

INTRODUCTION

Chronic kidney disease (CKD) is a growing health burden. CKD is defined as kidney damage for three or more months, as defined by structural or functional abnormalities of the kidney, with or without decreased glomerular filtration rate (GFR), manifested by pathologic abnormalities or markers of kidney damage, including abnormalities in the composition of the blood or urine or abnormalities in imaging tests. OR GFR less than 60 mL per minute per 1.73 m² (body surface area) for three months or more, with or without kidney damage **(Harper and Jacobson., 2008).**

Dyslipidemia is a primary risk factor for cardiovascular disease and a common complication of progressive kidney disease. Most patients with chronic kidney disease have an abnormal lipid panel that increases their risk for atherogenesis. **(Snively and Gutierrez, 2004).**

It has been known for a long time that chronic kidney disease (CKD) is associated with dyslipidemia, but the full extent of abnormalities has been appreciated only recently, because routine laboratory tests fail to disclose the entire spectrum of lipid abnormalities **(Ritz and Wanner.,2006).**

In ESRD, several studies reported an association between dyslipidaemia and surrogate cardiovascular endpoints, in chronic haemodialysis patients the progression of coronary arteries calcification was related to high concentrations of triglycerides (TG) and low concentrations of HDL (**Tamashiro et al ., 2006**). The same data were reported in peritoneal dialysis patients **(Stompor et al., 2006).**

High serum cholesterol is widely recognised as a cardiovascular risk factor in general population. However, in patients with ESRD high concentrations of cholesterol are associated with better survival. In patients with ESRD, it is now clear that this reverse epidemiology is explained by the confounding effect of malnutrition and chronic inflammation (**Liu et al., 2004**).

Despite the availability of different techniques for renal replacement therapy (haemodialysis, peritoneal dialysis, and renal transplantation), life expectancy of patients with ESRD on haemodialysis (HD) and peritoneal dialysis (PD) remains poor, with only a moderate amelioration after renal transplantation. The annual mortality rate in the dialysis population is about 20%. Approximately 50% of these deaths are caused by CVD. This makes cardiovascular mortality 10 to 30 times more prevalent in patients with ESRD compared with the general population (**Diepeveen et al., 2008**).

Several retrospective analyses that included patients with mild or moderate CKD documented benefit from lowering of cholesterol by statins. In contrast, the Die Deutsche Diabetes Dialyse (4D) study and a small Scandinavian study failed to show a benefit from lowering of cholesterol by statins in ESRD. It is possible that nonclassical pathomechanisms override statin-sensitive mechanisms as also suggested by the observation that statins fail to reduce carotid intima-media thickening. Although, experimentally, exposure to lipids (particularly oxidized lipids) aggravates progression, data on the effect of statins on progression in patients with CKD are not definite. The most likely explanation is that the impact of numerous confounders obscures their effect on progression (**Ritz and Wanner., 2006**).