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ORIGINAL CONTRIBUTIONS

Outcomes of Bariatric Surgery in Patients with Cirrhosis

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Abstract

Context Information concerning the risk–benefit profile of bariatric surgery in subjects with liver cirrhosis is scarce. Our aim was to describe the long-term outcomes of bariatric surgery in a cohort of patients with liver cirrhosis submitted to bariatric surgery.

Methods This was a multicenter, retrospective observational study performed by the Obesity Group of the Spanish Society of Endocrinology and Nutrition (GOSEEN), with a review of patients with cirrhosis who had undergone bariatric surgery during the period from April 2004 to March 2017 in ten public reference hospitals in Spain.

Results Data on 41 patients with cirrhosis submitted to obesity surgery were collected (mean age 53.8 ± 7.9 years, 46.3% women, presurgical BMI 45 ± 8.3 kg/m^2). All but one patient belonged to Child–Pugh class A, and sleeve gastrectomy was conducted in 68.3% of cases. Percentage of total weight loss (%TWL) was 26.33 ± 8.3% and 21.16 ± 15.32% at 1 and 5 years after surgery, respectively. This was accompanied by a significant reduction of type 2 diabetes, high blood pressure, and dyslipidemia and by an improvement of liver enzymes over time. Model for End-Stage Liver Disease (MELD) index increased from 7.2 ± 1.9 to 9.8 ± 4.6 after 5 years. Seven patients (17%) developed early postsurgical complications. No postsurgical mortality was observed. During follow-up, only five patients developed liver decompensation.

Conclusions Bariatric surgery in selected patients with liver cirrhosis has metabolic benefits that could have a positive impact on liver prognosis.

Trial Registration Controlledtrials.com Identifier: 10.1186/ISRCTN15009106

Keywords Obesity surgery · Liver · Cirrhosis

Introduction

While obesity is reaching epidemic proportions, one of its metabolic derangements, non-alcoholic fatty liver disease (NAFLD), is also increasing in frequency and it is present in up to 74% of patients with morbid obesity who are candidates for obesity surgery [1]. Patients with NAFLD may eventually progress to steatohepatitis (NASH) and cirrhosis [2], with a prevalence of advanced hepatic fibrosis in bariatric surgery candidates ranging from 1 to 7% [3].

Obesity is known to have a deleterious effect on the natural course of cirrhosis irrespectively of its origin [4], and obesity surgery could be beneficial for patients with cirrhosis through weight loss and improvement in metabolic comorbidities [5]. Furthermore, the prevalence of obesity among liver transplant candidates reaches almost 30% [6]. The weight loss obtained after obesity surgery could improve eligibility for liver transplantation among patients with end-stage liver disease, arising as an attractive treatment for morbid obese patients with cirrhosis. However, patients with liver cirrhosis undergoing surgery for any reason have a worse prognosis when compared to their non-cirrhotic counterparts [7–9] and information concerning the risk–benefit profile of bariatric surgery in subjects with liver cirrhosis is scarce and limited to small series of patients with a short follow-up. The aim of the present work is to describe the long-term outcomes of bariatric surgery in a cohort of patients with biopsy documented cirrhosis, focusing

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Materials and Methods

This was a multicenter, retrospective observational study performed by the Obesity Group of the Spanish Society of Endocrinology and Nutrition (GOSEEN), with a review of cases with liver cirrhosis who had undergone bariatric surgery during the period from April 2004 to March 2017 in ten public referral hospitals in Spain. Patients were included in the study if they had a minimum follow-up of 12 months after bariatric surgery and if cirrhosis was documented by a biopsy. Detailed clinical and biochemical data were retrospectively collected by reviewing the medical records. The study was conducted according to the principles of the Declaration of Helsinki, and all the patients signed an informed consent form approved by the institutional ethics committee. This trial was registered at www.controlledtrials.com as ISRCTN15009106.

