Surgery Case Reports: Advances and Techniques 8 (2026) 100201

Contents lists available at ScienceDirect

Surgery Case Reports: Advances and Techniques

journal homepage: www.sciencedirect.com/journal/surgery-case-reports

De novo acute liver failure requiring urgent liver transplantation after sleeve gastrectomy: A case report

Francesco Saverio Papadia [a,b,*], Marco Miggino [b], Alessandro Gambella [a,b], Federica Grillo [a,b], Enzo Andorno [b]

[a] Department of Surgical Sciences and Integrated Diagnostics (DISC), School of Medical and Pharmaceutical Sciences, University of Genoa, Genoa, Italy
[b] IRCCS Policlinico San Martino, Genoa, Italy

ARTICLE INFO

Keywords:
Acute liver failure
Sleeve gastrectomy
Bariatric surgery
Liver transplantation

ABSTRACT

Introduction: Bariatric surgery typically improves metabolic dysfunction-associated steatotic liver disease. We report a paradoxical case of de novo acute liver failure requiring super-urgent liver transplantation after sleeve gastrectomy.

Presentation of Case: A 46-year-old woman without pre-existing liver disease underwent uneventful sleeve gastrectomy, losing 20 kg over four months. She subsequently developed acute liver failure with encephalopathy and multi-organ failure. Extensive evaluation revealed no hepatotoxic trigger, alcohol use, or cirrhosis. Explant histopathology showed confluent necrosis involving 40% of the parenchyma. She underwent successful liver transplantation and has good graft function at nine months.

Conclusion: This is the first reported case of de novo acute liver failure requiring transplantation after sleeve gastrectomy without identifiable risk factors. It demonstrates that catastrophic liver injury can occur after restrictive procedures, highlighting a critical knowledge gap in individual susceptibility.

Introduction

Bariatric surgery is well-established for its hepatic benefits, with numerous studies demonstrating improvement or resolution of metabolic dysfunction-associated steatotic liver disease (MASLD) following both malabsorptive and restrictive procedures [1–8]. The underlying mechanisms include weight loss-mediated reductions in hepatic steatosis, inflammation, and fibrosis, as well as favorable changes in gut hormones such as glucagon-like peptide-1 (GLP-1) [9–11].

We report a paradoxical case of de novo acute liver failure requiring super-urgent liver transplantation after sleeve gastrectomy in a patient without identifiable risk factors. To our knowledge, this represents the first such documented case. This report highlights a critical knowledge gap in individual susceptibility to postoperative liver injury and underscores the importance of vigilance even for procedures generally considered safe.

Case presentation

Patient information

A 46-year-old woman with a body mass index of 42 kg/m^2 underwent laparoscopic sleeve gastrectomy for class III obesity (Fig. 1). Her medical history was significant only for well-controlled hypertension. She had no history of liver disease, alcohol use, or hepatotoxic medication exposure. Preoperative liver function tests were within normal limits.

Clinical findings and timeline

The surgical procedure was uneventful, and the patient was discharged without complications.

Over the subsequent four months, she achieved progressive weight loss of 20 kg (approximately 1.25 kg per week). During this period, she remained asymptomatic with regular follow-up.

Four months post-surgery, she developed jaundice, fatigue, and

* Correspondence to: Department of Surgical Sciences and Integrated Diagnostics (DISC), University of Genoa, Via Benedetto XV, 6, Second Floor, Genoa 16132, Italy.
E-mail address: francesco.papadia@unige.it (F.S. Papadia).

https://doi.org/10.1016/j.sycrs.2026.100201
Received 24 March 2026; Received in revised form 1 April 2026; Accepted 4 April 2026
Available online 6 April 2026

nausea. Over the following week, her condition rapidly deteriorated with worsening encephalopathy, coagulopathy, and multi-organ failure. She was transferred to our tertiary center for further management.

