

Successful Treatment of Severe Case of Lipid Overload Syndrome with Pancreatitis and Pneumonia: A Case Report

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Background: Fat overload syndrome is a rare and severe adverse reaction triggered by the infusion of a single source of lipid emulsion, resulting in elevated blood triacylglycerol (TG) levels. The majority of literature reports focus on cases of fat overload syndrome in patients with mild symptoms. This case is significant because it demonstrates the diagnostic and therapeutic experience and provide valuable insights for the management for severe fat overload syndrome.

Case Presentation: We present a case report of a female patient who developed fat overload syndrome following prolonged and excessive infusion of lipid emulsion after colon resection surgery. In the setting of compromised immune function and malnutrition, the patient's pulmonary infection and respiratory distress symptoms have further exacerbated. Hence, in addition to severe pancreatitis, the patient has also contracted severe pneumonia. Upon admission, tracheal intubation, plasma exchange and blood perfusion were performed. Subsequently, comprehensive treatment was provided, including anti-infection, antispasmodic, acid suppression, enzyme inhibition, as well as targeted supportive measures to stabilize electrolytes and nutritional status. After treatment, there was a progressive reduction in blood lipid levels. After assessing the relevant risks, it was deemed necessary to perform an emergency computed tomography (CT)-guided percutaneous drainage tube placement procedure targeting the necrotic area of the pancreas while the patient was still intubated. Finally, the patient was discharged from the hospital.

Conclusion: The case highlights the association between fat overload syndrome and pancreatitis as well as the use of lipid emulsions and suggests the treatment strategies for severe fat overload syndrome.

Keywords: lipid overload syndrome, plasma exchange, blood perfusion, pancreatitis, pneumonia

Introduction

Fat overload syndrome is a rare and severe adverse reaction triggered by the infusion of a single source of lipid emulsion. It occurs when the rate or dose of lipid emulsion exceeds the body's ability to clear fat, resulting in elevated blood triacylglycerol (TG) levels - a hallmark feature of this rare complication.¹ It is characterized by headaches, fever, jaundice, hepatosplenomegaly, respiratory distress, and spontaneous hemorrhage.² Other symptoms include anemia, leukopenia, thrombocytopenia, low fibrinogen levels, and coagulopathy. The diagnosis of fat overload syndrome lacks uniform criteria and mainly relies on medication history, clinical presentation, and laboratory tests. The prompt identification and cessation of lipid emulsion infusion are the primary principles in treating fat overload syndrome. Additionally, supportive measures such as respiratory and circulatory support should be provided, along with management for any associated complications. Acute pancreatitis refers to a condition characterized primarily by local inflammation of the pancreas and surrounding organs due to abnormal activation of pancreatic enzymes, leading to digestive action on the pancreas itself. In severe cases, it can result in organ dysfunction and acute abdominal pain. The diagnostic criteria for acute pancreatitis include the following three items: (1) Persistent upper abdominal pain; (2)

Serum amylase and/or lipase concentrations elevated more than three times the upper limit of normal; (3) Abdominal imaging findings consistent with radiological changes indicative of acute pancreatitis. Diagnosis requires meeting at least two of these criteria.³ The majority of literature reports focus on cases of fat overload syndrome in patients with mild symptom.^{4,5} In these cases, the prompt cessation of lipid emulsion infusion is typically all that is required for symptom improvement.⁶ Few literature reports have documented cases of severe fat overload syndrome, which often have a poor prognosis and can even result in fatalities.^{7,8} In this study, we successfully treated a critically ill patient with fat overload syndrome accompanied by pancreatitis and pneumonia applying medical interventions such as plasma exchange, hemoperfusion, and advanced life support. The diagnostic and therapeutic experience gained from this case provides valuable insights for the management of severe fat overload syndrome.

