RUPTURE OF THE ALLOGRAFTED KIDNEY – IS REPAIR POSSIBLE?

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Summary

Allograft rupture after transplantation is an uncommon but serious complication. A review of the literature suggests a significant mortality and very high incidence of graft loss. Data will be presented which suggests that conservative management of the allograft rupture is possible.

Introduction

Rupture of the kidney was first described by Wunderlich in 1856 [1] and since then approximately 360 cases have been reported.

Rupture of the allograft kidney was first reported by Murray et al [2] and a study of the literature of the last 10 years has revealed 96 further reports [3,4,5,6]. It would appear that renal allografts are more susceptible to spontaneous rupture than non-transplanted kidneys.

The incidence of allograft ruptures varies considerably between 0.3% [7] and 8.5% [8]. This significant complication of renal allografting, according to the case reports, has resulted in an overall mortality of 10% and a graft loss of 74% [7 and 9].

Is conservative management of allograft rupture a safe procedure for the patient and does it result in a viable kidney?

Material and Methods

Between September 1965 and December 1977, 419 renal transplants were performed in 372 patients, some patients receiving two or more allografts. Nineteen kidneys (4.5%) were from living donors and the remainder from cadavers. We encountered 13 major ruptures of the kidney in 12 patients (3.1%), one patient rupturing the same kidney twice! All ruptured kidneys were from cadaver donors and had had a capsulotomy and biopsy at the time of operation. There were six males and six females. The ages of the patients ranged from 23 to 53 years, with a mean age of 38 years. Two of the ruptures occurred in second grafted kidneys.
Clinical Presentation

Rupture usually occurred as an acute episode characterised by pain and tenderness over the site of the graft, oliguria, fever, hypotension and a fall in haematocrit. The clinical findings are summarised in Table I. In our survey nine ruptures occurred between the third and twenty-second post-transplant day, and in four patients delayed rupture had occurred; on days 30, 31, 39 and one at 41 months (this being a second spontaneous rupture) (Figure 1).

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>No. of Patients</th>
</tr>
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<tbody>
<tr>
<td>Pain at graft site</td>
<td>12</td>
</tr>
<tr>
<td>Tenderness at graft site</td>
<td>8</td>
</tr>
<tr>
<td>Swelling over allograft</td>
<td>8</td>
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<tr>
<td>Falling haematocrit</td>
<td>8</td>
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<tr>
<td>Non-functioning graft on haemodialysis</td>
<td>7</td>
</tr>
<tr>
<td>Fever</td>
<td>6</td>
</tr>
<tr>
<td>Oliguria</td>
<td>4</td>
</tr>
<tr>
<td>Shock</td>
<td>4</td>
</tr>
<tr>
<td>Haematuria</td>
<td>3</td>
</tr>
<tr>
<td>Local infection</td>
<td>2</td>
</tr>
</tbody>
</table>

The clinical features of delayed rupture were often less acute and local signs were more variable.

In the early postoperative period the cadaver kidneys were frequently oliguric and a number of investigations were carried out to determine the reasons for the poor function. These often included a Tizzardogram, diethylene triamine penta acetic acid scan, and biopsy or arteriography. However, none of these investigations could predict the sudden allograft rupture although they did frequently clarify its aetiology.

The diagnosis, however, remained essentially a clinical one, and was followed by prompt exploration of the allograft. The mean time between the commencement of symptoms and surgical exploration was 5.3 hours in patients rupturing their kidneys shortly after transplantation. In the patients with delayed rupture the mean time between the onset of symptoms and operation was 36 hours.

At exploration, eight patients were found to have perirenal haematoma and allograft rupture with the kidney enlarged, swollen and showing variable features of rejection. In three cases the ureter and pelvis were distended with distal obstruction, and in one of these cases the ureter was actually infarcted. In two patients there was urinary extravasation and in one patient kinking and obstruction of the renal vein was found.

One patient, at five days post-transplant, complained of sudden, acute abdominal pain and collapsed, and as resuscitation was being undertaken underwent a cardiac arrest. Resuscitation proved unsuccessful and at post-mortem a large perirenal haematoma was found with several litres of blood.
in the retroperitoneum and fresh clots of blood in the peritoneal cavity through a rupture in the peritoneum. The kidney was found to be grossly enlarged and swollen with an extensive fracture of the cortex. The histology confirmed acute severe rejection.

We have based our surgical management of spontaneous allograft rupture on the following features at operation.

1. An assessment of the gross appearance of the kidney and the severity of rejection.
2. The number of previous rejection episodes.
3. The patency of the renal vessels.
4. The findings of pelvic or ureteric obstruction.

The allograft was removed in four patients because the kidney appeared grossly rejected and histological examination confirmed extensive changes associated with severe rejection. In the remaining eight patients conservative management was possible with direct repair of the allograft rupture, and in half these patients a nephrostomy was also performed. In the patient with ureteric necrosis the allograft rupture was repaired, and the ureter splinted by Tizard catheter.

