



**INTERNATIONAL JOURNAL OF
PHARMACEUTICAL SCIENCES**
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: <https://www.ijpsjournal.com>



Case Study

Tinospora Cordifolia Induced Liver Injury: A Case Report

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ARTICLE INFO

Published: 06 Aug 2026

Keywords:

Herb Induced Liver Injury,
Giloy, Hepatotoxicity,
Herbal medicine

DOI:

10.5281/zenodo.21820443

ABSTRACT

Herb-induced liver injury (HILI) is a critical cause of acute liver dysfunction, which is defined as liver injury caused by the use of drugs, herbal products, or supplements. HILI has a broad range of clinical manifestations, ranging from asymptomatic increases in liver enzymes to acute liver failure. Herbal and traditional remedies have been increasingly recognized as a critical cause of drug-induced liver injury, especially in areas where their use is high. *Tinospora cordifolia*, also called Giloy, is a widely used Ayurvedic herb, which has been traditionally regarded as immunomodulatory as well as hepatoprotective. However, recent reports have emphasized its potential in causing hepatotoxicity, especially when it is used indiscriminately or in predisposed individuals. Giloy-induced liver injury has been recognized as immune-mediated, which can be comparable to autoimmune hepatitis in its clinical and biochemical presentation. A 52 Year old male patient presented to the outpatient department with history of high colored urine and yellowish discoloration of eyes of 1 week duration. History of alcoholism (+). No previous history of chronic liver disease. Had a past history of jaundice around 25 yrs back. He is a known case of Type II DM on herbal medication- Chittamrithu (giloy) for more than 3 months. No h/o any other comorbidities. On examination icterus (+). According to physical examination symptoms, laboratory investigation the patient was diagnosed with Herb induced liver injury and managed with IV antibiotics, Oral Steroids, and liver supportive medications.

INTRODUCTION

Drug-induced liver injury (DILI) is a major cause of acute liver dysfunction worldwide and remains

one of the most challenging diagnoses in hepatology because of its variable clinical presentation and the absence of specific diagnostic biomarkers.¹² The growing popularity of

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



complementary and alternative medicine (CAM) has led to an increasing number of reports of herb-induced liver injury (HILI), which is now recognized as an important subset of DILI.¹⁷

Tinospora cordifolia, commonly known as Giloy or Guduchi, is a climbing shrub belonging to the Menispermaceae family and has been extensively used in Ayurvedic medicine for centuries.³ It is traditionally prescribed for diabetes mellitus, fever, chronic inflammatory disorders, immune enhancement, and general well-being.³ The use of Giloy increased substantially during and after the COVID-19 pandemic because of its widespread promotion as a natural immune booster.¹

Although *Tinospora cordifolia* has been traditionally regarded as a safe herbal remedy, accumulating evidence suggests that prolonged or unsupervised consumption may result in clinically significant liver injury.⁵ Recent case series and observational studies have reported acute hepatitis, cholestatic liver injury, autoimmune-like hepatitis, and, in severe cases, acute liver failure associated with Giloy consumption.⁴

The pathophysiology of Giloy-induced liver injury is not completely understood and is likely multifactorial.⁸ Current evidence suggests that an immune-mediated mechanism may play a central role, wherein herbal constituents trigger an exaggerated immune response directed against hepatocytes.⁸ Experimental studies have demonstrated that *Tinospora cordifolia* possesses potent immunostimulatory activity, which may contribute to immune-mediated hepatic injury in genetically susceptible individuals.³ Some investigators have also proposed that contamination, adulteration, variable phytochemical composition, and herb-drug interactions may contribute to hepatotoxicity associated with herbal preparations.¹⁰

Clinical manifestations of herb-induced liver injury can range from asymptomatic elevations of liver enzymes to severe acute hepatitis and liver

failure.¹⁴ Patients commonly present with fatigue, malaise, anorexia, nausea, dark-colored urine, jaundice, scleral icterus, abdominal discomfort, and pruritus.¹³ Laboratory findings typically demonstrate elevated serum aminotransferases, hyperbilirubinemia, and varying degrees of coagulation abnormalities depending on the severity of hepatic injury.¹⁸

Several factors may increase the risk of developing herb-induced liver injury, including prolonged duration of herbal consumption, high cumulative exposure, underlying metabolic disorders, alcohol use, genetic susceptibility, and concomitant use of other medications.¹⁹ Establishing a causal relationship between a herbal product and liver injury remains challenging because herbal formulations often contain multiple bioactive constituents and may lack standardized manufacturing processes.⁹ The Roussel Uclaf Causality Assessment Method (RUCAM) is one of the most widely accepted tools for assessing causality in suspected cases of herb-induced liver injury.¹⁶

Recent reports from India and other countries have increasingly linked *Tinospora cordifolia* with hepatocellular liver injury, particularly among individuals consuming the herb for diabetes management and immune enhancement.⁷ These reports have strengthened concerns regarding the potential hepatotoxic effects of Giloy despite its traditional reputation as a hepatoprotective agent.⁶ We report a case of acute hepatocellular liver injury in a 50-year-old male with type 2 diabetes mellitus who presented with jaundice, scleral icterus, dark-colored urine, and marked elevation of liver enzymes following prolonged consumption of *Tinospora cordifolia* for glycemic control. Viral hepatitis and other common causes of acute liver injury were excluded through appropriate clinical and laboratory evaluation. The temporal relationship between prolonged Giloy



consumption and the onset of liver dysfunction strongly suggests *Tinospora cordifolia*-induced liver injury.

