

Renal Allograft Rupture: A Clinicopathologic Review

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RENAL ALLOGRAFT rupture (RAR) is a rare but very serious complication of renal transplantation, requiring emergency surgery. The most common cause is acute allograft rejection, but other causes such as renal vein thrombosis (RVT), acute tubular necrosis (ATN), renal biopsy, and lymphatic obstruction have been reported.^{1,2} We reviewed our experience with the aim of identifying RAR predisposing conditions.

PATIENTS AND METHODS

In a consecutive series of 934 renal transplants performed between July 1983 and September 1999, 11 patients (1.2%) had RAR. In these cases we studied donor and recipient characteristics, preservation conditions, clinical signs and symptoms, treatment, and pathology findings. This group of patients was then compared with their paired cohort. Data analysis was computer-based. In the statistical analysis *t* test and Fisher's exact test were used.

RESULTS

All 11 kidneys that suffered RAR were from cadaver donors, nine male and two female. The mean age was 29.5 years with good terminal serum creatinine (mean 1.1 mg/dL). All organs were stored in Eurocollins solution and the mean cold ischemia time was 21 hours and 25 minutes (range, 10 hours to 29 hours and 20 minutes).

Excluding one black patient, all recipients were Caucasian. Eight were female and 3 were male, with a mean age of 33.8 years. The mean HLA match was 1.7, and the mean peak panel reactive antibody (PRA) was 22% (range 0 to 93%) and current was 15% (range 0 to 67%). All patients had cyclosporine treatment, eight had delayed graft function requiring dialysis, and three underwent renal allograft biopsy. In two patients rupture occurred in the second allograft; the others were first transplants.

The day of RAR was a mean of 5.3 (range 2 to 13). All patients had new onset of severe allograft pain, eight had a drop in daily hematocrit, and six had hypotension. The four patients with more precocious ruptures had sudden onset of bleeding through the drainage tube.

Transplant nephrectomy was performed in 10 patients, and surgical conservative treatment with fibrin glue and collagen foam was performed in one. All patients survived RAR. Three had a second transplant and currently have functioning allografts.

Pathology examination revealed RVT in three patients and some degree of rejection in the remaining eight. One

patient had a rupture on the second day because of hyperacute rejection, and three had severe acute cellular rejection, but in four patients the dominant figure was ATN with minimal rejection. Excluding the patient with hyperacute rejection, the day of rupture was later for those with severe acute rejection, a mean of 9.6 days (range 6 to 13). In those with ATN, the day of RAR was a mean of 4.5 (range 3 to 6) and the patients with RVT had ruptures even sooner, on mean third day (range 2 to 4).

Variables associated with RAR were: sex mismatch ($P = .004$), current PRA ($P = .012$), and a need for dialysis ($P = .042$). Age of the recipient, transplant number, cold ischemia time, total HLA match, and peak PRA were not associated with RAR.

DISCUSSION

Higher current PRA and a need for dialysis are variables associated with rejection and ATN. Therefore they are expected to be related to rupture. The well-documented conditions that are associated with ATN and rejection³ must be the same, which in extreme conditions predispose to RAR. We find no explanation for the statistically significant association of sex mismatch and RAR, other than random error.

Acute allograft rejection is the most frequent cause of graft rupture in the literature (60 to 80%),³ but ATN has received little note. In our series, ATN was responsible for 36% of the ruptures, as much as severe acute rejection. ATN alone can cause RAR,⁴ because of interstitial edema and rise in intrarenal pressure. But when associated with rejection, it seems that these two conditions can act synergistically to cause allograft rupture.

Our data suggests that rupture occurs later when caused by rejection, rather than when RVT is responsible. To our knowledge this finding had never been reported in world literature. Perhaps the timing of RVT is related to technical problems, such as twisting and kinking of the vein or intima tear, although the thrombogenic effect of cyclosporine can also have a role in this process.⁵

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All these patients were on cyclosporine therapy, which may explain the small number of RAR caused by rejection alone and the significant number of patients that had RVT (27%). It appears that cyclosporine therapy is changing the etiology of the graft rupture.⁶

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