![Figure 1. Bar graph illustrating the time interval between transplantation and rupture of the allograft.](image)

All kidneys were biopsied at exploration and where obstruction was found a nephrostomy was performed using a small multi-perforated silastic catheter passed through the renal parenchyma into the pelvis and distal ureter.

**Pathological Findings**

In our patients rupture occurred on the convex border in eight patients, in several places in three, and in the upper pole alone in two cases. The rupture most commonly along the longitudinal border ranged from shallow fissures of several millimeters in depth to deep hemifracture of the kidney involving almost the entire cortical surface.
Histological examination of the kidney removed and biopsy of the kidneys left in situ in all cases revealed some degree of rejection.

**Outcome of the Conservatively Treated Ruptures**

Five of the ‘early’ ruptures occurred in the first week and where repair was performed the kidneys all recovered with mean kidney survival of 47.8 months. In the cases of delayed rupture at 30, 31, 39 days and then at 41 months, three of the grafts were treated conservatively and left in situ after repair, with good immediate renal function. However, two patients eventually died; one of pulmonary sepsis on the sixth post-operative day and the other after 70 days with gross systemic infection. The remaining kidney maintained adequate renal function for five months but later required removal because of chronic rejection.

**Conclusion**

We believe that an acute awareness of the possibility of allograft rupture in the early post-operative period would permit an early recognition of the ‘allograft rupture syndrome’ with prompt exploration. Providing the graft is viable and not severely rejected, conservative management may be possible although this may require a nephrostomy because of associated ureteric obstruction.

Delayed rupture, however, presents a more varied clinical picture which may result in delay in recognition of the syndrome, and while clearly the kidney may still be salvaged the impact of exploration and the delay in recognition may result in an increased morbidity and indeed mortality.

**References**


**Open Discussion**

BRYNGER (Göteborg) We surveyed our kidneys transplanted up to 1975 and
found almost exactly the same frequency of kidney rupture namely 19 out of 705 grafts, that is 2.7% which matches your 2.8% very well. There are differences anyhow with the management afterwards. We are more optimistic than you because of these 9 kidneys could be retained and only one of these kidneys had to be removed shortly afterwards. Of course we have later failures due to other reasons. But it was possible with conservative surgical treatment. We left the kidney in situ, removed the haematoma and if there was still bleeding we put in a drain. We have interpreted these kind of ruptures as mainly due to rejection and with vigorous anti-rejection therapy combined with conservative treatment it was possible to retain 18 out of the 19 kidneys. Why did you do this nephrostomy?

MONTES We found severe cases with evidence of ureteral obstructions. We’ve thought one of the factors influencing the rupture of the kidney is the rise in the ureteral pressure and this is the reason why we do nephrostomy.

BRYNGER We have not done that in any cases but it might be different causes of the rupture.

BARNES (Birmingham) I’d like to question the aetiology of these splits. We have roughly the same number as the two previous speakers describe. In fact I tend to regard these as one of the most benign complications you can get. As far as I know we have only lost one kidney and these kidneys seem to be remarkably free from rejection. They have not been associated with the treatment of rejection episodes. I’d also like to quibble with the suggestion that you would repair these. We certainly have not been able to repair them; we evacuate the haematoma, then stop bleeding and the whole thing settles down. I support your view that occasionally it is necessary to do something with urinary drainage because they can be associated with some infarction of the ureter, or some obstruction to the ureter. But that is not the primary aetiology of them. We don’t do capsulotomies. I gather that you do routine ones. But as far as I know from the literature there is no correlation between capsulotomy and rupture. For the beginner in transplantation it is a rather frightening thing to see one or two splits right across the kidney. You are tempted to take it out. But my advice is don’t. We recently had a patient who ruptured his kidney three times, and this kidney is functioning beautifully now.

MONTES I agree with you. We don’t mean that capsulotomy will prevent the rupture of the kidney, or the ATN, but we continue to use capsulotomy for historical reasons.

LINDSTROM (Helsinki) You didn’t mention another possible cause for renal rupture after transplantation. I think renal vein stasis is a cause of rupture. We saw it in 1967 in one of our first transplantations when we had problems with the vein anastomosis resulting in partial obstruction and later the kidney ruptured. What do you think about it?

MONTES Stasis in the venous outflow from the kidney could be one of the factors influencing rupture. In one of our cases there was kinking and obstruction of the renal vein.
BARNES  Can I ask Professor Lindström how he determines whether there is stasis in the renal vein? I presume you get a purple looking kidney. The ones that I see with this condition (i.e. rupture) are bright pink and it is arterial bleeding that is coming from them.

LINDSTROM  It is sometimes difficult to confirm that there has been obstruction to renal vein outflow caused by kinking or torsion of the renal vein when the wound is closed. At surgical exploration the compression is automatically relieved and may be overlooked.

GELIN (Chairman)  I think we have to agree that any cause of high parenchymal pressure might rupture the kidney. Either hypoxic damage or rejection or any other cause.