# Development of de novo Hemolytic Uremic Syndrome Post-Transplant and the Role of Donor Specific Antibodies: A Case Report and Review of Literature

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## Abstract

Introduction: Hemolytic uremic syndrome (HUS) following kidney transplantation is a devastating complication that may result in substantial morbidity and allograft loss. While calcineurin inhibitor-induced HUS has been well described, anti-donor specific antibody production may be an alternative pathway in the pathogenesis of HUS. We present a case of HUS following kidney transplantation, and will present evidence that donor specific antibodies (DSA) may be a factor in platelet activation and end-organ injury resulting in post-transplant HUS.

Case description: A 66-year-old female with diabetic nephropathy underwent a deceased donor renal transplant with a 5 HLA mismatched kidney. Her immediate course was uneventful with normalization of her creatinine (Cr). She was re-admitted with rising Cr, oliguria, proteinuria, anemia and thrombocytopenia. A peripheral smear revealed schistocytes, haptoglobin levels were depleted and an allograft biopsy was performed that was suggestive of thrombotic microangiopathy (TMA) with equivocal findings for AMR. Her ADAMTS 13 activity was 103% (normal). She concurrently developed a substantial de novo DSA burden. She underwent therapeutic plasma-pheresis, conversion from tacrolimus to cyclosporine, and received rituximab therapy. She had a complete clinical resolution and remains off of dialysis. Her laboratory markers improved and her antibody titers decreased.

Discussion: Post-transplant HUS requires immediate recognition and treatment. This clinical course suggestsDSA may be involved in an alternative mechanism of platelet activation leading to HUS and renal insult. Review of the literature suggests this is a rare cause of HUS and we postulate may be under-diagnosed in the transplant population and requires further study.