Acute Allograft Renal Vein Thrombosis: A Case Report

Saif Ahmed Khan, Mohammed Nasser Al-Huneini, Yasser Waad-Allah Al-Mula Abed, Saja Mahmood Mohammed, Dawood Ahmed Al-Riyami

Received: 23 Jun 2014 / Accepted: 19 Aug 2014
© OMSB, 2014

Abstract

Renal graft thrombosis is a serious and devastating complication of renal transplant that ultimately results in graft loss. It is associated with acute and hyper-acute rejections; however, the underlying cause in large proportion of patients remains unknown. We report a case of a young male who underwent live related kidney transplant but lost the graft on the operating table due to renal vein thrombosis (RVT).

Keywords: Kidney; Transplant; Graft; Loss; Thrombosis.

Introduction

Renal graft thrombosis accounts for one third of all early (within 90 days) graft failure in both adults and pediatric recipients. The North American Pediatric Renal Transplant Cooperative Study (NAPRTCS) reported that graft thrombosis was the major cause of graft failure in the first year in pediatric patients. It usually occurs as early as 48 hours to first 10 days of transplant. However, thrombus formation may be delayed until after the first few weeks. It typically presents with sudden onset of oliguria or anuria, graft swelling and dysfunction. In case of venous thrombosis, flank pain and hematuria are also associated.

Case Report

This is a 15-year-old boy who was diagnosed to have end-stage renal disease due to obstructive uropathy (vesico-uretric reflux with urethral stricture) three years ago. He was started on thrice-weekly haemodialysis in 2010 initially via tunneled catheter and later via arterio-venous fistula. His past medical history was significant for thrombosis of catheter followed by pulmonary embolism in 2010. Induction immune suppression included anti thymocyte globulin, methyleprednisolone and mycophenolate mofetil in standard dosage. Unfortunately, after clamps removal, the graft turned pink for few minutes then immediately it became blue and dusky. He remained anuric and bruit was appreciated. Immediately, Doppler study was done and the venous flow could not be detected at the renal hilum; however, there was a normal venous flow more distally at anastamosis site. This was further evaluated with Magnetic Resonance (MR) renal angiogram and its delayed phase showed non-opacification of the renal vein at hilum and its intra-renal tributaries. Patchy cortical enhancement with focal area of non-enhancement was noted in the transplanted kidney.

He remained anuric; a graft biopsy was performed after 24 hours, which revealed vascular and glomerular thrombosis. C4d stain was negative making hyper-acute rejection less likely but not entirely excluded. We do not have the facility of testing for donor specific anti-bodies; however, the repeated cross match anti bodies screen was persistently negative. At this stage thrombophilia screen was performed, which revealed heterozygous for prothrombin G20210A mutation.

The patient got started on plasma exchange, intravenous immunoglobulins and anti thymocyte globulin though the above picture was not compatible with hyperacute rejection. Therapeutic anti-coagulation was tried as well but not thrombolysis in view of the fresh surgery. Lately graft nephrectomy was done.

Discussion

The exact cause of RVT in a large proportion of patients remains unknown; however, several risk factors have been identified and are related to recipients, donors, operative and immunosuppression. Recipient factors include young age, membranous nephropathy, peritoneal dialysis as mode of pre-transplant dialysis and hypercoagulable status, including anti phospholipid anti bodies syndrome, anti-thrombin deficiency, mutation of factor V Leiden.
and prothrombin gene while the donor factors include female gender and old age. The operative risk factors of thrombosis risks are prolonged ischemia time, multiple graft vessels anastomosis, technical errors caused by vascular clamping. RVT can also be triggered by administration of monoclonal antibody like monoclonal antibody OKT3 that can induce procoagulant activity and risk is increased in patients treated with high dose of pulses methyleprednisisolone, which may activate the tissue factor/factor VII pathway.10

Genetic causes of venous thrombosis due to deficiencies of anti thrombin, protein C and S are found in less than 1% of the population.11 Even among patients with thrombosis, only a small percentage carries one of these defects. The most common genetic defect predisposing to thrombosis is FVL (factor V Leiden) with an overall prevalence in carriers of around 5%.12 Factor V causes thrombosis because of the protein resistance to inactivation of protein C.13

The protein gene mutation, which this patient has, is considered the second most common cause of inherited thrombophilia in Caucasians. It was first described in 1996.14 The mutation is found in the 32 untranslated region of prothrombin gene at position 20210 (G to A PT20210 A). It is found in about 3% of Caucasians with regional variation in prevalence ranging from 1 to 6%.15 Among patients with venous thrombosis enrolled in Leiden Thrombophilia Study, this mutation is present in 6%.16 It increases the risk of thrombosis about three folds, which appears to be mediated through elevated prothrombin levels.17

This patient was found to have the heterozygous form of the mutation i.e. only one gene is affected and this is more prevalent than the homozygous form, which has prevalence of 1% and a higher risk of thrombosis. Other tests showed FVL mutation negative, proteins S, C and antithrombin III were normal.

Conclusion
Allograft thrombosis generally causes irreversible damage and treatment options are usually disappointing. Few cases were reported in which the grafts were salvaged by early intra-arterial injection of anti fibrinlytic agents.18 We do believe that thrombophilia screening is mandatory in all patients with history of venous thromboembolism prior to transplant whether considered provoked or unprovoked. Screening should be also done for those with positive family history. Therapy with heparin for the above group in the pre-transplant period has been proved to be beneficial in preventing RVT.19

Acknowledgements
The authors reported no conflict of interest and no funding was received on this work.

References