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ENDOTHELIAL INJURY AND OXIDATIVE STRESS IN PATIENTS WITH HEPATITIS C VIRUS-RELATED CIRRHOSIS: RELATION TO RENAL FUNCTION AND HEMODYNAMICS.

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Background: Renal impairment is a common complication of cirrhosis. Endothelial injury plays an important role in the pathogenesis of chronic renal diseases and may be related to oxidative stress. The present work was designed to study markers of endothelial injury and oxidative stress in patients with hepatitis C virus-related cirrhosis in relation to renal function and hemodynamics.

Methods: Forty five treatment-naïve patients with HCV-related cirrhosis and 15 healthy subjects were included in the study. All patients had seropositivity of HCV antibodies and HCV RNA. The severity of liver disease was assessed using Child-Pugh scoring system and the Model for End Stage Liver Diseases (MELD). Glomerular injury was assessed by urinary albumin excretion rate and tubular damage was determined by measuring 24-hour urinary levels of leucine aminopeptidase (LAP). Endothelial injury was evaluated by plasma von Willibrand factor (vWF) activity and serum angiotensin converting enzyme (ACE) levels. Serum malondialdehyde (MDA) levels were measured as a marker for oxidative stress. Renal hemodynamics were assessed using Duplex-doppler ultrasonography by calculating the diastolic/systolic renal flow velocity ratio (d/s), intrarenal renal resistive index (RI) and hilar renal blood flow (RBF).

Results: Compared to healthy subjects, patients with HCV-related cirrhosis showed significant increases in plasma vWF activity, serum levels of ACE and MDA and urinary LAP levels ($P < 0.05$). Patients with cirrhosis also showed a significant increase in RI and significant decreases in d/s ratio and RBF compared with healthy subjects ($P < 0.05$). Plasma vWF activity and serum ACE levels were positively correlated with serum MDA levels, urinary albumin excretion rate, urinary LAP levels and RI and were inversely correlated with RBF ($P < 0.05$). No statistically significant correlations were found between severity of liver disease and plasma vWF activity, serum ACE levels and urinary LAP levels in patients with cirrhosis ($P > 0.05$)

Conclusion: Endothelial injury, possibly due to oxidative stress, may play an important role in the pathogenesis of renal dysfunction and increased renovascular impedence in patients with HCV-related cirrhosis. Pharmacological approaches to enhance endothelial function could have substantial benefits in improving renal function in these patients.

Keywords: Endothelial injury, LIVER CIRRHOSS, Oxidative stress, Renal Function, Renal hemodynamics