Obesity Surgery (2019) 29:828–834
https://doi.org/10.1007/s11695-018-3615-z

ORIGINAL CONTRIBUTIONS

Clinical Characteristics and Outcome of Morbidly Obese Bariatric Patients with Concurrent Hepatitis C Viral Infection

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Published online: 8 December 2018

Abstract

Background Hepatitis C virus (HCV) is an important cause of liver cirrhosis and its complications. The safety and efficacy of bariatric surgery in patients with HCV infection is not clear.

Methods Charts were reviewed to identify patients with HCV infection before bariatric surgery. Bariatric surgical patients with non-alcoholic steatohepatitis (NASH) and without NASH (non-NASH) were recruited as comparative groups. Demographic variables, perioperative data, follow-up, and HCV-related parameters were extracted and compared.

Results Forty-seven bariatric patients between 2000 and 2016 that suffered from HCV infection were identified. The mean age and body mass index (BMI) at baseline were 34.5 ± 9.9 years and 40.4 ± 7.7 kg/m^2, respectively. The HCV(+) group was associated with female sex, older age, lower BMI, and waist circumference than both NASH and non-NASH groups. Both HCV(+) and NASH groups had higher liver function tests and incidence of metabolic syndrome than non-NASH group. The HCV(+) group had lower uric acid and albumin level than the NASH group. Early major postoperative complication occurred in 1 (2.1%) patient of the HCV(+) group. At follow-up, the mean BMI decreased to 29.1 ± 7.1 kg/m^2 and total weight loss was 25% for the HCV(+) group at 5 years after surgery. The weight loss curves were similar between the HCV(+) group and NASH group. During follow-up, no patients died but one patient with HCV(+) developed flare up of hepatitis after gastric bypass. The mean liver transaminase level remained in normal range for the HCV(+) group.

Conclusion Co-existence of HCV infection does not influence the outcome of bariatric surgery but continued monitoring of the liver function is indicated.

Keywords Bariatric surgery · HCV infection · Weight loss · Liver transaminase

Introduction

Bariatric surgery has been used in the treatment of morbid obesity with a reduction in comorbidities [1]. Studies have shown that patients with morbid obesity are associated with many chronic liver diseases, such as non-alcoholic steatohepatitis (NASH), chronic viral hepatitis, or liver cirrhosis [2–7]. Among various chronic liver diseases, hepatitis C virus (HCV) infection is a common problem worldwide, affecting millions of people across population [8]. It is known that HCV-related hepatitis brings insidious hepatic parenchymal damage and 1 in 5 infected patients may develop cirrhosis and its complications [9]. Therefore, it brings our attention about the safety and efficacy of bariatric surgery in patients with HCV infection. As to our knowledge, data about bariatric surgery in patients with HCV infection is limited. After experiencing one patient with HCV infection developed extreme protein malnutrition and liver cirrhosis after bariatric surgery, we conducted this retrospective study to examine this issue. The aim of this study was to investigate and compare the clinical characters and outcomes of morbidly obese bariatric patients with the co-existence of HCV infection.

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Patients and Methods

This study was performed with the approval of the Ethics Committee of the Min-Sheng General Hospital. Between July 2000 and March 2016, 5387 patients that have undergone bariatric surgery for morbid obesity were studied. HCV infection was diagnosed as detectable HCV antibody (anti-HCV; Abbott HCV EIA 2.0; Abbott Laboratories, Abbott Park, IL, USA) in 47 (4.4%) out of 1063 patients (779 female, 284 male; mean age 30.9 ± 8.1 years; mean BMI 40.3 ± 7.5 kg/m^2). All patients received laparoscopic bariatric surgery at our department; 29 (61.7%) patients had laparoscopic gastric bypass procedures and the others received restrictive procedures. Among these HCV-infected patients, no one was diagnosed with liver cirrhosis by the findings of abdominal ultrasonography and laboratory data before bariatric surgery. Moreover, no patient had received anti-viral therapy before bariatric surgery.

