INTRODUCTION AND AIM OF WORK

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Atherosclerotic cardiovascular disease appears to be one of the major causes of premature death in patients with chronic renal failure and in renal transplant patients (Lacour et al., 1985).

Hypertriglyceridemia has been observed in patients with chronic renal failure both at predialytic uremic stage and on maintenance dialysis (Chan et al., 1981). Triglyceride elevation with its associated alterations in lipoprotein metabolism and decrease of high density lipoprotein (HDL) levels may be considered as one of the major determinants causing atherosclerosis and resulting in cardiovascular complications in these patients (Attman et al., 1984, Bagdade and Albers, 1977)

In 1977, Cramp et al., found significantly elevated plasma triglyceride concentrations persist during dialysis treatment. This elevated triglycerides have been attributed mainly to impaired catabolism of triglyceride-rich lipoproteins (Attman and Gustafson, 1979). In some cases may be further accentuated by an increased hepatic production of triglyceride-rich very low density lipoprotein (Bagdade et al., 1978).

Huttunen et al., (1978) suggested that, the elevation in serum lipids particularly triglycerides, in uremic patients on maintenance dialysis, might be related to a carnitine deficit.

Carnitine is required for the mitochondrial uptake of long chain fatty acids by skeletal and cardiac muscles which depend heavily on fatty acid oxidation for their performance (Bohmer et al., 1974), where it enables fatty acids to enter the inner mitochondrial compartment where beta oxidation occurs (Bremer, 1983).

In has been suggested that, the considerable loss of carnitine during dialysis results in a tissue carnitine deficiency with subsequent impairment of fatty acid oxidation (Bertoli, et al., 1981) and incorporation of free fatty acids into triglyceride synthesis (Gusmano et al., 1981).

Sustained hypertriglyceridemia following transplantation was reported by several reports (Cattran et al., 1979 and Savdie et al., 1980). In addition, development of hypercholesterolemia was reported as an another characteristic feature of transplant recipients (Kobayashi et al., 1983).

So, no definite answers have been given yet as to the effect of kidney transplantation on the derangement of lipid metabolism pre-existed in uremic patients before grafting.

In the present study the changes in plasma carnitine together with those of plasma lipids and lipoproteins will follow in uremic patients undergoing chronic hemodialysis and peritoneal dialysis as well as in patients with renal transplantation. Since apolipoproteins are the most probable determinants of the structural and functional specificity of plasma lipoproteins (Attman et al., 1984), apolipoprotein A and B will be estimated in this study.