Obesity Surgery, 13, 622-624

Hepatic Steatosis In Patients Undergoing Bariatric Surgery and its Relationship to Body Mass Index and Co-Morbidities

Myriam Moretto, MD[1]; Carlos Kupski, MD[1]; Cláudio Corá Mottin, MD[2]; Giuseppe Repetto, MD[3]; Marcelo Garcia Toneto, MD[2]; Jacqueline Rizzolli, MD[3]; Diovanne Berleze, MD[4]; Cesar Luis de Souza Brito, MD[5]; Daniela Casagrande, Nutr[6]; Fernanda Colossi, Nutr[6]

[1]Department of Internal Medicine (Gastroenterology), [2]Department of Surgery, [3]Department of Internal Medicine (Endocrinology), [4]Department of Internal Medicine (Cardiology), [5]Department of Internal Medicine (Psychiatry), [6]Department of Nutrition, Centro da Obesidade Mórbida, Hospital São Lucas da PUCRS, Porto Alegre, Brazil

Background: Although non-alcoholic hepatitis usually is asymptomatic and benign, this condition may progress to cirrhosis and hepatic failure. Some findings are similar to alcoholic hepatitis, but there is no history of excessive alcohol consumption. Among the factors associated with non-alcoholic hepatitis, obesity, diabetes and dyslipidemia are the most important.

Methods: 77 consecutive patients undergoing bariatric surgery had their liver biopsy compared to the presence of co-morbidities and BMI.

Results: 67 patients (87.1%) had an abnormal liver biopsy, mostly due to steatosis (83.1%), but also steatohepatitis (2.6%) and cirrhosis (1.3%). The degree of liver damage was related to higher BMI scores. Co-morbidities were present in 46.9% of the patients with hepatic steatosis.

Conclusions: The authors suggest that a liver biopsy should be performed in all patients at bariatric surgery, in order to evaluate possible liver damage and to assist postoperative care.

Key words: Steatohepatitis, cirrhosis, morbid obesity, bariatric surgery

Presented as a poster at the 7th Annual Meeting of the International Federation for the Surgery of Obesity, São Paulo, Brazil, August 28-31, 2002.

Reprint requests to: Myriam Moretto, MD, Centro da Obesidade Mórbida – COM - Hospital São Lucas da PUCRS, Av. Ipiranga, 6690/302, Porto Alegre, Brazil 90610-000.
E-mail: ccmottin@terra.com.br

Introduction

Non-alcoholic steatohepatitis (NASH) usually occurs without symptoms. Clinical findings include an enlarged liver, occasional elevated plasma transaminases, and histologic findings that resemble alcoholic hepatitis in patients without excess alcohol consumption. Histology shows hepatocytes with triglyceride excess – steatosis – and a lobular inflammatory infiltration, which may be associated with focal necrosis and various degrees of fibrosis – hepatitis.[1-7] The incidence of NASH, like obesity, appears to be increasing, and it may be considered as an important cause of hepatic disease in western countries.[2]

NASH usually presents as a benign condition, in contrast with the hepatitis induced by chronic alcohol consumption. However, patients with NASH may also evolve to cirrhosis, and in some cases, to hepatic failure.[1,8,9] The main features associated with NASH are obesity, diabetes and dyslipidemia. Obesity is the factor usually associated with NASH, although it can occur in patients with normal weight. Some studies have shown a relationship between the severity of obesity and the corporal distribution of fat, namely, centripetal obesity.[1]

The pathogenesis of NASH is not firmly estab-

lished. Associated factors, such as obesity, diabetes, hyperinsulinemia, high levels of triglycerides, drugs, and toxins may lead to increased fatty acids. These factors cause a higher availability of fatty acids to the liver, reduction in β-oxidation, increased synthesis of fatty acids by the liver, and a reduction in synthesis and secretion of LDL cholesterol.[1] The degree of steatosis may correlate with body mass index (BMI), and usually is more severe than in alcoholic hepatitis. The degree of lobular inflammatory infiltration in NASH shows a wide variation, mostly with lymphocyte, neutrophil and mononuclear cell infiltration.