A total of 370 patients underwent concurrent liver biopsy for the study of NASH in this period [6, 10]. The degree of non-alcoholic fatty liver disease (NAFLD) in each biopsy was scored using the National Institutes of Health-sponsored NASH Clinical Research Network NAFLD Activity Score (NAS) [11]. NAS score of ≥ 5 correlated with a diagnosis of NASH [12]. Among them, 80 patients of proven NASH were recruited for the comparative group. Another 135 bariatric patients proven non-NASH were recruited as normal control. Demographic and clinical comparisons were made between patients with HCV(+) and NASH and non-NASH groups.

Statistical Analysis

Data were expressed as mean ± SD and percentages. Statistical analyses were performed using chi-square test, Mann-Whitney *U* tests, and Student's *t* test. Logistic regression was used to assess the significance of associations between HCV infection and categorical or continuous predictors' variables. A P value < 0.05 was considered statistically significant. SPSS statistical software (SPSS Inc., Chicago, IL) was used for statistical analysis.

Results

Basic Characters

The demographic and clinical data of the study and comparative groups are summarized in Table 1. HCV(+) patients were associated with female sex, older age, lower BMI, and waist circumference than the NASH and non-NASH groups. Both HCV(+) and NASH groups had higher homeostatic model assessment-insulin resistance (HOMA-IR) index, liver function test levels, and incidence of associated metabolic syndrome than the non-NASH group. Compared to the NASH group, the HCV(+) group had a lower uric acid and albumin levels but no difference in the liver function test, HOMA-IR, and incidence of metabolic syndrome.

Perioperative Parameters

There was also no major difference in the perioperative outcomes between the HCV(+) group and the other two groups except a little more blood loss in the HCV(+) group (Table 2). However, only 29 (61.7%) out of 47 HCV(+) patients received gastric bypass procedure, significantly less than the 80% in the other two groups. Early major postoperative complication occurred in 1 (2.1%) patient out of 47 in the HCV(+) group but no re-operation was required. Perioperative major complications occurred in 6 (2.3%) of the whole 262 patients without mortality.

Weight Loss and Laboratory Data

Figure 1 shows the BMI reduction of the three groups after surgery. There was no difference in the weight reduction between the three groups. The mean BMI decreased to 29.1 ± 7.1 kg/m^2 and total weight loss was 25% for the HCV(+) group at 5 years after surgery.

The clinical data of the HCV(+) and NASH groups at 5 years following surgery are shown in Table 3. With weight loss, there was a significant improvement in obesity-related metabolic complications, including glucose level, blood pressure, cholesterol, triglyceride, AST, ALT, gamma-glutamyl transferase (GGT), uric acid, insulin, HbA1C, and LDL. There was no difference between the two groups except waist circumference is still higher in the NASH group. The postoperative changes of ALT levels in HCV(+), NASH, and non-NASH groups are shown in Fig. 2. The decrease of ALT levels for patients with HCV(+) and NASH groups was similar.

Long-Term Follow-up of HCV(+) Patients

To clarify the impact of procedure type on the outcome of bariatric surgery for HCV(+) patients, we listed in Table 4 the different types of bariatric surgery including demographics before and after surgery for the HCV(+) patients divided by procedure types as gastric bypass (GB) group and non GB group. The result revealed the GB group has a higher fasting glucose and lower creatinine and uric acid before surgery. Five years after surgery, the GB group has a lower BMI, total cholesterol, albumin, weight loss, and excess weight loss. There was no significant difference of liver function tests between the two groups. As for perioperative complications, 2 out of 29 patients (6.9%) had complications (one major complication and one minor complication); 2 out of 18 patients (11%) had minor complication and no major complication was noted.

