Clinical and Experimental HEPATOLOGY

Clinical and Experimental HEPATOLOGY 2015; 4: 127–130
DOI: 10.5114/ceh.2016.57758
Received: 19.11.2015, **Accepted:** 19.01.2016, **Published:** 04.02.2016

Case report

Severe fatty liver disease and acute pancreatitis: is there a correlation between them?

Xingshun Qi, Yue Hou, Xiaozhong Guo

Department of Gastroenterology, General Hospital of Shenyang Military Area, Shenyang, China

Abstract

In this manuscript, we present two cases of patients with severe fatty liver disease developing acute pancreatitis. They might suggest an association between severe fatty liver disease and acute pancreatitis.

Key words: pancreatitis, fatty liver disease, risk factor, etiology.

Address for correspondence

Xiaozhong Guo and Xingshun Qi, Department of Gastroenterology, General Hospital of Shenyang Military Area, No. 83 Wenhua Road, Shenyang, Liaoning 110840, China, e-mail: guo_xiao_zhong@126.com; xingshunqi@126.com

Many causes of acute pancreatitis have been identified. The most common causes are bile duct obstruction by gallstones and alcohol abuse followed by drugs, abdominal surgery, genetic mutation, bacterial and viral infection, hyperlipidemia, hypercalcemia, autoimmune diseases, pregnancy, and others [1, 2]. To our knowledge, an association of severe fatty liver disease with acute pancreatitis has never been reported. Here, we present two cases of patients with severe fatty liver disease developing acute pancreatitis.

Case 1

On November 1, 2015, a 33-year-old woman was admitted to our emergency department due to the sudden occurrence of persistent abdominal pain for four hours. She had two previous episodes of acute pancreatitis 6 years ago. She denied any recent history of alcohol or drug abuse. At her physical examinations, there was significant upper abdominal tenderness without any rebound or tension. Her weight was 77.5 kg, and height was 1.62 m. Body mass index was 29.5 kg/m^2. Laboratory test results are shown in Table 1. HBsAg was negative. No gallstone was observed at ultrasound. Abdominal non-enhanced computed tomography (CT) scans demonstrated that the volume of the pancreas was mildly enlarged and the ratio of liver versus spleen density in CT scans was 0.4 (Fig. 1). Thus, she was diagnosed with mild recurrent acute pancreatitis and non-alcoholic fatty liver disease (NAFLD). Routine treatment was given, including octreotide, antibiotics, and fluid infusion. After that, abdominal pain remarkably resolved. On November 12, she was discharged without any abdominal complaints. Laboratory tests were performed again (Table 1). Abdominal non-enhanced CT scans demonstrated that the condition of the pancreas had greatly improved (Fig. 2).

Case 2

On December 14, 2015, a 34-year-old man was transferred to our department due to the sudden onset of upper abdominal pain for one day. He had a history of hepatitis B virus infection. He denied any history of alcohol or drug abuse. At his local hospital, laboratory tests were performed on December 13, 2015, demonstrating that the white blood cell count was 10.3×10^9/l, neutrophil percentage was 81.3%, serum amylase was 67 U/l (reference range at his local hospital: 40-129 U/l), and serum lipase was 167.2 U/l (reference range at his local hospital: 0-60 U/l). Additionally, based on the findings of CT scans, acute pancreatitis was also suspected. Nasogastric tube insertion, antibiotics, protease inhibitors, and fluid therapy were given.

On admission, physical examinations demonstrated that his weight was 79 kg, height was 175 cm, heart rate was 76 times/min, temperature was 36.5°C, blood pressure was 143/97 mmHg, respiratory rate

