



Parenteral Nutrition-Induced Diffuse Nodular Hepatic Steatosis After Hepatocellular Carcinoma Resection: A Case Report

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Abstract

Introduction: Hepatic multifocal nodular fatty infiltration induced by parenteral nutrition is a rare complication, particularly in patients with a history of hepatocellular carcinoma who have undergone hepatectomy. Its imaging features may overlap with those of tumor recurrence, leading to misdiagnosis. Clinical data on hepatic multifocal nodular fatty infiltration in patients receiving long-term parenteral nutrition after hepatocellular carcinoma resection remain limited.

Case Presentation: We retrospectively analyzed the case of a 50-year-old woman with a 16-year history of hepatitis B virus infection who underwent laparoscopic left hepatectomy for hepatocellular carcinoma in April 2024. The patient received parenteral nutrition with C6 - 24 mixed fat emulsion for more than 1 month postoperatively. Clinical data, laboratory results, and computed tomography and magnetic resonance imaging findings were collected and analyzed, followed by observation of lesion changes after parenteral nutrition withdrawal. On postoperative day 25, imaging revealed multiple well-defined nodular hypodense hepatic lesions. Magnetic resonance imaging showed slightly increased signal intensity on T1- and T2-weighted images, signal reduction on fat-suppression sequences and opposed-phase imaging, and no enhancement on dynamic contrast-enhanced imaging. Liver function indices increased transiently. After parenteral nutrition was discontinued and nutritional support was adjusted for 40 days, partial regression of the lesions was observed.

Conclusions: Long-term parenteral nutrition with an excessive lipid emulsion, particularly a lipid energy supply of more than 50%, may induce hepatic multifocal nodular fatty infiltration in patients with metabolic disorders after hepatocellular carcinoma resection. Characteristic imaging features, including the absence of enhancement and fat-signal characteristics, can help distinguish this condition from tumor recurrence. Timely withdrawal of parenteral nutrition and nutritional adjustment may promote lesion regression.

Keywords: Magnetic Resonance Imaging, Hepatocellular Carcinoma, Hepatic Steatosis, Parenteral Nutrition, Computed Tomography

1. Introduction

Hepatic multifocal nodular fatty infiltration (HMNFI) is an uncommon benign entity that can mimic malignant hepatic lesions on imaging (1, 2). It typically presents as multiple hypodense nodules on unenhanced computed tomography (CT) without enhancement (1, 6). Magnetic resonance imaging (MRI) demonstrates characteristic signal dropout on fat-suppressed and opposed-phase sequences, confirming intracellular fat deposition (2, 6).

Patients who undergo hepatocellular carcinoma (HCC) resection often have metabolic disturbances. Prolonged parenteral nutrition (PN) is a well-recognized risk factor for hepatic steatosis, particularly when lipid supply is excessive (3-5). This case report describes PN-induced HMNFI after HCC resection and highlights

imaging features that may help avoid misdiagnosis as recurrence or metastasis (7).

2. Case Presentation

A 50-year-old woman with a 16-year history of chronic hepatitis B was admitted with dull pain in the right upper quadrant. Her body mass index was 23.1 kg/m². She had no diabetes, dyslipidemia, alcohol intake, or history of hepatotoxic medication use. Liver function was Child-Pugh class A. Imaging confirmed HCC in the left hepatic lobe (4, 8).

She underwent laparoscopic left hepatectomy in April 2024. Postoperatively, she developed gastrointestinal dysfunction and was unable to tolerate oral intake. Therefore, PN was administered from May 11 to May 30, 2024 (20 days). The PN regimen included C6 -