Table 1 Characteristics of included patients at baseline

	HCV(+)	NASH	*P* (1)	Non-NASH	*P* (2)
No.	47	80		135	
Male/female	8/39	36/44	0.001*	48/87	0.018*
Age (years)	34.5 ± 9.9	29.3 ± 7.9	0.006*	29.4 ± 8.6	0.003*
BMI (kg/m^2)	40.4 ± 7.7	44.4 ± 4.9	0.004*	45.3 ± 6.7	< 0.001*
Waist circumference (cm)	115.1 ± 19.8	126.7 ± 12.0	< 0.001*	125.0 ± 16.4	< 0.001*
SBP (mmHg)	131.7 ± 16.3	136.8 ± 16.2	0.249	132.2 ± 17.2	0.985
DBP (mmHg)	82.7 ± 10.8	83.6 ± 12.9	0.935	79.4 ± 13.1	0.289
Fasting glucose (mg/dL)	98.2 ± 14.5	98.6 ± 26.8	0.990	91.8 ± 9.4	0.094
HbA1c (%)	5.7 ± 1.2	5.7 ± 0.7	0.974	5.6 ± 0.4	0.724
HOMA-IR	6.6 ± 6.3	6.5 ± 5.9	1.000	4.6 ± 3.2	< 0.001*
Total cholesterol (mg/dL)	182.2 ± 41.8	187.5 ± 32.6	0.699	187.3 ± 31.6	0.676
Triglyceride (mg/dL)	135.4 ± 71.2	166.3 ± 87.3	0.094	137.4 ± 71.9	0.989
LDL (mg/dL)	125.3 ± 31.4	133.9 ± 26.3	0.684	127.3 ± 31.3	0.975
ALT (IU)	65.7 ± 57.6	78.5 ± 59.6	0.297	35.8 ± 23.3	< 0.001*
AST (IU)	48.8 ± 38.6	46.6 ± 30.5	0.999	24.7 ± 13.9	< 0.001*
GGT (IU/L)	66.9 ± 42.9	61.5 ± 44.2	0.777	32.8 ± 19.4	< 0.001*
Uric acid (mg/dL)	6.6 ± 1.4	8.0 ± 1.9	< 0.001*	7.2 ± 1.6	< 0.099
Albumin (gm/dL)	4.2 ± 0.4	4.4 ± 0.3	0.008*	4.3 ± 0.3	0.085
Creatinine (mg/dL)	0.75 ± 0.14	0.76 ± 0.21	0.977	0.74 ± 0.15	0.976
WBC ×10^3	8.2 ± 2.4	9.0 ± 2.0	0.158	8.9 ± 2.4	0.870
hsCRP	0.5 ± 0.5	0.8 ± 0.7	0.183	0.9 ± 1.1	0.085
Hemoglobin (gm/dL)	13.8 ± 1.5	14.4 ± 1.4	0.080	14.0 ± 1.7	0.600
Metabolic syndrome *n* (%)	27 (57.4%)	40 (50%)	0.417	41 (30.4%)	0.001*

Data are presented as the mean ± standard deviation. *BMI*, body mass index; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; *HbA1c*, glycated hemoglobin; *HOMA-IR*, homeostatic model assessment-insulin resistance index; *LDL*, low-density lipoprotein; *ALT*, alanine aminotransferase; *AST*, aspartate aminotransferase; *GGT*, gamma-glutamyl transferase; *WBC*, white blood cells; *hsCRP*, highly sensitive C-reactive protein. *$P < 0.05$ (1) as HCV(+) compared to NASH (2) HCV(+) compared to non-NASH

