

Delayed calyceal cutaneous fistula after renal transplantation

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Calyceal cutaneous fistula is a serious complication of renal transplantation. It is usually caused by segmental renal infarction resulting from an unrecognized ligated or severed accessory renal vessel during donor nephrectomy. Unless associated with trauma, a calyceal fistula usually develops within the first 3 months of transplantation. This is the first reported case of a calyceal cutaneous fistula in a renal transplant patient manifested 2 years after renal transplantation and associated with acute pyelonephritis and apparently newly developed amyloidosis. Prompt surgical intervention with primary closure of the fistula achieved a successful outcome.

Urinary extravasation is a perplexing problem of renal transplantation; its incidence varies between 8% and 17%.^{1,2} Although oliguria is a common finding in most of the cases, the initial diagnosis is usually unrecognized because of concomitant rejection or infection or both.³ We report a case of calyceal cutaneous fistula in a renal transplant patient with presenting symptoms of acute pyelonephritis.

Case report

A 51-year-old Caucasian man whose original renal disease was focal sclerosing glomerulonephritis received a second renal allograft. A Terasaki "C" matched renal allograft donated by a sibling had been rejected after 41

months of good function. The second renal allograft, from a cadaver donor, matched for only HLA antigen functioned immediately. By the 15th day the serum creatinine was 1.8 mg/dl.

The patient did well until 23 months later when he was admitted because of fever, chills, and pain over the allograft. Both urine and blood cultures grew *Escherichia coli* and his serum creatinine level increased to 4.4 mg/dl. Excretory urography, cystography, and retrograde pyelography were normal. Renal transplant angiography revealed patent renal vessels with a clearance time of 2 seconds and no evidence of infarction (*Fig. 1*). He was treated with a course of cephalothin intravenously for 2 weeks. At the time of discharge the urine was sterile and the serum creatinine level was 1.6 mg/dl.

One month later, the patient was readmitted because of spontaneous drainage of clear fluid from the medial aspect of the second transplant incision. Chemical analysis of the fluid showed it to be consistent with urine. Intravenous pyelography revealed prompt function, sharp calyces, and no demonstrable fistulous tract (*Fig. 2*). However,

intravenously injected indigo carmine extravasated through the fistula opening. Cystography was normal; methylene blue in the bladder did not extravasate through the fistula. A retrograde pyelogram demonstrated extravasation from the lower pole calyx (*Fig. 3*). At surgery, a healing cortical abscess cavity full of necrotic tissue was found to communicate with two calyces. The necrotic tissue was debrided, the calyces were closed primarily, and the renal cortex was approximated with mattress sutures. A soft rubber drain was placed, but no nephrostomy tube drainage was used. An excretory urogram 6 weeks later was normal (*Fig. 4*).

Five months later a rectovesical fistula with overwhelming sepsis developed and the patient died. At autopsy systemic amyloidosis was found that involved the renal allograft. There was no recurrence of the calyceal cutaneous fistula. Review of previous biopsy specimens of the patient's original and transplanted kidneys showed no evidence of amyloidosis.