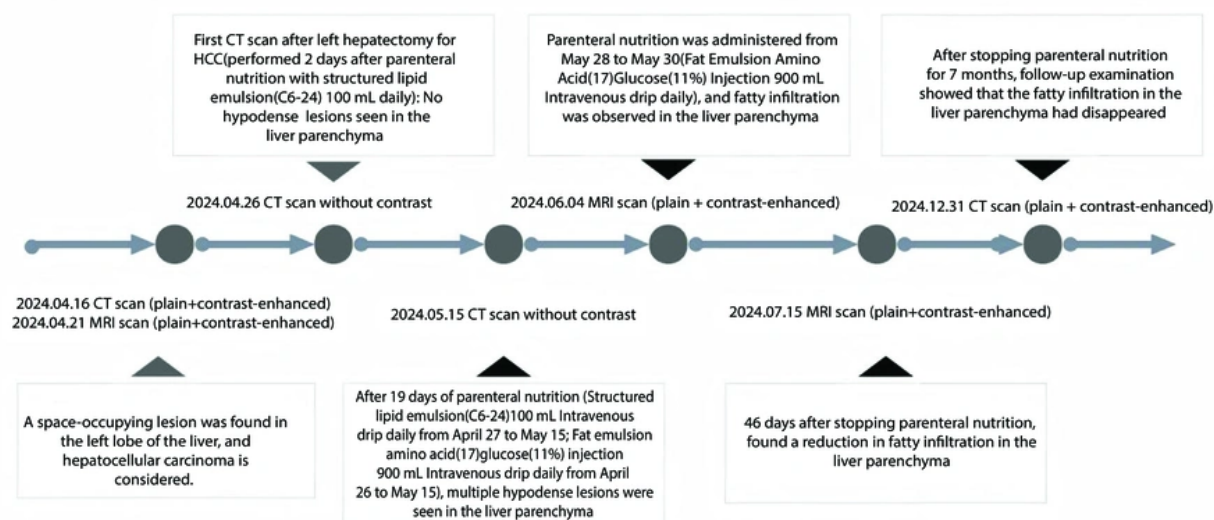


Figure 1. Patient treatment and imaging follow-up timeline

24 mixed lipid emulsion (medium- and long-chain triglycerides), 100 mL/d; lipid-amino acid 17-glucose (11%) injection, 900 mL/d; and compound amino acids, glucose, electrolytes, and vitamins.

The clinical timeline was as follows: laparoscopic left hepatectomy was performed in April 2024; PN was initiated on May 11, 2024; multiple nodules were detected on unenhanced CT on May 15, 2024 (postoperative day 25); MRI confirmed HMNFI on June 4, 2024; PN was discontinued on May 30, 2024; and follow-up MRI on July 15, 2024, showed partial regression of the lesions (Figure 1).

Unenhanced CT showed multiple well-circumscribed hypodense nodules. MRI revealed mild hyperintensity on T1-weighted imaging and T2-weighted imaging, signal loss on fat-suppressed T2-weighted imaging, signal reduction on opposed-phase imaging compared with in-phase imaging, no restricted diffusion, and no enhancement on dynamic contrast-enhanced sequences (Figure 2).

These findings were diagnostic of HMNFI (1, 2). Alternative diagnoses, including HCC recurrence, metastasis, hemangioma, cyst, and perfusion disorder, were systematically excluded. Biopsy was not performed because the imaging features were pathognomonic and the lesions regressed after PN withdrawal.

Serial liver function tests are shown in Table 1. Transient elevations in alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT) were observed. Total bilirubin, direct bilirubin, and albumin remained stable. After PN cessation, all markers improved, consistent with imaging regression.

2.1. Differential Diagnosis

HCC recurrence was excluded because recurrent HCC typically shows wash-in and washout enhancement, which was not observed in this case (4). Liver metastases were excluded because peripheral enhancement and the bull's-eye sign were absent (4). Hepatic hemangioma was excluded because peripheral nodular enhancement was not present (4). Liver cyst was excluded because the lesions did not show water attenuation and demonstrated fat-signal characteristics (4). Focal nodular hyperplasia was excluded because a central scar and characteristic flow features were absent (2).

3. Discussion

HCC resection disrupts hepatic metabolism, and prolonged PN may trigger fatty infiltration (3). Exogenous lipid overload may exceed the liver's oxidative capacity, leading to focal fat accumulation (3).

