# Spontaneous renal allograft rupture due to acute rejection in early post-transplant period – A case report

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## Abstract

Renal allograft rupture (RAR) is a rare but lethal complication of renal transplantation. This can be a threat to the graft and patient survival. Over the decades, allograft nephrectomy has been the standard treatment for renal allograft rupture. But now, recent evidences suggest that graft salvage treatment can be safely done in complicated cases also. We describe a case of 30-year-old male who received a living donor kidney transplant and had spontaneous graft rupture in early post transplant period due to acute rejection. Graft was successfully repaired surgically, along with medical management and now the graft is functioning well.

**Keywords:** Acute rejection, graft repair, nephrectomy, postoperative day, renal allograft rupture

## Introduction

Rupture of renal allograft is a rare and serious complication of transplantation that is usually attributed to acute rejection, acute tubular necrosis or renal vein thrombosis.[1-3] It is associated with a high incidence of graft loss.[4] The consequences are fatal in 6% of the cases and graft loss is the outcome in another 53%.[5] The incidence of allograft rupture has decreased due to the use of modern potent immunosuppressive medications.[6-8] Due to its devastating clinical course and outcome, recognition and prompt management of allograft rupture is important. Usually, nephrectomy is necessary treatment measure but conservative surgical intervention has also been attempted to preserve the renal allograft in certain cases.[2,9,10] We report an unusual case of early renal allograft rupture secondary to severe acute rejection. The graft was successfully salvaged by surgical repair and intensive medical management.

## Case Report

A 30-year-old male presented at our clinic with End stage renal disease secondary to Membranous glomerulopathy with a serum creatinine of 9 mg/dl. It was diagnosed through renal biopsy done 5 months back, which along with Anti PLA2R positive, showed significant global (43.7%) and segmental (15.6%) tuft sclerosis with significant interstitial fibrosis and tubular atrophy (50%). Patient had no history of thromboembolic episodes and was not on any anticoagulant. Patient was admitted and started on haemodialysis through right IJV catheter. He opted to undergo renal transplantation with mother as a prospective renal donor. On investigation blood group was found to be compatible and complete workup of donor and recipient was done. CT renal angiogram of donor showed bilateral accessory renal artery with bilateral single renal veins.

Pre-transplant immunological work up -

- HLA match was 3/6
- CDC cross match-Negative
- Flow cross match-Negative
- Single antigen test was not done.

Patient was posted for renal transplant.

### Transplant surgery details

Graft kidney was placed in right iliac fossa and open donor nephrectomy was done. Transplant surgery was uneventful.

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| Quick Response Code: | Website: www.ijtonline.in |
| | DOI: 10.4103/ijot.ijot_141_20 |

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**Received:** 10 November 2020; **Revised:** 20 March 2021; **Accepted:** 22 June 2021; **Published:** 31 March 2022



**How to cite this article:** Godara S, Saraf K. Spontaneous renal allograft rupture due to acute rejection in early post-transplant period – A case report. Indian J Transplant 2022;16:138-41.

After declamping graft was pink, turgid and well perfused with a good intraoperative urine output. Warm ischemia time was 5 min 30 sec. Prolongation of warm ischemia time was due to slippage of ligature. Cold ischemia time was 46 min and anastomosis time was 28 min.

Inj Antithymocyte globulin was given as an induction agent at a total dose 3 mg/kg body weight considering patient at a low risk. Intraoperatively, inj Antithymocyte globulin was given at a dose of 1.5 mg/kg body weight and rest dose of 1.5 mg/kg was given postoperatively. Intra operative Inj Methylprednisolone 500 mg IV stat dose was also given. The recipient was hemodynamically stable in the intraoperative and immediate post transplant period. Drain was inserted for collection of residual fluid at the graft site.

Following transplant urine output was 300 ml/hr and triple immunosuppression was started which included tab Tacrolimus at a dose of 0.08 mg/kg body weight, tab Mycophenolate sodium at a dose of 1400 mg/day and tab prednisolone 30 mg/day.

Patient was maintaining adequate urine output of 3-4 litres/24 hour and his serum creatinine was decreasing on each post operative days (POD). Drain was removed on POD 2.

On POD 6, patient had a rise in serum creatinine from 1.5 mg/dl to 2.1 mg/dl and urine output also started decreasing. Because of this sudden increase in serum creatinine, a suspicion of acute rejection arose. As Tacrolimus levels was also within normal limits, inj Methylprednisolone 500 mg IV stat was given in view of acute rejection and graft biopsy was planned for the next day.