CASEPRESENTATION

A 50-year-old male presented to the hospital with complaints of yellowish discoloration of urine, yellowish discoloration of the sclera, and excessive tiredness for one week. There was no history of pruritus, abdominal pain, fever, nausea, vomiting, altered sensorium, gastrointestinal bleeding, or significant weight loss. The patient reported recent travel to Tamil Nadu before the onset of symptoms.

The patient was a known case of type 2 diabetes mellitus diagnosed one year earlier but was not receiving regular anti diabetic medication. He had a history of chronic alcohol consumption, averaging approximately two pegs daily, with the last alcohol intake reported one week prior to presentation. Further history revealed the consumption of a complementary and alternative medicine (CAM) preparation consisting of the root of *Tinospora cordifolia* (Giloy/Chittamruthu) for glycemic control. The herbal preparation had been consumed regularly for more than three months before the onset of symptoms.

On physical examination, the patient was conscious, oriented, and hemodynamically stable. Scleral icterus was present, and clinical examination revealed icterus. No stigmata of chronic liver disease, hepatosplenomegaly, ascites, peripheral edema, or hepatic encephalopathy were noted.

Laboratory investigations demonstrated significant hepatic dysfunction with a total bilirubin level of 7.60 mg/dL, direct bilirubin of 5.33 mg/dL, and indirect bilirubin of 2.27 mg/dL. Liver enzyme analysis revealed markedly elevated aminotransferase levels with aspartate aminotransferase (AST) of 837 U/L and alanine

aminotransferase (ALT) of 1495 U/L. Alkaline phosphatase (ALP) was 156 U/L.

Coagulation studies showed an international normalized ratio (INR) of 1.760.

Glycemic assessment revealed an HbA1c of 7.3%.

Serological evaluation for common viral causes of acute hepatitis was negative.

Hepatitis A virus IgM, hepatitis B surface antigen (HBsAg), and hepatitis C virus (HCV) serology were non-reactive. Abdominal ultrasonography revealed a right-sided non-obstructive renal calculus, features suggestive of cystitis requiring clinical correlation, and grade I prostatomegaly. No significant hepatobiliary structural abnormalities were identified.

Based on the history of prolonged *Tinospora cordifolia* consumption, marked hepatocellular enzyme elevation, hyperbilirubinemia, and exclusion of common viral etiologies, a diagnosis of suspected herb-induced liver injury secondary to *Tinospora cordifolia* was considered. The herbal preparation was discontinued immediately upon admission.

The patient was managed conservatively with supportive therapy, including intravenous cefoperazone-sulbactam 1.5 g, intravenous esomeprazole 40 mg, vitamin K injection 10 mg, thiamine injection 100 mg, intravenous N-acetylcysteine 1.2 g, prednisolone 40 mg, ursodeoxycholic acid 300 mg, rifaximin 400 mg, hepatoprotective therapy (Heptagon), and probiotic supplementation with VSL#3.

Following clinical stabilization, the patient was discharged on 13 March 2026 with oral medications including esomeprazole 40 mg once daily before food, prednisolone 40 mg once daily, ursodeoxycholic acid 300 mg twice daily, Heptagon twice daily, Nacetylcysteine (Mucinac) 600 mg two tablets twice daily, and metformin sustainedrelease 500 mg twice daily. In addition, NPH insulin 15 units subcutaneously once daily was prescribed for five days, to be administered



along with the morning dose of prednisolone. The patient was advised strict avoidance of alcohol and herbal medications and was scheduled for outpatient follow-up with repeat liver function monitoring.

CONCLUSION

This case highlights the potential risk of herb-induced liver injury associated with prolonged use of *Tinospora cordifolia* (Giloy) for diabetes management. The temporal relationship between Giloy consumption and the development of jaundice, elevated liver enzymes, and hyperbilirubinemia, along with the exclusion of common viral causes, supports the diagnosis of Giloy-induced liver injury. Clinicians should consider herbal medicines as a possible cause of unexplained liver dysfunction and obtain a detailed history of complementary and alternative medicine use. Early recognition and prompt discontinuation of the suspected herbal product are essential for favorable clinical outcomes.

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HOW TO CITE: Krishna B, Aswathy J K, Drishya L, Neehar Shanavas, Shaiju Dharan, Tinospora Cordifolia Induced Liver Injury: A Case Report, *Int. J. of Pharm. Sci.*, 2026, Vol 4, Issue 8, 927-931, <https://doi.org/10.5281/zenodo.21820443>

