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From the Clinic

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Reverse autoperitoneal dialysis resulting in pseudo acute renal failure

A rise in serum creatinine without acute kidney injury (AKI) is possible. In the last 5 years, three patients presented to us for high serum creatinine levels after binge drinking. We already reported on two of these three patients [1, 2]. For the first one, dialysis was instituted as the initial diagnosis was AKI. With the experience gained, the next two patients were examined specifically for a particular cause. They were not dialyzed. The data of the third patient were as follows: a 44-year-old male, admitted after a fall following binge alcohol consumption. He complained of painful abdominal distension and was anuric. Serum creatinine was 6.5 mg/dL (575 µmol/L), blood urea: 66 mg/dL (23.5 mmol/L), serum sodium 134 mmol/L and serum potassium 4.3 mmol/L. An abdominal ultrasound examination showed two normal kidneys, a normal urinary tract and presence of peritoneal fluid. In view of these findings, a bladder rupture was suspected and confirmed by cystoscopy. With bladder drainage, serum creatinine diminished to 3.5 and then 1.4 mg/dL (309–123.7 µmol/L). Laparotomy was done and a 4-cm tear on the dome of urinary bladder was surgically repaired.

All three patients had similar histories: (i) an alcoholic binge, (ii) a trauma, (iii) sudden onset of abdominal pain and progressive distension (iv) anuria (v) elevated serum creatinine (vi) cystography revealing a tear in the bladder and (vii) improvement in serum creatinine on continuous bladder drainage. The data of these patients are given in supplementary Table S1.

In the majority of bladder ruptures, there is a history of significant abdominal trauma. Following heavy alcohol intake, the distended bladder is very susceptible to injury and can be ruptured by a minor trauma. Alcohol-induced polyuria and impaired sensorium that removes cues for voiding cause overdistension of the bladder and rupture. The intoxicated patient may fail to recall the trauma. In a review of 20 isolated intraperitoneal bladder ruptures, the mean time between presentation and diagnosis was 5.4 days [3]. The diagnosis was based on a compatible history: suprapubic pain, anuria, hematuria and rapidly increasing ascites. The diagnosis was strengthened by rapidly rising serum creatinine without features of hypercatabolism. Continuous bladder drainage normalized laboratory abnormalities. Urinary bladder injuries after blunt or penetrating trauma are rare, owing to the bladder's anatomical position [4]. Bladder ruptures can be extraperitoneal, intraperitoneal or both, depending on the site of the injury. Intraperitoneal bladder ruptures are usually associated with blunt abdominal trauma (excluding iatrogenic causes), and unlike extraperitoneal leaks, they are not usually associated with pelvic fractures. Intraperitoneal ruptures usually occur at the dome of the bladder in contrast to extraperitoneal leaks which are most often lateral [4]. Non-traumatic rupture is even rarer and causes may be essentially divided as increase in intravesical pressure or weakening of the bladder wall. In most cases, spontaneous rupture occurs in the presence of a urothelial neoplasm or after pelvic radiation therapy [2]. In neonates, peritoneal entry of urine is a rare complication. The major causes are congenital obstructive uropathy with urine leakage into the peritoneal cavity and bladder perforation as a complication of umbilical artery catheterization [5].

The expression 'reverse peritoneal dialysis' was first used in 1991 [5]. It is characterized by a flux of small molecules such as creatinine and urea from urine collected in the peritoneum along a concentration gradient opposite to conventional peritoneal dialysis where small molecules move from the blood to peritoneal cavity, a phenomenon mentioned as 'reverse autoperitoneal dialysis'. As a result, the blood values of creatinine and urea are elevated mimicking renal failure despite normally functioning kidneys. This was described as 'pseudo acute renal failure' in the literature.

Supplementary data

Supplementary data is available online at http://ndt.oxfordjournals.org.

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