium 2.25 mmol/l, phosphate 3.23 mmol/l, uric acid 0.72 mmol/l with normal haemoglobin, packed cell volume, acid and alkaline phosphatase and serum proteins. Chest X-ray, erect and supine abdominal X-rays and an electrocardiogram were normal. With a tentative diagnosis of acute pancreatitis he was treated conservatively. Due to progressive abdominal distension, diagnostic paracentesis was performed on the third day and a straw coloured fluid with an amylase content of 350 somogyi units/100 ml and negative cultures were obtained. By the fifth day in hospital the patient was anuric. Repeat abdominal X-rays revealed an adynamic ileus with intraperitoneal fluid. The diagnosis of a ruptured bladder was considered and repeat paracentesis revealed a straw coloured fluid with few lymphocytes and macrophages, numerous red blood cells, specific gravity 1.005, pH 7, sodium 133 mmol/l, potassium 12.1 mmol/l, chloride 100 mmol/l, urea 133 mmol/l with negative cultures. Simultaneously, serum levels were: sodium 133 mmol/l, potassium 7.7 mmol/l, urea 76 mmol/l, creatinine 1060  $\mu$ mol/l, chloride 105 mmol/l. The abdominal cavity was drained of what now seemed to be urine and over the next 24 hours, 12 litres of fluid were drained via a percutaneous catheter. Hyperkalaemia was treated and the patient's condition improved. On the sixth day cystography showed free flow of dye into the peritoneal cavity. At laparotomy a further 2.5 litres of

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straw coloured fluid were drained, a ruptured diverticulum in the dome was excised, and marked bladder wall trabeculation consistent with neurogenic outlet obstruction was noted. Over the next 72 hours the patient made a rapid recovery with reversion of biochemical abnormalities to normal. Further investigations of neurological function were refused by the patient who 5 years later remains well.

### Discussion

The ruptured bladder may present as an acute abdomen or insidiously following peritoneal autodialysis, as azotaemia, hyponatraemia, hyperkalaemia and acidosis with or without clinically detectable ascites. Diagnosis can sometimes be difficult and delayed, resulting in increased morbidity.<sup>6-10</sup> Recently, in a case where diagnosis was delayed, a haemodialysis had to be performed as a life saving measure.<sup>11</sup> Despite the presence of significant ascites in most of these patients analysis of ascitic fluid for urinary constituents has been overlooked as a diagnostic aid, a

practice noted even in recent reports.<sup>1,12,13</sup>

More than 30 years ago the fortuitous finding of spermatozoa and a uriferous odour in ascitic fluid led to cystographic recognition of bladder rupture.<sup>14</sup> In more recent practice, ascitic fluid analysis for urinary constituents has been confined to investigation of neonatal ascites. In neonatal urinary ascites the findings of a straw coloured fluid with a low protein level, the presence of erythrocytes, macrophages, and polymorphonuclear leucocytes; and urea electrolytes and creatinine levels intermediate to serum and urinary levels have been consistent with bladder rupture.<sup>5,15,16,17</sup> Some of these findings were noted in our patient. We suggest that in patients with ascites associated with azotaemia, hyperkalaemia and acidosis, early analysis of ascitic fluid for urea and electrolytes (and creatinine) and comparison with serum and urinary levels would lead to suspicion of bladder rupture and confirmatory cystography. This simple test would be of most value where radiological facilities are not readily available and conceivably may even preclude preoperative cystography.

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