

Fatty liver disease: predictors of nonalcoholic steatohepatitis and gallbladder disease in morbid obesity

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摘要

Abstract

Background Nonalcoholic steatohepatitis (NASH) and gallbladder disease (GD) are members of metabolic syndrome in morbidly obesity. Insulin resistance is a risk factor for NASH and GD. The aim of the present study was to investigate the relationship between insulin resistance (HOMA-IR), liver fibrosis, NASH and GD in morbidly obese patients who presented with fatty liver during preoperative abdominal ultrasonography examination.

Methods We studied 152 morbid obese patients with fatty liver disease including 54 with NASH, 11 with GD and two with concurrent NASH and GD that were undergoing laparoscopic bariatric surgery. Clinical data (gender, age, body mass index [BMI], and associated diseases), laboratory evaluation, and histopathology were obtained from the patient databases. We analyzed the relationship between clinical characteristics, histological parameters, HOMA-IR, and fibrosis stage associated with NASH and GD in morbid obese patients.

Results Among the 152 patients with fatty liver disease, 93 were females and 59 were males. The mean age was 30.3 ± 8.9 years and the mean BMI was 44.9 ± 5.4 kg/m². Fifty-four patients (54/152, 35.5%) were diagnosed as NASH and 11 patients (11/152, 7.2%) received concomitant laparoscopic cholecystectomy because of gallbladder disease (GD). Morbidly obese patients with fatty liver disease and GD were significantly older ($P = 0.020$), had higher serum levels of cholesterol ($P = 0.020$) and low-density lipoprotein (LDL)-cholesterol ($P = 0.044$), and had lower serum levels of total bilirubin ($P = 0.044$), C-peptide ($P = 0.023$), and insulin ($P = 0.039$) than the NASH group. Histopathology factors of hepatic steatosis ($P = 0.012$), ballooning degeneration ($P = 0.001$), lobular inflammation ($P = 0.019$), fibrosis ($P =$

0.026), and glycogenated nuclei ($P = 0.028$) were significantly different between NASH and GD groups. However, further multivariate analysis failed to demonstrate any independent clinicopathological factor. The prevalence of chronic hepatitis B and NASH was the same (18%) in all 11 GD patients. Besides, when we compared NASH patients ($n = 54$) with concurrent NASH-GD patients ($n = 2$), we found that waist ($P = 0.016$), waist/hip ($P = 0.039$), and HOMA-IR ($P = 0.040$) were independent associated factors. We further assessed the HOMA-IR distribution and the relationship between fibrosis stage in patients with NASH and GD. In the NASH group, HOMA-IR distribution progressively decreased when the severity of fibrosis was plotted as a function of insulin resistance.

Conclusion The prevalence of NASH in gallbladder disease was 18% in morbid obese population. We concluded that age, serum cholesterol, and low-density lipoprotein cholesterol levels were risk factors associated with gallbladder disease and fatty liver disease. Insulin resistance was more common in concurrent NASH and gallbladder disease. The mechanism between insulin resistance, fibrosis stage, NASH, and gallbladder disease is unknown

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