Questionnaire

A questionnaire was developed to collect demographic data and anthropometric characteristics of the patients. Clinical data concerning metabolic comorbidities of obesity and their treatment (diabetes, hypertension, dyslipidemia, and obstructive sleep apnea hypopnea syndrome (OSAHS)) as well as weight (measured in the absence of ascites) and blood pressure were also collected. Data regarding cirrhosis included its etiology, previous and postsurgical liver decompensations, hepatocellular carcinoma, clinically significant portal hypertension (defined by the presence of esophageal varices or portal hypertensive gastropathy, a previous ascitic decompensation or by a hepatic venous pressure gradient > or equal to 10 mmHg), and liver function assessed by MELD (Model for End-Stage Liver Disease) and Child–Pugh scores. Diabetes remission was defined as the presence of a HbA1c < 6.5% in the absence of pharmacologic treatment [10]. Laboratory tests including glycemia, glycosylated hemoglobin (HbA1c), lipid profile, liver enzymes, albumin, platelets, and international normalized ratio International Normalized Ratio (INR) were registered. Moreover, acute hepatic complications as well as general surgical complications after surgery were recorded. All data were collected preoperatively and at 12, 24, 36, 48, and 60 months after bariatric surgery.

Statistical Analysis

Descriptive statistics were used to analyze clinical characteristics using mean ± SD for continuous variables and number and percentage for categorical variables. Normality of data distribution was evaluated using the Kolmogorov–Smirnov test. The relationship between qualitative variables was assessed using the chi-square test. Additionally, *t* test and Mann–Whitney test were used to analyze independent samples. Paired *t* test and Wilcoxon test were used to analyze changes between baseline and subsequent data at 1, 2, 3, 4, and 5 years. *P* values < 0.05 were considered as statistically significant. Data were analyzed using SPSS (Statistical Package for the Social Sciences) version 20.0 for Windows.

Results

Baseline Characteristics

Forty-one patients with cirrhosis and morbid obesity were included in the study. Sleeve gastrectomy was performed in 68.3% of patients, gastric bypass in 26.8%, and biliopancreatic diversion (BPD) in 4.9%. Mean follow-up time was 3.2 ± 1.75 years. All patients completed 1 year of follow-up, 60.9% completed 2 years, 58.53% 3 years, and 41.46% 4 and 5 years of follow-up. No deaths were reported in the first year after bariatric surgery. Patient's baseline characteristics are summarized in Table 1.

The diagnosis of cirrhosis was confirmed by biopsy in all the patients, before surgery in 41.5% ($n = 17$) of patients and during bariatric surgery in the rest. The most frequent etiology of liver cirrhosis was NASH, and the second one was hepatitis C virus (Fig. 1). Concerning presurgical liver complications, two patients (4.9%) had been previously diagnosed and treated of hepatocellular carcinoma (one in remission 9 years after surgical resection and one of small size treated at the same time of bariatric surgery) and seven patients (17.1%) had presented a previous ascitic decompensation, compensated at the time of surgery. Eleven patients (26.8%) had clinically significant portal hypertension. Child–Pugh class was A in all patients except one patient who belonged to Child–Pugh class B. Mean MELD score in the whole cohort was 7.2 ± 1.9 (range 6–14). Concerning hepatic enzymes, 64.1%, 68.3%, and 70.3% had elevated AST, ALT, and GGT levels, respectively. Albumin values were normal before surgery in all the patients. No patient presented a presurgical INR above 1.5. On the other hand, 3 patients (7.3%) showed bilirubin values above 2 mg/dL and 17 patients (41.5%) presented a platelet count below 150×10^9/L.

Weight Evolution

Percentage of total weight loss (%TWL) was 26.33 ± 8.3% at 12 months and declined to 21.16 ± 15.32% 5 years after surgery. Weight loss evolution is shown in Fig. 2. %TWL and BMI evolution did not differ according to the cause of cirrhosis (NASH vs. other causes), the presence of a previous ascitic decompensation, or the type of surgery performed (sleeve vs. other types of surgery) (data not shown).

Table 1 Clinical characteristics at baseline

	Overall (n = 41)	By cause of cirrhosis*		By type of surgery*	
		NASH (n = 27)	Other (n = 14)	SG (n = 28)	Other (n = 13)
Gender (% females)	46.3	44.4	50	46.4	46.2
Age (years)	53.8 ± 7.9	54.3 ± 7.9	52.8 ± 8.2	53.1 ± 8.7	55.3 ± 6.3
BMI (kg/m^2)	45 ± 8.3	43.8 ± 7	47.4 ± 10.1	45.2 ± 9.3	44.6 ± 5.9
T2DM (%)	68.3	74.1	57.1	67.9	69.2
Hypertension (%)	63.4	59.3	71.4	60.7	69.2
Dyslipidemia (%)	36.6	33.3	42.9	39.3	30.8
OSAHS (%)	29.3	22.2	42.9	21.4	46.2

