Journal of Experimental and Clinical Medicine
https://dergipark.org.tr/omujecm

Case Report

J Exp Clin Med
2025; 42(1): 98-100
doi: 10.52142/omujecm.42.1.20

Postoperative hemodynamic stabilization with terlipressin: A case of liver transplantation

Neslihan ÜNAL AKDEMİR, Özkul Yılmaz ÇOLAK *, Melda İŞEVİ, Tuğçehan SEZER AKMAN, Fatma ÜLGER

Division of Intensive Care, Department of Anesthesiology and Reanimation, Faculty of Medicine, Ondokuz Mayis University, Samsun, Türkiye

Received: 13.01.2025 • **Accepted/Published Online:** 03.03.2025 • **Final Version:** 28.03.2025

Abstract

This report presents the successful management of a patient with acute liver failure on the background of chronic liver disease who underwent liver transplantation. Terlipressin, a vasoactive agent, managed hepatorenal syndrome and acute kidney injury by promoting splanchnic vasoconstriction and intrahepatic vasodilation, improving renal blood flow and hemodynamic stability. The case involves a 41-year-old woman with autoimmune hepatitis and primary biliary cirrhosis who required triple vasoactive therapy postoperatively. Bolus doses of terlipressin reduced the need for other vasopressors, stabilized renal function, and facilitated her recovery. This case underscores the potential of terlipressin as a valuable therapeutic option in the postoperative management of liver transplantation patients with refractory hemodynamic instability. Careful dosing and monitoring are essential to optimize outcomes and minimize risks.

Keywords: Hepatorenal syndrome, liver transplantation, terlipressin

1. Introduction

Acute liver injury developing on the background of chronic liver disease is defined as a severe clinical condition resulting in the sudden loss of liver functions (1). These patients may present with severe symptoms of liver failure as well as extrahepatic manifestations (2). Among extrahepatic organ failures, renal failure is the most common and is considered a significant prognostic factor independent of mortality (3). HRS typically develops in patients with decompensated liver cirrhosis. It can also emerge as a complication of fulminant liver failure and acute hepatitis. HRS is a syndrome characterized by severe acute kidney injury (AKI) resulting from renal artery vasoconstriction (4). The recommended pharmacotherapy for treating HRS-AKI includes using albumin combined with a vasoconstrictor agent (5).

Terlipressin, a vasopressin analog, exerts its effects primarily through two mechanisms. First, it binds to V1 receptors on the vascular wall, causing vasoconstriction. Second, it binds to V2 receptors, enhancing water reabsorption from the distal renal tubules and collecting ducts (6). By inducing splanchnic vasoconstriction, terlipressin reduces portal blood flow and lowers portal hypertension, playing a critical role in treating acute kidney injury observed in hepatorenal syndrome. Additionally, it decreases intrahepatic arterial resistance, leading to intrahepatic vasodilation and a subsequent reduction in hepatic venous pressure. This mechanism redistributes blood from the splanchnic region to the central vascular compartment, regulating renal blood flow and increasing mean arterial pressure (7). Compared to other vasoactive agents like norepinephrine, terlipressin has not demonstrated a significant difference in mortality rates but has been reported to have favorable effects in reversing hepatorenal syndrome (8). This article presents the successful management of a patient with acute liver failure on the background of chronic liver disease who underwent liver transplantation following treatment with terlipressin.

2. Case Report

A 41-year-old female patient with a 15-year history of autoimmune hepatitis and primary biliary cirrhosis was enrolled in a liver transplantation program. She presented to the emergency department with complaints of nausea, vomiting, poor oral intake, and fatigue. Due to rapidly rising bilirubin levels and changes in the MELD (Model for End-Stage Liver Disease) score, she was admitted to the internal medicine ward for further monitoring and management.

The patient's medical history included Hashimoto's thyroiditis, celiac disease, and a history of surgeries due to recurrent vertebral fractures. She regularly took ursodeoxycholic acid, furosemide, spironolactone, propranolol, and levothyroxine. On physical examination, her

*Correspondence: ozkulcolak@gmail.com

general condition was moderate, vital signs were stable, and she was alert, cooperative, and oriented. Systemic examination revealed widespread jaundice, tense ascites in the abdomen, and pretibial edema (+/+), with no other pathological findings noted.

