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Brief Note

Plasma Endotoxin and Serum Lipid Peroxide after Transcatheter Arterial Embolization in Liver Cancer

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Key words : endotoxin — lipid peroxide — transcatheter arterial embolization (TAE) — liver damage — mechanism

Transcatheter arterial embolization (TAE) has been widely used in the treatment of liver cancer. TAE is known to induce necrosis of tumor cells by the blockage of blood supply to the liver tumor. But TAE also induces a decrease in the reticuloendothelial function and an increase in endogenous endotoxin (Et).[1] It has been reported that administration of exogenous endotoxin in rats caused liver damage, in which the free radicals and lipid peroxidation played the major roles.[2–5] The present study was undertaken to elucidate the mechanism of liver damage after TAE from the standpoint of the plasma Et level and serum lipid peroxide (LPO) concentration.

MATERIAL AND METHOD

Thirteen New Zealand white rabbits, weighing 3 kg, were used for the examination. VX2 carcinoma[6] cells ($6–9 \times 10^5$ cells) were injected into liver parenchyma under general anesthesia with sodium pentobarbital. Two weeks after implantation, a 3-French catheter was introduced from the femoral artery and fixed at the proper hepatic artery. TAE was performed with gelfoam powder. Plasma Et and serum LPO were examined every hour up to six hours after TAE. Plasma Et was assayed by the Perchloric acid-Limulus colorimetric test (Toxicolor test®, Seikagaku Kogyo Co., Ltd)[7,8] and serum LPO was determined by thiobarbituric acid (Yagi's method[9]).

RESULTS AND DISCUSSION

Serial changes in the plasma Et level and serum LPO concentration after TAE are shown in Fig. 1. The plasma Et level began to increase abruptly from three hours after TAE and continued to increase until six hours. The serum LPO concentration also increased significantly and reached a plateau from three to six hours after TAE.

Exogenous endotoxin administration is known to increase intrahepatic LPO, in which Xanthine oxidase as a free radical producer increases and superoxide dismutase (SOD) as a scavenger of free radicals decreases.[4,5] The present study showed that both the plasma Et level and serum LPO concentration increased

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at the third hour after TAE. These findings suggest that TAE may induce an increase in endogenous Et, which stimulates free radical production of Kupffer cells[10] as a reaction to exogenous Et. Then the free radicals may provoke peroxidation of the unsaturated fatty acid in the plasma membrane, resulting in the production of LPO in the liver. Finally, the resulting LPO and the free radicals may cause liver cell injury.

Arii *et al.*,[10] however, reported that SOD activity in the liver was increased by injection of a low dose of Et. This study showed that the serum LPO concentration had increased significantly at the third hour after TAE and remained on a plateau from three to six hours after TAE. From these findings, it was proposed that the scavenging system of free radicals was increased by endogenous endotoxemia from three to six hours after TAE.

The results of this study seem to suggest that liver damage after TAE is caused by not only endogenous endotoxemia but also by LPO and the free radicals. In addition, endogenous endotoxemia might stimulate the scavenging system of free radicals after TAE. Further investigation of this point is needed.

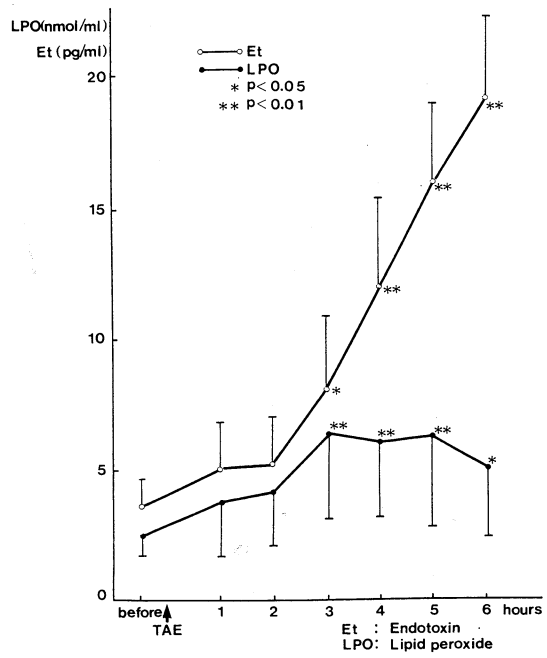

Fig. 1. The time course of the plasma endotoxin level and serum lipid peroxide concentration after experimental TAE.

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