Case Report

A 69-year-old elderly woman presented to the Bazhong Traditional Chinese Medicine Hospital with symptoms of abdominal pain and vomiting. She was diagnosed with intestinal obstruction and underwent laparoscopic exploration, removal of foreign body in the ileum, lysis of adhesions, and resection and anastomosis of the affected intestinal segment. After surgery, the patient was instructed to abstain from oral intake, thus she was treated with a 250mL infusion of C14-24 fat emulsion containing 75g (manufactured by Sichuan Kelun Pharmaceutical Co., Ltd.) once a day via intravenous drip. The postoperative review revealed a triglyceride (TG) level of 19.71 mmol/L. On the third day after surgery, the patient experienced worsening abdominal pain and black stools, prompting them to seek medical attention at our hospital (Bazhong Central Hospital). Upon admission, a comprehensive physical examination was performed on the patient. Specifically, auscultation of the lungs revealed decreased breath sounds and the presence of crackles, indicating the presence of pulmonary inflammation. Abdominal examination revealed abdominal distension, generalized tenderness, and muscle guarding. Further, an abdominal CT scan was performed on the patient, revealing acute pancreatitis with pancreatic swelling and slightly decreased enhancement of the local density. The specific performance might be peripancreatic fluid collection, thickening of the peritoneum and omentum, homogeneous decrease in splenic density, and homogeneous increase in gallbladder density (Figure 1). Moreover,



Figure 1 Abdominal CT scan upon admission for this case. (A) Acute pancreatitis is characterized by pancreatic swelling, which is more pronounced in the tail of the organ. Enhanced local density is slightly reduced, while widespread exudative fluid accumulates around the abdominal aorta, splenic artery, and anterior renal fascia. The fascia in front of the left kidney is thickened, and the interstitial spaces of abdominal fat are unclear. (B) The density of the spleen was significantly reduced, and enhancement was markedly decreased, suggesting splenic infarction. The interstitial spaces of abdominal fat were unclear.

Extensive fluid accumulation was observed surrounding the abdominal aorta, splenic artery, and anterior renal fascia, with blurred fat planes in the abdominal cavity (Figure 1). The chest CT scan indicated inflammation in the bilateral lower lobes of the lungs, minimal pleural effusion, and atelectasis in the lower lobes of both lungs (Figure 2). Further analysis of the complete blood count revealed a white blood cell count (WBC) of $33.68 \times 10^9/L$, with 89.1% neutrophils (Neu), hemoglobin (Hb) level of 146g/L, platelet count (PLT) of $242 \times 10^9/L$, procalcitonin (PCT) level of 1.17 ng/mL, and C-reactive protein (CRP) level of 12.03mg/L. The fecal occult blood test was positive, suggesting gastrointestinal bleeding likely induced by postoperative and prolonged fasting. It is worth noting that due to the severe hypertriglyceridemia, it was not possible to evaluate the liver and kidney function, as well as the lipid profile. Overall, blood purification, plasma exchange, oral lipid-lowering medications, CT-guided precise puncture drainage and catheter placement in the necrotic area of the pancreas, along with antimicrobial therapy, fluid resuscitation, and supportive care, are all considered beneficial in aiding the patient's recovery. On the first day of admission, the patient underwent plasma exchange, perfusion, antimicrobial therapy, antispasmodics, acid suppression, enzyme inhibition, and measures to stabilize electrolytes and provide nutritional support as symptomatic treatment. On the second day of admission, despite undergoing plasma exchange, the patient's lipid levels remained elevated, and an electrocardiogram revealed atrial fibrillation (Table 1). To address this, amiodarone was added to the existing treatment plan for rhythm control therapy. Nevertheless, on the third day of admission, the patient exhibited poor mental status and drowsiness. Despite undergoing another round of plasma exchange, both lipid levels and inflammatory markers remained elevated. At this point, the patient developed multi-organ dysfunction involving the cardiovascular, respiratory, and coagulation systems, necessitating endotracheal intubation. Blood perfusion with HA380 and HA330 was initiated to support hemodynamic stability. Following perfusion therapy, a subsequent examination revealed a significant decrease in lipid levels compared to previous measurements. Therefore, plasma exchange was discontinued and the patient was switched to oral lipid-lowering medications for continued treatment. By the fourth day of admission, coagulation function indicators had essentially returned to normal. On the fifth day of admission, there was a significant decrease in the patient's lipid levels compared to previous measurements. However, due to the patient's rapid respiratory rate, diminished breath sounds in both lungs, and chest CT findings suggestive of atelectasis, blood purification was implemented as an adjunct to the existing treatment plan to alleviate fluid overload. On the eighth day of admission, the patient's lipid levels showed significant improvement compared to previous measurements. Whereas, the patient developed recurrent fever, and after ruling out pulmonary infection, we considered acute necrotizing pancreatitis as the cause of fever. Hence, on the sixteenth day of admission, we performed CT-guided precise puncture drainage and catheter placement at the site of pancreatic necrosis, during which purulent fluid with a dark red color was obtained. Meanwhile, we continued with antimicrobial therapy and other treatments. On the twenty-fourth day of admission, approximately 5mL of purulent fluid was still draining from the catheter, but the color was lighter compared to previous drainage. A follow-up abdominal ultrasound showed normal pancreatic size and shape, clear contours,

