**IS LOW VITAMIN D LEVEL AN INFLAMMATORY MEDIATOR OF ANAEMIA IN KIDNEY TRANSPLANTATION?**

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**Introduction and Aims:** Anaemia remains a common finding after kidney transplantation. It is associated with inferior patient- and graft-survival. Hepcidin, the master regulator of iron homeostasis, prevents iron absorption and sequestration within the reticulo-endothelial system, thus inhibiting normal iron recycling for erythropoiesis. Previous study in kidney transplantation displayed an independent association between reduced haemoglobin (Hb) and elevated hepcidin levels, with hepcidin levels mostly driven by systemic inflammation and reduced renal function. Recently, vitamin D status was found to correlate with hepcidin and inflammation inversely in chronic kidney disease. However, its role in kidney transplantation remains undefined. The objectives of this study were to determine the prevalence of vitamin D deficiency among kidney transplant recipients (KTRs), and evaluate the impact of vitamin D on inflammation, hepcidin and Hb levels in this cohort.

**Methods:** This single-centre cross-sectional study enrolled 100 clinically stable KTRs at least 12 months post-transplantation. Mean age = 51±14 years, 54% male, and median time post-transplantation = 7 (2-13) years. Vitamin D deficiency was defined using the Kidney Disease Outcome Quality Initiative guideline, with 25-Hydroxyvitamin D concentrations ≤39nmol/L considered as deficient, this was further sub-divided into moderate (12-39nmol/L) and severe (<12nmol/L) deficiency. Fasting serum samples were collected for measurements of 25-Hydroxyvitamin D, high-sensitivity c-reactive protein (hsCRP), hepcidin-25, and Hb levels. Potential demographic, anthropometric and clinical predictors of inflammation, hepcidin and Hb levels were measured and assessed using regression analyses.

**Results:** Vitamin D deficiency was found in 49% of clinically stable KTRs, with 13% classified as moderate and 36% as severe hypovitaminosis D. Low vitamin D predicted raised hepcidin levels (β=−0.2, p<0.05) independently of other significant predictors including hsCRP (β=0.6, p<0.01), estimated glomerular filtration rate (eGFR) (β=−0.4, p<0.05), and transferrin saturation (β=−0.8, p<0.05). In turn, low vitamin D levels were associated with raised hsCRP (β=−0.4, p<0.05) independently of increased fat mass (β=−0.3, p<0.001), the only other independent predictor of hsCRP. Furthermore, there was a trend towards an association between vitamin D and Hb levels (β=−0.8, p=0.07). Other significant independent predictors of reduced Hb levels include raised hepcidin (β=−0.3, p<0.05), declining eGFR (β=−0.2, p<0.001), and female gender (β=0.9, p<0.001).

**Conclusions:** Vitamin D deficiency is highly common among clinically stable KTRs. It is closely associated with raised hepcidin and inflammation. Furthermore the relationship between vitamin D and hepcidin levels highlights possible inflammatory and non-inflammatory mechanisms of Hb reduction, setting the scene for future research and therapeutic strategies against anaemia among KTRs.