

# World Journal of *Clinical Cases*

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# Successful management of severe hypoglycemia induced by total parenteral nutrition in patients with hepatocellular injury: Three cases reports

Ling-Zhi Fang, Hui-Xin Jin, Na Zhao, Yu-Pei Wu, Ying-Qin Shi

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## Abstract

### BACKGROUND

Glucose imbalance is common in total parenteral nutrition (TPN). Hypoglycemia seems to be less frequent than hyperglycemia, but it influences the clinical outcome to a greater extent. Therefore, it should be effectively prevented and treated. However, there is no relevant report on how to treat hypoglycemia caused by TPN in patients with liver cell injury.

### CASE SUMMARY

We present three patients with liver cell injury who developed severe hypoglycemia during or after TPN infusion. The causes of severe hypoglycemia and glucose-raising strategies were discussed. According to the physiological characteristics of the hepatocellular injury, the ratio of nutrition components prescribed in TPN was appropriately adjusted for the three cases. We simultaneously reduced the dose of insulin and fat emulsion, and increased the dose of glucose in TPN. The blood glucose level was restored to normal range and clinical symptoms were eliminated.

### CONCLUSION

When hypoglycemia occurs during or after TPN in patients with hepatocellular injury, physicians need to simultaneously reduce insulin and fat emulsion, and increase glucose, and correct severe hypoglycemia in time to reduce its adverse consequences.

**Key Words:** Total parenteral nutrition; Hepatocellular injury; Severe hypoglycemia; Treatment; Causes; Case report

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**Core Tip:** Glucose imbalance is common in total parenteral nutrition (TPN). Hypoglycemia seems to be less frequent than hyperglycemia, but it influences the clinical outcome to a greater extent. Therefore, it should be prevented and treated. At present, there is no relevant report on how to treat hypoglycemia caused by TPN in patients with liver cell injury. We reported that three patients with liver cell injury developed severe hypoglycemia during or after TPN. The causes of severe hypoglycemia and the strategy of increasing glucose were discussed, which can provide a reference for reducing the adverse consequences of hypoglycemia.

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## INTRODUCTION

Parenteral nutrition is the intravenous supply of nutrients needed by patients, including glucose, fat emulsion, amino acids, vitamins, electrolytes, insulin and trace elements[1], which can maintain good nutritional status and survival of patients who cannot feed. It can be divided into total parenteral nutrition (TPN) and supplemental parenteral nutrition according to patients' needs. The purpose is to maintain good nutritional status when patients cannot eat normally or eat less than 60% of their normal intake[2]. Although parenteral nutrition can save the lives of patients who cannot eat, the improper prescription of parenteral nutrition can also lead to serious adverse consequences. We report three patients with hepatocellular injury who received a nutritional risk screening (2002-NRS screening scale) with scores > 3, indicating a high risk of malnutrition, who developed severe hypoglycemia during or after TPN.

## CASE PRESENTATION

### Chief complaints

**Case 1:** An 85-year-old male patient was admitted to hospital for hematemesis and hematochezia.

**Case 2:** A 75-year-old male patient was admitted to hospital for poor appetite, abdominal pain, hiccups, yellow staining of skin and sclera 3 d previously.

**Case 3:** A 55-year-old female was hospitalized for further treatment due to abdominal distention, poor food intake, and significantly reduced frequency of defecation and fart recently.

### History of present illness

**Case 1:** The patient complained of hematemesis and hematochezia.

**Case 2:** The patient underwent percutaneous transhepatic cholangial drainage (PTCD) in another hospital 2 wk previously because of obstructive jaundice. Postoperative bile drainage was smooth, with a daily average 300 mL. The patient complained of poor appetite, abdominal pain, hiccups, and yellow staining of skin and sclera 3 d previously.

**Case 3:** The patient complained of abdominal distention, poor food intake, significantly reduced frequency of defecation and fart recently due to recurrence and metastasis of breast cancer at > 4 years after surgery.

### History of past illness

None of the patients had any other medical history.

### Personal and family history

None of the patients had any remarkable personal or family history.

