reactive nitrogen species. However, the significant decrease in SOD and vitamin C levels in patients with significant glomerulonephritis might be the result of inactivation of enzyme by reactive oxygen species, could have been generated in excessive amount due to the rapid proliferation of the glomerular cells (mesangial cells, endothelial cells and podocytes) which are a source of ROS or could be due to infiltration of the macrophage and the neutrophils in the patients with significant change glomerulonephritis. Superoxide dismutase inactivation by hudrogen peroxide (H_2CO_2), a dismutaion product of O_2 through destruction of histidline residue has been reported by Bray and Cockle. Like in present study, significant increase in oxidative stress also has been supported by various other studies : Markan S et al[4] reported that mean serum MDA levels were significantly higher ($p<0.05$) and lower SOD levels ($P < 0.05$) in patients with proliferative glomerulonephiritis (MPGN and RPGN) as compared to non proliferative glomerulonephritis (MCD, MGN and FSGS).

Kuo HT et al[18] also reported increased plasma malondialdehyde (MDA) levels in the patients with FSGS as compared to patients with MCD which were associated with the degree of glomerulosclerosis, suggesting that oxidative stress occurs early and may play an important role in the pathogenesis of glomerulosclerosis. Hung Chun C et al[19] reported that plasma glutathione peroxidase levels were significantly lower (both $p < 0.01$) in FSGS patients than in either MCD patients or normal control subjects.

From the observations made in this study, it can be concluded that oxidative stress levels were significantly higher in idiopathic glomerulonephritis; the levels were much higher in significant change glomerulonephritis (membranous glomerulonephritis, membranoprliferative glomerulonephritis mesangioprliferative glomerulonephritis and focal segmental glomerulosclerosis) as compared to minimal change disease. Suggesting that more is the histopathological damage, higher were the levels and vice versa . Also the oxidative stress difference in different hishopathological types can be used for clinicopathological correlation and perhaps is a prognostic indicator in different hishopathological types. Thus future research should focus on decreasing oxidative stress by using various antioxidants, to halt the disease process and improve survival.

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LITERATURE REVIEW

Initiation of dialysis at higher GFRs: Is the apparent rising tide of early dialysis harmful or helpful?

Steven Jay Rosansky etal Kidney International 2009,76,257-261.

Over the past decade a trend of increasing estimated glomerular filtration rate (eGFR) at the initiation of dialysis for treatment of end-stage renal disease (ESRD) has been noted in the United States. In 1996, only 19% of patients began dialysis therapy with an eGFR of greater than 10 ml/min/1.73m^2 (denoted as 'early start'), but by 2005 the fraction of early start dialysis patients had risen to 45%. This review examines US dialysis data, national guidelines, and publications relevant to the early start phenomenon. It is not known whether early start of dialysis is beneficial, harmful or neutral with respect to the outcome of dialysis treatment for ESRD. Available data indicate that mortality while on dialysis therapy may be higher in those subjects with early start. Comorbidities present at the time of dialysis initiation do not appear to be a major driving force for early start patients. As well, residual kidney function in these patients is a major contributor to total urea or creatinine clearance. This can be a positive factor for patient outcomes and might be compromised by early start. Finally, we estimate the dollar cost of early start to the US Medicare-supported ESRD program. Properly designed, prospective and randomized studies may help to clarify the benefit or harm of early start of dialysis for ESRD.

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