Late ‘spontaneous’ kidney graft decapsulation with fluid collection: lymphocele or transudate?

Christiane Mousson, Gilbert Zanetta, Yves Tanter, Jean-Pierre Cercueil, Jean-Marc Chalopin, Serge Briet, Gérard Rifle

Departments of 1Nephrology–Intensive Care, 2Radiology and 3Urology, University Hospital, Dijon, France

Key words: acute renal failure; perirenal collection; transplantation

Introduction

The common aetiologies of perirenal fluid collections occurring after kidney transplantation include urinomas, haematomas, abscesses, and lymphoceles [1]. These complications generally occur early after transplantation and their causes are well defined. However, another possible cause of a lymphocele-like fluid collection may be observed: fluid leakage from kidney surface after decapsulation. We report two cases of such late subcapsular collections occurring in kidney transplant recipients.

Case reports

Case 1. A 31-year-old Caucasian male had undergone cadaver kidney transplantation 4 years before because of end-stage renal disease caused by glomerulonephritis. No surgical problem occurred either during organ harvesting or kidney transplantation. Renal function returned to normal values within a few days post-transplantation and remained in the normal range (serum creatinine = 1.3 mg/dl), without proteinuria throughout this time. The patient was admitted for evaluation of swelling over his graft which gradually increased (without any trauma) and decline of renal function (S-creatinine = 2.5 mg/dl) mimicking acute rejection. Immunosuppressive therapy consists of azathioprine (100 mg/day) and prednisone (10 mg/day). In fact, ultrasonographic examination showed a large amount of fluid around the kidney (over 1 litre). After puncture, examination of the fluid, which was yellow-coloured, showed that total protein was 2 g/l, creatinine level was 1.8 mg/dl, and cholesterol was 0.3 g/l.

White blood cell count was below 100/ml and fluid cultures were negative. Swelling reappeared twice after puncture and S-creatinine climbed each time to 2.5 mg/dl. Surgical intervention was carried out, firstly to explore this collection, and secondly to drain the collection. Surgical exploration showed that the collection was strictly subcapsular. Marsupialization, i.e. creation of a peritoneal window adjacent to the transplanted kidney, was therefore carried out to allow drainage of the collection into the peritoneum. Transplant biopsy showed a normal kidney on microscopic examination. Eleven years later, there has been no recurrence and renal function remains unchanged (S-creatinine = 1.3 mg/dl).

Case 2. A 30-year-old Caucasian male reached end-stage renal failure due to type I membranoproliferative glomerulonephritis. A first cadaver kidney transplantation was performed in the right iliac fossa, but the graft was lost by chronic rejection. A second cadaver kidney transplantation was performed 7 years later in the left iliac fossa, without surgical problem either during organ harvesting or kidney transplantation. After an early acute rejection episode, S-creatinine stabilized at 1.5 mg/dl. Immunosuppressive regimen comprised cyclosporin and prednisone. Five years after transplantation, a large swelling developed in the left iliac fossa. A few weeks earlier, the patient had fallen from a height of 2 metres. Renal function remained unchanged, without proteinuria. Ultrasonographic examination showed a perirenal collection which recurred in a few days after a puncture (volume = 1.2 l). Examination of the fluid, which was yellow-coloured, showed that total protein was 2.8 g/dl; cholesterol was 0.17 g/l, triglycerides were 0.02 g/l, creatinine level was 1.8 mg/dl and white blood cells were rare with negative fluid cultures. The volume of the collection varied from 0.6 to 2 litres, but after 4 punctures, the volume of the fluid tapered. Magnetic resonance imaging showed a subcapsular collection which took up gadolinium (Figure 1) and clearly confirmed the decapsulation. No lymphangiography was performed. It was decided not to use marsupialization.
Late ‘spontaneous’ kidney graft decapsulation with fluid collection

because the graft was functioning perfectly well and the patient did not experience any discomfort despite presence of the collection. Two years later, renal function remains stable and the graft is surrounded by a 500 ml collection without any clinical symptom.