Table 1. Laboratory tests

Variables	Reference range	1 November	2 November	4 November	12 November
White blood cells	4-10 × 10^9/l	9.9 × 10^9/l	10 × 10^9/l	7.6 × 10^9/l	NA
Percentage of neutrophil	50-70%	73.10%	74.10%	64.00%	NA
Red blood cells	3.5-5 × 10^{12}/l	5.15 × 10^{12}/l	4.71 × 10^{12}/l	4.971 × 10^{12}/l	NA
Hemoglobin	100-150 g/l	154 g/l	144 g/l	149 g/l	NA
Platelet count	100-300 × 10^9/l	240 × 10^9/l	232 × 10^9/l	243 × 10^9/l	NA
Serum amylase	31-110 U/l	277 U/l	NA	100 U/l	105 U/l
Serum lipase	23-300 U/l	2439 U/l	513 U/l	584 U/l	850 U/l
Alanine aminotransferase	9-72 U/l	173.08 U/l	164.92 U/l	155.48 U/l	73.38 U/l
Aspartate aminotransferase	8-50 U/l	90.01	96.53 U/l	100.86 U/l	46.67 U/l
Total bilirubin	0-20.5 μmol/l	10.5 μmol/l	25.3 μmol/l	27.4 μmol/l	37.6 μmol/l
Glucose		NA	8.94 mmol/l	8.31 mmol/l	6.37 mmol/l
Triglyceride	0.18-1.8 mmol/l	NA	2.12 mmol/l	2.57 mmol/l	1.76 mmol/l
Low-density lipoprotein	1.9-3.8 mmol/l	NA	4.12 mmol/l	4.06 mmol/l	3.16 mmol/l
High-density lipoprotein	0.91-3.8 mmol/l	NA	1.00 mmol/l	0.85 mmol/l	0.66 mmol/l
Blood urea nitrogen	2.5-8.1 mmol/l	NA	3.23 mmol/l	NA	NA
Serum calcium	2.13-2.88 mmol/l	NA	2.26 mmol/l	2.41 mmol/l	NA
Prothrombin time	11.5-14.5 s	NA	12.7 s	NA	NA