On POD 7, patient started complaining of severe pain around graft site. A doppler ultrasound was done which showed a collection of fluid at upper pole of graft kidney of approximately 450 cc and a raised resistive index (RI) of 0.8. Gradually the patient's blood pressure started decreasing and patient also developed tachycardia. In view of these changes, Iv fluids (normal saline) was given. On further investigation, it was found that heamoglobin (Hb) levels had also dropped from 7.2 gm/dl to 5.7 gm/dl. Since patient did not responded to Iv fluids, Ionotrope support was started and urgent transplant surgeon's opinion was taken after which re-exploration of graft kidney was planned and patient was immediately shifted to operation theatre.

### Intra-operative findings

A hematoma with large amount of blood clots was evacuated. A gross rupture measuring approximately 8 cm along the convex border of graft kidney was seen and repaired surgically by suturing with vicryl 1.0, cauterization was done with Argon laser and covered with surgical [Figures 1 and 2]. Two units of packed red blood cells were transfused during procedure. There was no leak at the anastomosis site and no other source of bleeding was found. Intra-operatively graft biopsy was also taken and tissue was sent for histo-pathological examination.

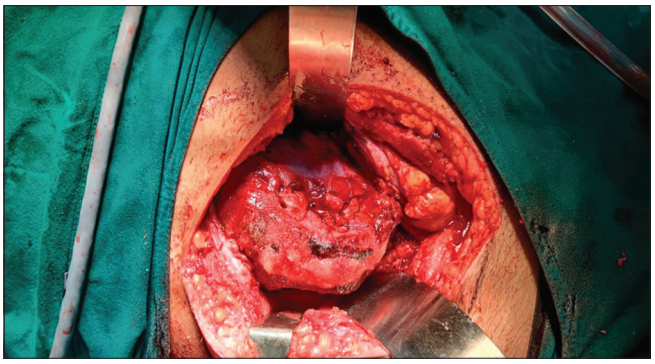

**Figure 1:** Image showing rupture of graft along the convex border with repair done surgically by suturing

Following graft repair, patient's vitals stabilized and ionotrope support was stopped. But urine output did not increase and was constant at a rate of approximately 20 ml/hr. On the same evening patient developed signs of volume overload and one session of haemodialysis was done.

Meanwhile we received the renal biopsy report the next day which showed evidence of active tissue injury in the form of glomerulitis, peritubular capillaritis (microvascular inflammation) and intimal arteritis. Focal areas of interstitial inflammation and tubulitis were also observed [Figures 3 and 4]. Features suspicious for co-existing active cellular and antibody (humoral) rejection were seen.

In view of acute rejection patient was given Antithymocyte globulin at a dose of 1.5 mg/kg body weight.

On subsequent POD's plasmapheresis was done of 1.5 volume. Total four sessions of plasmapheresis were done on alternate days.

Injection IVIG was also given. A total of 50 gms of IVIG was given on subsequent POD's.

As a result, urine output started increasing gradually and serum creatinine started declining on succeeding post operative days. On repeat doppler ultrasound RI also decreased to 0.6.

The patient was discharged in a stable condition with a urine output of approximately 4 litres/day and a serum creatinine of 1.6 mg/dl.

On follow up patient's serum creatinine further decreased to 1.4 mg/dl.

## Discussion

Renal allograft rupture (RAR) is defined as a superficial or deep tear of the renal capsule as well as renal parenchyma.[9] The prevalence of renal allograft rupture (RAR) varies from 0.3% to 3%.[9] It normally occurs in the first few weeks after transplantation. It is associated with severe graft pain, hypotension, and a drop in haemoglobin. The most common cause of allograft rupture is acute rejection.[2]

The rupture most frequently occurs longitudinally, along the convex border of the kidney. Immediate ultrasound evaluation

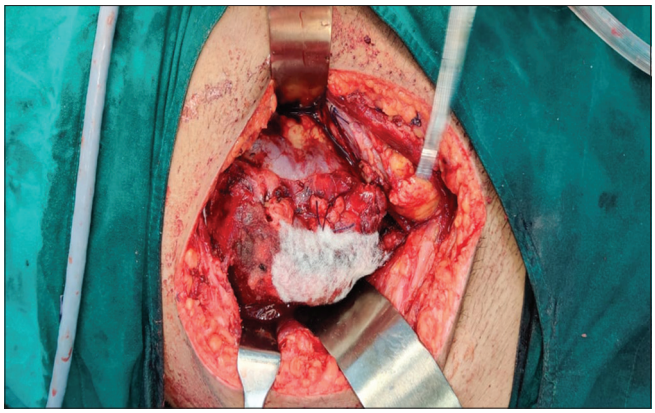

**Figure 2:** Image showing rupture of graft along the convex border with repair done surgically by suturing with abgel applied