### Physical examination

**Case 1:** Temperature 38.3 °C, respiratory rate 19 breaths/min, pulse rate 60 beats/min, and blood pressure 93/61 mmHg. Physical examination showed clear consciousness, listlessness, clear speech, erythema palmare, no spider nevus, conjunctiva was pale and sclera was slightly yellow.

**Case 2:** Temperature 35.8 °C, respiratory rate 20 breaths/min, pulse rate 107 beats/min, and blood pressure 95/73 mmHg. Physical examination showed clear consciousness, fluent language, yellow skin and sclera, normal abdominal appearance, right midaxillary line of the fifth intercostal PTCD drainage tube, smooth drainage, and well fixed. Abdominal tenderness was obvious, mainly under the xiphoid process, without radiating pain, rebound pain or any abdominal mass.



**Case 3:** Temperature 37.1 °C, respiratory rate 18 breaths/min, pulse rate 63 beats/min, and blood pressure 95/60 mmHg. Physical examination showed listless spirit, lack of left breast, abdominal distension, no gastric pattern and peristaltic waves, abdominal hardness, induration of the right side of the middle abdomen, positive shifting dullness, hyperactive bowel sounds, and sound of air passing through water could be heard through auscultation.

### Laboratory examinations

**Case 1:** Blood biochemical studies: albumin 29.5 g/L (normal 40–55 g/L), total and direct bilirubin 33 µmol/L (normal 0–23 µmol/L) and 19.9 µmol/L (normal 0–4 µmol/L) respectively, alanine transaminase (ALT) 174.7 U/L (normal 9–50 U/L), aspartate transaminase (AST) 156.8 U/L (normal 15–40 U/L), blood glucose 5.5 mmol/L. Glycated hemoglobin 5%. Blood analysis results: white blood cell count  $17.1 \times 10^9$ /L, neutrophil ratio as 73.4, hemoglobin 100 g/L.

**Case 2:** Blood biochemical studies: hemoglobin 97 g/L, total and direct bilirubin 220.7 µmol/L and 113.8 mmol/L respectively, ALT 96.4 U/L, AST 109.5 U/L, γ-glutamyltransferase 98.6 U/L, alkaline phosphatase 134.5 U/L, K 3.2 mmol/L, Na 135 mmol/L, Cl<sup>-</sup> 90 mmol/L, P 0.69 mmol/L, Mg 0.60 mmol/L, Ca 1.79 mmol/L, blood glucose 5.0 mmol/L. Glycated hemoglobin 4.4%.

**Case 3:** Blood biochemical studies: albumin 25 g/L, hemoglobin 96 g/L, K 2.9 mmol/L, Na 125 mmol/L, Cl<sup>-</sup> 85 mmol/L, P 0.60 mmol/L, Mg 0.48 mmol/L, Ca 1.68 mmol/L, glucose 3.39 mmol/L, AST 125 U/L, ALT 96 U/L, blood glucose 5.2 mmol/L. Glycated hemoglobin 4.5%.

### Imaging examinations

**Case 1:** Abdominal computed tomography (CT) revealed possible abdominal abscess.

**Case 2:** Upper abdominal CT showed dilatation of extra- and intrahepatic bile ducts and common bile duct. Obstruction of the common bile duct was considered.

**Case 3:** Upright abdominal plain film showed that some intestinal lumens in the abdominal cavity were dilated and gas was accumulated, and the gas–liquid plane was found in the left lower abdomen, which was considered as incomplete intestinal obstruction.

## FINAL DIAGNOSIS

The final diagnosis of Case 1 was decompensated cirrhosis, hepatic pleural effusion, abdominal effusion, and upper gastrointestinal bleeding. The final diagnosis of Case 2 was cholangiocarcinoma (with query retroperitoneal lymph node metastasis), obstructive jaundice, acute liver injury, malnutrition, electrolyte metabolism disorder. The final diagnosis of Case 3 was chest wall metastasis and liver metastasis of left breast cancer after postoperative radiotherapy and chemotherapy, malnutrition, electrolyte metabolism disorder, incomplete intestinal obstruction.