Comments

Calyceal cutaneous fistulas are un-

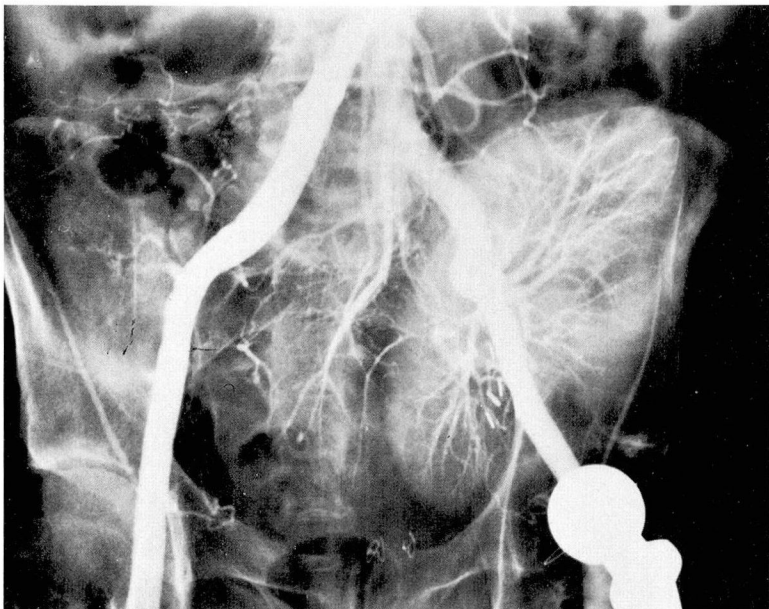


Fig. 1. Renal transplant angiogram reveals multiple patent arteries and no evidence of segmented renal infarction.

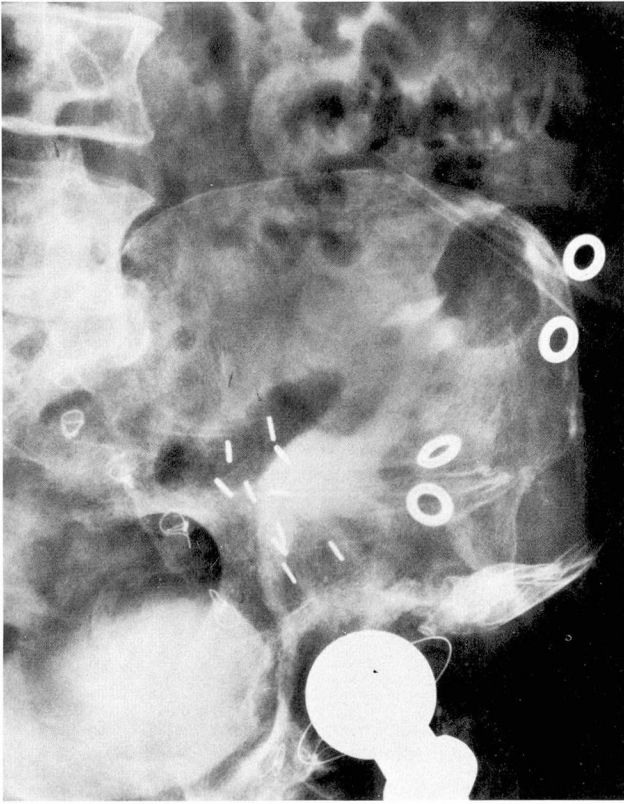


Fig. 2. Excretory urogram shows prompt function with sharp calyces and no demonstrable fistulous tract.

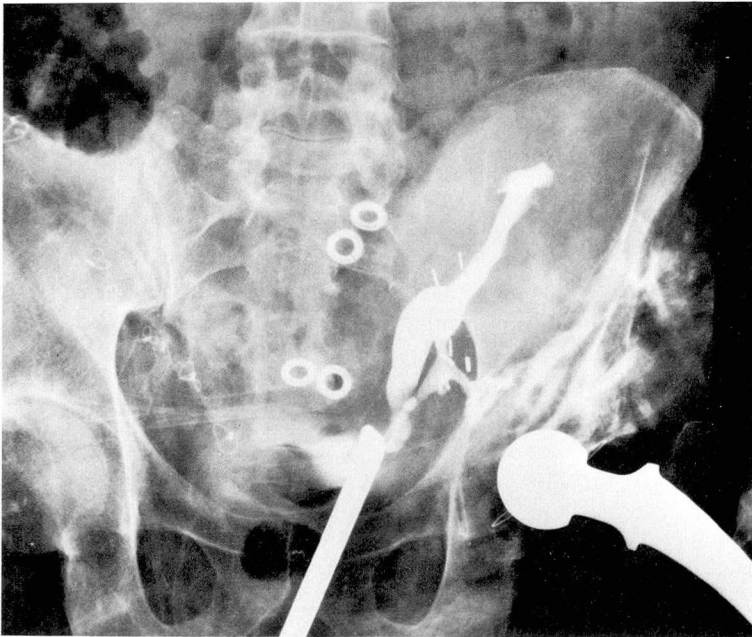


Fig. 3. Retrograde pyelogram demonstrates extravasation of contrast media from the lower pole calyx.