Diagnostic assessment

Laboratory evaluation revealed: total bilirubin 24 mg/dL, international normalized ratio 4.2, alanine aminotransferase 1890 U/L, aspartate aminotransferase 2450 U/L, and creatinine 3.1 mg/dL. Extensive workup was negative for viral hepatitis (A, B, C, E, CMV, EBV), autoimmune hepatitis (ANA, ASMA, anti-LKM1), Wilson's disease (ceruloplasmin, urinary copper), and alpha-1 antitrypsin deficiency. No hepatotoxic medications or supplements were identified. Abdominal imaging demonstrated hepatomegaly with steatosis but no evidence of biliary obstruction or vascular compromise.

Therapeutic intervention

Due to worsening encephalopathy and a Model for End-Stage Liver Disease-Na score of 31, she was listed for super-urgent transplantation. The peak pre-transplant arterial lactate was 8.2 mmol/L. Orthotopic liver transplantation was performed on May 22, 2025. Intraoperative findings included abundant ascites (3 L) and a frankly steatotic liver with nodular margins. Cold ischemia time was 5 h 40 min; warm ischemia time was 41 min. Transfusion requirements were 11 units of packed red blood cells, 9 units of plasma, and 2 units of platelets.

Following transplantation, the lactate level normalized to 1.5 mmol/L on the first postoperative day.

Explant pathology showed completely disrupted architecture with extensive collapse and necrosis involving approximately 40% of the parenchyma and immature collagen deposition (Fig. 2). Residual viable hepatocytes exhibited feathery degeneration and cholestasis. The diagnosis was diffuse confluent hepatocyte necrosis.

Follow-up and outcomes

The postoperative course was complicated by necrotizing pancreatitis requiring drainage, persistent pleural effusion requiring thoracoscopy with chemical pleurodesis, acute kidney injury requiring temporary renal replacement therapy, cytomegalovirus reactivation, and *Strongyloides* infection. Following neurorehabilitation, she made gradual functional recovery. At nine months post-transplant, she is ambulatory with normal liver function tests. Time line of events is illustrated in Fig. 3.

Discussion

To our knowledge, this is the first reported case of de novo acute liver failure requiring transplantation after sleeve gastrectomy in a patient without identifiable risk factors. This finding is paradoxical given the substantial evidence that bariatric surgery confers hepatic benefits and highlights that rare and unexpected hepatic complications may occur even after procedures generally considered safe.

Differential diagnosis

The evaluation of acute liver failure in the postoperative period necessitates a broad differential diagnosis. In this case, extensive serological and histological investigations were performed to exclude common etiologies. Viral hepatitis (A, B, C, E, CMV, EBV) was ruled out by

