Dear Sir,

Spontaneous renal allograft rupture is a life-threatening surgical emergency. The reported incidence of this unusual complication of renal transplantation varies between 0.3 and 9.6%, most occurring in the first 3 weeks following surgery [1-3]. The pathogenesis of graft rupture has been extensively studied but it still remains controversial. The principal cause involved is severe acute rejection with increasing intrarenal pressure in over 80% of cases [4, 5]. The incidence of this catastrophic complication has been reported to have decreased following the introduction of cyclosporine as the primary drug in the immunosuppressive protocol [6]. We report a case of renal allograft rupture following severe acute rejection in a 26-year-old woman who had received a well-matched kidney from her mother and was on cyclosporine therapy.

The patient, a case of chronic interstitial nephritis with end-stage renal disease, was on maintenance haemodialysis since January 1993. The patient’s mother, a healthy 56-year-old lady, was the donor. The mother and daughter had a one, one, one mismatch on the HLA A, B and Dr loci, respectively. The panel-reactive antibody was negative against a panel of forty.

She was admitted for a live related renal transplant. The donor nephrectomy revealed an additional lower polar artery. The main renal artery was anastomosed end to end with the internal iliac artery. Following the removal of the vascular clamps the lower polar artery was anastomosed to the inferior epigastric artery using the microscope in another 22 min. The graft had good primary function.

The patient was on triple drug immuno-suppression comprising cyclosporine, aza-thioprine and prednisolone. She did well in the immediate postoperative period and was producing adequate urine. However on the 5th postoperative day she complained of excessive pain over
the graft site accompanied by a drop in her urine output. An urgent ultrasound revealed blood around the transplanted kidney. The patient was immediately explored. There was fresh blood and clots around the kidney. There was a 3.5-cm linear rupture on the convex surface of the kidney which was actively bleeding. The vessels were normal. The clots were evacuated and the rupture repaired with a muscle patch. A biopsy was taken from the site. She was antirejected with three pulses of methylprednisolone. She responded and her renal parameters are now normal. The patient is currently on triple drug immunosuppression. The kidney biopsy was compatible with acute tubulointerstitial rejection. There was marked focal lymphoid cellular infiltration in the interstitium associated with tubular involvement in these areas.

Renal allograft rupture is a well-known complication of renal transplantation. The dramatic and life-threatening nature of this catastrophe demands an immediate diagnosis and aggressive therapy. The main predisposing factor involved is acute rejection with increasing intraluminal pressure [4, 5]. Other aetiological factors reported are ureteral obstruction [7], acute tubular necrosis [8], renal biopsy [9], heparin therapy [10], complete lymphocytic ligation [1], renal vein obstruction [11] and renal trauma [8].

Most ruptures are seen in cadaveric renal allografts. It is interesting to note that of nearly two hundred published cases of spontaneous rupture, only 2 patients had received cyclosporine [6]. The current consensus is that the incidence of graft rupture has decreased following the inclusion of cyclosporine in the immunosuppressive protocol.

Renal allograft rupture requires immediate surgery on diagnosis. The control of haemorrhage and evacuation of the haematoma is mandatory. Earlier series recommended transplant nephrectomy as the treatment of choice [7, 12]. However most series now indicate that graft repair is worthwhile if bleeding can be controlled [2, 13-15]. The long-term prognosis of the repaired allograft is decided by the success of treating acute rejection [6].

Our case highlights the fact that the inclusion of cyclosporine in immunosuppression does not preclude the occurrence of this serious condition. Clinicians in the cyclosporine era should be aware of this possibility as an early diagnosis and treatment improve graft salvage. Conservative therapy should be the mainstay of management as in countries with a live related programme, saving the graft is of paramount importance.

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References


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Spontaneous Renal Allograft Rupture: Still a Threat