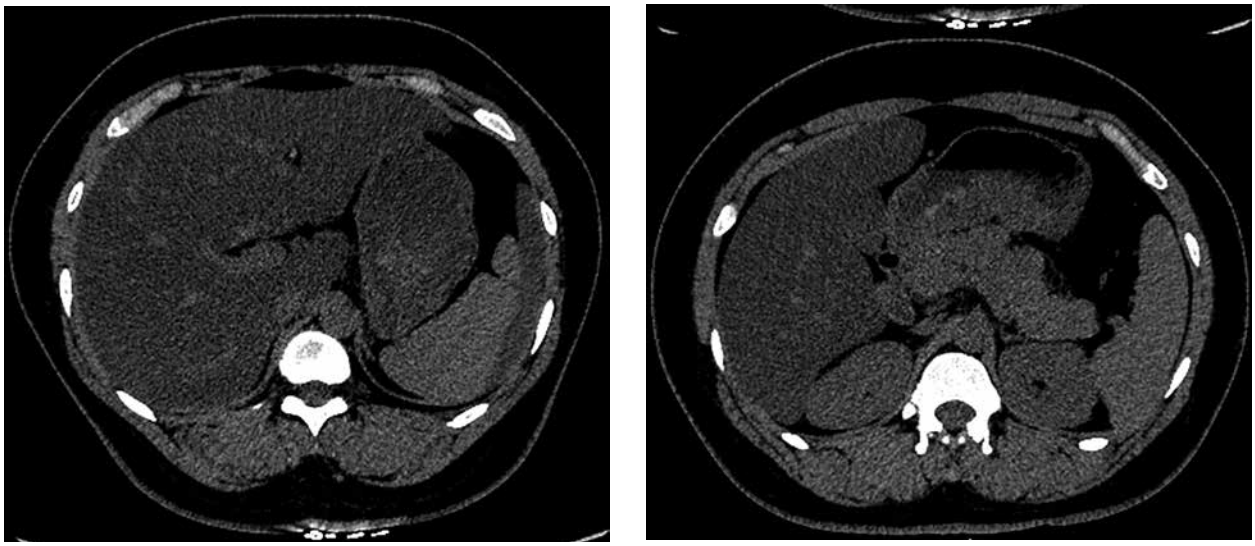

Fig. 1. Abdominal computed tomography scans on admission in the first case

was 17 times/min, and upper abdominal tenderness was positive without any rebound or tension. On December 14, 2015, blood tests were performed. White blood cell count was 7.6 × 10^9/l, neutrophil percentage was 61.0%, platelet count was 139 × 10^9/l, serum amylase was 38.6 U/l (reference range: 22-80 U/l), serum lipase was 235 U/l (reference range: 23-300 U/l), C-reactive protein was 73.6 mg/l (reference range: 0-8 mg/l), fasting glucose was 14.25 mmol/l (reference range: 3.9-6.1 mmol/l), triglyceride was 15.03 mmol/l (reference range: 0.45-1.7 mmol/l), cholesterol was 7.71 mmol/l (reference range: 2.85-5.7 mmol/l), procalcitonin was 0.091 ng/ml (reference range: 0-0.05 ng/ml), total bilirubin was 14.9 μmol/l (reference range: 5.1-22.2 μmol/l), alanine aminotransferase was 14.9 μmol/l (reference range: 5.1-22.2 μmol/l), alkaline phosphatase was 73.00 U/l

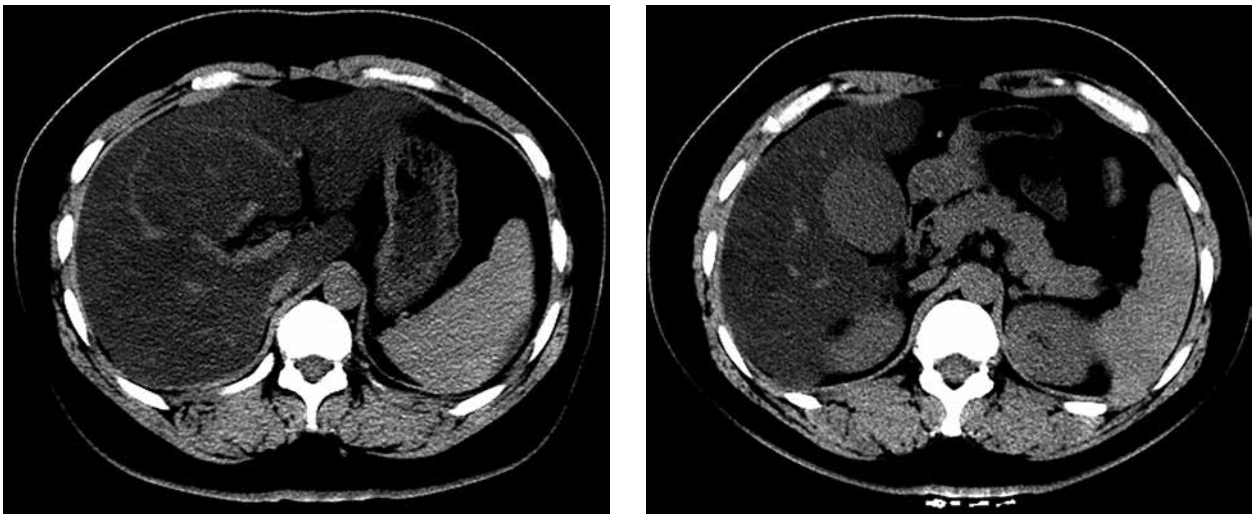

Fig. 2. Abdominal computed tomography scans after treatment in the first case

(reference range: 45-125 U/l), albumin was 43.7 g/l (reference range: 40-55 g/l), creatinine was 73.39 μmol/l (reference range: 44-133 μmol/l), and prothrombin time was 12.2 s (reference range: 11.5-14.5 s). HBsAg, HBeAb, and HbcAb-IgG were positive, and HBV DNA was 3.2×10^3 copies/ml (reference range: $< 1.0 \times 10^3$ copies/ml). Non-enhanced CT scans demonstrated that the pancreatic edge was coarse and peripancreatic fat was vague, suggesting mild acute pancreatitis related to hypertriglyceridemia. Additionally, the ratio of liver to spleen density in CT scans was less than 0.5, suggesting the presence of severe fatty liver disease (Fig. 3). Lipid- and glucose-lowing treatment was also added.