BMI body mass index, *T2DM* type 2 diabetes, *OSAHS* obstructive sleep apnea hypopnea syndrome

*Comparisons between groups were not different

Progress of Metabolic Comorbidities and Cirrhosis

Progress of metabolic comorbidities, metabolic parameters, and parameters of liver function is shown in Table 2. There were no differences in the evolution of comorbidities and of metabolic and hepatic parameters according to the etiology of cirrhosis or the type of obesity surgery performed (data not shown). Overall, 53.6% of patients showed type 2 diabetes remission at 1 year after surgery that was maintained during the follow-up. Insulin use in patients with type 2 diabetes was 64.3% at baseline and 46.1% after 1 year of surgery.

Regarding the development of liver decompensations during follow-up, two patients presented ascites in the early postsurgical period (< 30 days). Three additional patients developed late liver decompensations (ascites) at 2, 4, and 5 years post surgery. Patients who developed liver decompensations during follow-up were older (61.4 ± 11.1 vs. 52.7 ± 6.9 years; $p = 0.021$) than patients that did not develop decompensations. No other differences in baseline characteristics where found in this group of patients, including the presence of a previous ascitic decompensation or portal hypertension.

Six patients (14.6%) were diagnosed of hepatocellular carcinoma postsurgically, and a seventh one is currently being studied for the presence of hepatic nodules.

With respect to Child–Pugh score Index, the patient with presurgical Child–Pugh class B remained as Child–Pugh B after 1 year of follow-up. From the remaining Child–Pugh A patients, eight of them (20%) showed a deterioration between 2 and 5 years after obesity surgery. One of the 41 patients included in the study is currently being evaluated for the possibility of liver transplantation. Patients with deterioration of Child–Pugh score during follow-up presented higher BMI indexes at baseline (50.93 ± 10.57 vs. 43.57 ± 7.22 kg/m^2; $p = 0.024$) and at 1 year (38.44 ± 6.94 vs. 31.73 ± 5.21; $p = 0.006$), 2 years (38.10 ± 7.16 vs. 32.17 ± 5.17; $p = 0.038$), and 3 years of follow-up (39.01 ± 7.41 vs. 32.97 ± 4.76; $p = 0.038$), but no differences were found concerning %TWL. No differences were found concerning either baseline MELD index, the

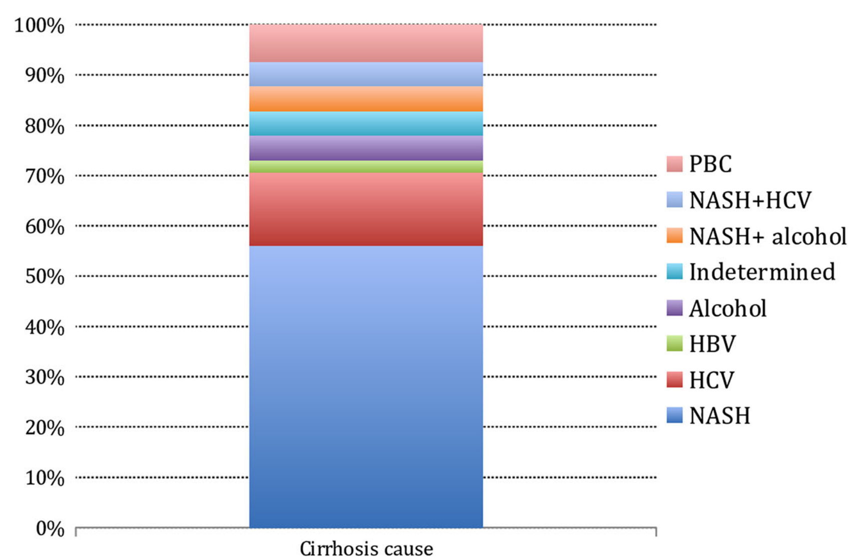

Fig. 1 Etiology of cirrhosis. NASH non-alcoholic steatohepatitis, HCV hepatitis C virus, HBV hepatitis B virus, PBC primary biliary cirrhosis

