Differentiation of Diabetes Mellitus Caused by Liver Cirrhosis from Other Type 2 Diabetes Mellitus

Background: The liver plays important roles in the homeostasis of glucose metabolism as a target organ for insulin and as a site for gluconeogenesis and glycogen storage. Diabetes mellitus (DM) commonly develops in patients with liver cirrhosis as the result of hepatocyte dysfunction and/or inadequate mass. **Aim:** To assess differences between DM due to liver cirrhosis (hepatogenous DM) and other type 2 DM (genuine type 2 DM), we compared the patterns of hyperglycemia and hyperinsulinemia in hepatogenous DM with those observed in genuine type 2 DM. **Methods:** 18 diabetic patients with liver cirrhosis (caused by alcohol, n=8; HBV, n=5; HCV, n=2; others, n=3) were matched with 18 type 2 diabetic patients without liver cirrhosis for age and gender. None of the patients or controls had been treated with insulin or β-blockers. The level of glycosylated hemoglobin (HbA1C), fasting blood sugar (FBS), postprandial blood sugar (PP2h), fasting levels of plasma C-peptide and insulin were measured. **Results:** The ratio of PP2h/FBS (2.27 vs. 1.69) and fasting plasma insulin/C-peptide ratio (8.47 vs. 4.26) were significantly higher in hepatogenous DM than genuine type 2 DM (p<0.05). In addition, the HOMA-IR index (8.38 vs. 3.52) was also higher in cirrhotics but FBS, PP2h, fasting insulin and HbA1C in cirrhotics were not significantly different from those of controls. **Conclusions:** The ratio of PP2h/FBS and fasting plasma insulin/C-peptide differentiated hepatogenous DM from genuine type 2 DM. Insulin resistance in liver cirrhosis was higher than genuine type 2 DM, and impaired hepatic insulin degradation was important to hyperinsulinemia in liver cirrhosis. A larger case–controlled study will be required about the clinical implication of hepatogeneous DM as well as cellular mechanisms of hyperinsulinemia and insulin resistance in liver cirrhosis.

색인단어: Liver cirrhosis, DM, HbA1C, Insulin resistance, C–peptide