On December 16, 2015, abdominal pain was rapidly alleviated. Blood tests were performed again. White blood cell count was 7.0×10^9/l, neutrophil percentage was 66.7%, serum amylase was 42.6 U/l (reference range: 22-80 U/l), serum lipase was 78 U/l (reference range: 23-300 U/l), fasting glucose was 8.21 mmol/l (reference range: 3.9-6.1 mmol/l), triglyceride was 5.48 mmol/l (reference range: 0.45-1.7 mmol/l), and cholesterol was 7.13 mmol/l (reference range: 2.85-5.7 mmol/l). Liver and renal function remained within the normal range.

On December 21, 2015, abdominal discomfort completely disappeared. Serum amylase was 49.5 U/l (reference range: 22-80 U/l), serum lipase was 171.0 U/l (reference range: 23-300 U/l), fasting glucose was 7.70 mmol/l (reference range: 3.9-6.1 mmol/l), triglyceride was 3.19 mmol/l (reference range: 0.45-1.7 mmol/l), and cholesterol was 6.43 mmol/l (reference range: 2.85-5.7 mmol/l). Thus, he was discharged without any complaints.

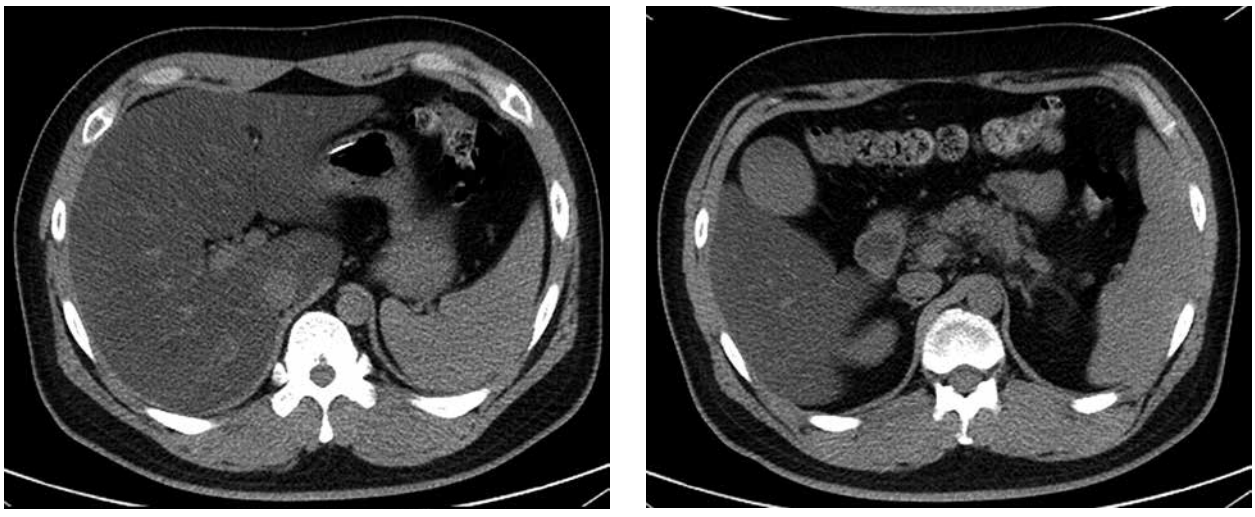

Fig. 3. Abdominal computed tomography scans on admission in the second case

Discussion

Serum triglyceride level > 1000 mg/dl (i.e., 11.3 mmol/l) is an identifiable risk factor for acute pancreatitis [3]. Hypertriglyceridemia is rare in Western patients with pancreatitis (1.3-3.8%) [4], but is relatively frequent in Chinese patients with pancreatitis (12.3%) [5]. Because the highest serum triglyceride level was 2.57 mmol/l in the first case, the etiology of acute pancreatitis could not be attributed to hypertriglyceridemia. By comparison, because the highest serum triglyceride level was 15.03 mmol/l in the second case, the major etiology of acute pancreatitis should be hypertriglyceridemia. Additionally, other known causes, such as alcohol abuse or gallstone, had been excluded. Notably, magnetic resonance cholangiopancreatography (MRCP) was not performed in the two cases, because liver function was well preserved and no gallstone in the bile duct was observed.