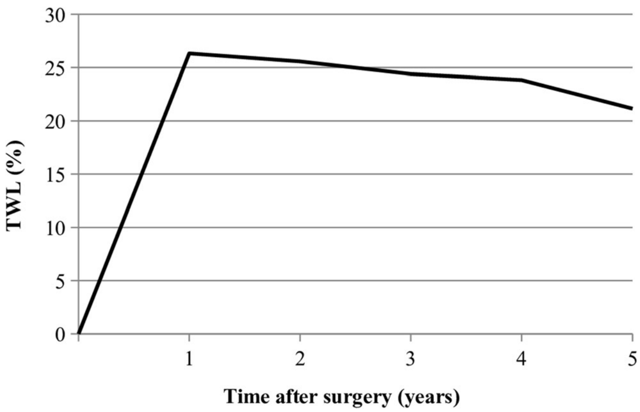

Fig. 2 Weight loss after bariatric surgery in cirrhosis patients **a** evolution of TWL **b** evolution of EWL **c** evolution of BMI

etiology of cirrhosis, the presence of baseline portal hypertension, or previous ascitic decompensation or concerning liver enzymes, except for bilirubin, which was higher in patients that presented deterioration of Child–Pugh score across follow-up (1.91 ± 1.75 vs. 0.72 ± 0.29; $p = 0.001$).

During the 5 years of maximum follow-up, only one patient died of undetermined cause at year 5, after 4 years of being lost to follow-up.

Surgical Complications

Early postoperative complications were present in seven patients (17%). One presented septic shock due to an anastomotic leak and required multiple reinterventions in the late postsurgical period due to the presence of a gastric fistula. Other early postsurgical complications were hemoperitoneum, upper gastrointestinal bleeding due to an anastomotic ulcer, wound infection, and portal thrombosis in a patient found to be a carrier of the mutation G20210A in the prothrombin gene. Two other patients developed ascitic decompensation, one of whom needed to be readmitted to hospital due to pneumonia and hyponatremia in the late postoperative period. Postsurgical mortality was 0% in this series. No baseline differences could be found between patients that presented postsurgical complications and those who did not (Table 3). Nutritional complications were not found during follow-up in any patient.

Discussion

We describe the largest series of patients with liver cirrhosis who have undergone obesity surgery finding that, in selected patients, the benefits of bariatric surgery in terms of weight loss, improvement in metabolic comorbidities, and liver prognosis outweigh the risks. Almost all cases in our series presented a Child–Pugh class A (mainly because more severe liver disease is considered a contraindication in many centers), the main cause of cirrhosis was NASH, and the most frequent surgery performed was sleeve gastrectomy.

Table 2 Clinical and laboratory parameters after bariatric surgery

	Baseline	1 year ($n = 41$)	3 years ($n = 24$)	5 years ($n = 17$)
AST (U/L)	58 ± 33.3	41.3 ± 25.3*	43.6 ± 31.4	47.9 ± 47.9
ALT (U/L)	60.3 ± 36	38 ± 27.2*	37.3 ± 22.6*	46.6 ± 59.4
GGT (U/L)	238.1 ± 311.1	129.2 ± 163.4*	199.7 ± 481.1	99.6 ± 75.2
ALP (U/L)	175.2 ± 148.5	164.2 ± 123.8	162.2 ± 88.6*	173.9 ± 108.4
Bilirubin (mg/dL)	1 ± 0.91	1 ± 1.4	1.2 ± 1.2	1.6 ± 2
Albumin (g/L)	4.2 ± 0.4	4 ± 0.4	4 ± 0.5	3.8 ± 0.7
INR	1.1 ± 0.1	1.1 ± 0.2	1.1 ± 0.3	1.2 ± 0.4
Platelet count (× 10^9/L)	183.6 ± 75.1	184.4 ± 77.9	163.4 ± 73.5	155.2 ± 79.2*
MELD score	7.2 ± 1.9	7.8 ± 2.2*	8.4 ± 3.1	9.8 ± 4.6*
Glycemia (mg/dL)	153.3 ± 60.3	105.3 ± 24.3*	113.4 ± 33.1*	105.5 ± 20.5*
HbA1c (%)	7.4 ± 2.1	5.8 ± 1.2*	5.7 ± 1.4*	5.7 ± 0.8*
TC (mg/dL)	184.8 ± 40.2	184.2 ± 43.7	180.9 ± 40.3	162.7 ± 52.3*
LDLc (mg/dL)	105 ± 30	102.3 ± 33.3	91.1 ± 21.6*	88.8 ± 29.7
HDL-c (mg/dL)	52.5 ± 18.8	57.4 ± 22.4	64 ± 24.1*	55 ± 32.3
T2DM (%)	68.3	31.7*	29.2*	29.4*
Hypertension (%)	63.4	36.6*	37.5*	35.3*
Dyslipidemia (%)	36.6	24.4*	29.2*	23.5*
OSAHS (%)	29.3	4.9	0	2.4