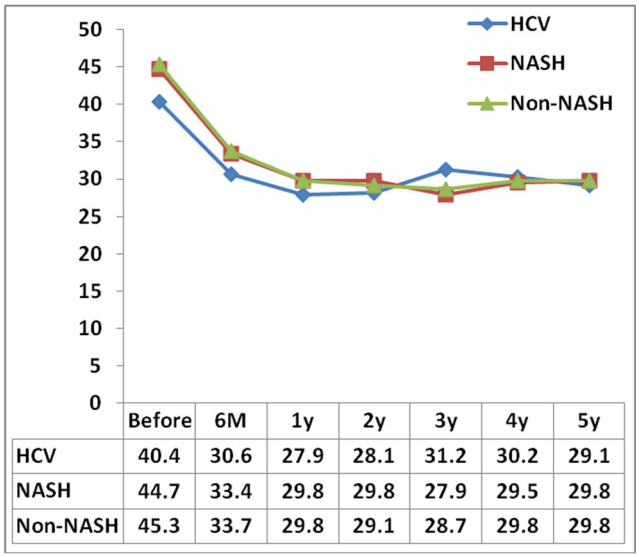
At a mean follow-up of 9 years (1 to 15 years), no patients died. One patient with HCV infection developed flare up of hepatitis. This is a 46-year-old male, who developed flare up of hepatitis 12 months after gastric bypass. The patient recovered after anti-viral therapy with peginterferon and ribavirin for 48 weeks.

Up to now, six patients received revision surgery. Four patients with restrictive surgery were converted to gastric bypass surgery. Two patients with bypass surgery were converted to sleeve gastrectomy surgery for malnutrition, one for intractable anemia and the other one for severe hypoproteinemia with liver cirrhosis. All the revision surgery was uneventful.

Table 2 Comparison of perioperative data

	HCV(+)	NASH	*P* (1)	Non-NASH	*P* (2)
No.	47	80		135	
Operative time (min)	116.3 ± 35.2	104.1 ± 28.3	0.123	116.7 ± 33.6	0.997
Hospital stay (day)	5.3 ± 5.4	5.4 ± 3.0	0.546	6.1 ± 5.0	0.779
Blood loss (mL)	46.4 ± 55.4	31.4 ± 19.3	0.041*	34.3 ± 24.4	0.089
Flatus passage (day)	1.6 ± 0.7	1.7 ± 0.7	0.965	1.7 ± 0.8	0.788
Complication (overall)	4 (8.5%)	2 (4.0%)	0.092	13 (9.6%)	1.000
Minor	3 (6.4%)	1 (1.3%)	0.069	8 (5.9%)	1.000
Major	1 (2.1%)	1 (1.3%)	1.000	5 (3.7%)	1.000
Bypass procedure	29 (61.7%)	64 (80%)	0.037*	117 (86.7%)	0.001*

*P value < 0.05 (1) as HCV(+) compared to NASH (2) HCV(+) compared to non-NASH



	Before	6M	1y	2y	3y	4y	5y
HCV	40.4	30.6	27.9	28.1	31.2	30.2	29.1
NASH	44.7	33.4	29.8	29.8	27.9	29.5	29.8
Non-NASH	45.3	33.7	29.8	29.1	28.7	29.8	29.8

Fig. 1 Change of body weight index of morbidly obese patients after surgery in patients with HCV(+), NASH, or non-NASH