According to the CT findings, severe fatty liver disease could be diagnosed in both cases [6]. As is well known, fatty liver diseases involve hepatic fat accumulation, which is a type of metabolic abnormality. Common causes of secondary hepatic steatosis include excessive alcohol consumption, genotype 3 hepatitis C virus infection, Wilson's disease, starvation, parenteral nutrition, medications, and hereditary diseases, among others [7]. After excluding the above-mentioned causes, NAFLD can be clearly diagnosed by liver histology. However, considering potential procedure-related complications, both patients refused liver biopsy. Additionally, HBV infection was not found in the first case, but was in the second case. Thus, the diagnostic criteria of NAFLD are not fully met in either case.

To our knowledge, no guideline or consensus has reported the role of severe fatty liver disease in the development of acute pancreatitis. However, several investigators have reported the occurrence of pancreatitis as a consequence of acute fatty liver of pregnancy. In Canada, Apiratpracha *et al.* reported a 34-year-old woman developing chronic pancreatitis secondary to acute fatty liver of pregnancy [8]. In Romania, Cruciat *et al.* also reported a 26-year-old pregnant woman who developed fatal acute pancreatitis subsequent to acute fatty liver of pregnancy [9]. In Puerto Rico, Collado Ferrer *et al.* reported a 17-year-old female patient with acute fatty liver of pregnancy who was complicated by acute pancreatitis and liver and renal failure after caesarean section [10]. In Brazil, de Oliveira *et al.* reported a case of acute fatty liver of pregnancy associated with severe acute pancreatitis in a 26-year-old woman [11]. Given that pancreatitis is a potentially life-threatening complication, all patients with acute fatty liver of pregnancy should be screened for pancreatitis [12].

Based on our cases, we also suspected the possibility that severe fatty liver disease might be a risk factor for acute pancreatitis. However, we had to acknowledge that the evidence from the case reports was very weak. In future, case-control studies should be performed to confirm this correlation.

Disclosure

Authors report no conflict of interest.

References

1. Frossard JL, Steer ML, Pastor CM. Acute pancreatitis. Lancet 2008; 371: 143-152.
2. Lankisch PG, Apte M, Banks PA. Acute pancreatitis. Lancet 2015; 386: 85-96.
3. Yadav D, Pitchumoni CS. Issues in hyperlipidemic pancreatitis. J Clin Gastroenterol 2003; 36: 54-62.
4. Fortson MR, Freedman SN, Webster PD 3rd. Clinical assessment of hyperlipidemic pancreatitis. Am J Gastroenterol 1995; 90: 2134-2139.
5. Chang MC, Su CH, Sun MS, et al. Etiology of acute pancreatitis – a multi-center study in Taiwan. Hepatogastroenterology 2003; 50: 1655-1657.
6. Fan JG, Jia JD, Li YM, et al. Guidelines for the diagnosis and management of nonalcoholic fatty liver disease: update 2010 (published in Chinese on Chinese Journal of Hepatology 2010; 18: 163-166). J Dig Dis 2011; 12: 38-44.
7. Chalasani N, Younossi Z, Lavine JE, et al. The diagnosis and management of non-alcoholic fatty liver disease: practice Guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. Hepatology 2012; 55: 2005-2023.
8. Apiratpracha W, Yoshida EM, Scudamore CH, et al. Chronic pancreatitis: a sequela of acute fatty liver of pregnancy. Hepatobiliary Pancreat Dis Int 2008; 7: 101-104.
9. Cruciat G, Stamatian F, Puscas M, et al. Acute pancreatitis in a pregnant woman with acute fatty liver dystrophy. A case report. J Gastrointestin Liver Dis 2007; 16: 193-196.
10. Collado Ferrer M, Collado Ferrer L, Serrano A. Acute fatty liver of pregnancy. Bol Asoc Med P R 2010; 102: 51-53.
11. de Oliveira CV, Moreira A, Baima JP, et al. Acute fatty liver of pregnancy associated with severe acute pancreatitis: A case report. World J Hepatol 2014; 6: 527-531.
12. Moldenhauer JS, O'Brien JM, Barton JR, Sibai B. Acute fatty liver of pregnancy associated with pancreatitis: a life-threatening complication. Am J Obstet Gynecol 2004; 190: 502-505.