SPONTANEOUS RUPTURE OF RENAL PELVIS
AFTER RENAL TRANSPLANTATION

BARRY A. KOGAN, M.D.
JOHN W. KONNAK, M.D.
ROBERT J. MACGREGOR, M.D.
DARRELL A. CAMPBELL, JR., M.D.

From the Sections of Urology and General Surgery, Department of Surgery,
University of Michigan Medical Center, Ann Arbor, Michigan

ABSTRACT — Spontaneous rupture of the renal pelvis occurred in 4 cases following renal trans-
plantation. The diagnosis was difficult, and all 4 patients were septic at the time
of treatment. Three
patients survived, but preservation of the transplant was possible in only 1 case. The cause is un-
certain but may be related to functional obstruction or vascular insufficiency.

Urinary extravasation occurred in 31 of 585 renal transplants performed at the University of
Michigan Medical Center, and of these, 4 suffered spontaneous rupture of the renal
pelvis. Previously, this complication has been
reported only rarely, therefore, a review of the
diagnosis, treatment, outcome, and possible
origin of this complication appeared warranted.
Our experience is contrasted with that reported
for spontaneous rupture of the renal pelvis in
the general population and that reported for uri-
inary fistulas after renal transplantation.

Material and Methods

Table 1 is a summary of the clinical data in
our 4 cases. Extravasation from the renal pelvis
occurred in 1 cadaver and 3 living related
recipients. It developed from five to forty-six
days post-transplant and was noted in patients
who had undergone ureteroureterostomy, trans-
vesical ureteroneocystostomy, and external
ureteroneocystostomy using the techniques
described by Freier et al.1 The exact diagnosis
was not made preoperatively in any case. Treat-
ment was transplant nephrectomy in 2 cases,
repair and drainage of the renal pelvis in 1, and
pyelostomy in the fourth. Although 3 patients
survived, graft preservation was possible in only
1 case.

Comment

The diagnosis of rupture of the renal pelvis
was elusive in all 4 cases, as evidenced by the
delay of from six to thirty-five days from onset
of symptoms until treatment. In 2 cases a
wound infection was the initial abnormality
noted. Later this developed into an obvious uri-
inary fistula, requiring surgical exploration. In
the other 2 patients acute peritonitis developed
as a result of transperitoneal rupture of an
infected urinoma. Again, surgical exploration
was required. Despite the use of excretory
urography, ultrasonography, and nuclear
scanning, the site of extravasation was not
identified preoperatively in any case. Retro-
grade pyelography is difficult after ureteroneo-
cystostomy especially when an external uretero-
neocystostomy technique is used. Antegrade
pyelography is not performed safely unless the
renal pelvis is dilated. Dilatation was not
present in any of the cases and is unlikely after
rupture. Diagnosis is dependent on a high index
of suspicion. When clinical judgment suggests
operative intervention, a careful inspection of
the renal pelvis should be included.

All the patients in the group under considera-
tion were ill at the time of presentation. In 2
cases (Cases 1 and 4), the septic status of the
patient required that transplant nephrectomy be
performed. Both patients made an uneventful recovery. In 1 patient (Case 3) the transplant was left in place, a pyelostomy performed, and immunosuppression continued; this patient died of progressive sepsis. The fourth patient (Case 2) also presented with septic symptoms. He was treated with extensive drainage of the retroperitoneal space, and the kidney was left in situ. Immunosuppression was temporarily withdrawn, and the patient's sepsis was reversed. Subsequently, low doses of immunosuppressive drugs were administered following repair of the renal pelvis and nephrostomy drainage. Because there was a 2 haplotype match between the sibling donor and recipient, normal transplant function was preserved, and the patient made an uneventful recovery.

The origin of this complication was not clearly elucidated in any case. No mechanical obstruction occurred in our patients, and no major vascular injury could be implicated. All patients in the series were infected at the time of treatment; but it is impossible to determine if this was the cause or the result of the extravasation. Several other theories may explain this occurrence but are difficult to document. Functional obstruction may have played a role in 3 cases. Case 3 had the visual appearance of an acquired ureteropelvic junction obstruction at the time of surgical exploration. Two other patients (Cases 1 and 2) had a brisk diuresis (300–1,000 cc/hr) in the immediate postoperative period. Because of the edema and decreased peristalsis at the ureteral anastomosis, the urine output may have exceeded the capacity of the collecting system, creating a functional obstruction and high intrapelvic pressures. This is impossible to document retrospectively. Another theory would be that there was interference with the blood supply to the renal pelvis. This is unlikely since the renal pelvis has a rich capillary blood supply and there was no dissection of the renal pelvis, and in particular, no dissection near the transverse artery at the pyelocortical junction. Perhaps high intrapelvic pressures may decrease capillary blood flow in the renal pelvis. The fact that 3 patients who received kidneys from living related donors had the onset of symptoms four to five days post-transplantation is suggestive of a vascular origin, but there is no explanation of why the renal pelvis and not the ureter was affected in our cases. The role of rejection and/or poor healing due to immunosuppression is also impossible to document.

Several reports of ruptures of the renal pelvis in the general population have been presented. Abeshouse reviewed the literature on this subject in 1935. His article remains a definitive work on this subject. He noted that most cases are traumatic and those which are “spontaneous” usually occur in an abnormal upper urinary tract. Predisposing factors include infections of the renal pelvis, hydronephrosis, obstruction, and instrumentation of the urinary tract. Symptoms and signs are varied and range from local irritation of nearby gastrointestinal organs to generalized sepsis. Although this was the preantibiotic era, prognosis varied directly with time until diagnosis and treatment. Repair and drainage, drainage alone, and nephrectomy have all been employed successfully depending on the clinical circumstances.
Several authors have also addressed the topic of urinary fistulas after renal transplantation. Infection, poor healing in the immunosuppressed host, and errors in techniques of harvesting or transplantation are believed to be responsible for most cases. Vascular insufficiency and ureteral necrosis is an end result in many cases. Symptoms and signs are varied, but a mass overlying the kidney and wound drainage are often seen. Early diagnosis and prompt institution of therapy is emphasized by all authors. Depending on the clinical circumstances, therapy may be nephrectomy, drainage and repair, or observation.

Spontaneous rupture of the renal pelvis after renal transplantation has been mentioned only three times in the literature. The diagnosis was made only after considerable urinary extravasation had resulted in a fluctuant mass. Treatment consisted of successful repair, attempted repair followed by nephrectomy, and transplant nephrectomy. The authors believed that the rupture was secondary to obstruction of the ureteroneocystostomy in 1 case and to erosion of an infected hematoma in another.

Our data suggest that this complication, while unusual, is more common than previously appreciated (4 cases in 585). The diagnosis is often delayed, and as a consequence the patient will often show signs of systemic sepsis. Adequate debridement and tensionless reapproximation of the renal pelvis is not always feasible. Transplant nephrectomy is the treatment of choice in most cases. However, the clinical condition of each patient is different, and an occasional graft may be salvaged if the patient is clinically well or if the degree of histocompatibility is such that immunosuppression can be dramatically reduced.

Urology Section Box 03
University Hospital
1405 E. Ann Street
Ann Arbor, Michigan 48103
(Dr. Konnak)

References