Laboratory tests revealed elevated total bilirubin levels with a predominance of direct bilirubin (19.31–19.77 mg/dL), hyperammonemia (98 µmol/L), and an increased INR (2.06). Abdominal computed tomography (CT) scans showed no new pathology that could explain the patient's current complaints. The MELD (Model for End-Stage Liver Disease) score was calculated as 25, and the patient was included in the organ transplantation waiting list.

The patient underwent orthotopic liver transplantation with a cadaveric donor liver. In the postoperative period, she was admitted to the intensive care unit (ICU) on triple inotropic and vasopressor therapy (norepinephrine, dopamine, and dobutamine). The need for triple vasoactive agents persisted during postoperative hemodynamic monitoring, and reduced urine output was observed. The patient was administered a 1 mg intravenous bolus of terlipressin. Subsequently, the vasoactive agents were gradually tapered and discontinued, and the patient was extubated. Due to the positive effects of terlipressin, it was decided to administer 1 mg every 6 hours with a plan to taper the dose based on the patient's clinical condition. The patient's vasoactive drug requirements, hemodynamic parameters, and fluid management within the first 24 hours are shown in **Fig. 1**.

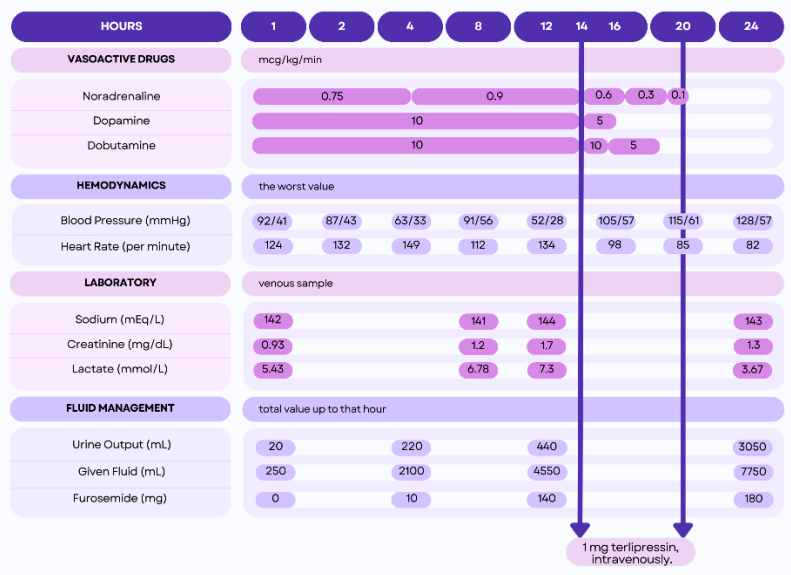

Fig. 1. First 24 hours after liver transplantation

Terlipressin was administered via slow intravenous push following a gradual dose reduction protocol. On the first day, the patient received 1 mg every 6 hours (total 4 mg/day); on the second day, 1 mg every 8 hours (total 3 mg/day); on the third day, 1 mg every 12 hours (total 2 mg/day); and on the fourth day, 1 mg once daily (total 1 mg/day). The medication was discontinued, and treatment was terminated on the fifth day.

The patient's creatinine levels, which were normal upon admission to the intensive care unit, progressively increased in the postoperative period as urine output decreased. Consequently, a furosemide infusion was initiated. The dose of furosemide was titrated to maintain a urine output rate of 0.5 mg/kg/hour. By the third day of follow-up, the patient's urine output and creatinine levels stabilized, and renal replacement therapy was not required.

The patient's concurrent prophylactic antifungal and immunosuppressive therapies were continued without interruption. The patient achieved clinical stability on the fifth intensive care unit follow-up day and was transferred to the general ward.

3. Discussion

Terlipressin has been shown to reduce the incidence of postreperfusion syndrome (PRS), particularly in cases of cadaveric donor liver transplantation, highlighting its potential to mitigate complications such as severe PRS (9). In our case, the patient required multiple vasopressor therapies in the postoperative period, and bolus terlipressin administration successfully restored hemodynamic perfusion, thereby reducing the need for other vasopressor agents.

Although some studies suggest that continuous terlipressin infusion may be more effective than bolus administration, in our case, adequate blood pressure was achieved with intermittent bolus administration of terlipressin (10). Terlipressin is considered a promising agent for managing hemodynamic instability and renal dysfunction during and after transplantation. However, despite its efficacy for the intended use, it carries significant risks of serious side effects, such as cardiac and ischemic complications and electrolyte imbalances (11).

