Glycogen and Liver Dysfunction in Anorexia Nervosa

Anorexia nervosa is one of the most well-known eating disorders and the patients manifest fear of weight gain and subsequent refusal of food intake. Recently, various neuroendocrine abnormalities have been reported in patients with anorexia nervosa, which may be related to the etiology and/or pathogenesis of this disorders. In addition, patients with anorexia nervosa have been demonstrated to be associated with dysfunction of various organs, including those related to malnutrition or starvation. Liver dysfunction was previously reported in patients with anorexia nervosa. Nordgren and Scheele (1) first reported two patients of anorexia nervosa associated with evidence of liver and pancreas injury in 1977. In these cases, both amylase and liver enzymes became normal in association with gain of body weight. Therefore, they proposed that nutritional pancreas and liver dysfunction represent one of the complications of patients with anorexia nervosa. Wada et al (2) also reported a 17-year-old girl with anorexia nervosa with liver dysfunction and rhabdomyolysis resulting in renal failure. Yamada et al (3) recently reported a 22-year-old patient with anorexia nervosa with recurrent hypoglycemic coma and cerebral hemorrhage associated with hepatic dysfunction. However, the histological features of the liver of patients with anorexia nervosa, which are essential to understanding the pathophysiology and pathogenesis of liver function in these patients, have not been reported in detail. In this issue, Komuta et al (4) reported an interesting case of anorexia nervosa with liver dysfunction and described the morphological features of the liver including the electron microscopic features.

See also p 678.

The histological feature of the liver in this patient was the accumulation of glycogen in the cytoplasm of hepatocytes, confirmed by PAS stain and electron microscopy. It is well known that malnutrition causes various histological changes such as fatty metamorphosis, non-specific periportal inflammatory cell infiltration and glycogen accumulation (5, 6). This glycogen accumulation in hepatocytes in patients with anorexia

nervosa does not necessarily provide information directly related to the pathogenesis and/or pathophysiology of liver dysfunction of these patients. However, this histological finding and its possible biological significance may provide new insight into the adaptative or defensive mechanism in response to starvation or malnutrition such as that observed in anorexia nervosa. Profound hypoglycemia may occur in anorexia nervosa and is sometimes lethal. However, hypoglycemic coma is a relatively rare event in anorexia nervosa and only seven cases have been reported (7). Depletion of glycogen and defective gluconeogenesis have been proposed as the pathogenesis of profound hypoglycemia in the patients with anorexia nervosa (7). Therefore, glycogenic accumulation in the hepatocytes is considered to play an important role in the prevention of these fatal hypoglycemic episodes and may represent an adaptative or defensive response to malnutrition.

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