**Enteric hyperoxaluria secondary to pancreatic insufficiency – an unusual cause of renal allograft dysfunction**

**Introduction**

Oxalate deposition is rarely identified in native kidneys and calcium oxalate deposition in renal transplant kidney without history of primary hyperoxalauria is poorly understood. Incidences of oxalate deposits in transplanted kidney have been identified which range from 4% to 52% in biopsies and up to 82% in transplant nephrectomies. Very few reports illustrated the impact of oxalate deposition on renal graft function and graft survival of kidney transplant recipients.

We would like to present a case of renal transplant recipient with end stage renal failure secondary to diabetic nephropathy who experienced graft dysfunction secondary to calcium oxalate crystal deposition. The latter was due to enteric hyperoxalauria secondary to pancreatitic insufficiency.

**Methods**

Literature review and review of case notes carried out.

**Results**

Graft dysfunction was identified during routine clinic visit which lead to further investigations including renal transplant biopsy. This identified acute tubulointerstitial nephritis and some oxalate crystals deposition and no evidence of rejection. A trial of steroids and increased immunosuppression did not lead to improvement in renal function and a second transplant biopsy performed at eight weeks revealed significant oxalate crystals deposition.

Further investigations showed marginally elevated 24 hours urinary oxalate excretion of 0.44mmol/l (normal range 0.04 to 0.34mmol/l). Also noted low faecal elastase 114ug/g (100-200ug/g suggest moderate pancreatic insufficiency) and a MRI of the pancreas identified an atrophic pancreas. This lead to a diagnosis of enteric hyperoxalauria secondary to pancreatitic insufficiency

**Discussion**

Oxalate is produced by various metabolic pathways although these pathways of oxalate synthesis are not fully understood.Dietary hyperoxalauria can occur with high oxalate content consumed in excess. Enteric hyperoxalauria can be observed in small bowel malabsorption, chronic pancreatitis or antibiotics use and high ingestion of vitamin C. Oxalate deposition can lead to tubular atrophy, interstitial inflammation and interstitial fibrosis.  Our case report emphasised enteric hyperoxalauria secondary to pancreatic insufficiency leading to renal allograft dysfunction.

Pinheiro et al highlighted high incidence of calcium oxalate deposition in renal allograft with early dysfunction and almost 30% loss of graft function. However, Bagnasco et al emphasized that presence of oxalate does not appear to be associated with increased occurrence of acute rejection in first few months post-transplant and, long term impact of oxalate deposition in transplanted kidney is not clear.

In summary we present a case of allograft dysfunction secondary to calcium oxalate deposition due to enteric hyperoxaluria. This is an unusual cause of allograft dysfunction and we would like to raise awareness of this condition. In addition, we plan to review renal transplant biopsies from regional transplant centre to identify other similar cases and study the natural history of this condition.