Surgical Correction of Nephrogenic Ascites in a Renal Transplant Recipient

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Abstract

The unusual development of massive ascites, 3 years after renal transplant, caused by undefined, innate renal allograft pathology is described. Challenges of surgical correction of this problem, allowing for salvage of the allograft, are reviewed.

Key words: *Nephrogenic ascites, Renal transplant, Renal allograft pathology, Ascitic fluid*

Introduction

The occurrence of ascites after renal allograft transplant in an otherwise healthy individual generally raises concern for an ominous process in the recipient, and oncologic, hepatic, or cardiac pathology is commonly found. Analysis of the ascitic fluid can often shed light on the possible cause in most individuals; however, when laboratory studies are unrevealing, other diagnostic tests may be needed. Despite multiple diagnostic tests, an exploratory laparotomy was necessary to identify the cause of tense ascites in our renal transplant recipient, 80 months after renal transplant.

Case Report

A 24-year-old woman with type 1 diabetes for 22 years underwent a deceased donor, combined kidney and pancreas transplant in August 2002, with intraperitoneal placement of organs. She had good dual allograft function for 39 months, after which she developed polyuria and polydipsia. Evaluation revealed a nonfunctioning pancreas allograft and the patient returned to insulin use. Over the next 24 months she had poor glycemic control with a gradual increase in serum creatinine. A percutaneous renal allograft biopsy performed in February 2007 revealed changes consistent with early diabetic glomerulosclerosis. She was evaluated for a second pancreas allograft and subsequently underwent a successful pancreatic allograft retransplant in October 2007. She showed chronic renal allograft dysfunction with a serum creatinine of 141.4 μmol/L (1.6 mg/dL) at the time of surgery.

Four months later, the patient presented with abdominal distension, hypotension, and anemia. The results of admission laboratory workup showed creatinine 137 μmol/L (1.55 mg/dL), glucose 5.2 mmol/L (94 mg/dL), and a normal liver profile. Diagnostic paracentesis revealed hazy fluid with 7279 red cells and 11 white cells per high-power field with a differential of 14 segmented neutrophils, 46 lymphocytes, and 39 monocytes. Cytologic evaluation revealed no malignant cells. The results of biochemical analyses of fluid showed an amylase level of 0.10 μkat/L (6 U/L), protein 14 g/L (1.4 g/dL), creatinine 159.12 μmol/L (1.8 mg/dL), lactate dehydrogenase isoenzymes 0.58 μkat/L (35 U/L), and triglycerides 0 mmol/L (0 mg/dL). The serum-ascites albumin gradient was elevated at 20 g/L (2.0 g/dL) consistent with a transudate. Results of culture, smears, and Gram stain were all negative for pathogens. The results of a computed tomography scan of the abdomen were remarkable only for ascites.

The patient required frequent volume resuscitation owing to hypotension from the recurrent massive ascites and was maintained on fludrocortisone and ProAmatine (midodrine hydrochloride) to support a systolic blood

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pressure >100 mm Hg. She underwent a right hepatic venogram, with measurements of mean free right hepatic and hepatic wedge venous pressures, which were 3 mm Hg and 4 mm Hg. The corrected systolic sinusoidal gradient was 2 mm Hg. A transjugular hepatic biopsy was without pathology. A two-dimensional transthoracic echocardiogram revealed a left ventricular ejection fraction of 62% with no other abnormalities.

Over the ensuing weeks, she continued to reaccumulate ascitic fluid requiring frequent large volume paracentesis, with removal of over 4 liters at each sitting, with multiple, repeated analyses of the fluid remaining unchanged. She began to develop recurrent ventral incisional hernias requiring surgical repair with drainage of clear fluid during surgery. She was anorexic and lean body mass began to slowly decrease.

With no clear cause for her ascites uncovered, fluid leak from lymphatic disruption occurring during the course of her second pancreatic allograft transplant procedure was considered a possibility. She was taken to the operating room for abdominal exploration with preparations for a right leg methylene blue lymphangiogram. During surgery, no fluid was identified emanating from the pancreas transplant or the right lower abdomen; however, a copious amount of fluid was draining from the left lower quadrant of the abdomen where her renal transplant was positioned. Upon close inspection of the renal allograft, multiple spongiform exophytic masses were noted on the surface of the kidney and were weeping fluid (Figure 1). A biopsy of 1 of the masses revealed moderate to marked interstitial edema with focal fibrosis; viable and intact glomeruli and tubules, and unremarkable vessels on pathological evaluation (Figure 2). Abdominal drains were placed with daily collection of 300 mL of fluid over the next several weeks. The drainage then began to diminish slightly and abruptly ceased though her ascites began to reaccumulate.