Discussion

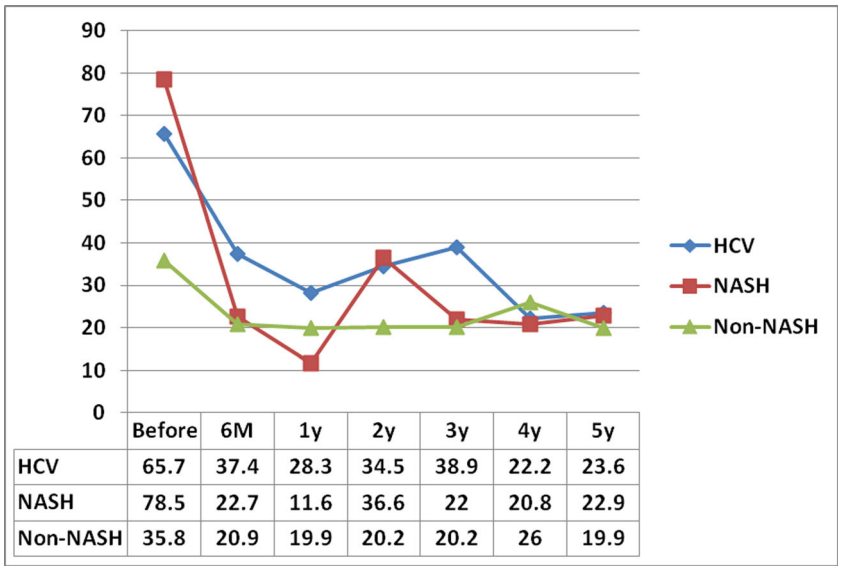
NASH is the most commonly seen liver pathology in patients with morbid obesity [3, 6]. Central obesity and insulin resistance were the important mechanisms for the development of steatosis and steatohepatitis [2, 5]. Elevated liver enzymes, AST, ALT, and GGT, are predictive of the progressive type of fatty liver, NASH [2, 13]. In the current study, HCV(+) patients have similar levels of AST, ALT, and GGT as patients with NASH. In addition, HCV(+) patients also had increased insulin resistance as NASH comparing to other morbidly obese patients. These data were consistent with previous reports that HCV(+) induced insulin resistance by blocking intracellular insulin signaling [14]. However, the HCV(+) group had a significant older age, lower BMI, and lower level of albumin than the NASH group that reflected the more severe hepatic necroinflammatory status of HCV infection than NASH [15].

HCV is an important etiologic factor of chronic liver disease with an incidence of about 2.2–3.0% in general population [8]. In our previous study, the incidence of HCV(+) in morbid obese patients was around 1.3% [6]. However, the incidence of HCV(+) in this study was 4.4% which might be attributed to the different ways of patient selection in the two studies. The chronic and persistent necroinflammatory injury in HCV was considered to be an important cause of end-stage liver disease and hepatocellular carcinoma in the USA [9]. To our knowledge, there is no published literature on the outcome of bariatric surgery in HCV(+) patients. This study is the first to confirm the efficacy and safety of bariatric surgery in patients with morbid obesity and HCV infection.

Table 3 Comparison of clinical and laboratory data 5 years after surgery

	HCV positive ($N = 39$)	NASH ($N = 58$)	P value
BMI (kg/m^2)	28.1 ± 7.1	29.6 + 5.1	0.441
Waist circumference (cm)	85.1 ± 9.9	96.6 ± 11.8	0.028*
SBP (mmHg)	125.7 ± 19.1	119.8 ± 19.6	0.760
DBP (mmHg)	75.3 ± 9.9	71.7 ± 9.5	0.665
Fasting glucose (mg/dL)	85.5 ± 10.2	87.8 ± 7.6	0.808
HbA1c (%)	4.9 ± 0.7	4.8 ± 0.8	0.728
HOMA-IR	1.2 ± 0.9	0.9 ± 0.6	0.906
Total cholesterol (mg/dL)	162.1 ± 47.7	141.1 ± 26.9	0.545
Triglyceride (mg/dL)	83.7 ± 61.6	66.4 ± 17.8	0.826
LDL (mg/dL)	93 ± 22.2	88.9 ± 22.8	0.377
ALT (IU/L)	34.7 ± 26.4	34.1 ± 26.0	0.999
AST (IU/L)	34.5 ± 26.7	36.6 ± 30.8	0.984
GGT (IU/L)	50.9 ± 74.5	57.1 ± 78.2	0.830
Uric acid (mg/dL)	5.3 ± 1.0	5.2 ± 1.3	0.999
A1bumin (gm/dL)	4.0 ± 0.5	3.9 ± 0.6	0.910
Creatinine (mg/dL)	0.7 ± 0.1	0.8 ± 0.4	0.247
WBC (10^3/μL)	5.9 ± 2.1	5.7 ± 1.8	0.789
hsCRP	0.144 ± 0.18	0.124 ± 0.17	1.000
Hemoglobin (gm/dL)	11.4 ± 1.9	11.8 ± 1.2	0.714
Metabolic syndrome *n* (%)	1 (2.6%)	2 (3.5%)	0.678

