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Brief Note

Serum Lipoperoxide in Liver Diseases

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ABSTRACT. Lipoperoxide is known to be related not only to the direct damage of biomembranes but also to cancer and arteriosclerosis. In this study, the serum lipoperoxide level in patients with chronic liver diseases (52 cases) and hepatocellular carcinoma (HCC) after transcatheter arterial embolization (TAE, 11 cases) was measured and compared with the arterial ketone body ratio (AKBR). Serum lipoperoxide increased significantly in liver cirrhosis and alcoholic liver disease and HCC. A negative correlation ($P<0.01$) was found between lipoperoxide and AKBR. Serial changes in lipoperoxide and AKBR showed reciprocal changes in HCC after TAE. Therefore, simultaneous measurement of lipoperoxide and AKBR could be useful in the evaluation of the prognosis of TAE.

Key words : AKBR — MDA — chronic liver diseases

The toxicity of lipoperoxide in the animal body has been widely discussed. A high level of lipoperoxide has been related not only to direct damage of biomembranes but also to cancer and arteriosclerosis.[1-4] We showed that transcatheter arterial embolization (TAE) for hepatocellular carcinoma (HCC) induced a decrease in the arterial ketone body ratio (AKBR).[5,6] In this study, serum lipoperoxide was measured in patients with chronic liver diseases and HCC after TAE.

Six healthy subjects and 52 cases of chronic liver diseases (10 cases of chronic hepatitis, 8 cases of liver cirrhosis, 28 cases of HCC and 6 cases of alcoholic liver disease) were selected. The serum lipoperoxide level was measured using the modified method of Yagi.[1-4] Serum lipid was precipitated along with serum protein and made to react with thiobarbituric acid. The reaction product was assayed by fluorometry with excitation at 515 nm and emission at 553 nm. The lipoperoxide values were expressed in terms of malondialdehyde (MDA, nmol/0.5 ml of serum). AKBR was measured according to the method

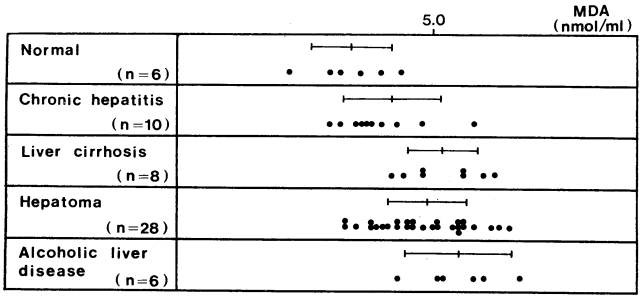

Fig. 1. Distribution of MDA in chronic liver diseases

山本亮輔，山本晋一郎，井手口清治，大元謙治，高取敬子，大海庸世，日野一成，平野　寛

of Williamson[7] (Ketorex®, Sanwa Chemical Corporation).

Fig. 1 shows the serum lipoperoxide levels in chronic liver diseases. Normal controls had a level of 3.4±0.78 nmol/ml of MDA, while the mean MDA levels were 4.2±0.95 nmol/ml in chronic hepatitis, 5.2±0.67 nmol/ml in liver cirrhosis, 4.9±0.77 nmol/ml in HCC, and 5.5±1.05 nmol/ml in alcoholic liver diseases. Statistically significant differences were observed between normal controls and liver disease patients with the exception of chronic hepatitis.

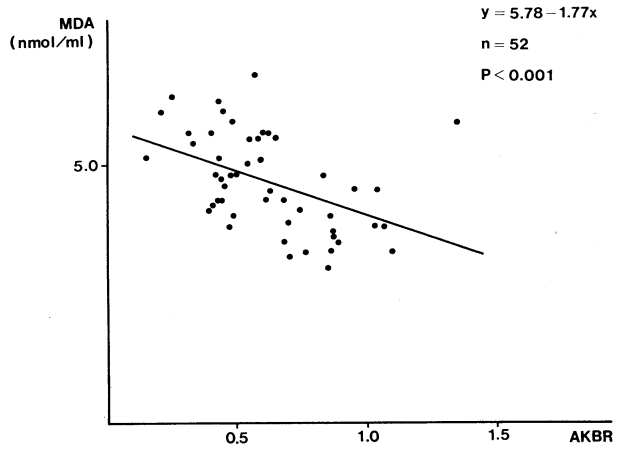

Fig. 2. Correlation between AKBR and MDA

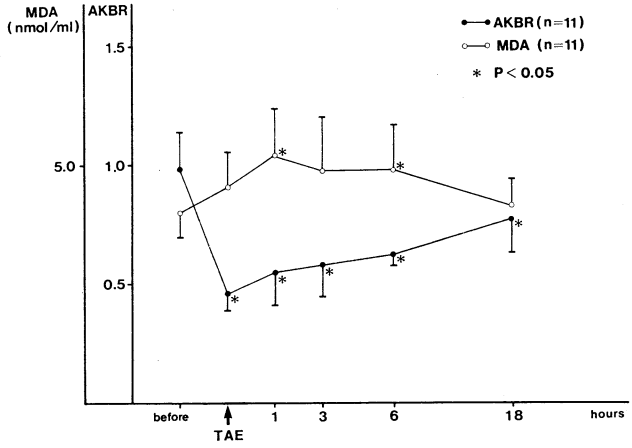

Fig. 3. Serial changes of AKBR and MDA in HCC after TAE

Fig. 2 shows the correlation of MDA with AKBR in 52 cases of liver diseases. There was a negative correlation ($p<0.01$) between MDA and AKBR. Fig. 3 shows serial changes in MDA and AKBR after TAE in 11 cases of HCC. MDA increased from 4.0±0.52 nmol/ml to 5.2±0.98 nmol/ml one hour after TAE, but it decreased to 4.15±0.57 nmol/ml 18 hrs later. On the other hand, AKBR decreased from 0.98±0.16 to 0.46±0.07 immediately after TAE, but it recovered to 0.78±0.15 18 hrs later. The above results indicate that reciprocal changes in MDA and AKBR occurred after TAE.

There have been few reports concerning lipoperoxide in liver diseases. Suematsu *et al.*[1] reported that serum lipoperoxide increased due to parenchymal disorders of the liver. Chiba *et al.*[8] reported that an increase in lipoperoxide was noted in 50% of liver cirrhosis cases. We obtained similar results in liver cirrhosis and alcoholic liver disease. The reason for high levels of lipoperoxide in chronic liver diseases has not yet been clarified, but experimental hemorrhagic shock[9] and endotoxin shock[10] induce a decrease in superoxide dismutase and a subsequent increase in lipoperoxide. A transient decrease in the arterial blood flow of the liver by TAE might correlate with an increase in lipoperoxide. Therefore, simultaneous measurement of AKBR and MDA could be useful in the evaluation of the prognosis of TAE.

Ryosuke YAMAMOTO, Shinichiro YAMAMOTO, Seiji IDEGUCHI, Kenji OHMOTO, Keiko TAKATORI, Tsuneyo OHUMI, Kazunari HINO and Yutaka HIRANO

Division of Gastroenterology I, Department of Medicine, Kawasaki Medical School, Kurashiki 701-01, Japan

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