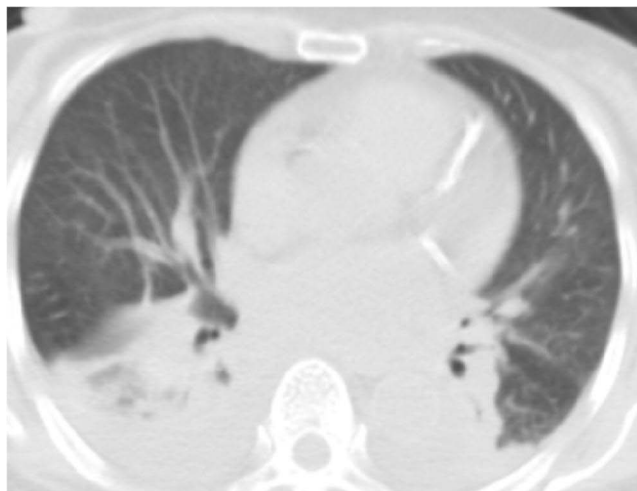


Figure 2 Chest CT scan upon admission for this case. The inflammation and partial consolidation in the bilateral lower lobes of the lungs, along with bilateral pleural effusion.

Table 1 Blood Lipid Levels of Patients at Different Time Points

| Date | CHOL (mmol/L) | TG (mmol/L) | HDL (mmol/L) | LDL (mmol/L) |
|---|---------------|--------------|--------------|--------------|
| June 6, 2022 (Testing in Bazhong Hospital of Traditional Chinese Medicine) | 4.87 | 3.47 | 0.91 | 2.5 |
| June 15, 2022 (Testing in the first day of admission to hospital of Bazhong City) | Undetectable | Undetectable | Undetectable | Undetectable |
| June 16, 2022 (Testing after the first plasmapheresis) | 15.3 | 30 | 0.2 | 4.65 |
| June 17, 2022 (Testing after the second plasmapheresis) | 10.2 | 23.6 | 0.15 | 4.29 |
| June 18, 2022 (Testing after two plasmapheresis and two hemoperfusion) | 6.65 | 2.04 | 0.63 | 3.96 |
| June 21, 2022 (Testing for reexamination) | 7.45 | 1.75 | 0.64 | 4.85 |
| June 24, 2022 (Testing at discharge) | 5.18 | 1.34 | 0.61 | 3.28 |

and uniform echo in the parenchyma, with no dilation of the main pancreatic duct. The results of the follow-up abdominal CT scan are consistent with the ultrasound findings (Figure 3). Further review of the chest CT reveals improved pneumonia bilaterally, with minimal pleural effusion in both lungs (Figure 4). Considering the significant improvement in the patient's condition, the patient was discharged on the twenty-fourth day of admission. Moreover, we have conducted long-term follow-up of the patient. The prognosis is favorable, with repeated measurements indicating normal lipid levels.

