Introduction

Patients with advanced cirrhosis and ascites show a hemodynamic disturbance characterized by low arterial pressure, high cardiac output and plasma volume, low peripheral vascular resistance and a marked stimulation of renin-angiotensin-aldesteron and sympathetic nervous system and antidiuretic hormone (Bosch et al., 1988 and Schrier et al., 1988).

Studies using specific antagonists of angiotensin-II and antidiuretic hormone had shown that the stimulation of these endogenous vasoactive system is a homostatic response to maintain arterial pressure within normal or near to normal limits. (Claria et al ., 1993).

Based on these data, peripheral arteriolar vasodilatation has been proposed as the initial event in the activation of these endogenous vasoactive system and in the pathogenesis of renal sodium and water retention and ascites formation in cirrhosis (Asbert et al., 1993).

Numerous investigations have shown that in addition to the above mentioned vasoconstrictor system, arterial pressure homostasis is a slow regulated by vasoactive substances synthesized in the vascular endothelium, namely the vasodilator nitric oxide and the vasoconstrictor endothelin (Moncada et al., 1991).

Endothelin is a bioactive polypeptide, which has a strong action promoting vascular contraction, proliferation and hypertrophy of vascular smooth muscle cell (Moncada et al., 1991).

Lerman et al.,(1991) and Veglio et al.,(1992) had reported normal or reduced plasma level of endothelin in cirrhotic patients with ascites. On the contrary, plasma endothelin concentration was found to be increased in other studies.