While terlipressin reduces the incidence of PRS and improves renal function, its effects on portal pressure and potential side effects, such as increased pulmonary capillary wedge pressure, require careful evaluation. In our case, no adverse effects associated with terlipressin use were observed. To optimize liver transplantation outcomes, terlipressin administration should be closely monitored and tailored to individual patient needs, with consideration of potential side effects. In our case, a bolus dosing strategy combined with gradual tapering led to successful clinical improvement without developing adverse effects.

The severity of liver disease significantly impacts treatment response; however, it has been reported that a response to terlipressin can be achieved regardless of disease severity, leading to better outcomes in these patients (11). Additionally, terlipressin has been shown to have a more favorable effect on survival than norepinephrine (4). In our case, the preoperative MELD score was calculated as 25, consistent with severe liver disease. Nevertheless, the patient responded well to terlipressin therapy, achieving clinical improvement. Initially requiring triple vasopressor therapy, the patient experienced gradual discontinuation of other vasopressors following the initiation of terlipressin, resulting in hemodynamic stability.

This case report highlights the effectiveness of terlipressin in managing hemodynamic instability postoperatively in a patient requiring liver transplantation due to autoimmune hepatitis and primary biliary cirrhosis. Compared to other vasopressor agents, terlipressin provides a superior hemodynamic profile, particularly in patients with hepatorenal syndrome, and is associated with improved renal blood flow. However, further clinical experience and research are needed to evaluate terlipressin's benefits and potential risks comprehensively.

Informed consent

Informed consent was obtained from the patient's legal guardian, and ethical standards were followed.

Conflict of interest

The authors declare that they have no conflict of interest.

Funding

There is nothing to declare.

Acknowledgments

There is nothing to declare.

Authors' contributions

Concept: M.İ., F.Ü., Design: Ö.Y.Ç., Data Collection or Processing: M.İ., T.S.A., Analysis or Interpretation: Ö.Y.Ç., F.Ü., Literature Review: M.İ., T.S.A., Drafting: M.İ., Ö.Y.Ç.

References

1. Moreau R, Jalan R, Gines P, Pavesi M, Angeli P, Cordoba J, et al. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. Gastroenterology. 2013;144(7):1426-1437.
2. Sarin SK, Choudhury A. Acute-on-chronic liver failure: terminology, mechanisms and management. Nat Rev Gastroenterol Hepatol. 2016;13(3):131-149.
3. Maiwall R, Sarin SK, Moreau R. Acute kidney injury in acute-on-chronic liver failure. Hepatol Int. 2016;10(2):245-257.
4. Gifford FJ, Morling JR, Fallowfield JA. Systematic review with meta-analysis: vasoactive drugs for the treatment of hepatorenal syndrome type 1. Aliment Pharmacol Ther. 2017;45(5):593-603.
5. Wong F. Terlipressin for hepatorenal syndrome. Curr Opin Gastroenterol. 2024;40(2):156-163.
6. Jamil K, Pappas SC, Deverakonda KR. In vitro binding and receptor-mediated activity of terlipressin at vasopressin receptors V1 and V2. J Exp Pharmacol. 2017;10:1-7.
7. Villanueva C, Planella M, Aracil C, López-Balaguer JM, González B, Miñana J, et al. Hemodynamic effects of terlipressin and high somatostatin dose during acute variceal bleeding in nonresponders to the usual somatostatin dose. Am J Gastroenterol. 2005;100(3):624-630.
8. Israelsen M, Krag A, Allegretti AS, Jovani M, Goldin AH, Winter RW, et al. Terlipressin versus other vasoactive drugs for hepatorenal syndrome. Cochrane Database Syst Rev. 2017;9(9):CD011532.
9. Zhang L, Tian M, Sun LY, Zhu ZJ. Prophylactic terlipressin infusion for severe postreperfusion syndrome in patients undergoing deceased donor liver transplantation: the TIPS-DDLT randomized controlled trial. Int J Surg. 2023;109(7):1923-193.
10. Gifford FJ, Morling JR, Fallowfield JA. Systematic review with meta-analysis: vasoactive drugs for the treatment of hepatorenal syndrome type 1. Aliment Pharmacol Ther. 2017;45(5):593-603.
11. Weinberg EM, Wong F, Vargas HE, Curry MP, Jamil K, Pappas SC, Sharma P, Reddy KR. Decreased need for RRT in liver transplant recipients after pretransplant treatment of hepatorenal syndrome-type 1 with terlipressin. Liver Transpl. 2024;30(4):347-355.