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Case Report

Alcohol Consumption for Consecutive Twenty-Six Days in a Patient with Metabolic Dysfunction–Associated Steatohepatitis under Tirzepatide Treatment: Alanine Aminotransferase Levels Remained Normal

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Abstract

Glucagon-like peptide-1 (GLP-1) receptor agonists (RAs) and dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonists are potential therapies for metabolic dysfunction-associated steatohepatitis (MASH). In a MASH patient, who achieved biochemical normalization of ALT levels after tirzepatide therapy, simultaneous weekly injection of tirzepatide 2.5 mg and daily drinking of alcohol beverages did not incur ALT abnormality. Besides, GLP-1 RAs would induce delayed gastric emptying and were found to mitigate ethanol-induced upregulation of liver metabolizing enzymes in mouse models. However, in this MASH patient under tirzepatide 2.5 mg per week, the blood ethanol concentration 7 hours after ingestion of 5% alcohol beverage 350 mL was undetectable.

Keywords: Tirzepatide; Alcohol; Metabolic Dysfunction-Associated Steatohepatitis; Alcohol-Associated Hepatitis; Ethanol.

Introduction

Glucagon-like peptide-1 (GLP-1) receptor agonists (RAs) and dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonists, such as tirzepatide (Mounjaro), are therapies for type 2 diabetes mellitus, obesity and metabolic dysfunction-associated steatohepatitis (MASH)[1][2]. Loomba, et al. revealed that the therapy with tirzepatide for 52 weeks for patients with MASH and moderate or severe fibrosis, the tirzepatide group was more effective than the placebo group[1]. No worsening of fibrosis was observed[1]. In this article, a MASH patient received tirzepatide treatment, and the alanine aminotransferase (ALT) levels fell into normal ranges after tirzepatide treatment. Afterwards, the patient voluntarily adopted a protocol, which

included consecutive drinking of alcohol beverages once daily and continuation of weekly tirzepatide therapy.

Case Presentation

A 33-year-old male anesthesiologist without history of alcohol use disorder was diagnosed with MASH. His past medical history included gout only. From 2010 to 2025, his ALT levels fluctuated between 45 and 165 U/L. Prior to the initiation of tirzepatide therapy on September 10, 2025, laboratory exams revealed aspartate aminotransferase (AST) 47 U/L and ALT 96 U/L, with all other routine laboratory parameters within normal limits. The patient began subcutaneous tirzepatide treatment at the dose of 2.5 mg weekly since September 10, 2025. He continued the same dosage at intervals of every 7–8 days without interruption. On December 26, 2025, follow-up blood tests showed a significant reduction in ALT to 20 U/L. Since the date, the patient had voluntarily began daily alcohol consumption of 300–350 mL of alcoholic beverages containing 4%–5% alcohol by volume (manufactured by SUNTORY or KIRIN corporation in Japan). Tirzepatide therapy was continued without dose adjustment or interruption. ALT level on January 19, 2026, mildly elevated to 33 U/L, which was still within normal ranges.

Besides, at 0:00 AM on December 26, the patient voluntarily ingested a 5% alcohol beverage 350 mL (manufactured by KIRIN corporation in Japan). Later at 7:00 AM on December 26, blood was drawn to exam the ethanol concentration. The result of blood ethanol was undetectable.

Discussion

This literature illustrates a substantial improvement in hepatic ALT levels following tirzepatide therapy in a patient with long-standing MASH. Notably, ALT normalization occurred within approximately three and a half months of therapy, despite a prolonged history of elevated ALT levels. Afterwards, despite consecutive daily alcohol consumption for 26 days, ALT level was still within normal ranges.

GLP-1 RAs have been shown to reduce hepatic fat contents, improve insulin resistance and decrease hepatic inflammation [1,2]. On the opposite, alcohol consumption or alcohol use disorder could lead to alcohol-associated hepatitis [3]. The noteworthy aspect of this article is the maintenance of normal ALT levels despite the initiation of moderate daily alcohol ingestion after biochemical normalization of ALT in a MASH patient. While alcohol consumption is generally discouraged in patients with steatohepatitis, this observational study suggests that tirzepatide might provide metabolic improvements.

On the other way, no human study to date about the blood ethanol concentrations of patients under GLP-1 RAs was published. Zahrawi, et al. developed mouse models and found that GLP-1 RAs mitigate ethanol-induced upregulation of liver metabolizing enzymes, including Cyp2e1 [4]. Besides, the effect of delayed gastric emptying [5, 6] might influence the absorption of alcohol in patients treated with GLP-1 RAs. Nevertheless, in our study, the blood ethanol concentration 7 hours after alcohol consumption was undetectable. That is, perhaps mouse models by Zahrawi, et al. could not totally applied to humans. Also, delayed gastric emptying might have small influences on alcohol absorption.

However, the long-term outcomes cannot be inferred from this single-patient study. Further larger scale studies of tirzepatide on patients with alcohol use disorder are warranted.

Conclusion

First, in a MASH patient, who achieved biochemical normalization of ALT levels after tirzepatide therapy, simultaneous weekly injection of tirzepatide 2.5 mg and daily drinking of alcohol beverages did not incur ALT abnormality. Second, in the MASH patient under tirzepatide 2.5 mg per week, blood ethanol concentration 7 hours after ingestion of 5% alcohol beverage 350 mL was undetectable.

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