Factors Associated with Osteonecrosis in the Femoral Head in Chronic Alcoholism

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Summary: While alcohol abuse is a possible etiologic factor in osteonecrosis in the femoral head (ON), the relationship between alcoholic liver dysfunction and ON is uncertain. Among 336 patients with alcoholic liver dysfunction who had radiographic examination of the hip at two hospitals for alcohol abuse treatment in southern Japan, the records for 291 men and 1 woman (mean age, 47.8 years; range, 24 to 72 years) had adequate information available concerning daily and cumulative alcohol intake, duration of intake, serum concentrations of liver enzymes, and platelet count. These variables were investigated for any correlation between the 8 patients with radiographic evidence of ON and the 284 without. Liver biopsy was performed in 223 patients. Except for alanine aminotransferase, liver enzyme concentrations were significantly lower in patients with ON than in those without. Histologically, 2 patients with ON were diagnosed with cirrhosis; 1 with pre-cirrhotic changes; and 2 with fibrosis. These results suggested that ON occurred in the late stages of liver disease when serum enzyme concentrations had returned to normal or were only mildly elevated.

Key words osteonecrosis in the femoral head, alcoholism, liver dysfunction, pathological diagnosis, roentgenographic survey

INTRODUCTION

Alcohol abuse is a possible risk factor for osteonecrosis in the femoral head (ON) [1-5], but a causal relationship between alcoholic liver dysfunction and ON is uncertain. We have investigated this relationship with respect to clinical and laboratory parameters and the cause of hepatic disease.

MATERIALS AND METHODS

During a 70-month period (August 1986 to June 1992), 336 patients with alcoholic liver dysfunction were admitted to two hospitals for alcohol abuse treatment in southern Japan. During their stay, hip roentgenography was performed using both anterior-posterior and Lauenstein views. Of these patients, 291 men and 1 woman (mean age, 47.8 years; range, 24 to 72 years) presented detailed information available concerning daily alcohol intake, duration of alcohol intake, and cumulative intake; serum activities of aspartate aminotransferase (AST), alanin aminotransferase (ALT), and $\gamma$-glutamyl transferase (GGT); AST/ALT ratios; and platelet counts. These 292 cases were grouped radiographically according to the presence or absence of ON. Liver biopsy was performed in 223 of the 292 cases. Patients with the following conditions were excluded: ON due to trauma such as dislocation of the hip joint; a history of systemic corticosteroid use; alcoholic psychosis; and caisson disease.

RESULTS

Of the 292 patients, 8 (2.7%) were diagnosed with ON according to roentgenograms. Less severe changes were noted in 100 patients (34.2%), who had irregularly calcified areas or translucent or cystic areas. The 292 subjects consumed a mean of 148.0 g/day of alcohol (range 43 to 536 g), and had con-
Alcohol increases fat storage in hepatocytes, resulting eventually in the morphological characteristics of fatty liver. The fatty liver can release fat globules into the circulation that are sufficiently large to act as showers of emboli, producing intraosseous vascular occlusion leading to ON [6-9]. While disturbed liver function has been reported to be an important etiological factor in ON [10], Mori et al. [11] have disagreed, finding that a history of liver disease was not significantly associated with occurrence onset of ON. As idiopathic ON can occur without alcohol abuse, ON can develop in the absence of alcoholic liver disease. All patients in this study had both chronic alcoholism and disturbed liver biopsy specimen, including cirrhosis in 2, pre-cirrhotic changes in 1, and fibrosis in the other 2. These pathological diagnoses in patients with ON represented late stages of liver disease. When age, daily alcohol intake, duration of intake, cumulative intake, liver enzyme parameters, and platelet count were compared between patients with and without ON using Student’s t test, values for AST, AST/ALT, and GGT were significantly (p<0.05) less elevated in patients with ON than in those without (Table 1). Other variables showed no significant difference.

**Discussion**

Alcohol increases fat storage in hepatocytes, resulting eventually in the morphological characteristics of fatty liver. The fatty liver can release fat globules into the circulation that are sufficiently large to act as showers of emboli, producing intraosseous vascular occlusion leading to ON [6-9]. While disturbed liver function has been reported to be an important etiological factor in ON [10], Mori et al. [11] have disagreed, finding that a history of liver disease was not significantly associated with occurrence onset of ON. As idiopathic ON can occur without alcohol abuse, ON can develop in the absence of alcoholic liver disease. All patients in this study had both chronic alcoholism and disturbed liver biopsy specimen, including cirrhosis in 2, pre-cirrhotic changes in 1, and fibrosis in the other 2. These pathological diagnoses in patients with ON represented late stages of liver disease. When age, daily alcohol intake, duration of intake, cumulative intake, liver enzyme parameters, and platelet count were compared between patients with and without ON using Student’s t test, values for AST, AST/ALT, and GGT were significantly (p<0.05) less elevated in patients with ON than in those without (Table 1). Other variables showed no significant difference.
liver function, and all required hospitalization. We considered this population to be informative regarding relationships between ON and liver dysfunction. Age, daily alcohol intake, intake duration, cumulative intake, ALT, and platelet count were not related to ON, but relatively low values for AST, AST/ALT, and GGT showed a statistically significant correlation to ON (p<0.05). In the late stage of liver disease such as cirrhosis or fibrosis, serum enzyme concentrations reportedly show milder elevations than in early stages. In the present study, late changes were confirmed in liver specimens from patients with ON.

While only 8 patients had ON, 100 (29.8%) others had less severe radiographic abnormalities including irregularly calcified areas or translucent or cystic areas in the hip. While abnormalities in hip roentgenogram have been reported to be frequent in alcoholic patients, Smith et al. [12] found comparison of hip roentgenograms between alcoholic and nonalcoholic subjects to be uninformative, with abnormalities apparent in 45.3% of alcoholic patients and in 49% of controls. More studies are necessary.

CONCLUSION

Of 292 cases of alcoholic liver dysfunction, 8 (2.7%) were associated with ON. Relatively low liver enzyme concentrations and late changes in biopsy specimens suggested that ON occurred in the late stages of alcoholic liver disease.

ACKNOWLEDGMENTS: The author thanks Professor Akio Inoue for kind advice on an earlier manuscript.

REFERENCES