Probable Drug-Induced Liver Injury Caused by Tinospora species: A Case Report

We read with interest the article by Nagral A et al about the hepatotoxicity of Tinospora and its presentation as autoimmune hepatitis. It is in keeping with our own experience of multiple cases of deterioration in liver function with the use of Tinospora and subsequent recovery, once it is stopped.

We would like to reinforce Dr. Nagral’s experience by briefly describing two of our cases. The first case we saw was a 66-year-old diabetic, nonobese, nonalcoholic male with a history of daily consumption of homemade extract of Tinospora plant stem (grown in near vicinity) for 4 weeks prior to the development of jaundice and ascites. The viral markers, Hepatitis A virus serology, Hepatitis E virus serology, HbsAg, and HCV serology were nonreactive. Autoimmune markers, including ANA, ASMA, AntiLKM1, were negative. RUCAM scale score was 9 (highly probable). Transjugular liver biopsy (Figure 1) revealed parenchyma hepatocytes showing ballooning, giant cell formation,

![Figure 1](https://doi.org/10.1016/j.jceh.2021.10.002)

**Figure 1** Liver biopsy of the first patient - Core showing (200 x) — (A) Bridging necrosis B) Cholestatic plugs, Ballooned hepatocytes (C) Giant Cells (D) CK 7 immunostain highlighting bile duct proliferation.

**Abbreviations:** HbsAg: Hepatitis B surface antigen; HCV: Hepatitis C virus; ANA: Antinuclear antibody; ASMA: Anti smooth muscle antibody; AntiLKM1: liver kidney microsome type 1 antibody; RUCAM: Roussel Uclaf Causality Assessment Method; HEV: Hepatitis E virus; HAV: Hepatitis A virus; HSV: Herpes Simplex Virus; TCP: Tinospora crispa; TCF: Tinospora cordifolia

feathery degeneration, cholestatic plug, intracellular bile, spotty to confluent necrosis, and two foci of portal-to-portal bridging necrosis. These histopathologic features are those of acute hepatitis with bridging necrosis. In view of the above and the laboratory findings of the tests conducted, a probable diagnosis of Drug (Tinospora) induced acute liver injury was made. He then made a gradual recovery and currently doing well.

The second patient was a 33-year-old female who was referred to our hospital as a case of acute liver failure with complaints of progressive jaundice and encephalopathy with a significant medical history of consumption of Tinospora pellets for a month prior to her illness. She purchased these pellets from a local chemist shop. Serology for common hepatotropic viruses HBV, HCV, HEV, HAV, HSV 1 and 2 were negative. Markers of autoimmune hepatitis ANA, ASMA, Anti LKM1 were nonreactive. Serum ceruloplasmin and 24 hour urine copper were within the normal range. Toxicology screen of blood and urine also did not reveal anything significant. RUCAM scale score was 6 (probable). An urgent liver transplant was carried out, and she recovered uneventfully. The liver biopsy from the explanted liver (Figure 2) showed distorted liver architecture, submassive necrosis, regenerative tubules (stem cell reaction), and moderate lymphocytic infiltrate along with neutrophils.

Liver toxicity of Tinospora crispa (TCP) has been identified in a study done by Langrand et al. The protective effect of Tinospora cordifolia (TCF) in gastrointestinal and hepatotoxicity has also been described in moderate and chronic alcoholism. However, a web-based image search of Guduchi, and T. cordifolia showed mostly the images of blunt stems of the plant. Since the stems of both species have a similar appearance, it may misguide people to misidentify TCP as TCF. Our experience suggests hepatotoxicity from the use of Tinospora may go on to acute liver failure requiring liver transplantation. We suggest that Tinospora products should definitely not contain T. crispa and till it is firmly established that T. cordifolia consumption is safe, they should not be used.

**REFERENCES**

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