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Late Spontaneous Rupture in a Homografted Kidney

A Case Report

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An unusual case of late spontaneous parenchymal perforation of an allogenic kidney transplant was successfully treated by nephrostomy drainage.

Spontaneous parenchymal rupture in renal homografts causing hemorrhage is not rare. The incidence is about 3% to 6%.[1] It happens usually during an early acute rejection period.[1-6]

Both pelvic and ureteral necrosis, common in both transplant and nontransplant patients, are related to infection, rejection, trauma, and technical problems.[2,7-12]

This is a case report of an unusual delayed spontaneous parenchymal rupture in the lower pole of a homografted kidney that caused no hemorrhage. It appeared to be due to focal necrosis causing localized abscess formation. The complication was successfully treated by nephrostomy drainage.

REPORT OF A CASE

The patient is a white woman born in 1942. In 1963 she had several episodes of urinary tract infections. At that time she was pregnant and had signs and symptoms of mild toxemia of pregnancy. In 1966 a biopsy examination of the kidney showed chronic glomerulonephritis. In February 1970, she was started on regular hemodialysis therapy twice a week. In June 1973, she had preformed cytotoxic antibodies against 58% of the lymphocytes from a panel of voluntary blood donors.

On Nov 2, 1973, she received a cadaveric kidney transplant (D match; mixed lymphocyte culture stimulation index, 1.37%; negative cross match). The organ had a single renal artery by arteriography (Fig 1). Simultaneously, she underwent bilateral nephrectomy and splenectomy. A regimen of cyclophosphamide (Cytoxan), azathioprine (Imuran), prednisone, and antilymphocyte globulin were started on the day of operation. The transplanted kidney functioned satisfactorily (on the fifth day the blood urea nitrogen [BUN] value was 38 mg/100 ml; creatinine value, 1.7 mg/100 ml). A summary of the clinical course is shown in Fig 2. Two intravenous pyelograms done on the seventh and 27th postoperative days were normal. Postoperatively, a urine culture showed substantial growth of *Escherichia coli*. She was given nitrofurantoin (Furadantin) for two weeks. Subsequent urine cultures were negative. The patient was discharged in satisfactory condition with excellent renal function 28 days after transplantation.

Four days after discharge she was readmitted because of rejection, and prednisone dosage was raised to 200 mg and then de-

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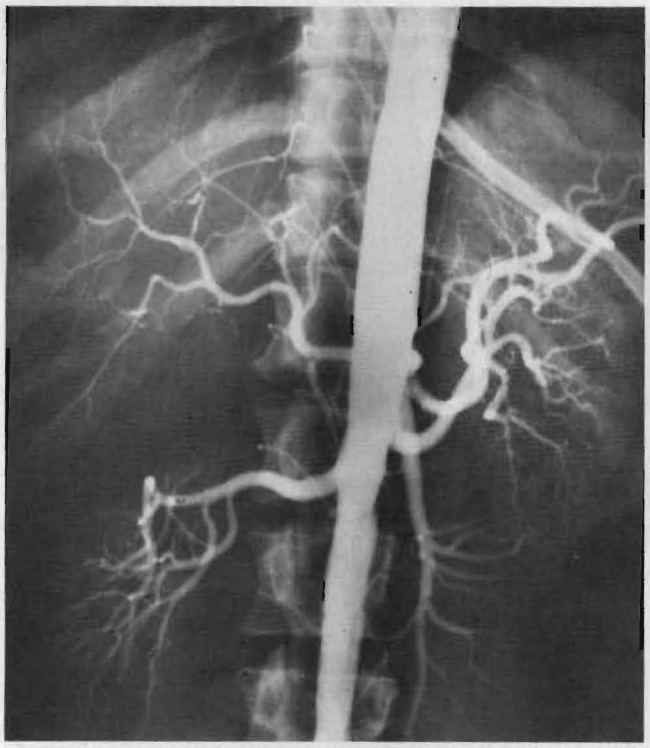

Fig 1.—Angiogram of donor kidney; no anatomical abnormality.