Discussion

Perirenal collections after kidney transplantation are detected by routine ultrasound follow-up in 20–50% of patients [1]. However, about 50% of these collections are estimated to be less than 50 ml and of minor clinical importance. The most common causes are lymphoceles, haematomas, abscesses, and urine collections. Examination of the aspirate fluid is an essential method for specifying the kind of perirenal collection. Perirenal haematomas occur during the postoperative period or after traumatisms, and diagnosis is easily made with fluid composition. Abscesses are also easily identified by aspirate WBC composition and bacterial cultures. Analysis of fluid composition is also helpful in identifying urine leakage, since higher creatinine and potassium concentrations and lower sodium concentrations are detected in urine than in lymphocele fluid. Indeed, urine fistulae may be responsible for perirenal collection, particularly early after surgery by ureteral or bladder leak.

Lymphoceles seem the most frequent collections, because they develop in about 20% of patients but only 10% are symptomatic [1]. Two possible sources are implicated: iliac lymphatic vessel dissection during surgery or disrupted lymphatics in the renal hilum during harvesting. These factors might therefore explain the early appearance of lymphoceles after grafting. Moreover, some circumstances increase lymphatic flow as acute rejection, which could explain the increase of the collection volume during rejection crisis [2]. Lymphocele fluid studies reveal that the concentrations of creatinine, urea nitrogen, sodium, potassium are not significantly different from serum values. But total protein and cholesterol levels are significantly lower in lymphatic fluid than they are in serum [2].

In our two cases, the biochemical composition of the collections does not clearly differ from those of a lymphatic fluid as in the five other similar cases previously reported in the literature (Table 1). Sodium, potassium, cholesterol, protein and creatinine levels are not different from plasma or lymph. In patient 2, protein level seems relatively high (2.8 g/dl), but for Braun et al. [2] protein levels in lymphoceles are 2.2 ± 1.1 g/dl. Yet the cases reported in the literature and our own two patients all reveal some unusual characteristics for lymphoceles:

late occurrence (4 and 7 years after transplantation), whereas lymphoceles are generally detected between 1 and 34 weeks (mean = 7.8) after surgery [2];
subcapsular site of the collection, demonstrated by CT scan or MRI (as in our second case) or by surgical exploration ([3,4], our first case).

Moreover, a common point is found in all these subcapsular collections: the large amounts of collected fluid and very rapid recurrence after puncture as in our first case and in the two cases reported by Koeue et al. [3]. In the latter two cases, despite several attempts to control fluid leakage, grafts functioning perfectly well had to be removed because of excessive fluid loss. Large amounts of fluid can cause discomfort and/or renal failure. Large volume collection is not a specific characteristic because, in a few cases, large lymphoceles may be observed.

These features, in line with the five previously reported cases (Table 1), suggest that these late subcapsular collections should be distinguished from usual lymphoceles. We did not include in Table 1 the case reported by Sollinger et al. [5] 8 years after transplantation, because of the presence of very particular lesions, multiple cysts on the renal surface, dilated lymphatic channels in the cortex. In the seven cases of decapsulation with subcapsular collections, renal biopsies were normal and there is no mention of cysts or dilated lymphatics vessels. By virtue of these anatomical lesions, this case seems to us very different from other late subcapsular collections because of these anatomical lesions.

The physiopathology of such collections remains unclear. Fluid composition could suggest transudation from the kidney cortex after decapsulation or lymphocele by rupture of the subcapsular lymphatics of the graft. An indirect argument for this hypothesis is the success of the treatment in the case of Tiggeler et al. [4]: the formation of a new capsule by infrared contact coagulation led to disappearance of the leak-
Table 1. Main characteristics and outcome of the seven cases of late subcapsular collections after kidney transplantation