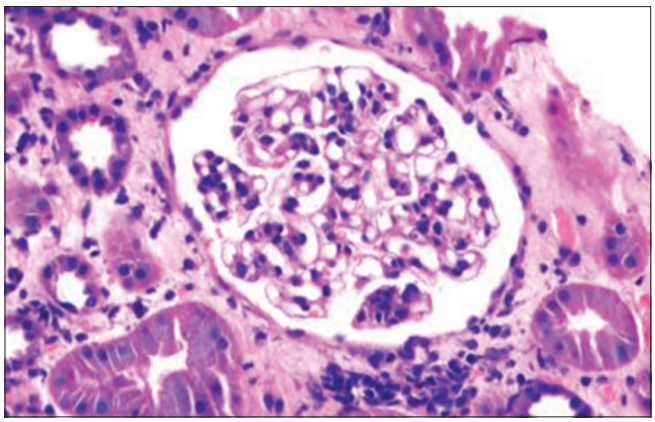

**Figure 3:** Histopathological image of graft biopsy showing active tissue injury in the form of glomerulitis

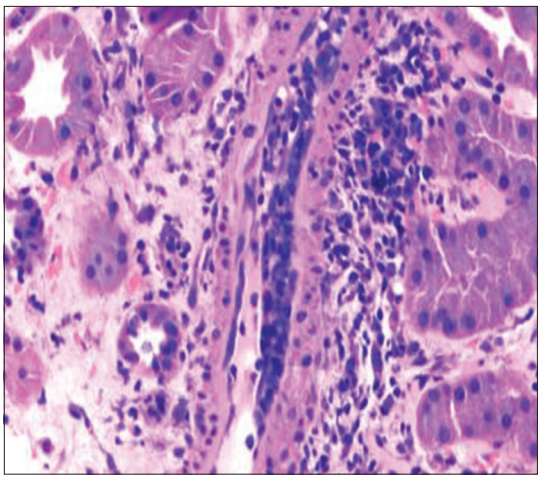

**Figure 4:** Histopathological image of graft biopsy showing active tissue injury in the form of interstitial inflammation, peritubular capillaritis, intimal arteritis and tubulitis

can rapidly and safely confirm the diagnosis with 87% sensitivity and 100% specificity.[11]

The pathological mechanism of RAR is not fully understood. Cortical and capsular ischemia resulting from interstitial oedema and cellular inflammatory cell infiltration in the setting of acute rejection is considered the major cause of RAR by exerting capsular tension, tearing, and rupture. This may occur even many years after transplantation.[12] Rarely RAR can be triggered by graft biopsy.[13] Recipients of non-heart-beating donor kidneys are at a greater risk of developing graft rupture, and this has been attributed to the higher rate of acute tubular necrosis in this type of grafts.

Other risk factors are high peak panel reactive antibodies (PRA) and younger recipient, probably due to more vigorous immunological responsiveness.[1] On the other hand, the use of ATG was found to be associated with a lower incidence of RAR, presumably due to reduced frequency and severity of rejection.[1] Clinical diagnosis may be confirmed by ultrasound or CT scan.[14] There is new evidence showing superiority of multi-detector computed tomography (MDCT) in the diagnosis of RAR.[15] The most common course of management in RAR is urgent graft nephrectomy.

However, recent reports suggest that ruptured kidney grafts are potentially salvageable by conservative surgical repair of the rupture, with good success rates.[6,9,16-18] Current reports demonstrate that ruptured grafts can be saved with a success rate as high as 80%.

Moreover, recipients undergoing successful repair have long-term outcomes similar to the general transplant population. Recurrent rupture occurs in only 5% patients.[19] With salvage rates between 40 and 100% and variable long-term complications and performance of the repaired graft, it is worth trying to salvage spontaneously ruptured grafts. Only in those patients whose hemodynamic status cannot be stabilized by appropriate aggressive hemodynamic support, graft nephrectomy should be considered as a definite treatment.[13,20]

## Conclusion

RAR is still encountered in clinical practice. In spite of advances in immunosuppression, most of the cases are due to acute rejection. When spontaneous renal transplant rupture occurs, nephrectomy is justified only in case of refractory hemodynamic instability or compromised kidney viability. If irreversible graft damage is ruled out and the patient can be readily resuscitated, transplant salvage should always be attempted.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

### Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

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