
Case Report

Haemoperitoneum originating in renal cyst in a patient with ADPKD not treated by dialysis

Jacques A. Bagon
Néphrologie, Clinique Générale Saint-Jean, Bruxelles, Belgium

Introduction

Intracystic haemorrhage is a frequent feature in patients affected by autosomal dominant polycystic kidney disease (ADPKD). Its clinical manifestations are protean. They include gross haematuria which may occur at any time in 30–50% of cases, abdominal and flank pain (50–60% of cases), renal colic and perirenal haematoma [1]. Haemoperitoneum as a complication of renal cyst in ADPKD has been described only twice (one dubious case) and only in patients on peritoneal dialysis (PD). We present here such a complication affecting an ADPKD patient not treated with dialysis.

Case

In a male patient, aged 35 years, the diagnosis of ADPKD was made in 1987. CAT scan showed a typical polycystic right kidney with congenital absence of left kidney and no liver cyst (Figure 1). The solitary kidney measured 19 × 10 cm. The largest cyst, located at the upper pole, had a diameter of 4 cm.

Over the years the patient became hypertensive. His blood pressure was well controlled with an ACE inhibitor and furosemide, while his creatinine clearance decreased progressively, reaching 19 ml/min in 1998. He had an episode of pyelonephritis in 1987 and two episodes of prostatitis. An echographic control in 1994 showed a 20 cm long kidney. The largest cyst located at the upper pole had then a diameter of 5 cm.

In May 1996 an episode of right lumbar pain without haematuria or fever, associated with signs of a moderate inflammatory syndrome, was interpreted as a renal cyst haemorrhage, and ultrasonography demonstrated two large cysts at the upper pole, measuring 8.5 and 11 cm, respectively, with internal echos. The same clinical picture recurred in April 1998, with fever and an elevated serum CRP. The patient was treated with analgesics and made a good recovery. On CT scan the diameter of the largest upper pole cyst was 14–19 cm, reflecting a volume of about 1.7 l (Figure 2).

In August 1998, he was admitted at the emergency department with a diffusely painful abdomen which was tender and slightly tense. He had no fever but microscopic haematuria. Serum CRP was normal, creatinine was 4 mg/dl and creatinine clearance 20 ml/min. Haemoglobin had decreased from 13.5 to 10.9 g/dl. Ultrasonography demonstrated a giant cyst of the upper renal pole with intracystic echos. A CT scan was performed and showed massive ascites with complete rupture of the upper pole cyst’s wall which floated freely in the ascites (Figure 3). The length of the kidney was almost 24 cm. Hyperdensity was present in some cysts, suggesting haemorrhage. There were a few deep seated liver cysts, less than 1 cm in diameter. Magnetic resonance imaging (MRI) showed signs of haemorrhage in a few renal cysts, no evidence of neoplastic lesion and absence of renal tissue on the left side. Drainage of the ascites, which was grossly bloody, revealed the presence of 380 000/mm³ red cells, with signs of haemolysis, and 18 000/mm³ white cells. Culture was negative for common bacteria and Mycobacterium tuberculosis. The treatment included rinsing of the peritoneal cavity with unwarmed solution of 1.5% glucose and ampicillin-clavulanate was also administered because of the high leucocyte count in the peritoneal aspirate. The peritoneal drainage volume was 31 on day 1 (not including the rinsing volume) and 0.9 l on day 2. It became negligible and clear on day 3, when the drain was removed. Three weeks later blood haemoglobin had risen spontaneously to 12.4 g/dl. None of these intracystic bleeding episodes were secondary to trauma or to unusual physical effort.

In July 1999, intracystic haemorrhage recurred with a decrease of haemoglobin of 3 g. Control CT scan and MRI again showed the haemorrhage to be located in the large upper pole cyst without any solid lesion. The patient made an uneventful recovery.