Data are presented as the mean ± standard deviation. *BMI*, body mass index; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; *HbA1c*, glycated hemoglobin; *HOMA-IR*, homeostatic model assessment-insulin resistance index; *LDL*, low-density lipoprotein; *ALT*, alanine aminotransferase; *AST*, aspartate aminotransferase; *GGT*, gamma-glutamyl transferase; *WBC*, white blood cells; *hsCRP*, highly sensitive C-reactive protein. *$P < 0.05$



	Before	6M	1y	2y	3y	4y	5y
HCV	65.7	37.4	28.3	34.5	38.9	22.2	23.6
NASH	78.5	22.7	11.6	36.6	22	20.8	22.9
Non-NASH	35.8	20.9	19.9	20.2	20.2	26	19.9

Fig. 2 Change of liver enzymes (ALT) in morbid obese patients after surgery with HCV(+), NASH, or non-NASH

Although both NASH and HCV infection are important etiologic factors of chronic liver diseases, our data implicated that the co-existence of HCV infection and obesity had a similar liver injury with morbid obese patients

Table 4 Comparison between different types of bariatric surgery in HCV-positive patients

	Before			After		
	GB ($N=29$)	Non GB ($N=18$)	P	GB ($N=26$)	Non GB ($N=13$)	P
BMI (kg/m^2)	34.4 (11.2)	34.5 (8.0)	0.844	26.5 (6.3)	29.8 (7.9)	0.01*
Waist (cm)	115.2 (18.1)	114.3 (23.1)	0.831	85.7 (10.2)	105.3 (8.5)	0.05
SBP (mmHg)	134.0 (17.6)	127.9 (13.4)	0.151	123.8 (20.4)	110.0 (8.5)	0.96
DBP (mmHg)	85.3 (12.1)	81.9 (8.9)	0.843	74.2 (9.2)	82.7 (10.9)	0.19
Fasting glucose (mg/dL)	94.1 (13.4)	81.9 (8.9)	0.013*	74.2 (9.2)	82.7 (10.9)	0.545
HbA1c (%)	5.8 (1.3)	5.6 (0.6)	0.446	4.9 (0.6)	5.5 (0.3)	0.067
HOMA-IR	5.8 (3.2)	0.62 (0.4)	0.827	8.9 (12.0)	2.8 (3.4)	0.065
Total cholesterol (mg/dL)	178 (46.8)	188.9 (32.2)	0.437	139.2 (29.5)	211.6 (54.9)	0.02*
Triglyceride (mg/dL)	140.7 (79.9)	126.8 (55.4)	0.607	78.3 (41.5)	131.8 (102.9)	0.228
LDL (mg/dL)	130.5 (28.1)	123.5 (33.6)	0.571	88.4 (19.3)	126.5 (91.2)	1
ALT (IU/L)	45.9 (39.6)	48.3 (38.1)	0.558	40.9 (40.3)	33.5 (27.3)	0.229
AST (IU/L)	67.6 (58.7)	62.8 (57.4)	0.922	40.1 (45.6)	21.5 (13.4)	0.122
GGT (IU/L)	68.2 (41.3)	63.3 (50.0)	0.701	48.5 (66.3)	21.4 (13.4)	0.666
Uric acid (mg/dL)	6.2 (1.2)	7.2 (1.5)	0.016*	5.4 (1.2)	6.9 (1.6)	0.079
A1bumin (gm/dL)	4.2 (0.4)	4.2 (0.4)	0.501	3.9 (0.5)	4.5 (0.3)	0.017*
Creatinine (mg/dL)	0.7 (0.1)	0.96 (0.15)	0.018*	0.7 (0.1)	1.1	0.061
WBC (10^3/μL)	8.7 (2.0)	7.5 (2.1)	0.155	5.8 (2.3)	5.8 (1.9)	0.777
hsCRP	0.5 (0.4)	0.1 (0.1)	0.305	0.6 (0.6)	0.1 (0.04)	0.396
Hemoglobin (gm/dL)	13.7 (1.6)	11.0 (1.9)	0.293	14.0 (1.4)	13.2 (2.8)	0.112
Metabolic syndrome n (%)	5 (17.2%)	4 (22.2%)	0.673	0 (0.0%)	0 (0.0%)	–
Weight loss (%)	–	–	–	31.7 (7.5)	21.9 (10.4)	0.006*
Excess weight loss (%)	–	–	–	81.3 (22.7)	45.0 (19.8)	0.002*

