Late onset-lymphocele after renal transplantation

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ABSTRACT

Symptomatic lymphoceles that occur early after renal transplantation are usually managed by surgical marsupialization with drainage into the peritoneal cavity. Late-onset lymphocele, however, is more uncommon and is etiologically different from the former type. We report a patient, who was detected to have a lymphocele 11 years after renal transplantation. The treatment of which presented one of the biggest challenges in the management of postrenal transplant complications.

Key words: Denver shunt, Lymphocele, Postrenal transplant

CASE REPORT

A 48-year-old man presented in 1985 with uremia and was confirmed to have bilaterally contracted kidneys by ultrasonogram. He was started on hemodialysis for 9 months before he received a one haplotype matched living-related kidney transplant from his sister. The procedure was uneventful though the best serum creatinine of the patient hovered between 200 µmol/L to 250 µmol/L. The immuno-suppressive regime included prednisolone and azathioprine and the patient was HBsAg carrier. He remained well till July 1996 when a routine graft kidney ultrasonogram showed a subcapsular collection at the upper pole. Computed tomogram (CT) confirmed similar findings but the nature of the interior content was unknown (Fig. 1). Ultrasound-guided aspiration of the collection showed the following biochemical and cytological findings: urea 15 µmol/L (serum: 19.9 µmol/L), protein 9 g/L (serum: 83 g/L), cell count 0.75 x 109/L (neutrophil 75%, lymphocyte 7%, macrophage 18%). Analysis for chyle and bacterial culture were negative. Late-onset post-transplant lymphocele was diagnosed. The collection quickly recurred after the aspiration and laparoscopic fenestration of the lymphocele wall was performed. A lymphangiogram showed that the lower limb lymphatics were normal and there was no lymphatic leakage in the retroperitoneum and the pelvis. The thoracic duct was also unremarkable. In view of the intractable ascites, regular abdominal tapping was required for symptomatic relief. An upper endoscopy, performed for suspected gastrointestinal bleeding, showed evidence of grade 1 esophageal varies and it was surmised that portal hypertension may be contributory to the ascites. After consultation with the hepatologists, transjugular intrahepatic porto-systemic shunt (TIPS) was planned. The procedure was subsequently abandoned because the hepatic venous pressure and hepatic vein wedge pressure were both 6 mmHg only. Transjugular liver biopsy revealed near normal histology. An albumin scintiscan also failed to locate the exact source of the leakage. At the same time, the patient developed pleural effusion and the content of the pleural aspirate closely resembled that of lymphocele.
in the abdomen. After 3 months of repeated abdominal tapping, an abdominoatrial shunt was considered for symptomatic relief and conservation of body proteins. In September 1998, a Denver\textsuperscript{®} shunt was inserted (Fig. 2,3). Intraoperatively, 15 L of clear peritoneal fluid was released. The graft kidney, which was located in the left iliac fossa, appeared edematous and there was continuous oozing of clear fluid from its surface. The procedure was smooth but it was later complicated by deep vein thrombosis and pulmonary embolism 1 week postoperatively. He was started on warfarin and the ascites improved after the operation. Though chest x-ray showed slight improvement in the effusion, it never completely subsided. Two months afterward, the rate of fluid reaccumulation intensified again. Abdominal and pleural tapping was required at a frequency of once to twice per week. A "shuntogram" showed contrast hold-up at superior vena cava (SVC), suggestive of either a SVC thrombosis or stenosis. Abdominopleural communication was also documented by rapid diffusion of radio-labeled tracer from the abdominal cavity to the right hemithorax. The source of the ascitic fluid was still difficult to define accurately. To relieve the patient symptomatically, ascitic fluid ultrafiltration was attempted. Two Tenckhoff catheters were inserted so as to create a continuous circuit for ascitic fluid ultrafiltration using a high flux hemodialyzer with an area of 1.3 m\textsuperscript{2}. The treatment was regarded as a temporizing measure to relieve the ascites and delay the need for dialysis.

After 3 months of intermittent treatment, there was little
improvement in the effusion and the frequency of pleural tapping increased to almost daily. In view of the risk of repeated pleural tapping, nephrectomy was suggested. The patient underwent a laparotomy in September 1999. Intraoperatively, the graft kidney was found to be in good shape with minimal oozing. The peritoneal window was wide open. The relatively preserved architecture of the graft kidney precluded an immediate nephrectomy and the peritoneal window was sutured. His renal function, however, deteriorated progressively after the operation and peritoneal dialysis was started in early October. His condition remained precarious, with frequent need for chest tapping. In the succeeding weeks, his liver function also showed progressive derangement with incremental serum transaminases. The serum ammonia also rose rapidly with clouding of mental state, eventually necessitating ventilatory support. Multi-organ failure superseded promptly with renal, respiratory and hepatic decompensation. His condition continued to deteriorate and finally succumbed as a result of multiorgan failure and fulminant sepsis 3 weeks after resuming dialysis. Post-mortem examination revealed massive hepatic necrosis. The gross and microscopic findings in the liver were consistent with postnecrotic scarring after massive hepatic necrosis. The latter was likely attributable to fulminant hepatitis B reactivation as suggested by the extensive cytoplasmic immunostaining for HBcAg. Histological examination of the transplanted kidney only showed chronic transplant nephropathy with marked tubular atrophy and interstitial fibrosis. Abnormal lymphatic channels could not be delineated in the postmortem specimens.