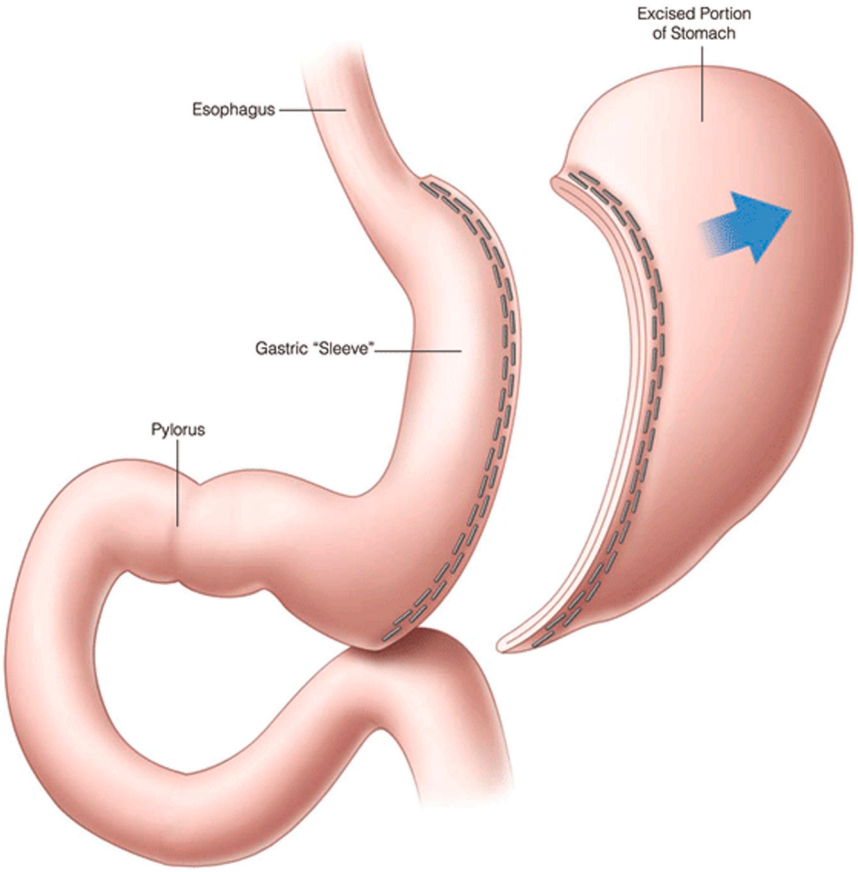

Fig. 1. Surgical diagram of sleeve gastrectomy. Schematic representation of the stomach following sleeve gastrectomy, illustrating the tubular gastric remnant ("gastric sleeve") along the lesser curvature with the staple line, and the resected greater curvature portion shown separately. Key anatomical landmarks including the esophagus and pylorus are indicated.

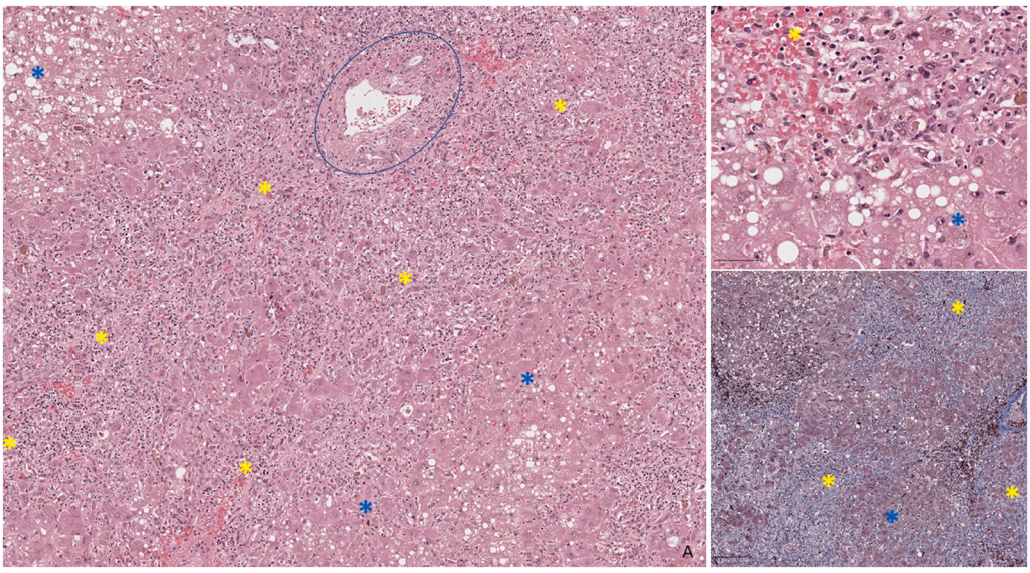

Fig. 2. Histopathology of explanted liver. (A) Section of explanted liver with acute massive hepatic necrosis; yellow asterisks show collapsed liver parenchyma with complete loss of hepatocytes, ductular reaction, cholestasis and non-specific inflammation; blue asterisks show viable areas of hepatocytes with reactive features and macrovesicular large droplet steatosis; the blue oval highlights a preserved portal tract with little inflammation and preserved portal structures. Haematoxylin and eosin; scale bar = 200 μm. (B) Higher magnification of an area with acute collapse with numerous bile-stained Kupffer cells and ductular reaction (yellow asterisks); reactive hepatocytes with steatosis (blue asterisks). Haematoxylin and eosin; scale bar = 50 μm. (C) Trichrome stain showing recent collapse (yellow asterisk) with little to no collagen deposition.