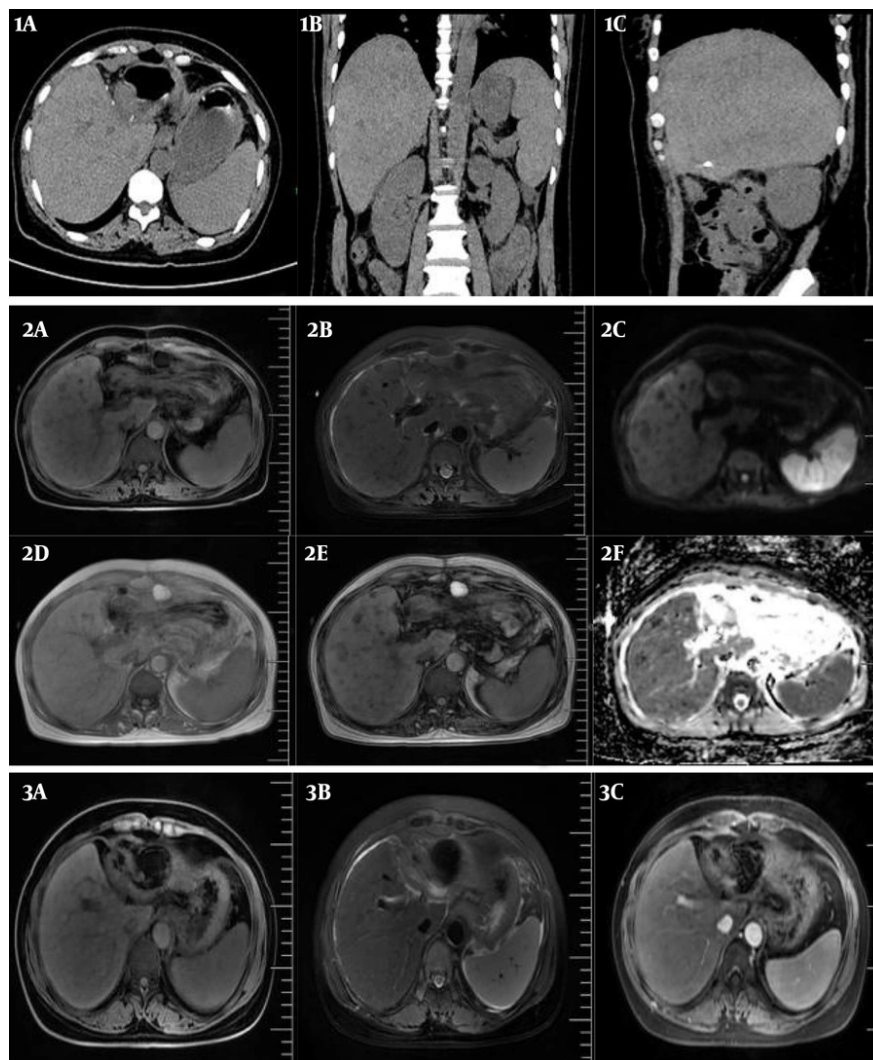


Figure 2. Imaging findings. (1A-1C) Unenhanced computed tomography shows multiple hypodense nodules; (2A) T1-weighted imaging shows hyperintensity; (2B) T2-weighted imaging shows hyperintensity; (2C) diffusion-weighted imaging shows hypointensity; (2D-2E) opposed-phase imaging shows signal reduction consistent with fat; (2F) apparent diffusion coefficient mapping shows no restricted diffusion; and (3A-3C) follow-up magnetic resonance imaging after parenteral nutrition discontinuation shows partial lesion regression.

HMNFI exhibits typical MRI features that allow for a confident diagnosis (1, 2). Unlike metabolic dysfunction-associated fatty liver disease, PN-related HMNFI is reversible after lipid withdrawal (3, 4).

We recommend monitoring hepatobiliary function 2 weeks after PN initiation to enable early detection of steatosis (1).

Prolonged PN with high lipid content can induce HMNFI after HCC resection. Its characteristic imaging

features allow for reliable differentiation from malignant lesions. Early discontinuation of PN and nutritional adjustment can lead to lesion regression.

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Table 1. Liver Function Parameters During the Treatment Course ^a

Date	ALT (U/L)	AST (U/L)	ALP (U/L)	GGT (U/L)	TBIL (μmol/L)	DBIL (μmol/L)	ALB (g/L)
April 24, 2024	204	196	76	36	22.6	7.8	33.6
April 29, 2024	91	46	65	72	37.1	29.1	31.2
April 30, 2024	61	43	53	45	38.7	30.1	32
May 3, 2024	51	51	66	57	33.9	19.4	30.7
May 7, 2024	46	49	73	40	60.2	36.6	39.7
May 9, 2024	50	54	80	41	78.1	41	41.3
May 11, 2024	55	63	108	54	80.1	53.1	39.6
May 14, 2024	106	140	251	277	50.5	34.3	37.9
May 17, 2024	74	57	153	180	44.7	29.8	34.4
May 19, 2024	51	36	157	136	34.2	23.1	34.5
May 21, 2024	53	45	135	134	34.0	23.5	34.5
May 23, 2024	54	50	145	131	36.5	23.3	35.4
May 27, 2024	72	63	145	126	40.3	26.2	37.0
May 30, 2024	108	80	136	191	45.8	26.0	32.4
June 2, 2024	107	111	176	250	25.2	17.4	34.5
June 8, 2024	176	100	336	434	17.2	11.5	33.1
June 12, 2024	82	59	227	266	15.1	9.5	32.8
June 16, 2024	53	50	188	213	13.6	8.0	34.5
June 20, 2024	43	44	192	191	15.2	8.2	34.2
July 14, 2024	31	44	127	160	10.7	6.5	42.4
July 22, 2024	39	52	125	143	11.7	4.7	44.7
December 30, 2024	45	46	75	79	14.7	10.8	39.3

^a Abbreviations: ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; DBIL, direct bilirubin; GGT, gamma-glutamyl transferase; TBIL, total bilirubin.

Footnotes

AI Use Disclosure: The authors declare that no generative AI tools were used in the creation of this article.

Authors' Contribution: Study concept/design and supervision: P. T.; Data acquisition: A. W., X. W.; Data analysis/interpretation: X. X. and P. G.; manuscript drafting: A. W.; critical revision: P. T.; statistical analysis: X. X.; Administrative/technical/material support: X.W. and P. G.

Conflict of Interests Statement: The authors do not declare any conflicts of interests for this study.

Data Availability: The data supporting the findings of this study are not publicly available due to ethical restrictions (to protect patient confidentiality) but are available from the corresponding author upon reasonable request, subject to institutional review board approval.

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Informed Consent: Written informed consent was obtained from the patient for publication of this case report and all accompanying images. The informed consent form has been uploaded as a separate file in the submission system.

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