[Innovative use of the vascular endothelial growth factor in an experimental model of acute liver failure]

[Article in Italian]

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Abstract

Vascular Endothelial Growth Factor (VEGF) is an endothelial cell mitogen and an important stimulator of sinusoidal endothelial cell proliferation. The aim of this research was to study the effects of exogenous VEGF in a rat model of acute liver failure. The study was conducted on 64 rats (240-300 g). All rats underwent intraperitoneal injection (5 ml/kg) of 25% carbon tetrachloride (CCl4) and 75% paraffin oil. This dosage of CCl4 was devised to induce nonfatal acute liver failure with spontaneous recovery in 7 days. The animals were randomly divided into 2 groups. Group B animals underwent i.v. injection of 200 ng of VEGF165 one hour following intra-peritoneal injection of CCl4. To obtain daily liver functional tests (LFTS) and histological liver samples, 4 rats in each group were sacrificed daily up to 8 days. In group A, the liver histology showed massive periportal hepatocyte necrosis associated with portal lymphocytic infiltrates. The peak of the damage was documented at 72 hours following CCl4. Group B showed minimal necrosis, moderate periportal edema and a minimum periportal steatosis. At 48 hours steatotic changes had disappeared and the periportal edema was resolving. LFTs demonstrated severe liver damage in rats in group A. In group A the peak AST (mean 322.5 IU/L) and ALT (mean 250.25 IU/L) were recorded at 72 hours. In group B, at 72 hours the mean AST was 137 IU/L (normal < 95 IU/L) and ALT 68 IU/L(normal < 45 IU/L). The maximum levels of AST and ALT, in group B, were 152.3 IU/L and 72.3 IU/L, at 24 hours. According to our results exogenous VEGF successfully protects the liver from CCl4 induced acute liver failure. Further studies will demonstrate if exogenous VEGF can be effective in other liver injuries.