Discussion

Multiple studies indicate that the adverse reactions triggered by clinical administration of lipid emulsions are mostly allergic reactions, whereas the occurrence of fat overload syndrome is relatively rare.^{9,10} Fat overload syndrome primarily occurs in two situations. The first situation is the administration of excessive amounts of lipid emulsion to patients with normal lipid metabolism. The second situation is when there are disorders in the patient's internal lipid metabolism mechanism, which can result in fat overload syndrome, even with the administration of a normal amount of lipid emulsion. The patient's outpatient examination indicated acute severe pancreatitis, along with a history of 11 days of normal-dose parenteral nutrition infusion. It can be inferred that the patient already had a pre-existing disorder in fat metabolism and was administered a normal dose of

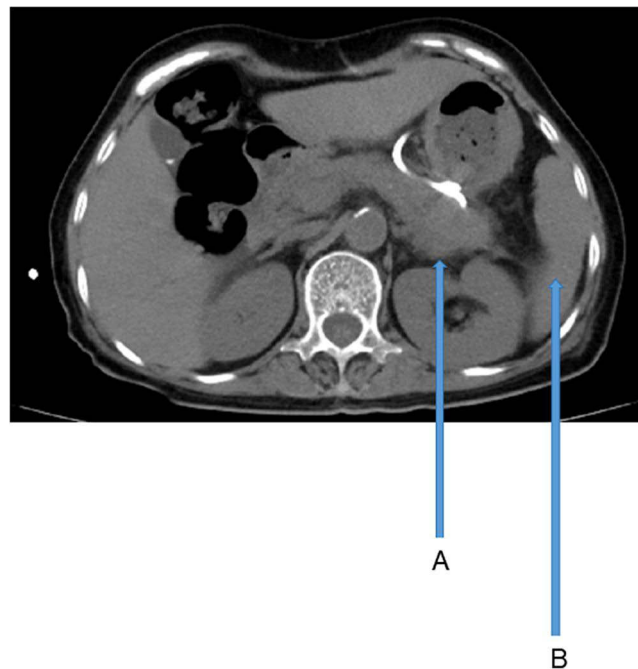


Figure 3 Abdominal CT scan at the time of discharge for this case. (A) The pancreas demonstrates an enlarged morphology with heterogeneous internal density. There are patchy areas of slightly lower density with indistinct borders. A local drainage tube has been implanted. (B) The spleen appears normal, with no apparent abnormalities in density.

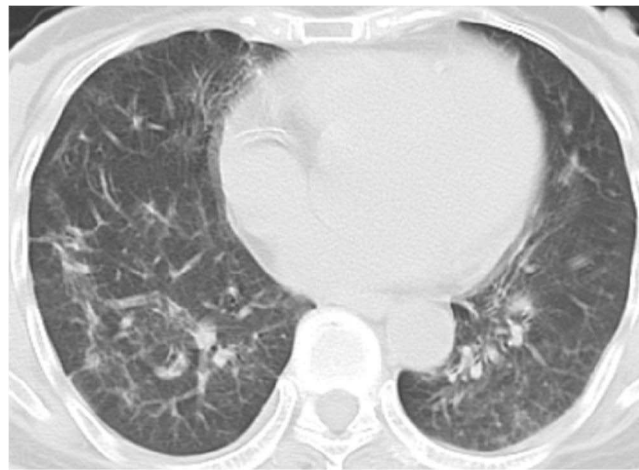


Figure 4 Chest CT scan at the time of discharge for this case. Bilateral pulmonary inflammation shows significant absorption, with minimal pleural effusion observed bilaterally.