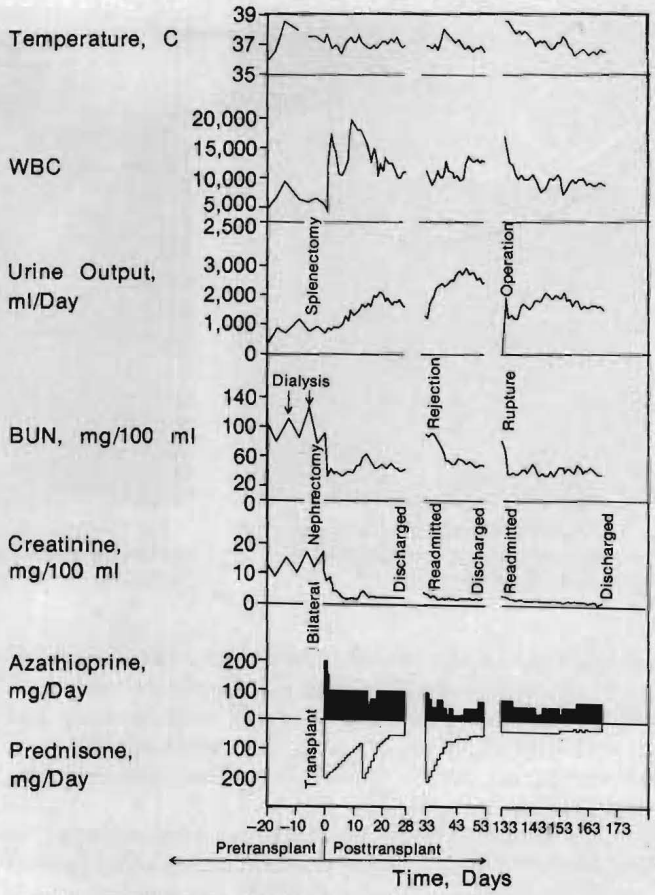

Fig 2.—Clinical course of patient.

creased by 20 mg per day. She developed steroid-induced diabetes (fasting blood glucose levels over 500 mg/100 ml), which was treated first with insulin injection and later with tolbutamide (Orinase). The kidney recovered satisfactorily.

She was discharged with a BUN value of 44 mg/100 ml, a creatinine value of 1.3 mg/100 ml, and a fasting blood glucose level of 138 mg/100 ml. She was well until she again returned to the hospital with severe acute abdominal pain. Her temperature was 37.5 C (99.5 F); blood pressure, 140/100 mm Hg; pulse, 84 beats per minute. The lower part of her abdomen was severely tender, with mild guarding of the right lower quadrant just over the graft and the suprapubic region. The graft was not swollen. Bowel sounds were normal. Rectal examination disclosed minimal tenderness of the right rectal shelf; no masses were felt.

Laboratory studies disclosed the following values: white blood cell count (WBC), 16,500/cu mm; hematocrit reading, 39%; electrolytes, normal; fasting blood glucose, 155 mg/100 ml; BUN, 81 mg/100 ml; creatinine, 2.6 mg/100 ml; amylase, 114 units (normal). An electrocardiogram and chest roentgenogram were normal. An intravenous pyelogram showed mild hydronephrosis. There was extravasation in the area of the lower pole of the graft. The renal shadow was not enlarged (Fig 3).

Hospital Course.—On admission, a Foley catheter was inserted to the bladder. Only 10 ml of urine was obtained. In view of the roentgenographic findings, graft exploration was done under general anesthesia. The graft was found to be of normal size and color. There was a perforation with localized abscess formation in the lower pole. Urine appeared through the perforation. The vascular anastomoses were normal. Gram stain from the abscess material showed numerous gram-negative rods. Pockets of serous-containing fluid were seen to compress the distal part of the ureter. Otherwise the ureter looked normal. An 8N Foley catheter was inserted through the perforated area to a lower major calix of the kidney. An intraoperative nephrostomogram verified proper positioning of the nephrostomy tube and absence of leakage or ureteral obstruction (Fig 4). The wound was irrigated with antibiotic solution. Drains were placed around the kidney. Ampicillin and karamycin sulfate therapy was started and later replaced by clindamycin (Cleocin). The bacteria seen on Gram stain failed to grow in cultures and an anaerobic infection was suspected.

Postoperative intravenous pyelogram and nephrostomogram, done on days 7 and 12, respectively, showed no leakage or obstruction. On the 24th postoperative day, the nephrostomy catheter was removed. Ten days later, the intravenous pyelogram was normal (Fig 5). Thirty-five days after surgery she was discharged well, with good renal function (BUN level, 46 mg/100 ml; creatinine level, 1.1 mg/100 ml). Urine cultures were sterile.

