Introduction

For patients with end stage renal failure, transplantation remains the treatment of choice. However, the ever increasing success of renal transplantation, graft failure remains a major problem after transplantation ***(Dallman, 2003)***. Renal allograft failure can be caused by large variety of diseases, among them acute rejection is both frequent and clinically significant ***(Colvin and Mauiyyedi, 2002)***.

Acute rejection can be mediated by allo-reactive inflammatory cells (acute cellular rejection "ACR") or specific antibodies (antibody mediated rejection "AMR") ***(Nickleit and Andreoni, 2007)*** and the distinction between them is therapeutically important but pathologically difficult ***(Collins et al, 1999)***.

Acute humoral rejection occurs in 20-30% of acute rejection cases with poorer prognosis than acute cellular rejection and is refractory to conventional immunosuppressive therapy ***(Mauiyyedi et al, 2002).***

The recent rediscovery of peritubular capillary deposition of C4d, a complement split product (typically activated during classical antibody dependent classical pathway and covalently bound to the endothelium), has lead to better recognition of AMR as well as pathogenesis, diagnosis, and treatment of this entity ***(Troung et al, 2007)***.

Detection of C4d staining in peritubular capillaries, together with positive post transplant cross matching (donor specific antibody "DSA"), and evidence of histological damage (Polymorph nuclear leukocytes or lymphocytes in dilated peritubular capillaries &/or glomeruli, transmural arteritis, fibrinoid necrosis and thrombosis) have been incorporated into the revised Banff '97 classification system as criteria for diagnosis of AMR in renal allografts ***(Racusen et al, 2003)***.

Fibrin is the end product of the coagulation cascade. Fibrin clots compromise glomerular capillary flow (leading to focal ischemia and necrosis) and attract both macrophages and leukocytes ***(Hertig and Rondeau, 2004)***. Fibrin is noted in arteries with fibrinoid necrosis/arteritis and is specific for antibody mediated rejection ***(Troung et al, 2007)***.