## TREATMENT

### Case 1

In addition to the conventional treatment of cirrhosis complicated with gastrointestinal bleeding, the patient was deprived of food and water and received TPN support. The nutritional plan is shown in Table 1. On day 1, the peripheral blood glucose was 7.4 mmol/L at 2 h after receiving TPN, but 1 h after infusion of TPN, the patient suddenly developed palpitations, cold sweat and hunger. The peripheral blood glucose was 2.3 mmol/L, and the symptoms were gradually relieved by intravenous infusion of 40 mL 50% glucose injection. On day 2, the TPN program was adjusted, and the insulin dose was reduced from 16 to 8 U. One hour after infusion of TPN, the patient showed the symptoms of hypoglycemia again. On day 3, the TPN regimen was adjusted again, that is, no insulin was added into the nutrient solution and the drip rate was slowed from 2 to 1 mL/min, but the patient still showed symptoms of hypoglycemia. The nutrition plan was adjusted on day 4: the dosage of 50% glucose injection was increased by 50 mL, while the dosage of fat emulsion was reduced by 50 mL, and the infusion speed was adjusted to 2 mL/min. No hypoglycemia occurred in the patient 1 h after infusion of TPN.

### Case 2

The patient was unable to eat due to hiccups and abdominal pain after eating, and received TPN support. The nutritional plan is shown in Table 1. The patient suddenly had palpitations and cold sweat at 2 h after infusion of TPN on day 1. Peripheral blood glucose was 3.9 mmol/L, and the symptoms were gradually relieved by intravenous infusion of 40 mL 50% glucose injection. The blood glucose returned to normal 1 h after completion of TPN. On day 2, the dosage of insulin was reduced from 16 to 6 U, and the postprandial peripheral blood glucose was 4.3 mmol/L. The patient had low blood glucose but no symptoms of hypoglycemia. On day 3, the nutrition plan was adjusted: no insulin was added to the nutrition solution, the dosage of 50% glucose injection was increased by 50 mL, and the dosage of fat emulsion was reduced by 100 mL, the blood glucose was 7.2 mmol/L at the beginning of 2 h of infusion of TPN, which was within the



Table 1 Partial parenteral nutrition preparation prescription and treatment plan adjustment and blood glucose								
Patient	Time	50% glucose (mL)	Medium and long chain fat emulsion (mL)	Compound amino acid 3AA (mL)	Insulin (U)	Blood glucose of 2 h after TPN initiated (mmol/L)	Blood glucose of 1 h after TPN (mmol/L)	Drip rate (mL/min)
Case 1	Day 1	300	250	400	16	7.4	2.3	2
	Day 2	300	250	400	8	7.2	2.6	2
	Day 3	300	250	400	0	7.4	3.4	1
	Day 4	350	200	400	0	7.8	6.4	2
Case 2	Day 1	300	200	500	16	3.9	7.1	2
	Day 2	300	200	500	6	4.3	7.6	2
	Day 3	350	100	550	0	7.2	7.3	2
Case 3	Day 1	250	250	750	12	3.8	6.0	2
	Day 2	250	250	750	0	5.6	6.6	2
	Day 3	300	150	800	0	7.6	7.8	2

TPN: Total parenteral nutrition.

normal range.

Case 3

The patient was given TPN support due to incomplete intestinal obstruction and fasting and water deprivation. The nutritional plan is shown in Table 1. On day 1, 2 h after receiving TPN, the patient suddenly felt palpitations, and peripheral blood glucose was 3.8 mmol/L. After intravenous infusion of 40 mL 50% glucose injection, the peripheral blood glucose was 5.0 mmol/L, indicating low blood glucose. The blood glucose returned to normal 1 h after completion of TPN. On day 2, no insulin was added to the TPN, and the peripheral blood glucose was 5.6 mmol/L 2 h after receiving TPN, but the patient did not show symptoms of hypoglycemia. The nutrition plan was adjusted on day 3: no insulin was added to the nutrition solution, the dosage of 50% glucose injection was increased by 50 mL, and the dosage of fat emulsion was reduced by 100 mL. Peripheral blood glucose was 7.6 mmol/L 2 h after receiving TPN, which was within the normal range.

In addition to the composition shown in Table 1, the nutritional regimens also included 10% potassium chloride, fat-soluble vitamin, water-soluble vitamin, calcium gluconate, magnesium sulfate, sodium chloride, and a variety of trace elements. Case 1 also received alanyl glutamine injection. During infusion of TPN, the ratio of insulin to glucose in nondiabetic patients was 1 U to (6-10 g), and postprandial glucose was observed from the beginning of infusion of TPN solution to 2 h later.