COMMENT

Spontaneous rupture of transplanted kidneys may occur early or late after surgery. The first early spontaneous ruptures of transplanted kidneys were reported by Starzl[10] and Murray et al.[6] They reported ruptures that occurred within a few days after surgery in both cadaveric and sibling recipients. Afterward others[1-3,5] also reported early spontaneous rupture of the transplanted kidney. Early rupture usually occurs within the first week after surgery.[1,6] The clinical picture of a ruptured kidney is uniform and characteristic; the patient complains of pain over the side of the transplant and a swelling can be noticed, the blood pressure falls, the pulse rate increases, and diuresis stops.[1,4-6] These complications seem to occur

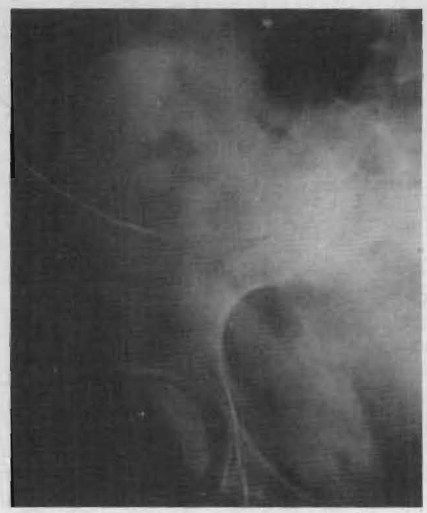

Fig 3.—Mild hydronephrosis due to distal ureteral obstruction and extravasation in right side of pelvis.

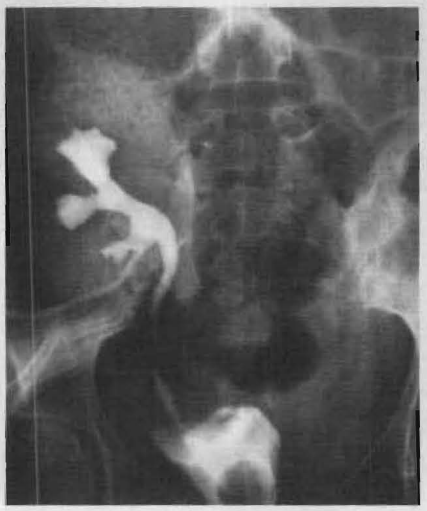

Fig 4.—Intraoperative nephrostomogram, perforated lower calix, nonobstructive and functional ureter, and normal bladder.

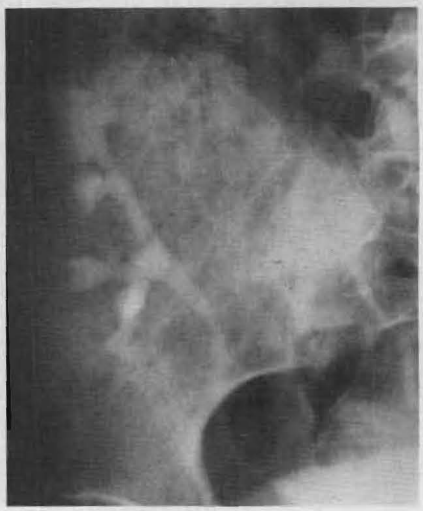

Fig 5.—Postoperative 34th day; normal pyelogram.

during a period of rejection or soon after rejection, and almost all authors describe very similar clinical and operative findings: rupture of the graft with bleeding and clots around it and bleeding on the peritoneal wall or in the peritoneal cavity.[1-5,13] The line of rupture is usually sagittal.

Late rupture with hemorrhage has been reported by Lord et al[4] 170 days after transplantation. The patient herein reported presented a different and unusual complication in that a delayed spontaneous rupture of a localized area in the lower pole of the graft occurred 133 days after transplantation. There was no bleeding. Although there was a mild rise of the BUN (81 mg/100 ml) and creatinine (2.6 mg/100 ml) values, these promptly returned to normal after nephrostomy drainage, without adjustment in immunosuppression. The kidney was of normal size at surgery. Review of the history revealed a mild episode of rejection 3½ months before this acute symptom. Pyelograms at this time were normal. There was no anatomical abnormality (such as a polar artery) or technical difficulty at the time of donor nephrectomy. However, there had been recurrent postoperative urinary tract infections.

A biopsy examination of the ruptured area showed necrosis and acute and chronic inflammation. The genesis of the complication is not clear. Possibly an intrarenal arterial branch was thrombosed during an earlier rejection episode. The infarcted area of the kidney then could be silent until an infection occurred and caused a tissue breakdown. This type of complication had been reported by Jeppesen,[13] Potampa,[7] and Shaw[9] in nontransplanted kidneys.

The patient was treated successfully with nephrostomy, wound drainage, and antibiotics without any complications. However, Jeppesen,[13] Potampa,[7] and Shaw[8] have suggested nephrectomy for similarly affected kidneys in nontransplanted patients. In transplant patients with good graft function, conservative treatment should be tried in both noninfected and infected spontaneous parenchymal rupture[1,3,6] as well as in pelvic and ureteral complication.[2,6,8,10,11,14] We believe that early diagnosis and conservative treatment are very important in transplant patients to save the graft.

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