CHARACTERISTIC CHANGE IN SERUM AMINO ACID LEVELS IN DIFFERENT TYPES OF HEPATIC ENCEPHALOPATHY

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Summary

Serum amino acid patterns in patients with different types of hepatic encephalopathy were investigated. Marked elevations in most of serum amino acids observed in untreated patients with acute type of fulminant hepatitis were not remarkable in the patients who have already treated; particularly branched chain amino acids (BCAA), phenylalanine and tyrosine were much lower in the latter group. However, elevation of serum methionine levels and lower ratio of BCAA/(phenylalanine + tyrosine) were similarly observed in both groups. In encephalopathic patients with decompensated cirrhosis, many amino acids such as phenylalanine, tyrosine and methionine were elevated with a slight depressed levels of serum BCAA. Highly significant decrease in serum BCAA levels and no elevation of phenylalanine and methionine with a minimal increase of tyrosine were observed in patients with chronic type of hepatic encephalopathy; other amino acids except for glutamine and arginine were much lower as compared to those in decompensated cirrhotics and even to the control values.

Key Words: serum amino acid, branched chain amino acid, hepatic encephalopathy, fulminant hepatitis, liver cirrhosis.

Introduction

Plasma amino acid abnormality has been described in patients with severe liver diseases such as fulminant hepatitis and liver cirrhosis. The change includes the elevated levels of aromatic amino acids, phenylalanine, tyrosine, and free tryptophan, and of methionine and the depressed concentrations of branched chain amino acids (BCAA), leucine, valine and isoleucine. There is some evidence that these abnormal patterns of plasma amino acids might be causally related to the pathogenesis of hepatic encephalopathy, since normalization of these impaired aminograms by infusing a BCAA-rich solution to comatose patients has resulted in improvement of neuropsychiatric abnormalities.

The amino acid patterns in the circulating blood are variable depending upon the pathophysiologic state of injured livers, nutritional and hormonal conditions during clinical courses of even the same patient. These facts prompted us to determine serum aminogram of
encephalopathic patients with liver disease much more frequently during their clinical courses by using a rapid procedure for measuring serum neutral amino acid levels. The impaired amino acid patterns in liver disease could be used as one of clinical measures to diagnose the disease, to evaluate the efficiency of therapies performed and to predict urgency of hepatic encephalopathy and prognostic significance. Therefore, aminogram abnormalities in patients with different etiologies of hepatic encephalopathy might be of clinical importance for judging an appropriate therapeutic modality and predicting its efficacy.

This communication was thus undertaken to examine whether serum amino acid patterns in patients with different types of hepatic encephalopathy can be differentiated from each other.

**Subjects and Methods**

Thirty-two patients with fulminant hepatitis (14 cases) and liver cirrhosis (18 cases), who admitted to Okayama University Hospital from 1978 to 1980, were examined in this study. There were 21 male and 11 females. Their ages ranged between 15 and 65 with a mean age of 52. Patients with hepatic encephalopathy were divided into the following five groups. Group A consisted of 7 patients with early stage of fulminant hepatitis (acute type) who have received no treatments yet. Group B consisted of 5 cases with fulminant hepatitis (acute type) who have already received various therapies mostly including daily infusion of a large dose of glucose. Direct hemoperfusion and hemodialysis therapy, which have serious effects on serum amino acid levels, were excluded from both groups. Group C comprised of only 2 patients with fulminant hepatitis (subacute type). Group D consisted of 10 patients with decompensated stage of liver cirrhosis, which are most frequently encountered as hepatic failure. Group E comprised of 8 cases with well compensated stage of liver cirrhosis and repeated type of hepatic encephalopathy. Ten subjects (6 male and 4 female with an average age of 41) are selected as healthy controls and have no history of liver disease, and all were clinically well nourished. At the time when serum amino acids were determined, all the patients were under Grade III or IV of hepatic encephalopathy. Mean grade of encephalopathy was not essentially different in these groups of patients. They did not receive any nutrients via a nasogastric tube.

Blood samples from encephalopathic patients were obtained before any specific treatment for encephalopathy including continuous intravenous infusion of glucose and/or amino acids was started. Serum was separated and deproteinized with 3% sulfosalicylic acid, and amino acid concentrations were measured according to our previous method.

Results of routine liver function test in five groups of patients are summarized in Table 1.