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ACUTE RENAL ALLOGRAFT RUPTURE – A GOOD PROGNOSTIC SIGN?

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Summary

Acute renal allograft rupture is a well recognised though uncommon complication of transplantation. Previous reports suggest a close association with rejection and record a high incidence of graft loss and significant mortality. In a series of 11 consecutive allograft ruptures, no graft required removal at initial exploration and eight are still functioning with a mean follow-up of 20 months. This experience shows acute renal allograft rupture is a relatively benign complication and that conservative management leads to a satisfactory outcome in the majority of patients.

Materials and methods

During a 44 month period (July 1978 to February 1982) 305 cadaveric renal transplants were performed at one centre and complications were recorded prospectively. Acute renal allograft rupture occurred in 11 patients (seven male, four female: age range 16–39, mean age 24 years). The ruptures occurred two to nine days after transplantation and all were characterised by acute graft pain, swelling and tenderness and usually accompanied by hypovolaemia and fall in urine output. The number of mismatched A+B human leucocyte antigens was between none and three with an average of 1.5. Renal preservation was by cold storage after perfusion with hypertonic citrate and total ischaemic times varied from 5.6 to 25.1 hours with a mean of 17.5 hours. Only two patients had been dialysed post-operatively both for presumed acute tubular necrosis. Three patients had been treated with methylprednisolone prior to graft rupture for clinically diagnosed rejection episodes. All grafts were placed in the iliac fossa and a small wedge cortical biopsy was taken prior to wound closure which was approximately 30 minutes after re-establishment of the renal circulation. Capsulotomy was not routinely performed. Once allograft rupture was suspected, urgent exploration was undertaken.

TABLE I

Case number	Age	HLA A+B mismatch	Total ischaemic time (hours)	Post-operative day	Pre-rupture rejection	Pre-rupture dialysis	Pathology	Outcome
1	21	1	10.4	6	+	no	Mild rejection	Nephrectomy day 14: renal vein thrombosis
2	31	2	8.1	4	–	no	Mild rejection	Nephrectomy day 35: rejection
3	17	3	23.3	3	–	no	Not biopsied	Functioning graft 40 months
4	20	2	18.3	9	+	no	Mild rejection	Functioning graft 38 months
5	27	2	20.4	2	–	no	Mild rejection	Nephrectomy day 12: renal vein thrombosis
6	39	2	25.1	4	–	no	No rejection	Functioning graft 20 months
7	16	0	24.1	7	–	yes:ATN	No rejection	Functioning graft 18 months
8	20	1	23.0	9	+	no	Mild rejection	Functioning graft 17 months
9	34	2	21.5	9	–	no	No rejection	Functioning graft 14 months
10	21	2	5.6	6	–	yes:ATN	No rejection	Functioning graft 8 months
11	16	0	12.2	5	–	no	Mild rejection	Functioning graft 5 months

Results

At exploration all patients had a perigraft haematoma arising from cortical ruptures which were usually situated along the convex border. Though the grafts were swollen and oedematous, all were pink with no macroscopic features of rejection or evidence of vascular or ureteric obstruction. Surgical management was evacuation of the haematoma with control of bleeding by simple pressure, suture or packing with cellulose gauze or muscle patch. No graft required removal at the time of initial exploration. Ten of the ruptured grafts were biopsied at exploration and histology revealed no evidence of rejection in four cases and mild rejection in the other six. Subsequently three grafts were removed, one for irreversible rejection at 35 days and two for renal vein thrombosis at 12 and 14 days. The remaining eight patients all have satisfactory graft function with a mean follow-up of 20 months (Table I).

Discussion

The first report of renal allograft rupture was by Murray et al [1] who described four cases occurring in the first post-operative week. Rejection did not seem to be involved and three kidneys recovered function following repair. Indeed our longest surviving cadaveric graft maintains a serum creatinine of 83μmol/L thirteen years after a spontaneous rupture managed by surgical evacuation of the haematoma. Over one hundred cases have now been reported in the literature and the incidence has varied between 0.4 and 8.5 per cent [2, 3]. The mechanism of graft rupture is not well understood and postulated aetiological factors have included ischaemia, biopsy, heparinisation during dialysis, ureteric obstruction and renal vein thrombosis [2, 4]. Though cortical biopsies were taken routinely at transplantation in our cases, the site of biopsy did not usually correspond to that of the subsequent cortical rupture. Two patients had undergone needle biopsy during suspected rejection episodes both four days prior to the diagnosis of graft rupture. A further two patients were dialysed post transplantation both being anuric from presumed tubular necrosis, though in neither case was rupture diagnosed during or immediately following dialysis. No case was found to have ureteric obstruction. The subsequent development of renal vein thrombosis in two cases may be due to disturbance of the renal vein at reoperation rather than a cause of the rupture.

Matas et al [5] reported a high initial nephrectomy rate with poor chronic performance of ruptured grafts that were not removed and Homan et al [6] recommended that, in most instances, exploration should be accompanied by nephrectomy. More recently Montes et al [7] and Dryburgh et al [8] recommended conservative management if bleeding can be controlled. We support this latter view, particularly as the absence of significant rejection in our cases has been striking. Only three patients had been treated for rejection prior to rupture and biopsies taken at exploration revealed mild rejection only in six cases with no evidence of rejection in the remaining four. Moreover with only one graft subsequently lost to rejection we feel that immunological factors are unlikely to be involved. An explanation of the findings of Matas et al [5] and

Homan et al [6] is that the renal allografts in their patients had developed end-stage rejection which with present knowledge might be removed earlier, before rupture had occurred.

In evaluating possible aetiological factors, it is relevant to compare this present series with our earlier experience as a number of changes have taken place in the practice of cadaveric transplantation. In the years between our first case and the current series (1969–1978) we encountered several other cases of allograft rupture and the prognosis again was usually good. The use of heart beating cadaveric donors during this series has significantly reduced initial warm ischaemic times compared with our earlier cases. In contrast the total ischaemic times have considerably lengthened with a mean of 17.5 hours in the present series of ruptures. It therefore seems unlikely that graft rupture is closely related to ischaemic time. Nor is there any clear connection with perfusion fluids as rupture was observed before and since the use of hypertonic solutions. A consequence of the changes in kidney retrieval and preservation is that the majority of grafts now achieve immediate function whereas in the earlier series tubular necrosis usually occurred. Again this seems unrelated to graft rupture. Finally the practice of capsulotomy appears to have no influence as units which perform capsulotomy routinely have reported similar graft rupture rates to our own [7].

We conclude that the aetiology of renal allograft rupture is unknown and unlikely to be due to any single causative factor. However, providing allograft rupture is diagnosed and repaired at an early stage, it carries little morbidity and can lead to a satisfactory outcome in the majority of patients. We have come to regard its occurrence as a good prognostic sign.

References

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