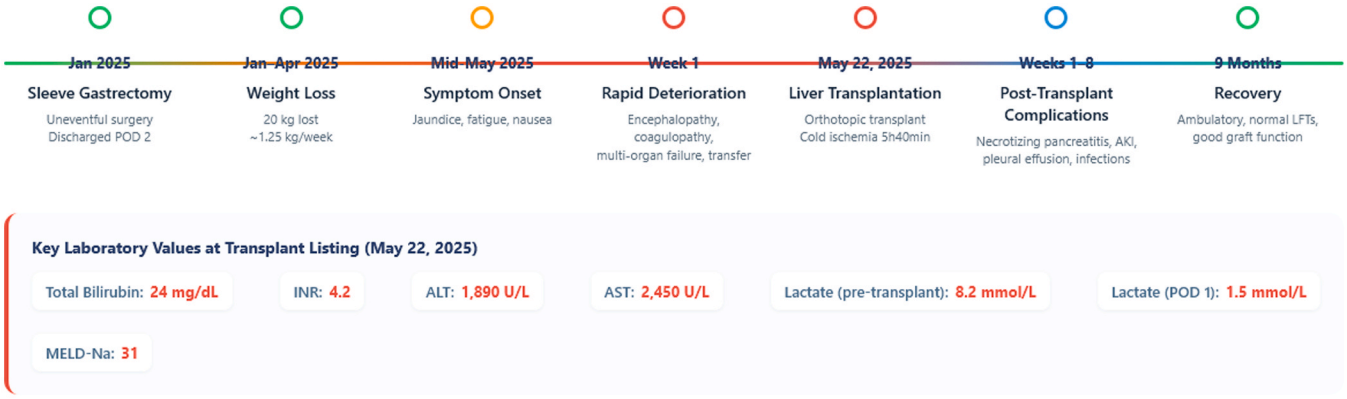

Fig. 3. Timeline of clinical course. Key events from sleeve gastrectomy through nine months post-transplant, demonstrating the rapid progression of liver failure and subsequent recovery after transplantation. POD: postoperative day; AKI: Acute kidney injury; LFTs: liver function tests; MELD: model for end-stage liver disease.

negative serologies and polymerase chain reaction. Drug-induced liver injury was considered; however, the patient was not taking any known hepatotoxic medications, including acetaminophen, and had no history of herbal supplement use. Autoimmune hepatitis was excluded by negative autoantibodies (ANA, ASMA, anti-LKM1) and the absence of characteristic histological features. Ischemic hepatitis was deemed unlikely given the absence of a hypotensive event or cardiac failure in the perioperative period. Wilson's disease and alpha-1 antitrypsin deficiency were excluded by appropriate laboratory studies. The absence of these alternative diagnoses, coupled with the temporal association with surgery and the unique histopathological findings, supports the unusual nature of the hepatic injury.

Rapid weight loss and nutritional stress

Rapid weight loss has been proposed as a potential contributing factor to hepatic injury through mobilization of free fatty acids, inducing lipotoxicity and oxidative stress [12]. Protein malnutrition may also limit hepatic synthetic capacity [13]. Kroh et al. [14] demonstrated in a murine model that the early postoperative period after sleeve gastrectomy is characterized by a massive stress response in the liver proteome, with upregulation of apoptosis-related proteins. While this typically resolves, a causal role for rapid weight loss in the present case cannot be established; our patient's weight loss of 1.25 kg/week was not extraordinary, which may instead point to individual susceptibility as a critical factor.

GLP-1: Protection Versus Susceptibility

While GLP-1 receptor activation typically exerts hepatoprotective effects [9–11], rare cases of idiosyncratic liver injury associated with GLP-1 agonists have been documented, including biopsy-proven acute hepatitis with necrosis [15,16]. Kalsi et al. [15] reported a patient with recurrent jaundice after semaglutide whose liver biopsy demonstrated acute hepatitis with necrosis that resolved upon drug discontinuation.