lipid emulsion, which could be the main cause of triggering fat overload syndrome. Although there have been other reported cases of fat overload syndrome, this patient experienced persistent abdominal pain and respiratory distress on top of fat overload syndrome. The patient even developed multiple organ failure. Blood samples collected from the patient after admission showed lipemic appearance, appearing abnormally pink. Even after dilution, the lipid levels exceeded the detection range and could not be measured. Indeed, there are also studies indicating that the patient developed fat overload syndrome due to a failure to recheck blood lipids and timely discontinue lipid emulsion.¹¹ Furthermore, after the diagnosis, no effective measures were taken, leading to the exacerbation and acceleration of the underlying disease, ultimately resulting in death. Therefore, the successful management experience reported in this case can serve as a reference for the treatment strategies of fat overload syndrome patients with critical illnesses in the future.

The main principles of treating fat overload syndrome are to provide supportive care for the gastrointestinal, respiratory, and circulatory systems, while also preventing the development of complications. Previous studies have demonstrated that plasma or volume exchange is an effective treatment for fat overload syndrome.^{12,13} Furthermore, previous studies have indicated that heparin administration enhances the activity of circulating lipoprotein lipase and hepatic lipase, thereby facilitating the clearance of hyperlipidemia.¹⁴ Furthermore, insulin that activates lipoprotein lipase leading to decrease in triglycerides. The previous study has revealed that improving insulin sensitivity and mitochondrial oxidative capacity can reverse the effects of lipid overload.¹⁵ For blood lipid abnormalities, there are currently various treatment options. For example, fenofibrate can activate peroxisome proliferator-activated receptor alpha (PPAR- α), thereby increasing the activity of tissue lipoprotein lipase and the breakdown of triglycerides in very low-density lipoprotein (VLDL), thus regulating abnormal lipid levels.¹⁶ Niacin can also lower levels of lipoprotein(a) [Lp(a)], thereby regulating lipid abnormalities.¹⁷ It's worth noting that consuming 2–4 grams of Omega-3 fatty acids per day can also lower triglyceride levels.¹⁸ Additionally, when statin therapy is intensified, inflammation and hyperlipidemia can be controlled, potentially altering their relative contribution to future cardiovascular event risks.¹⁹ In this study, following the definitive diagnosis of fat overload syndrome based on the patient's medical history and relevant examinations, in addition to discontinuing lipid emulsion, we initially focused on maintaining respiratory and circulatory function. Subsequently, anti-inflammatory measures were employed, followed by precise percutaneous drainage and replacement of necrotic pancreatic tissue guided by CT. As a result, the patient's clinical symptoms significantly improved, leading to discharge after recovery. As a commonly used nutritional supplement in clinical practice, lipid emulsions are widely used. However, through the treatment and management of this case, we have implemented a stricter assessment of the indications for lipid emulsion therapy. In particular, for patients with normal TG levels but potentially impaired lipid clearance or metabolism, healthcare providers should focus on careful evaluation. Additionally, caution should be exercised when using lipid emulsions, especially in elderly individuals, decompensated diabetes mellitus, obesity, patients with impaired liver and kidney function, as well as those with hyperlipidemia.²⁰

Conclusion

This case report highlights the association between fat overload syndrome and pancreatitis as well as the use of lipid emulsions. Improper application of lipid emulsions can result in significant harm to patients, as demonstrated in this case. It is crucial to emphasize that during the administration of lipid emulsions, meticulous control of the infusion rate and vigilant monitoring of the patient's vital signs are paramount. In the event of adverse reactions, the infusion should be promptly discontinued and appropriate symptomatic treatment initiated. When confronted with fat overload syndrome, expeditious assessment of the patient's condition is imperative, followed by early implementation of plasma exchange and blood perfusion. In cases of concurrent multiple organ dysfunction, prompt respiratory and circulatory support should be administered without delay.

Ethics Statement

The patient and her family allowed her case to be presented and submitted, and written consent was obtained. The patient also agreed to be published. Institutional Review Board was not required for the publication of this case report.

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Author Contributions

All authors made a significant contribution to the work reported, took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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