During removal of the drain in the office, a tongue of the omentum was extracted through the drain site requiring surgical removal of the drain in the operating room where the ascites also was evacuated. With mobilization of the allograft and removal of an adhesed fallopian tube to the lower pole of the allograft, a lower pole artery was injured requiring repair. As the majority of the surface of the allograft was being cauterized, the exophytic lesions

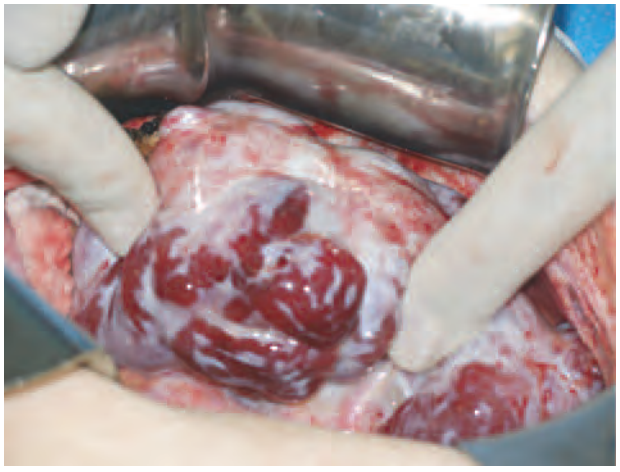

Figure 1. Exophytic spongiform lesions covering the renal allograft as a source of the ascitic fluid.

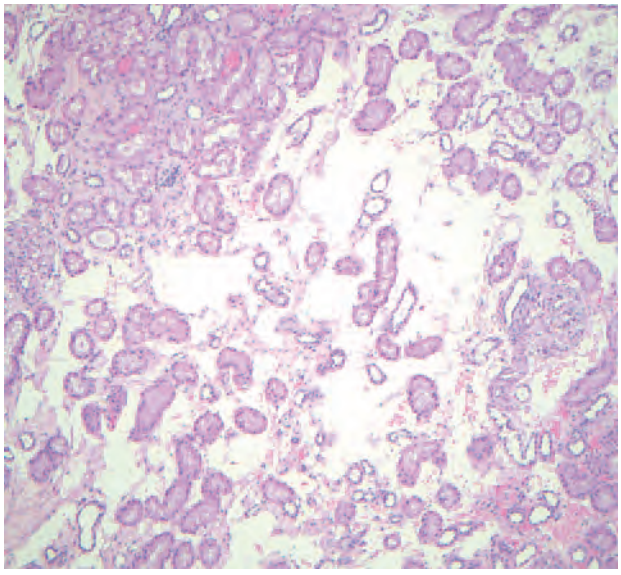

Figure 2. Microscopic evaluation of the exophytic allograft lesions revealing chronic fibrosis with significant interstitial edema.

began to involute, resulting in a normal surface contour of the kidney. The left side of the colon was then placed over the allograft, and a drain was placed beneath the colon adjacent to the allograft. Over the next 2 months, drainage progressively diminished and the drain was subsequently removed. The ascites was then reasonably well controlled with diuretic use for 6 months and then began to reaccumulate at a rapid pace necessitating weekly large volume paracentesis.

The patient was offered allograft nephrectomy but she requested another attempt to salvage her allograft. She was returned to the operating room in June 2010 and again underwent cauterization of the surface of the allograft. The lateral abdominal peritoneum was then elevated and the allograft

placed beneath the peritoneum. A drain was placed exiting from the retroperitoneum, not traversing the peritoneal cavity as had been the case previously. The peritoneum was then sewn to the edge of the left colon, completely retroperitonealizing the allograft. The drain output approximated a liter of fluid daily immediately after surgery. By 2 months, there was minimal fluid output from the drain and her abdomen was not distended. The drain was removed at 8 weeks with no diuretic requirement and no subsequent reaccumulation of abdominal fluid over 6 months. Follow-up ultrasound of her renal allograft revealed no perinephric fluid (Figure 3). She continues to have chronic renal allograft dysfunction with a current serum creatinine of 247.5 μmol/L (2.8 mg/dL).

