ISSN: 2689-8616

Case report

Advancements in Journal of Urology and Nephrology

The Silent Risk in 'Natural' Health Products

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Submitted: 2025, Dec 29; **Accepted:** 2026, Jan 22; **Published:** 2026, Feb 02

Citation: Siripurapu, A., Kankanala, S. (2026). The Silent Risk in 'Natural' Health Products. *Adv J Uro Nephro, 8*(1), 01-03.

Abstract

Drug induced liver injury (DILI) is a rapid loss of hepatic function in a person and is clinically diagnosed by jaundice and elevated liver enzymes; It is caused by hepatotoxic medications such as acetaminophen. This case report is of a patient who is a 60 year female with a history of positive ANA who presents the clinic with jaundice and full body pruritic rashes. She has been to urgent care twice in the last couple of months where medrol dose packs and injections were given. She admits this helps, but symptoms resume once the steroid course is completed. Laboratory evaluation showed cholestasis with elevations in alkaline phosphatase (911 U/L), total bilirubin (6.2 mg/dL), and alongside macrocytosis (MCV 106.8 fL). Despite positive autoimmune markers (ANA/AMA 1:160) which initially raised concern for autoimmune etiology, a liver biopsy confirmed DILI. Further interrogation revealed that she is taking several herbal supplements and protein powder, which were discontinued. Most recent visit to the office her symptoms improved but liver function tests were still abnormal and currently being monitored in consultation with Hepatology. This case highlights the critical importance of obtaining a comprehensive medication history that includes unregulated herbal supplements which can contribute to potential health hazards.

Keywords: Drug-Induced Liver Injury, Herbal Supplements, Cholestasis

1. Introduction

Drug-induced liver injury (DILI) is a spectrum of hepatic injury resulting from the ingestion of pharmaceutical drugs, herbal medications, or dietary supplements. While often associated with prescription medications, the incidence of liver injury secondary to herbal and dietary supplements is a concern as the consumption of unregulated products increases globally [1].

DILI is categorized into intrinsic (dose-dependent) or idiosyncratic (unpredictable) mechanisms. In cases involving herbal supplements, the injury often manifests as hepatocellular damage, cholestasis, or a mixed pattern. Clinically, patients may present with a wide range of symptoms, from asymptomatic elevations in liver enzymes to fatigue, nausea, and jaundice. Severe cases can progress to acute liver failure [2].

Diagnosing DILI is predominantly a diagnosis of exclusion that establishes a clear temporal relationship between agent ingestion and the onset of injury. This clinical evaluation rules out alternative causes of liver dysfunction, including viral hepatitis, autoimmune hepatitis, and biliary obstruction by eliminating suspected agents and observing symptom resolution to confirm the diagnosis [3]. However, this process is frequently obscured by comorbidities, such as obesity and the presence of non-specific autoantibodies. Differentiating DILI from autoimmune hepatitis (AIH) is particularly challenging, as certain drugs can trigger immune-mediated responses characterized by positive antinuclear antibodies (ANA) or antimitochondrial antibodies (AMA), mimicking the serological profile of AIH [4]. Consequently, a liver biopsy is often required to differentiate between these etiologies based on histological features, such as prominent cholestasis or eosinophilic infiltration [5].

As seen in our patient, a 62-year-old female, presented with jaundice & symptoms of cholestasis, physical examination revealed generalized jaundice and scleral icterus. The diagnosis was further obscured by a history of positive ANA and current lab results showing high titer ANA/AMA, suggesting an autoimmune etiology. However, a liver biopsy confirmed drug-induced liver injury. This case underscores the necessity for clinicians to

maintain a suspicion for supplement toxicity.

2. Case Presentation

A 62-year-old female presented to the outpatient clinic on August 25, 2025, with a three-week history of jaundice and scleral icterus. Associated symptoms included generalized pruritus (worsening at night), abdominal pain, dark urine, and changes in stool color and caliber. She reported no recent changes in lifestyle or environment prior to symptom onset.

Review of systems was negative for fever, night sweats, unintentional weight loss, acute vision changes, or headaches. The patient denied recent travel or the use of new medications, vitamins, alcohol, tobacco, or illicit drugs. Her past medical history was notable for abnormal liver enzymes (elevated GGT and Alk Phos) in 2023, suggestive of fatty liver disease; however, no follow-up testing was performed. Additionally, she reported a history of severe heartburn, which resolved following dietary modifications (avoidance of spicy foods and late-night eating), increased exercise, and subsequent weight loss.

On physical examination, the patient appeared in no acute distress. Significant findings included frank jaundice, scleral icterus, and macular spots on the upper extremities and chest.

Her past medical history was significant for a known positive antinuclear antibody (ANA) status and a recent *Helicobacter pylori* infection, which had been treated with triple therapy and was currently maintained on a proton pump inhibitor (PPI). The patient denied alcohol consumption and illicit drug use. A review of her medication history revealed the use of occasional cetirizine and the regular consumption of unspecified herbal supplements.

Initial laboratory evaluation (August 25, 2025) revealed a cholestatic pattern of liver injury. Significant findings included an elevated alkaline phosphatase (ALP) of 613 U/L (Reference: 40–140 U/L) and total bilirubin of 7.28 mg/dL (Reference: 1.2 mg/dL). Transaminases were also elevated, with an aspartate aminotransferase (AST) of 221 U/L and alanine aminotransferase (ALT) of 150 U/L.

