INTRODUCTION

The most important complication of ESRD is cardiovascular disease, in which the prevalence of ischemic heart disease among haemodialysis patients is 10-20 times higher than normal population ***(Mallamaci et al, 2002).***

In dialysis patients, cardiovascular diseases were accounting for about 40% of death in these patients ***(Brunner et al. 1988).*** Also 42% of the patients undergoing haemodialysis have myocardial infarction or coronary revascularization; in addition the survival after myocardial infarction is much lower than general population ***(Mallamaci et al, 2002).***

Heart failure is common in patients under haemodialysis, the prevalence rates of coronary heart disease and left ventricular hypertrophy are approximately 40% and 70% of these patients respectively ***(Zoccali et al, 2001).***

Attention has focused on new markers of cardiac injuries like the cardiac hormones natriuretic peptide, myocardial proteins released during myocardial ischemia and necrosis like troponin T and troponin I, however the measurement of these substances is still scarcely applied in clinical practice in dialysis patients ***(Deegan et al, 2001).***

Cardiac isoform of troponin T, which in adults is ordinarily expressed exclusively in the heart, may be reexpressed in injured or diseased skeletal muscle ***(Adams et al, 1993)***, as has been observed in animals and in humans with polymyositis. It is unknown whether cardiac troponin T is expressed in skeletal muscle during uremia ***(Kobayashi et al, 1992).***

The cardiac troponin T is not only associated to all causes and cardiovascular mortality, but also is strongly related to left ventricular mass , furthermore , myocardial function is an important determinant of circulating troponin because specific isoform of cardiac troponin are over expressed in failing human heart ***(Mallamaci et al, 2002).***

Cardiotoxicity in patients with chronic renal failure undergoing dialysis may be due to recurrent volume expansion and contraction and changes in osmolarity and /or ion fluxes, which are factors specifically related to metabolic environment of dialysis patients. Other potential causes of non-ischemic cardiac injury include elevated parathyroid hormone concentration, calcium and oxalate crystals deposition in the heart and uraemic pericarditis ***(Frankal et al, 1996).***

The increase in serum troponin level may be associated with possible subclinical myocardial injury (ischemia due to coronary

artery disease, left ventricular hypertrophy, and/or fluctuation in blood volume) and/or abnormality of troponin catabolism induced by renal failure and /or haemodialysis itself***. (Iliou et al, 2001)***.

Patient with uraemia often have elevated serum cardiac troponin T even without clinical cardiac damage. Cardiac damage, indicated by either elevated cardiac troponin or low left ventricular contractility, is related to uraemic, deranged calcium phosphrous metabolism and sodium bicarbonate level***. (Lipshultz et al, 2003).***

Cardiac troponin T predicts death and cardiovascular outcome in clinically stable patient with end stage renal disease. Because this protein is synthesized exclusively in myocardial cell, so, in haemodialysis patient, plasma cardiac troponin T is independently related to left ventricular mass and predicts all causes and cardiovascular mortality***. (Mallamaci et al, 2002).***

Cardiac troponin may be an important independent prognostic marker in patient with CRF under haemodialysis ***(Choy et al, 2003).***