Abstract

BACKGROUND AND AIMS: Subacute hepatic failure (SHF) is a fatal complicaton of acute viral hepatitis. Renal failure has been implicated as the main cause of death in this disease. However, renal functional and structural evaluation in such patients have not been performed. The present prospective study evaluated the renal functional and structural abnormalities in patients with subacute hepatic failure. METHODS: Fourteen consecutive patients with SHF, 11 with acute liver failure (ALF) and 15 with cirrhosis of the liver (Child's B or C) were included in the present study. All 40 patients had liver disease caused by hepatitis viruses. The glomerular filtration rate (GFR) and effective renal plasma flow (ERPF) estimations were measured in all patients by the use of technetium-99m diethylenetriaminepentaacetic acid and [131I]-labeled ortho-iodohippuric acid, respectively. Ante-mortem or post-mortem liver biopsies were performed in all patients. In three patients with SHF, post-mortem kidney biopsies were also performed. RESULTS: Thirty six percent (5/14) of patients with SHF, 18% (2/11) of patients with ALF and 20% (3/15) of patients with cirrhosis had renal failure. Seven patients with SHF, seven with ALF and nine with cirrhosis died. All the patients with renal failure in each of the three groups were among the deceased patients. Glomerular function was markedly affected among patients with SHF, which was shown by significantly higher ($P < 0.05$) proteinuria levels (0.367 +/- 0.38 g/24 h) compared to levels in patients with ALF (0.178 +/- 0.11 g/24 h) and cirrhosis (0.212 +/- 0.133 g/24 h). The GFR in SHF (56 +/- 27 mL/min per 1.73 m2) and cirrhotic patients (58 +/- 36 mL/min per 1.73 m2) was significantly lower compared to those in ALF patients (102 +/- 51 mL/min per 1.73 m2; $P < 0.05$). A significantly higher proportion ($P < 0.05$) of patients with SHF and cirrhosis (64 and 73%, respectively) had a GFR below 80 mL/min per 1.73 m2 compared to patients with ALF (18%). The GFR value among the deceased SHF patients (46 +/- 26 mL/min per 1.73 m2) was significantly lower ($P < 0.05$) than those SHF patients who survived (65 +/- 25 mL/min per 1.73 m2). However, similar features could not be documented among patients with ALF or cirrhosis. Subtle structural changes in the glomerulus were also noted in patients with SHF. These included mesangial proliferation and thickening, basal membrane thickening and increased cellularity with interstitial edema. The ERPF was markedly reduced ($P = 0.058$) among patients with SHF (347 +/- 131 mL/min per 1.73 m2) and cirrhosis (395 +/- 137 mL/min per 1.73 m2) in comparison to ERPF documented among patients with ALF (436 +/- 217 mL/min per 1.73 m2). Such a reduction in renal tubular blood flow, along with histologic documentation of hyaline presence, bile and grannular cast in the tubule, indicated a possible tubular dysfunction in patients with SHF. CONCLUSION: It is concluded that glomerular and tubular dysfunction with subtle structural abnormalities does occur in patients with SHF. These are similar to renal changes in cirrhosis and may have similar pathogenetic mechanisms that require further evaluation.