Fig. 4. Normal excretory urogram 6 weeks following primary closure of the fistula.

sual complications of renal transplantation. Williams et al³ reported two cases in 170 transplants; Anderson et al,⁴ one case in 125 transplants; and Schiff et al,⁵ three cases in 134 transplants. The overall incidence of calyceal fistulas is 1.3%.

The most important contributing factor is segmental renal infarction resulting from severing an unrecognized accessory renal artery or inadvertently ligating a small polar vessel. Since calyceal fistulas occur more commonly in patients receiving renal allografts supplied by two or more renal arteries, failure to revascularize successfully the renal segment supplied by the vessel leads to renal infarction and production of the fistula. Although our patient had

two renal arteries supplying the allograft, both were proved to be patent by angiography and no infarction was demonstrated. Localized spasm of the renal artery during perfusion has been suspected to cause segmental renal ischemia and subsequent fistula formation,⁶ but with the use of systemic phenoxylbenzamine and local procaine infusion, vascular spasm has been ameliorated.^{7,8} In essence, calyceal fistulas typically are early complications of renal transplantation and are most likely to occur during the first 3 months of surgery.⁴⁻⁶ A delayed calyceal fistula of 1 year's duration developed after an automobile accident that had caused blunt trauma to the allograft.⁹

Although in humans, renal cortical

necrosis has been observed after hyperacute rejection,¹⁰ neither that early form of rejection nor chronic rejection was evident in this transplanted kidney. The infusion of certain bacterial toxins has also been demonstrated to cause cortical necrosis similar to that seen in the rejection graft.¹¹ It is likely that cortical ischemia in our patient was due to severe acute pyelonephritis that occurred 2 years following transplantation and caused cortical necrosis and fistula formation. Amyloidosis also may have contributed to development of the fistula.

It is important to be alerted to this complication when recurrent upper urinary tract infection occurs in renal transplant patients. Prompt surgical intervention is mandatory. Although nephrostomy tubes have been used by others,^{3-6,9} debridement and primary closure of the calyces appear to be more feasible because foreign bodies are eliminated, the incidence of urinary leakage is reduced, and early recovery is promoted.

References

1. Palmer JM, Kountz SL, Swenson RS, et al: Urinary tract morbidity in renal transplantation. *Arch Surg* **98**: 352-356, 1969.
2. Prout GR Jr, Hume DM, Lee HM, et al: Some urological aspects in 93 consecutive renal homotransplants in modified recipients. *J Urol* **97**: 409-425, 1967.
3. Williams G, Birtch AG, Wilson RE, et al: Urological complications of renal transplantation. *Br J Urol* **42**: 21-28, 1970.
4. Anderson EE, Glenn JF, Seigler HF, et al: Urologic complications in renal transplantation. *J Urol* **107**: 187-192, 1972.
5. Schiff M Jr, McGuire EJ, Weiss RM, et al: Management of urinary fistulas after renal transplantation. *J Urol* **115**: 251-256, 1976.
6. Fox M, Tottenham RC: Urinary fistula from segmental infarction in a transplanted kidney; recovery following surgical repair. *Br J Urol* **44**: 336-338, 1972.
7. Belzer FO, Kountz SL: Preservation and transplantation of human cadaver kidneys; a two-year experience. *Ann Surg* **172**: 394-404, 1970.
8. Belzer FO, Reed TW, Pryor JP, et al: Cause of renal injury in kidneys obtained from cadaver donors. *Surg Gynecol Obstet* **130**: 467-477, 1970.
9. Schiff M Jr, McGuire EJ, Webster J: Successful management of caliceal fistulas following renal transplantation. *Arch Surg* **110**: 1129-1132, 1975.
10. Williams GM, Hume DM, Hudson RP Jr, et al: "Hyperacute" renal-homograft rejection in man. *N Engl J Med* **279**: 611-618, 1968.
11. Klassen J, Milgrom F: Studies on cortical necrosis in renal grafts. *Transplant Proc* **3**:598-601, 1971.