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Ischemia-Reperfuson Injury and Oxidative Damage in Human Liver Transplantation

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ABSTRACT

The aim of this study was to evaluate oxygen-dependent hepatic injury in human being following liver transplantation. Total 30 patients underwent liver transplantation. Blood samples were taken at different stages of transplantation for determination of the levels of serum lipid peroxide and serum nitric oxide. It was observed that the level of serum lipid peroxide and serum nitric oxide increased maximum at the stage of reperfusion than that of pre- reperfusion and post- reperfusion stages. These findings indicate that significant generation of ROS during I/R injury plays crucial role in the pathogenesis of liver damage in human liver transplantation.

Key words : Lipid peroxide, Nitric oxide, ROS, I/R injury

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Liver transplantation is a surgical replacement of the diseased liver with a healthy liver.Indication for this operation is end-stage liver disease (ESLD). Throughout the 1970s, liver transplantation remained a hazardous procedure that frequently failed. However, since then the results have progressively improved due to result of number of factors. These include better patient selection, improved immunosuppression and chemotherapy, better organ preservation, refinements in the operative techniques etc (Russell and Bailey).

However, even with major improvements in the logistic of organ transplantation, every transplantation starts with an inevitable insult on the graft: ischemia and reperfusion (I/R). A reperfusion injury occurs in liver transplantation model when oxygen is introduced via the blood to ischemic explant upon completion of portal vein anastomosis. Reperfusion of previously ischemic tissue may lead to an aggravation of injury (Henry *et al.*, 1992). This reperfusion injury occurs in addition to other ischemic injury which may already exist.

A variety of oxygen derived free radicals are produced during I/R injury which can cause direct oxidative damage to lipids, proteins and DNA (Davies *et al.*, 1989 and Sakamoto *et al.*, 1991). Superoxide ($O2^-$) is generated by the activation of xanthine oxidase (Marzi *et al.*, 1989) and in the presence of free iron, forms the damaging hydroxyl radical (Beckman *et al.*, 1990). Nitric oxide($NO\cdot$), itself a free radical is also liberated and reacts with superoxide (O^-2) to form peroxynitrite, eventually decomposing to the hydroxyl radical (OH^-) (Webster and Nunn, 1988). Thus, reperfusion injury is characterized by loss of endothelial cell viability, which occurs after cold ischemic storage and reperfusion of liver at transplantation

Therefore, in the present study it was planned to evaluate effect of I/R injury on oxidants.

MATERIALS AND METHODS

The present study was carried out in the Department of Biochemistry, Global Hospitals, Hyderabad and Dr. V.M. Govt. Medical College, Kolhapur. Liver transplantations were performed at Global Hospitals, Hyderabad. Transplantations were carried out using both the techniques as orthotopic liver transplantation and live liver transplantation. Total 34 patients were included in present study. The age group of patients was between 7-52 yrs.

Blood samples were taken into non-heparinised vacutainer to study serum lipid peroxide by Kei Satoh's method (Satoh, 1978)