Hepatic fibrosis may present with varying degrees of intensity, surrounding small veins or forming thick and dense septa, altering liver architecture.[1,2]

The aim of this study was to show the hepatic architectural changes in morbidly obese patients undergoing bariatric surgery, and their relation to BMI and co-morbidities.

Matherial and Methods

At the Centro da Obesidade Mórbida do Hospital São Lucas da PUCRS, 161 morbidly obese patients underwent bariatric surgery in a 22-month period. Starting in July 2001, liver biopsy became routine during the operation to evaluate possible histologic changes. Seventy-seven patients were eligible for study from July 2001 to March 2002. The liver biopsies were studied and compared with BMI and the presence of two co-morbidities, dyslipidemia and diabetes. Patients were divided in three categories: BMI 35-40, 40.1-50, and >50 kg/m^2.

Regarding co-morbidities, patients were considered as having diabetes when at least two fasting glycemic levels were >126 mg/dl. Dyslipidemia was considered present when total cholesterol and/or triglycerides were >200 mg/dl.

Histologic studies of liver biopsies classified patients as having a normal biopsy, steatosis, steatohepatitis or cirrhosis. Patients with steatosis were further divided as mild, moderate or severe steatosis.

The statistic was descriptive, with variables expressed as frequency percentage.

Results

Over the 9-month period, 77 consecutive patients underwent bariatric surgery with liver biopsy. There were 58 females (75.3%) and 19 males (24.7%), with average age 36.1 years (range 18-60 y). Average BMI was 48.1 kg/m^2 (range 37.1-82.5).

In the liver biopsies, 67 patients (87.1%) had abnormal findings in hepatic structure. Most had steatosis (64 patients, 83.1%), with the others having steatohepatitis (two patients, 2.6%) and cirrhosis (one patient, 1.3%) (Figure 1). Ten patients had a normal biopsy (12.9%).

Regarding patients with steatosis, there were 18 patients classified as having mild steatosis (28.1%), 17 with moderate steatosis (26.5%) and 29 with severe steatosis (45.3%). When analyzing BMI and severe steatosis, three patients (10.3%) had BMI 35-40, 11 patients (37.9%) had BMI 40.1-50, and 15 patients (51.7%) had BMI >50 (Figure 2).

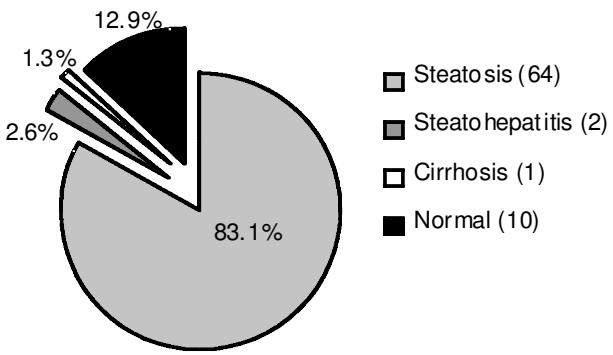

Figure 1. Liver biopsies at bariatric surgery.

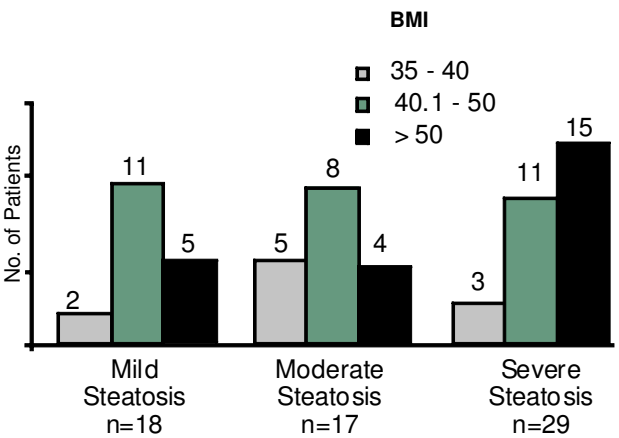

Figure 2. Hepatic steatosis amd BMI.

In the co-morbidities studied, of the 64 patients with steatosis, 30 (46.9%) had at least one co-morbidity and 34 patients (53.1%) had none (Figure 3).