TC total cholesterol, *T2DM* type 2 diabetes, *OSAHS* obstructive sleep apnea hypopnea syndrome

*The comparisons with the baseline were different, p value < 0.05

Table 3 Baseline characteristics of patients according to the occurrence of postsurgical complications

	Patients with complications (*n* = 7)	Uncomplicated patients (*n* = 34)
Age (years)	57.6 ± 9.4	53.0 ± 7.6
Gender (%females)	57.1	44.1
Type of surgery (%sleeve gastrectomy)	85.7	64.7
Cirrhosis etiology (%NASH)	71.4	64.7
BMI (kg/m^2)	40.8 ± 5.0	45.9 ± 8.6
AST (U/L)	45.7 ± 29.7	60.2 ± 33.8
ALT (U/L)	42.6 ± 32.6	63.9 ± 36.0
GGT (U/L)	122.0 ± 127.3	63.9 ± 36.0
ALP (U/L)	151.4 ± 67.2	180.1 ± 160.5
Bilirubin (mg/dL)	0.8 ± 0.2	0.9 ± 1.0
Platelet count (× 10^9/L)	143.7 ± 44.0	191.7 ± 77.9
INR	1.0 ± 0.1	1.1 ± 0.1
MELD score	6.7 ± 1.2	7.3 ± 2.0
Portal hypertension (%)	42.9	17.6
Fasting plasma glucose (mg/dL)	124.8 ± 50.6	159.1 ± 61.1
HbA1c (%)	6.5 ± 2.2	7.6 ± 2.1
T2DM (%)	42.9	73.5
Hypertension (%)	42.9	67.6
Dyslipidemia (%)	28.6	38.2
OSAHS (%)	14.3	32.4

Comparison between groups were not different

NASH non-alcoholic steatohepatitis, *BMI* body mass index, *MELD* model for end-stage liver disease, *T2DM* type 2 diabetes, *OSAHS* obstructive sleep apnea–hypopnea syndrome

Postsurgical mortality in our series was 0%, which is not higher than the 0.28–0.35% described in general patients undergoing a bariatric procedure [11]. Mosko et al. [12] published data extracted from a nationwide inpatient sample found that mortality rates of patients with compensated cirrhosis were slightly superior than in patients without cirrhosis, but it increased considerably in those with decompensated cirrhosis (16.3% vs. 0.3% and 0.9% in patients without cirrhosis or with compensated cirrhosis, respectively). Our mortality results may therefore be a reflection of the relatively preserved liver function of the patients included, as most of them were Child A with a mean presurgical MELD of 7.2 ± 1.9 and only 17.1% had presented previous decompensations of cirrhosis. However, we found 17% of early postsurgical complications such as anastomotic leak, anastomotic ulceration or wound infection, and early hepatic decompensations, mainly in the form of ascites. Previous published series of patients with cirrhosis have shown similar results, as summarized in the metaanalysis published by Jan et al. in 2015 [13], which concludes that the higher overall risk of complications is acceptable. It is noteworthy that we were not able to detect any specific characteristic of cirrhotic patients that would lead to a higher complication rate. Consequently, studies with a higher number of subjects are certainly needed to be able to select which cirrhotic patients show the best risk-to-benefit profile.

Obesity surgery in our patients with cirrhosis had a significant and relevant effect on weight loss and on resolution of comorbidities such as diabetes, hypertension, and dyslipidemia. Metabolic parameters related with glucose and lipid metabolism also showed a clear postsurgical improvement. These effects are similar to those found for non-cirrhotic patients [14–16] and to those found in previous studies with fewer numbers of patients with cirrhosis [17–20]. It is noteworthy that, although type 2 diabetes remission is higher than 50%, there was a high prevalence of insulin use after obesity surgery when compared to studies in patients without cirrhosis [21]. We speculate this may be a result of the limitation of use of oral drugs because of the presence of liver disease. The baseline prevalence of diabetes, hypertension, and dyslipidemia in our cohort of patients with liver cirrhosis is higher than in the general bariatric population [22] and concordant with previous series of bariatric patients with cirrhosis [17]. This may reflect the predominance of patients with NASH, which is regarded as the liver manifestation of the metabolic syndrome [2].