Mechanistically, Rahman et al. [17] showed that C/EBP homologous protein (CHOP), a key endoplasmic reticulum stress protein, is essential for GLP-1-mediated protection. In CHOP knockout mice, liraglutide fails

to ameliorate steatohepatitis, rendering hepatocytes susceptible to death [17]. This provides experimental evidence that genetic variability in stress-response pathways could determine whether GLP-1 receptor activation is protective or harmful.

Our patient's confluent necrosis resembles the biopsy-proven hepatitis in GLP-1 agonist-associated injury. It is therefore conceivable that supraphysiologic endogenous GLP-1 elevation after sleeve gastrectomy could, in genetically susceptible individuals, contribute to an abnormal cellular stress response. This hypothesis is speculative and cannot be inferred from a single observation, but it may represent one of several possible mechanisms to be explored in future studies.

Comparison with previously reported cases

Two previously reported cases differ fundamentally from ours. Abusabeib et al. [18] described acute liver failure after sleeve gastrectomy in a patient with preoperative mild liver derangement, multiple nutritional deficiencies, and concomitant acetaminophen use who recovered without transplantation. Peverelle et al. [19] reported decompensation in a patient with pre-existing non-alcoholic steatohepatitis cirrhosis treated with semaglutide who recompensated with drug cessation. Our patient had no pre-existing liver disease, no identifiable trigger, and progressed to multi-organ failure requiring transplantation in temporal association with sleeve gastrectomy.

Strengths and limitations

Strengths include comprehensive documentation and histopathology. Limitations include inability to establish causation definitively, as the temporal association with surgery may be coincidental and not causative, and lack of pre-operative liver biopsy. Despite extensive investigation, we could not identify a specific susceptibility factor.

Conclusion

We report the first case of de novo acute liver failure requiring urgent liver transplantation after sleeve gastrectomy in a patient without identifiable risk factors. This case challenges the assumption that restrictive procedures are uniformly safe and highlights a critical knowledge gap: why do the overwhelming majority of patients experience hepatic improvement while rare individuals may develop unexpected liver failure after surgery? The answer may lie in as-yet-unidentified genetic or metabolic susceptibility factors. This case underscores the importance of postoperative monitoring and the urgent need for research to predict and prevent rare but devastating outcomes.

Research registration unique identifying number (UIN)

Not applicable. This case report does not involve a clinical trial, systematic review, or other research requiring prospective registration. The case is reported in accordance with SCARE guidelines without a registration number.

CRediT authorship contribution statement

Enzo Andorno: Writing – review & editing, Visualization, Validation, Data curation. **Alessandro Gambella:** Writing – review & editing, Visualization, Validation, Investigation, Formal analysis, Data curation. **Federica Grillo:** Writing – review & editing, Visualization, Validation, Investigation, Formal analysis, Data curation. **Marco Miggino:** Writing – review & editing, Visualization, Investigation, Data curation. **Francesco Saverio Papadia:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Ethics

Ethical approval for this case report was waived by the institutional ethics committee of IRCCS Policlinico San Martino, Genoa, Italy, due to the retrospective nature of the report. Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Funding

No funding was received for this case report.

Declaration of Competing Interest

The authors declare no financial or personal relationships with other people or organizations that could inappropriately influence (bias) their work. No funding was received for this case report. The authors have no employment, consultancies, stock ownership, honoraria, paid expert testimony, patent applications/registrations, or grants to disclose.

Acknowledgements

The authors thank the patient for providing consent for publication. We also thank the nursing staff and allied health professionals involved in the patient's perioperative and post-transplant care. During the preparation of this work, the authors used ChatGPT to assist with language editing and grammar refinement. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Data availability

Data will be made available on request.