Data are presented as the mean ± standard deviation. *GB*, gastric bypass; *BMI*, body mass index; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; *HbA1c*, glycated hemoglobin; *HOMA-IR*, homeostatic model assessment-insulin resistance index; *LDL*, low-density lipoprotein; *ALT*, alanine aminotransferase; *AST*, aspartate aminotransferase; *GGT*, gamma-glutamyl transferase; *WBC*, white blood cells; *hsCRP*, highly sensitive C-reactive protein. *$P<0.05$

with NASH. Central obesity and insulin resistance play a dominant role in the parenchyma liver disease of morbid obese patients, either with HCV infection or NASH. The co-existence of HCV does not put an additive effect on the liver injury of our patients. In previous studies, HCV infection was also found to correlate with the presence of steatohepatitis [16, 17]. On the contrary, the presence of another kind of viral hepatitis, hepatitis B viral infection, reduced the risk of NASH [18]. This finding reflects a different nature of liver injury from different types of viral hepatitis, but bariatric surgery can be performed safely in both [19].

In this study, we confirm that weight loss following bariatric surgery is likely to indicate significant improvement in obesity-related metabolic complication, including remission of fatty liver diseases [2, 20–25]. A fall in liver enzymes is predictive of improved fatty liver disease [26, 27]. However, the role of HCV infection in liver disease will emerge after the resolution of morbid obesity. There is a persistent tendency of elevated AST and ALT levels in morbid obese patients concurrent with HCV infection. The ALT level of patients with HCV infection was continually mildly elevated within the postoperative 6 months to 3 years that implicated a persistent liver parenchymal damage in HCV(+) patients. In this study, there was one patient that developed flare up of hepatitis 1 year after surgery. This patient was rescued by anti-viral therapy. Another patient developed intractable hypoalbuminemia and liver cirrhosis after gastric bypass and was then converted back to sleeve gastrectomy [28]. These findings indicate that after resolution of morbid obesity and fatty liver, HCV infection is persistent as an etiology of chronic liver disease in these patients. We should pay special attention to the liver function of those with HCV infection in the follow-up. Anti-viral drugs may be considered if deterioration of liver function was detected in this special group of patients. Continuing follow-up of liver enzymes along with markers of liver cirrhosis and hepatocellular carcinoma is also mandatory in these patients.

Although this study is retrospective, there were several limitations, such as unavailable data of HCV viral load and liver biopsy in HCV(+) patients and loss of follow-up 5 years after surgery for part of the participants. Further prospective and longitudinal studies are needed to test the association between viral activity and long-term outcome after bariatric surgery.

In conclusion, the results of this study demonstrate that the existence of HCV seemed to have no influence on the outcomes of bariatric surgery in this special subset of patients. However, HCV infection remains to be a cause of chronic liver disease with possible acute exacerbation and adverse result after resolution of morbid obesity and fatty liver disease. This knowledge should assist in the appropriate tracking of this special subset of patients.

Funding Information This work was supported by grant NSC 90-2314-B-385-001 from the National Science Council, R.O.C.

Compliance with Ethical Standards

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

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent This article does not contain any studies with human participants or animal performed by any of the authors. For this type of study, formal consent is not required. Informed consent does not apply to the submission.

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