	Value	Reference Range
HEP B CORE TOTAL AB	NON-REACTIVE	NON-REACTIVE -
HEPATITIS B SURFACE AB	NON- REACTIVE	NON-REACTIVE -
HEPATITIS C ANTIBODY	NON-REACTIVE	NON-REACTIVE -

Table 1: An acute viral hepatitis panel on August 25, 2025 was non-reactive for Hepatitis A IgM, Hepatitis B Surface Antigen/ Core Antibody, and Hepatitis C Antibody.

Abdominal ultrasound on August 27, 2025 demonstrated a coarse liver texture without focal lesions and an incidental finding of an ectopic right kidney; no biliary obstruction was identified. The diagnostic picture was obscured by the patient's serological profile. Previous autoimmune workup revealed positive ANA and antimitochondrial antibodies (AMA) at titers of 1:160. Given the overlap between DILI and autoimmune hepatitis (AIH) in serological presentation, a liver biopsy was performed on September 9, 2025, to differentiate the etiology [4].

Examination of the liver biopsy specimen showed cholestatic hepatitis of moderate activity with parenchymal collapse but no definitive fibrosis. The portal tracts contained mild-to-moderate mixed inflammatory infiltrates, including lymphocytes, plasma cells, neutrophils, and focally abundant eosinophils. The background lobules displayed prominent canalicular and hepatocellular cholestasis with associated feathery degeneration. Although the findings were non-specific, the presence of prominent cholestasis combined with eosinophilic infiltration strongly favored a diagnosis of Drug-Induced Liver Injury (DILI) rather than AIH [6].

Measured	Reference Range & Units	10/3/2025	11/5/2025	11/26/2025
Protein, Total	6.0 - 8.5 g/dL	6.8	6.6	6.3
Albumin	3.9 - 4.9 g/dL	3.5	3.4	3.3
Bilirubin, Total	0.0 - 1.2 mg/dL	6.5	9.3	9.1
Alkaline Phosphatase (ALP)	49 - 135 IU/L	613	568	501
Aspartate Aminotransferase (AST)	0 - 40 IU/L	221	211	211
Alanine Aminotransferase (ALT)	0 - 32 IU/L	150	106	93
Bilirubin, Direct	0.00 - 0.40 mg/dL	(Not Reported)	7.28	7

Table 2: Time-Course of Liver enzymes and bilirubin levels following the discontinuation of the all supplements (post-August 25, 2025). Note the consistent downward trend in Alanine Aminotransferase (ALT) and Alkaline Phosphatase (ALP) from October 3rd to November 26th, which strongly supports the diagnosis of Drug-Induced Liver Injury (DILI) and indicates a positive clinical response to the withdrawal of the supplement. Total Bilirubin peaked on 11/5/2025 before starting to decline/plateau.

Based on the temporal relationship with supplement use and the histological findings, a diagnosis of DILI secondary to herbal supplements was established. The patient was instructed on August 25, 2025, to immediately discontinue all herbal supplements and cetirizine. Management for pruritus was initiated with topical lotions. The clinical plan anticipated a gradual normalization of liver enzymes and resolution of jaundice following the withdrawal of the supplements [3].

3. Discussion

Consumption of herbal and dietary supplements (HDS) can lead to hepatotoxicity . Unlike prescription medications, herbal products are often unregulated, containing variable concentrations of active ingredients or undisclosed contaminants. In this case, the patient's exposure to hepatotoxic substances via herbal supplements resulted in cholestatic injury, mimicking the clinical presentation of chronic autoimmune liver disease.

The differentiation of DILI from autoimmune hepatitis (AIH) is difficult, particularly when serological markers are confounding. Our patient presented with high ANA and AMA (1:160), markers associated with AIH and Primary Biliary Cholangitis respectively [7]. Literature suggests that up to 23% of DILI cases may present with autoantibodies, a condition often termed "drug-induced autoimmune-like hepatitis" [4,8]. Misdiagnosis in this context is a significant risk; treating DILI with the immunosuppression required for AIH exposes the patient to unnecessary side effects.

The patient's histology revealed prominent cholestasis and eosinophilic infiltration. Eosinophils are a footprint of hypersensitivity reactions associated with DILI and are less common in AIH [8]. The absence of significant fibrosis further supported an acute condition rather than the chronic progressive course of untreated AIH.

Management relied on the withdrawal of the supplements. The laboratory trends in this case (Figure 1) illustrate the gradual resolution of DILI. While ALT normalized rapidly, cholestatic markers (Bilirubin/ALP) can exhibit a "lag," as seen in our patient's bilirubin peaking in November despite the cessation of supplements in September. This pattern confirms the diagnosis and highlights the need for prolonged monitoring.

Finally, this case emphasizes the silent nature of herbal supplement use in medical histories. Patients often do not categorize supplements as medications and may overlook them during routine intake. The incidental discovery of jaundice in this patient, who presented for a foot callus, underscores that liver pathology can remain unreported until advanced stages.

4. Conclusions

This case highlights the necessity of maintaining a broad differential diagnosis in patients presenting with unexplained liver injury. While positive ANA and AMA titers suggest autoimmune etiology, they do not preclude a diagnosis of Drug-Induced Liver Injury (DILI). A comprehensive history specifically targeting herbal and dietary supplement use is essential, as these products are under-reported causes of hepatotoxicity. In ambiguous cases, liver biopsy remains helpful, with features such as eosinophilic infiltration and prominent cholestasis favoring DILI. Ultimately, the resolution of liver injury following the withdrawal of the suspected supplement provides the definitive confirmation of the diagnosis.

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