Introduction

The very high risk of cardiovascular death in patients with end-stage renal disease (ESRD) has become increasingly apparent over the last several years (***Afshin, et al. 2005)*** .

Despite advanced dialysis techniques, patients with end stage renal disease (ESRD) frequently have complications of cardiovascular diseases. The risk of cardiovascular death is 10–20 times higher in dialysis patients than in the general population. Vascular calcification is also very common in dialysis patients and this induces the development of cardiovascular disease ***(Osamu, et al. 2007 and Peter, et al. 2003).***

It has been shown that the calcification process as seen in calcifying atherosclerosis is an active cellular process, which is controlled by calcification inhibitors and inducers ***(Hermans, et al. 2007).*** Physiological inhibitors of calcification, fetuin-A, osteoprotegerin (OPG) and undercarboxylated- matrix-carboxyglutamic acid protein (uc-MGP) may play a role in preventing the development and progression of ectopic calcification ***(Rukshana, et al. 2008 and Katsuhito, et al. 2007).***

Fetuin was first isolated in 1944 by **Pedersen K. O.** as a major plasma protein in fetus**.** Fetuin is produced by multiple organs (including liver, kidney and brain) during fetal development ***(Dziegielewska, et al. 2000).*** It is more abundant in fetal blood, hence the name fetuin *(from Latin*

word fetus). Fetal calf serum contains more fetuin than albumin, while adult serum contains more albumin than fetuin ***(Ketteler, et al. 2003).***

Fetuin-A, a glycoprotein with a molecular weight of about 60 kDa ***(Hermans, et al. 2007).*** It is produced primarily by the liver in the adult. The circulating levels of fetuin and its human homologue (α 2 -HS-glycoprotein) are significantly lower in adults than fetus, and can be further decreased during inflammation **(*Sciacca*, et al. 2008).** Low levels of fetuin-A are associated with higher C-reactive protein levels and increased cardiovascular mortality in adults on haemodialysis. It is suggested that chronic low serum fetuin-A contributed to ectopic calcification observed in some of the patients in that study ***(Marhaug, et al. 2008).***

So, Fetuin-A acts as inhibitor of vascular calcification; it reversibly complexes with calcium and phosphorus and increases their respective serum solubility in a mechanism reminiscent to that by which apolipoproteins solubilize lipids. Incubation of fetuin-A with calcium and phosphorus at physiologic pH prevents crystallization for more than 9 days, whereas the minerals crystallized within hours in its absence. Similar effects have been demonstrated in human serum. Among populations with end stage renal disease (ESRD), serum fetuin-A concentrations are inversely correlated with vascular and cardiac valvular calcification. ***(Shlipak, et al. 2007).***