

Case report - donor derived allergy

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For successful organ transplantation, immunosuppressive drugs are administered to suppress cells of the immune cell, primarily T and B lymphocytes. Consequently, some of the same drugs applied in transplantation medicine are used in severe allergic diseases as well. Therefore, one would expect that transplant recipients would not show clinical signs of type I allergy and do not develop any kind of sensitization or clinical symptoms of allergy because the IgE pathway is being suppressed.

We present a case which we suggest is the first report of donor derived drug allergy in solid organ transplantation. A 37yr old man developed ESRD secondary to Alport's COL4A5 mutation. He was started on CAPD in 2002 and then received his first transplant in 2004. This failed in 2009, he was restarted on haemodialysis with graft nephrectomy soon after. Histology of the graft showed a well encapsulated (6mm) type 2 papillary renal carcinoma pT1a which was completely excised. He recently received his second transplant from a 20 year old DCD donor, MM 111 who died from bacterial meningitis. The donor was treated with ceftriaxone 3 days before death but developed a rash within 24 hours and switched to chloramphenicol and metronidazole; creatinine 90 at organ harvest. Our recipient received ceftriaxone starting in theatre and continued post transplant for 5 days. He developed no systemic reaction to the drug. Our patient had delayed graft function, had bleeding around the graft which required exploration in theatre on day 5 hence a renal biopsy was taken. The biopsy showed acute tubular injury but no tubulitis. There was granulomatous tubulointerstitial nephritis with diffuse mononuclear cell infiltrate and occasional eosinophils most prominent in the renal medulla; no evidence of rejection. The ceftriaxone was already stopped and he made a sustained recovery.

The most likely aetiology and mechanism was proposed as passive transfer of sensitized passenger cells from donor transplanted tissue. These cells are likely to be resident for a short time, hence donor derived drug allergy not being a major clinical problem. Other causes of granulomatous TIN such as infection, sarcoid and de-novo anti GBM disease were excluded. Donor derived food allergy has been commonly reported in solid organ transplants, particularly in paediatric liver. The factors explaining the failure of immunosuppressive therapy to prevent type I hypersensitivity in transplant recipients are not entirely clear.