Discussion

Although alcoholic steatohepatitis and NASH share some common histologic findings, the prognosis seems to be very different.[7] The long-term outcome of NASH is not known, but a 5-year follow-up has shown a 67% survival rate for NASH patients compared to 38% for alcoholic hepatitis.[1] Current data suggest that NASH may be the major cause for the "cryptogenic cirrhosis".[1,2]

Obesity is a well known cause of hepatic steatosis, and in the present study we found that in a sample of 77 patients, 83.1% had steatosis, a result that is similar to the literature. Gholam et al[10] found steatosis in 84% of morbidly obese patients at time of gastric bypass. In almost half of our patients (45.3%), the steatosis was severe. One of the main factors relating NASH with fibrosis is the severity of steatosis.[11] We found that the severity of liver damage was greater in patients with BMI >50.

We found no relationship in the finding of severe steatosis and the presence of dyslipidemia or diabetes. This suggests that the pathophysiology of steatosis is not related only to the presence of co-morbidities, and that obesity *per se* is an important factor.

We believe that it is important to perform a liver biopsy during bariatric surgery, to verify the presence and degree of liver damage, the behavior of liver architecture after weight loss, and the relevance of NASH in the evolution to cirrhosis.

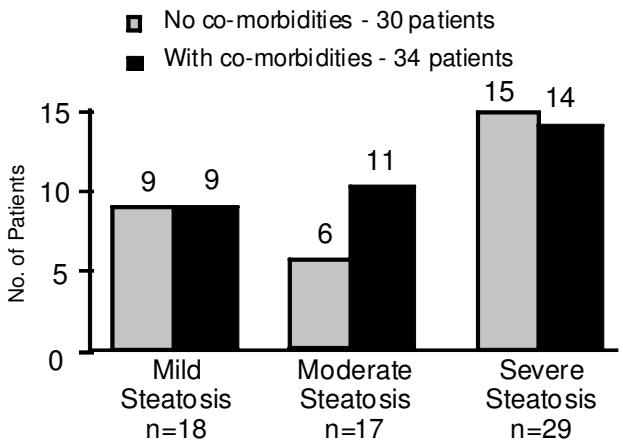

Figure 3. Association of steatosis and co-morbidities.

References

1. Reid AE. Nonalcoholic steatohepatitis. Gastroenterology 2001; 121: 710-23.
2. James OFW. Nonalcoholic steatohepatitis. In: Arroyo V, Bosch J, Rodès J et al, eds. Therapy in Hepatology. Barcelona: Ars Medica 2001: 381-6.
3. Del Gaudio A, Boschi L, Del Gaudio GA et al. Liver change in obese patients. Obes Surg 2000; 12: 802-4.
4. Spaulding L, Trainer T, Janiec D. Prevalence of non-alcoholic steatohepatitis in morbidly obese subjects undergoing gastric bypass. Obes Surg 2003; 13: 347-9.
5. Crespo J, Fernández-Gil P, Hernández-Guerra M et al. Are there predictive factors of severe liver fibrosis in morbidly obese patients with non-alcoholic steatohepatitis? Obes Surg 2001; 11: 254-7.
6. Dixon JB, Bhatal PS, O'Brien PE. Nonalcoholic fatty liver disease: predictors of nonalchoholic steatohepatitis and liver fibrosis in the severely obese. Gastroenterology 2001; 121: 91-100.
7. Burt AD, Mutton A, Day CP. Diagnosis and interpretation of steatosis and steatohepatitis. Sem Diagnos Pathol 1998; 15: 246-58.
8. Shet SG, Gordon FA, Chopra S. Non-alcoholic Steatohepatitis. Ann Intern Med 1997; 126: 137-45.
9. Marchesini G, Brizi M, Margeli – Labate A, et al. Association of nonalcoholic fatty liver disease with insulin resistance. Am J Med 1999;107: 430-3.
10. Gholam PM, Kottler DP, Flancbaum LJ. Liver pathology in morbidly obese patients undergoing Roux-en-Y gastric bypass surgery. Obes Surg 2002; 12: 49-51.
11. Day CP, James OFW. Hepatic steatosis: innocent bystander or guilty party? Hepatology 1998; 27: 1463-6.

(Received August 30, 2002; accepted December 27, 2002)