Discussion

Through a manual and Medline search we were unable to retrieve any case of haemoperitoneum secondary to
renal cyst haemorrhage in ADPKD patients not treated with peritoneal dialysis. This is also the experience of renal units with special interest and wide experience in ADPKD [2–5]. Two cases originating in liver cyst and two in renal cyst have been published in patients on peritoneal dialysis (PD) or haemodialysis (HD), the latter from a liver cyst (Table 1).

Peritoneal dialysis is a ‘window to the peritoneum’ [10] and is thus a ‘sensitizer’ revealing peritoneal pathologies which might otherwise go undiagnosed. One or two ml of blood in 2 l peritoneal effluent will tinge it a distinctive red [11,12]. Haemodialysis-associated heparin therapy is a promoter of otherwise spontaneously occurring haemorrhage [13].

Even in the clinical settings of DP or HD the incidence of haemoperitoneum complicating intrarenal cyst haemorrhage in ADPKD is extremely low. This is not surprising because a retroperitoneal haemorrhage should theoretically not find its way through the peritoneal membrane. A review of the causes of haemoperitoneum by Nace mentioned mostly intraperitoneal pathologies although it does include some extraperitoneal sources [12].

Episodes of haemoperitoneum secondary to spontaneous perirenal haemorrhage (Wunderlich syndrome) have been reported in very few cases (outside the special setting of peritoneal dialysis). They were secondary to adenocarcinoma [14], angiomyolipoma [15,16] and simple cyst [14].

In our patient, MRI excluded a possible remnant left kidney (multicystic dysplastic kidney or multicystic nephroma) as the source of bleeding and did not show malignancy in the solitary right kidney.

The clinical presentation of our case seems to leave no doubt that the haemoperitoneum originated in the ruptured renal cyst. A liver origin is highly improbable,
Table 1. Published cases of haemoperitoneum originating in hepatic or renal cyst in ADPKD patients

<table>
<thead>
<tr>
<th>Source of bleeding</th>
<th>Possibly promoting conditions</th>
<th>Age</th>
<th>Gender</th>
<th>Treatment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatic</td>
<td>PD</td>
<td>40</td>
<td>M</td>
<td>oversewing of cyst</td>
<td>6</td>
</tr>
<tr>
<td>Hepatic</td>
<td>HD</td>
<td>76</td>
<td>F</td>
<td>marsupialization of cyst</td>
<td>7</td>
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<tr>
<td>Renal</td>
<td>previous intracystic haemorrhages, PD</td>
<td>39</td>
<td>M</td>
<td>transfusion→ nephrectomy</td>
<td>8</td>
</tr>
<tr>
<td>Renal</td>
<td>PD</td>
<td>?</td>
<td>F</td>
<td>rinsing of peritoneal cavity</td>
<td>9c</td>
</tr>
<tr>
<td>Renal</td>
<td>previous intracystic haemorrhages, previous APN</td>
<td>46</td>
<td>M</td>
<td></td>
<td>present study</td>
</tr>
</tbody>
</table>

ADPKD, adult dominant polycystic kidney disease; PD, peritoneal dialysis; HD, haemodialysis; APN, acute pyelonephritis. *Abstract, case dubious.

since the hepatic cysts were sparse, small and deep-seated without sign of haemorrhage on the imaging procedures. Haemoperitoneum could be due to a deficient peritoneum [14], of which we have no proof in this case. More probably it should be ascribed to adhesion between the cyst’s wall and the peritoneum, favoured by their anatomical proximity and inflammation secondary to intracystic haemorrhage, with rupture of these adjoining structures as a result of the rising intracystic pressure.

Treatment of such a haemoperitoneum is not standardized. Rinsing the peritoneal cavity with unwarmed solution is a recommended procedure in case of severe persistent haemoperitoneum in patients treated with DP [11]. It is possible that a conservative attitude would have been as efficient.

We conclude that haemoperitoneum, albeit very rare, must be added to the already long list of renal complications of ADPKD, even in patients not treated by HD or PD. Accordingly, in the presence of a haemoperitoneum in an ADPKD patient, not only a hepatic but also a renal cyst must be looked for as a possible source of bleeding.

References

2. Pirson Y, Brussels, Belgium (personal communication, August 1998)

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