<table>
<thead>
<tr>
<th>Case-report authors</th>
<th>Time/grafting (years)</th>
<th>Collection volume</th>
<th>Fluid composition</th>
<th>Treatment</th>
<th>Results/follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Koene (1979) [3]</td>
<td>5</td>
<td>Range 4000–12000 ml</td>
<td>Na = 144 mEq/l; K = 4 mEq/l</td>
<td>Attempt of formation of a new capsule with butylcyanoacrylate or lyophilized human dura</td>
<td>Nephrectomy 3 months later</td>
</tr>
<tr>
<td>Koene (1979) [3]</td>
<td>3</td>
<td>More than 900 ml</td>
<td>Na = 135 mEq/l; K = 4 mEq/l</td>
<td>Le Veen shunt</td>
<td>Nephrectomy 2 months later</td>
</tr>
<tr>
<td>Nghiem (1982) [6]</td>
<td>7</td>
<td>Mean 2000 ml</td>
<td>Na = 134 mEq/l; K = 4 mEq/l</td>
<td>Marsupialization</td>
<td>Success (Follow-up unknown)</td>
</tr>
<tr>
<td>Tiggeler (1986) [4]</td>
<td>3</td>
<td>Mean 2440 ml (1440–3600 ml)</td>
<td>Na = 139 mEq/l; K = 4 mEq/l; Creatinine = 0.9 mg/dl</td>
<td>Formation of a new capsule by infrared contact coagulation</td>
<td>Success/9 months</td>
</tr>
<tr>
<td>Brown (1990) [7]</td>
<td>4</td>
<td>'Massive ascites'</td>
<td>Na = 139 mEq/l; K = 3.7 mEq/l; Proteins = 0.5 g/dl; Cholesterol = 0.3 g/l</td>
<td>'Diuretics'</td>
<td>Success/6 years</td>
</tr>
<tr>
<td>Personal case 1</td>
<td>4</td>
<td>More than 1000 ml</td>
<td>Na = 139 mEq/l; K = 3.8 mEq/l</td>
<td>Marsupialization</td>
<td>Success/11 years</td>
</tr>
<tr>
<td>Personal case 2</td>
<td>5</td>
<td>Range 600–2000 ml</td>
<td>Na = 139 mEq/l; K = 3.8 mEq/l; Protein = 2.8 g/dl; Cholesterol = 0.17 g/l; Creatinine = 1.3 mg/dl</td>
<td>Punctures</td>
<td>Success/2 years</td>
</tr>
</tbody>
</table>
Late ‘spontaneous’ kidney graft decapsulation with fluid collection

A trauma could be a potent predisposing factor of decapsulation in our second case but the decapsulation seemed spontaneous in the literature cases [3,4,7]. Data about frequency (0.4% in our series) and causes of late decapsulation are lacking, so it seems difficult to draw definite physiopathological conclusions. However, Nghiem et al. [6] failed to demonstrate the presence of the radiotracer in the fluid after a 99mTc sulphur colloid lymphangioscan of the ipsilateral foot, suggesting that the fluid did not originate from iliac lymphatics.

Another interesting point for discussion is the choice of therapy in such cases. The decision to treat or to withhold treatment is based largely on patient clinical presentation. In our second case, the collection was asymptomatic and after some punctures, fluid production gradually decreased and no further treatment was needed. But other treatments have been used in the literature. Brown et al. [7] obtained a progressive reduction of the collection volume by diuretics and the outcome was favourable. Conversely, the accumulation of fluid with compression and deterioration of renal function, as in our first case, may need more active therapy. In comparison with lymphoceles, one should also consider such therapeutic methods as prolonged duration of drainage or instillation of sclerosing agents (tetracyle sulphate, povidone iodine) [8]. But we cannot exclude a deleterious effect of cortical instillation of sclerosing agents in such subcapsular collections. Koene et al. [3] failed to create another capsule with butylcyanacrylate or lyophilized human dura in one case. However, Tiggeler et al. [4] successfully opted for another approach: the formation of a new capsule by infrared contact coagulation of the kidney surface, with precise and careful technical conditions. He then covered the kidney with a slip of omentum and the fluid leakage gradually decreased. Nevertheless, we prefer to propose treatment by marsupialization, which seems easier from a technical point of view. Marsupialization consists of an internal drainage, which allows free access of fluid from the peritransplant space into the peritoneal cavity, where it is resorbed.

Marsupialization successfully treated subcapsular collection in the patient of Nghiem et al. [6] and in one personal case, as well as in treating lymphoceles [9]. In these two cases, marsupialization, because of large collections, seems to have eliminated the need for graft nephrectomy, as performed by Koene et al. [3] whereas creatinine clearance was in the normal range. This would therefore seem to be a very interesting result.

In conclusion, these collections seem to us to have distinctive characters which set them apart from usual lymphoceles: late occurrence, subcapsular localization, and large volumes of lymph-like fluid. This complication seems rare but we have to bear in mind the fact that marsupialization appears to resolve such large collections and to eliminate the need to resort to functional graft nephrectomy.

References


Received for publication: 3.11.97
Accepted in revised form: 22.12.97