When focusing on the liver effects of obesity surgery, we found an initial improvement on liver enzymes (AST, ALT and GGT) which was seen in the first postoperative year and is concordant with some but not all previous studies [5, 17, 18, 20]. This change in liver enzymes, although not significant due to the reduced number of patients, seemed to maintain up to 5 years postsurgery. Contrary to the effects of bariatric surgery on liver enzymes, data at 5 years postsurgery showed a significant decline in platelet number and an increase in MELD scores, which reflect the tendency for liver disease to deteriorate. It is difficult, however, to compare this progression with the natural progression of liver disease in patients not undergoing bariatric surgery, but the slow progression of MELD scores over 5 years of follow-up suggests that obesity surgery may slow down liver deterioration, in concordance to what is found on liver enzymes.

The observation, in the present study, that patients with a deterioration of Child–Pugh score across time presented higher BMI at baseline and during follow-up reinforces the negative impact of obesity on cirrhosis outcomes. On the other hand, bariatric surgery has a slight impact on cirrhosis. In this sense, in spite of 17.1% of patients with previous decompensated cirrhosis, only three patients presented clinical liver decompensation manifested as ascites during follow-up after surgery, apart from two other cases of ascites in the early postsurgical period. This contrasts with data from Berzigotti et al. [4] that reported that clinical decompensation occurred in 43% of obese, compensated cirrhotic patients with portal hypertension but without gastroesophageal varices, and not submitted to obesity surgery during a 5-year follow-up. Regarding hepatocellular carcinoma, nine patients (14.6%) developed it after bariatric surgery. Although the rate of progression to hepatocellular carcinoma depends on the degree and the cause of fibrosis, our incidence is similar to patients diagnosed with NASH, in which a 12.8% of incidence is reported during a mean follow-up of 3.2 years [23]. Taken altogether, our data indicate a beneficial effect of obesity surgery on liver disease prognosis. Furthermore, the performance of bariatric surgery on a patient with cirrhosis may improve eligibility for transplantation in case of deterioration of liver function over time. Our results are concordant with the study performed by Bromberger et al. [24] which concludes that in patients with compensated cirrhosis, obesity surgery may have a greater benefit in overall survival than diet and contrast with the ancient survey performed by Brolin et al. [25] which states that 40% of surgeons would not perform a bariatric procedure after the incidental finding of hepatic cirrhosis.

Most of the patients in our series underwent a sleeve gastrectomy, which is the recommended bariatric technique in these patients. The performance of a sleeve gastrectomy allows endoscopic access to the remaining gastric tube in case of variceal bleeding, and avoids the deleterious effects that malabsorptive procedures can have on patients with cirrhosis [26], even leading to liver failure as previously reported with jejunoileal bypass or BPD [27]. In case of need for liver transplantation, sleeve gastrectomy allows endoscopic access to the biliary system and minimally affects the absorption of medications [28]. Furthermore, sleeve gastrectomy is less challenging, requires less intraoperative time, and no mortality has been found in cirrhotic patients undergoing sleeve gastrectomy [13, 29]. Our work did not find differences in terms of weight outcomes, comorbidity resolution, or complications when comparing SG with other techniques, although power to detect such differences was limited by the number of patients.

Current guidelines focusing on the management of NASH recommend obesity surgery only in the presence of Child A, compensated cirrhosis [30, 31]. However, seven patients (17.1%) in our series had a previous ascitic decompensation. These patients did not differ in terms of weight outcomes, the occurrence of postsurgical complications, deterioration of Child–Pugh scores or the occurrence of liver decompensation during follow-up. Thus, it may be possible that obesity surgery may also be recommended in some patients with previous liver decompensations. More data is needed to determine in which patients the benefits of obesity surgery outweigh the risks.

Limitations of our work include its retrospective nature and the lack of postsurgical biopsies that could reinforce the findings related to liver disease evolution. However, main strengths are the number of patients included and the long-term follow-up, being the study that describes the largest number of patients up to 5 years of follow-up after obesity surgery. Furthermore, all patients included had a liver biopsy that confirmed the presence of cirrhosis, either before or at the time of surgery.

In conclusion, obesity surgery in selected patients with cirrhosis shows a beneficial metabolic effect and may positively impact on their liver prognosis. Further prospective studies, with a control group of obese cirrhotic patients not undergoing obesity surgery, are needed to more clearly elucidate the benefits of obesity surgery in patients with cirrhosis.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

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