Case Report

Hyponatraemic renal pseudofailure—don’t forget the possibility of uroperitoneum

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Key words: bladder diverticulosis; bladder rupture; ascites; hyponatraemia; renal pseudofailure

Introduction

Spontaneous or non-traumatic rupture of the bladder is a rare condition [1]. Following the initial report of urinary ascites in the neonate [2] no more than 25 cases of this condition have been described [1,3,4]. Despite better recognition of this disease and advances in diagnostic procedures in cases without recent history of abdominal trauma or urological intervention the diagnosis remains difficult. Symptoms such as abdominal pain, dysuria, haematuria and renal failure accompanied by profound fluid and electrolyte disturbances may eventually lead to diagnosis, but in all cases reported until 1970 the correct diagnosis was established only at autopsy [3]. We report a case of spontaneous rupture of a bladder diverticulum with massive urinary ascites, severe hyponatraemia, and biochemical pattern and clinical course resembling that of acute renal failure.

Case Report

A 42-year-old man was referred with a 2-week history of general malaise, abdominal pain and abdominal enlargement, nausea, vomiting, diarrhoea, and dysuria. Those symptoms prompted him to seek medical advice and he was admitted to a local hospital, where increased serum creatinine (460 μmol/l) was found. Ultrasound examination revealed normal size and structure of the kidneys. During the patient’s first days in the hospital despite intensive fluid replacement and administration of high doses of loop diuretics there was a steady decrease in urine output followed by complete anuria by day 11, further enlargement of the abdomen, an increase of serum creatinine up to 1010 μmol/l, and serum potassium to 5.9 mmol/l, and a decrease of serum sodium to 98 mmol/l. Metabolic acidosis (pH 7.27, HCO₃ 11 mmol/l) was also observed.

The patient was transferred to our clinic with a diagnosis of acute renal failure and a suspected syndrome of inappropriate antidiuretic hormone secretion (SIADH). Upon admission he was found to have massive ascites causing respiratory problems. His blood pressure was 120/70 mmHg, pulse rate 80/min. On X-ray, upward displacement of the diaphragm was seen. The kidneys appeared normal by ultrasonography and an enormous amount of fluid was seen in the peritoneum. Following initial examination, puncture of the peritoneal cavity was carried out. Instantly about 1500 ml of light-yellow fluid were obtained. A fluid sample was sent for biochemistry and the following results were obtained: sodium 89 mmol/l, potassium 9.1 mmol/l, creatinine 2.5 mmol/l, glucose 7.0 mmol/l, total protein 2.1 g/l, and amylase 80 Caraway units. Fluid sediment contained mainly erythrocytes. Since such biochemical composition suggested the presence of urine in the peritoneal cavity, the bladder was catheterized. In a short time 7500 ml of urine was obtained from the catheter and ascites disappeared. The composition of urine was nearly identical to that of fluid obtained from the peritoneal cavity (Table 1). To normalize the biochemistry quickly, a short haemodialysis was performed. After a 2-h haemodialysis, serum creatinine was 370 μmol/l, sodium

Table 1. Composition of serum, peritoneal fluid, and the urine obtained from bladder catheterization

<table>
<thead>
<tr>
<th></th>
<th>Serum</th>
<th>Peritoneal fluid</th>
<th>Urine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osmolality (mOsm/kg H₂O)</td>
<td>261</td>
<td>Not measured</td>
<td>303</td>
</tr>
<tr>
<td>Na + (mmol/l)</td>
<td>101</td>
<td>89</td>
<td>85</td>
</tr>
<tr>
<td>K + (mmol/l)</td>
<td>6.8</td>
<td>9.1</td>
<td>12.0</td>
</tr>
<tr>
<td>Creatinine (mmol/l)</td>
<td>0.95</td>
<td>2.5</td>
<td>3.0</td>
</tr>
<tr>
<td>Protein (g/l)</td>
<td>75</td>
<td>2.1</td>
<td>3.5</td>
</tr>
<tr>
<td>Amylase (Car.)</td>
<td>63</td>
<td>77</td>
<td>77</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>8.8</td>
<td>7.1</td>
<td>6.6</td>
</tr>
<tr>
<td>Sediment: erythrocytes</td>
<td>–</td>
<td>++</td>
<td>++</td>
</tr>
</tbody>
</table>

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Fig. 1. Cystogram showing the bladder diverticulum and a contrast media leakage (arrow) into the peritoneal cavity.

117 mmol/l, potassium 3.5 mmol/l. The next day serum creatinine was 123 µmol/l. Cystography was performed, revealing a large diverticulum of the bladder with contrast media leakage into the peritoneal cavity (Figure 1). Bladder endoscopy confirmed inflammation and perforation of the diverticulum. The patient was transferred to the Department of Urology where the ruptured diverticulum was removed. The perioperative period was without complications.

Discussion

Urinary ascites is usually caused by post-traumatic rupture of the bladder, less often by inflammation of the bladder wall, neoplasmatic infiltration, or results from urological interventions or spontaneous perforation of a bladder diverticulum [3,4]. In such cases absorption or rather reverse autodialysis of urine across the peritoneum causes an increase in serum urea and creatinine and profound electrolyte and acid–base disturbances [2–5]. Hyponatraemia, hyperkalaemia and metabolic acidosis can be found as early as after 24 h of urine leak into the peritoneal cavity [6]. Severe hyponatraemia in our patient was not merely a consequence of the above mechanism. The other determinants of hyponatraemia in this case were long-term intravenous administration of solute-free fluids in the absence of a free water excretion by the kidneys, diarrhoea and vomiting. Although hyponatraemia is a salient biochemical feature of uroperitoneum it was not mentioned in a recent review of hyponatraemic conditions [7] most probably because of the rarity of this condition. In our patient, and in most cases of spontaneous bladder rupture, the biochemical pattern of changes in the serum resulting from equilibration of chemicals between serum and urine resembles that of acute renal failure, which may be a source of diagnostic errors. The differential diagnosis is particularly difficult in cases without a recent history of trauma. As proposed in such cases, diagnostic procedures should involve cystography followed by sonography and computer tomography [8]. We also propose that peritoneal fluid should be routinely checked for creatinine concentration, and a low serum to peritoneal fluid ratio of creatinine raises a strong suspicion of the presence of urine in the peritoneum. The case presented shows that in every patient with ascites, hyponatraemia, and biochemical pattern of renal failure, uroperitoneum should be taken into consideration.

References


Received for publication: 25.7.96
Accepted: 31.7.96