**DISCUSSION**

Lymphocele formation is not an uncommon occurrence after renal transplantation. The reported incidence varies between 2% to 18% (1,2). With the availability of routine ultrason sound, the overall incidence was up to 50% (3). Most cases, however, are subclinical and resolve spontaneously (4). The biochemical characteristics of lymphocele fluid are similar to that of serum except for a lower protein content and cell count. The lymph is thought to come from unligated lymphatic vessels of the recipients during the early postoperative period. Most lymphoceles measure less than 3 cm in diameter on ultrasonogram and contain less than 100 mL lymph fluid. Larger collections usually occur between 2 weeks and 6 months and may result in deterioration in renal function and urine output due to ureteric compression.

The occurrence of a lymphocele several years after transplantation is an extremely rare event, and by itself, suggests an unusual etiology. In six retrievable cases in the literature, the lymphocele content was thought to originate from the lymphatics of the transplanted kidney (5-7). The cardinal feature was diffuse oozing of lymph fluid from the kidney surface. The explanation would require a brief review of the anatomy of the lymphatic drainage in kidneys. In normal native kidneys, lymphatic drainage occurs via hilar lymphatic vessels and to a lesser extent via capsular channels, where they proceed through six to eight channels, to the cisterna chyli. In the transplanted kidney, however, the pathway is less clear. It is postulated that in late-onset lymphocele, an obstruction to the normal hilar flow of lymph produced a retrograde flow toward the capsular surface. This obstruction was considered to be due to fibrosis of perihilar lymph vessels, possibly as a result of chronic rejection process (8). Retrograde flow of lymph could result in accumulation of fluid within the kidney and between the kidney surface and capsule, if present. Internal drainage results in shifting of lymph into the abdominal cavity. Abnormalities in the thoracic lymphatic drainage, for example due to previous pulmonary tuberculosis, will result in gross ascites and pleural effusion (6).

In one case report (8), a 40-year-old man developed spontaneous lymphocele 7 years after cadaveric renal transplantation. Marsupialization of the lymphocele into the peritoneal cavity resulted in persistent right pleural effusion and intractable ascites. Abdominoatrial shunting (Denver® shunt) resulted in symptomatic relief. In the current case, despite the absence of a definite history of pulmonary tuberculosis, obstruction to thoracic drainage after un-noticed subclinical infection may still be possible. After consultation with the surgeon, an abdominoatrial shunt was inserted which resulted in prompt symptomatic relief. It must, however, be admitted that further measures to definitively deal with the
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lymphatic drainage have to be sought in view of the limited lifespan of the shunt. The optimal management option was still unanswered and required imaginative and varied treatment. In the report by Sollinger (6), attempt was made to treat the patient by excising the perirenal tissue in the hope that the weeping surface would granulate and obliterate the lymphatic channels. This failed, and the problem was solved by transplant nephrectomy.

Continuous ultrafiltration of ascitic fluid has been reported to be useful in symptomatic relief in patients with resistant ascites (9). The procedure, however, did not rectify the underlying lymphatic abnormalities and the failure to relieve the pleural effusion, in effect, nullified the benefit of the treatment. The deterioration in the graft function was attributable to a combination of factors including the hemodynamic stress from the gross ascites, repeated para- and pleurocentesis and systemic sepsis. The pressure effect of the ascites and the constant loss of nutrients through tapping further jeopardized the nutritional well-being of the patient. The acute flare of hepatitis B infection cannot only be linked to the generalized immuno-compromised state of the patient, which triggered a series of fatal complications.

In conclusion, the case presented stresses the important difference between early and late onset postrenal transplant lymphocele formation. The possibilities of ascites and pleural effusion after the drainage operation obligate a detailed assessment of the lymphatic drainage before the procedure. When definitive treatment to deal with the repeated fluid recollection fails, a graft nephrectomy may be the only option to relieve the lymphatic recollection. This late complication of renal transplantation may become more frequent as graft survival increase beyond 5 years. If this is the case, difficult management decisions, as illustrated by this case, may be seen more frequently in the future.

REFERENCES