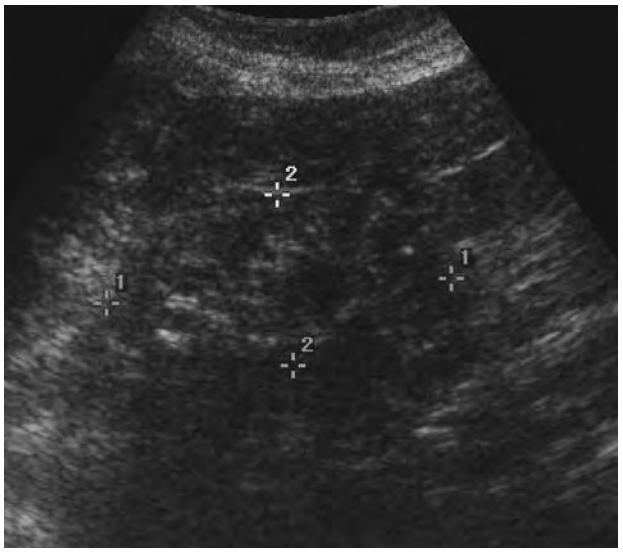

Figure 3. Renal allograft ultrasound showing no fluid accumulation 4 months after drain removal.

Discussion

The term *nephrogenic ascites* was coined to describe a syndrome associated with refractory ascites occurring in patients with end-stage renal disease in whom infectious, malignant, hepatic, and cardiac causes have been excluded.[1] The source of fluid is unclear in such cases and has not been shown to be directly derived from the kidneys. The source of the ascites in our patient was clearly the renal allograft. Although the patient had renal allograft dysfunction, end-stage renal disease was not present. Renal transplant has been used to correct preexisting nephrogenic ascites.[1, 2] Interestingly, ascites can develop after transplant in the absence of this preexisting condition.[3] Ascites may accumulate with or without renal allograft dysfunction.

De novo ascites may develop after renal transplant from multiple causes. Transudation from a renal allograft with massive ascites accumulation was first described by Clark in 1976.[3] Treatment in this case consisted of placement of abdominal and perirenal drains accompanied by fluid resuscitation. Long-term follow-up was not available, as the patient died of a pulmonary embolus within 5 days. Abdominal accumulation of fluid after renal transplant in 1 case was felt to be the result of volume expansion during allograft rejection, causing cardiac dysfunction and hepatic congestion in 1 case report.[4] No hepatic, oncologic, or cardiac diseases were present in our patient.

Allograft urine leak as a cause for the ascites was excluded by analysis of the ascitic fluid. Development of ascites owing to a small urine leak as a complication of intraperitoneal drainage of a lymphocele has been described.[5] Analysis of the ascitic fluid in our patient suggests a preponderance of the fluid was lymph. The leak and ascites resolved after placement of a percutaneous nephrostomy tube. Spontaneous drainage of a subcapsular lymphocele occurred, requiring retroperitonealization of the kidney and drainage to eliminate the ascitic fluid accumulation.[6] In addition to causing intravascular volume contraction, cachexia, and renal allograft dysfunction, the ascitic fluid can be prone to spontaneous bacterial peritonitis.[7]

Unlike patients with renal failure, the ascitic fluid after transplant was found to be weeping directly from the surface of the renal allograft. This became acutely manifest 80 months after renal transplant, 12 months after a percutaneous renal allograft biopsy, and 4 months after pancreatic allograft retransplant. Treatment options for our patient seemed limited. She declined allograft nephrectomy knowing that the waiting time for another allograft may be prolonged with the possible development of sensitizing antibodies further extending her wait.

Peritoneovenous shunting has been used in patients with end-stage renal disease with nephrogenic ascites as well as in a transplant recipient with spontaneous decapsulation of his allograft 5 years after transplant.[8, 9] Attempts to control an intractable lymphocele using multiple surgical procedures was eventually achieved with

use of a Denver shunt (Care Fusion, Waukegan, IL) .[10] However, peritoneovenous shunts are prone to blockage and disseminated intravascular coagulation and may pose an infectious risk in the immunocompromised patient.

Retroperitonealization of a renal allograft was used successfully by Kulkarni.[6] This was felt to be the only reasonable means to attempt control of the fluid accumulation. Cauterization of the surface of the allograft was done with the intention of promoting a fibrotic reaction around the allograft; however, complete retroperitonealization was necessary to achieve the desired result. One can only speculate on the cause of this unusual renal allograft pathology.

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