References

[1] Powell EE, Wong VW, Rinella M. Non-alcoholic fatty liver disease. Lancet 2021;397(10290):2212–24.
[2] Sjöström L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med 2004;351(26):2683–93.
[3] Schauer PR, Bhatt DL, Kirwan JP, et al. Bariatric surgery versus intensive medical therapy for diabetes – 5-year outcomes. N Engl J Med 2017;376(7):641–51.
[4] Adams TD, Davidson LE, Litwin SE, et al. Weight and metabolic outcomes 12 years after gastric bypass. N Engl J Med 2017;377(12):1143–55.
[5] Lassailly G, Caiazzo R, Buob D, et al. Bariatric surgery reduces features of nonalcoholic steatohepatitis in morbidly obese patients. Gastroenterology 2015;149(2):379–88.
[6] Verrastro O, Panunzi S, Castagneto-Gissey L, et al. Bariatric-metabolic surgery versus lifestyle intervention plus best medical care in non-alcoholic steatohepatitis (BRAVES): a multicentre, open-label, randomised trial. Lancet 2023;401(10390):1786–97.
[7] Aminian A, Al-Kurd A, Wilson R, et al. Association of bariatric surgery with major adverse liver and cardiovascular outcomes in patients with biopsy-proven nonalcoholic steatohepatitis. JAMA 2021;326(20):2031–42.
[8] Aminian A, Aljabri A, Wang S, et al. Long-term liver outcomes after metabolic surgery in compensated cirrhosis due to metabolic dysfunction-associated steatohepatitis. Nat Med 2025;31(4):988–95.
[9] Sanyal AJ, Newsome PN, Kliers I, et al. Phase 3 trial of semaglutide in metabolic dysfunction-associated steatohepatitis. N Engl J Med 2025;392(21):2089–99.
[10] Havranek B, Loh R, Torre B, Redfield R, Halegoua-DeMarzio D. Glucagon-like peptide-1 receptor agonists improve metabolic dysfunction-associated steatotic liver disease outcomes. Sci Rep 2025;15(1):4947.
[11] Peterli R, Wolnerhanssen B, Peters T, et al. Improvement in glucose metabolism after bariatric surgery: comparison of laparoscopic Roux-en-Y gastric bypass and laparoscopic sleeve gastrectomy: a prospective randomized trial. Ann Surg 2009;250(2):234–41.
[12] Verna EC, Berk PD. Role of fatty acids in the pathogenesis of obesity and fatty liver: impact of bariatric surgery. Semin Liver Dis 2008;28(4):407–26.
[13] Moxley RT, Pozefsky T, Lockwood DH. Protein nutrition and liver disease after jejunoileal bypass for morbid obesity. N Engl J Med 1974;290(17):921–6.
[14] Kroh A, Schmitz S, Kohne S, et al. Sleeve-gastrectomy results in improved metabolism and a massive stress response of the liver proteome in a mouse model of metabolic dysfunction-associated steatohepatitis. Heliyon 2024;10(21):e38678.

[15] Kalsi D, Patel V, Singhal S, et al. Semaglutide-induced acute hepatitis: a case report and review of the literature. Eur J Gastroenterol Hepatol 2024;36(2):245–8.
[16] Garg R, Williams K, Ayala E, et al. Compounded semaglutide-associated hepatotoxicity: a case series. Am J Gastroenterol 2025;120(1):89–92.
[17] Rahman K, Liu Y, Kumar P, et al. C/EBP homologous protein (CHOP) is critical for the hepatoprotective effects of glucagon-like peptide-1 receptor activation. Lab Invest 2016;96(9):942–54.
[18] Abusabeib A, El Ansari W, Alobaidan J, Elhag W. First case report of fulminant hepatitis after laparoscopic sleeve gastrectomy associated with concomitant maximal therapeutic dose of acetaminophen use, protein calorie malnutrition, and vitamins A and D, selenium, and glutathione deficiencies. Obes Surg 2020;30(12):5125–9.
[19] Peverelle M, Ng J, Peverelle J, Hirsch RD, Testro A. Liver decompensation after rapid weight loss from semaglutide in a patient with non-alcoholic steatohepatitis-associated cirrhosis. World J Gastroenterol 2023;29(44):6165–7.