OUTCOME AND FOLLOW-UP

The blood glucose was returned to a normal level at follow-up in all three cases.

DISCUSSION

The main function of insulin in concert with other hormones such as glucagon is the control of blood glucose levels[3]. The ratio of insulin to glucose in TPN in nondiabetic patients is 1 U to (6-10 g)[4]. In patients with hepatocellular injury, glucose oxidation rate and storage rate are decreased, and liver glycogen storage and production are reduced[5,6], and energy metabolism shifts from carbohydrate predominance to more fat oxidation[7,8]. When fat and glucose are supplied together, patients with hepatocellular injury tend to oxidize glucose rather than fat. When energy supply is insufficient or not timely, patients are prone to severe hypoglycemia. Therefore, it is necessary to increase the amount of glucose in TPN

solution and reduce the insulin dose, or do not add insulin, to better maintain blood glucose level.

The liver is closely related to the digestion, absorption, decomposition, metabolism, storage and transportation of fat [9]. Medium- and long-chain fat emulsion injection of TPN is a mixture of long-chain fat emulsion and medium-chain fat emulsion at the ratio of 1 to 1. Medium-chain fat emulsion can be oxidized readily by all body tissues and little is redeposited in the liver[10]. Long-chain fat emulsion provides essential fatty acids, linoleic acid and linolenic acid, but transport of the emulsion into mitochondria depends on carnitine as a carrier[11]. When liver cells are damaged, the synthesis of carnitine and apolipoprotein in liver decreases, affecting the transportation and utilization of lipids, especially the oxidation of long-chain fatty acids, resulting in a decrease in the clearance rate of long-chain fatty acids. If the synthesis of carnitine is reduced, the long-chain fat emulsion cannot enter the mitochondria for oxidative decomposition, resulting in the accumulation of fat in the liver and further damage to the liver[12]. Obstructive jaundice can cause abnormal bile secretion and cholestasis, which results in reduced bile acid salt secretion, and reduced absorption and oxidative decomposition of fat. Bile obstruction and increased biliary pressure cause damage to the structure and function of liver cells, causing liver dysfunction or aggravating liver cell damage. When bilirubin level is > 200  $\mu\text{mol/L}$ , fat emulsion should be reduced and not used in TPN, which reduces the burden on the liver and the impact of bilirubin metabolism[13]. Therefore, when patients with hepatocellular injury receive TPN support, the dose of fat emulsion should be appropriately reduced to reduce the burden on the liver and relieve the symptoms of severe hypoglycemia during and after TPN infusion.

No matter what causes liver cell damage, glucose and fat metabolism and blood glucose will be affected. Our three cases had liver cell damage with different causes, and all of them had normal blood lipids, kidney function, heart function and lung function. However, they all had severe hypoglycemia during or after TPN infusion. When adjusting the nutritional regimen, blood glucose could not be restored to normal levels by reducing insulin dose, not adding insulin, or slowing the drip rate. However, blood glucose was restored to normal by appropriately reducing the amount of insulin, reducing the amount of fat emulsion, and increasing the amount of glucose at the same time.

Not all patients with hepatocellular injury will suffer from severe hypoglycemia, which may be related to different degrees of cirrhosis, bile duct obstruction, hepatocyte injury, and size of liver metastasis. Therefore, for timely detection of severe hypoglycemic events during or after parenteral nutrition infusion, it is recommended that physicians closely monitor peripheral blood glucose of patients with hepatocellular damage on the first day of TPN infusion. If hypoglycemia is found, physicians can reduce the dose of fat emulsion and insulin, and increase the dose of glucose at the same time, to reduce the burden of fat metabolism in the liver, and compensate for the reduction in glycogen storage in the liver, to rapidly restore blood glucose level.

## CONCLUSION

It is rarely reported that severe hypoglycemia is caused by TPN in patients with hepatocellular injury. By reporting the three cases, we aim to raise awareness among physicians and enhance glucose monitoring in these patients, and provide a reference for treatment, to reduce the adverse consequences caused by severe hypoglycemia.

## FOOTNOTES

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