

# Tea not Tincture: Hepatotoxicity Associated with Rooibos Herbal Tea

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## Abstract

A 52-year-old male presented with signs of acute hepatitis and liver failure. Laboratory investigations for common etiologies were unrevealing, but history suggested liver injury secondary to ingestion of a traditional South African herbal tea made with rooibos and buchu. Livery biopsy confirmed a toxin-mediated liver injury. The patient recovered liver function after stopping the herbal tea. Although hepatotoxicity associated with rooibos and buchu has rarely been reported, anecdotal correspondence with South African physicians confirmed suspected cases. Hepatotoxicity may be due to the heterogeneous composition of herbal teas due to small-batch manufacturing. Our case clearly outlines the need to suspect herbal causes of idiopathic liver injury.

## Introduction

Drug-induced liver injury is a common etiology of hepatotoxicity, but the diagnosis is often delayed or missed when patients and clinicians do not consider herbal supplements as drugs. The following case of acute hepatitis and liver failure in a 52-year-old male with recent daily ingestion of rooibos and buchu herbal tea illustrates the importance of a careful herbal drug history when presented with a case of hepatotoxicity.

## Case Report

We describe a patient with hepatotoxicity related to herbal tea and highlight the importance of reviewing herbal consumption in such cases. A 52-year-old man presented to the emergency room with new-onset jaundice and malaise. He had scleral icterus, diffuse pruritus, dark urine, and had sought consultation with his physician, who drew labs showing acute hepatitis prior to referral. He had a history of hyperlipidemia and stage III chronic kidney disease secondary to IgA nephropathy. Medications included oral steroids and long-term statin use, but no other hepatotoxic drugs. He noted rare alcohol consumption, but he had daily ingestion of buchu and rooibos tea from South Africa in the last month.

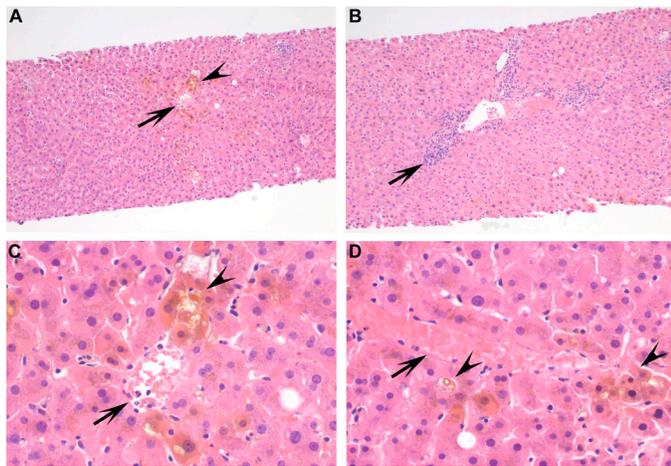
On examination he was afebrile and jaundiced, with icteric sclera, dermal excoriations, and a non-tender abdomen. He had no asterixis, spider angiomas, hepatosplenomegaly, or ascites. Laboratory tests included alanine amino transferase (ALT) of 2,589 IU/L, aspartate amino transferase (AST) of 1,438 IU/L, total bilirubin of 12.1 mg/dL, direct bilirubin of 8.3 mg/dL, and alkaline phosphatase of 359 IU/L. His albumin was 3.0 g/dL with normal platelet count and INR. An abdominal ultrasound showed a normal liver without ascites. Tests for hepatitis A, B, C, E, as well as HSV, CMV, EBV, HIV, VZV, anti-nuclear antibody, anti-mitochondrial antibody,

*ACG Case Rep J* 2013;1(1):58–60 doi:10.14309/crj.2013.20. Published online: October 8, 2013.

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and anti-smooth muscle antibody were negative. On hospital day 6, a liver biopsy showed predominantly centrilobular cholestasis without evidence of large duct obstruction, bile ductular proliferation, or portal edema. There was focal mild portal chronic inflammation and rare apoptotic cells (Figure 1). Peri-portal plasma cells were absent and there was no significant steatosis. Immunohistochemical staining for HSV I and II was negative. The results were consistent with drug-induced liver injury (DILI) secondary to herbal tea ingestion. Although statin-related hepatotoxicity was considered, a review of the literature showed long-term statin use to be a rare cause of liver failure.<sup>1</sup> Our case met all the diagnostic criteria



**Figure 1.** Histopathologic findings. (A) Low-power view of central vein (arrow) with pericentral cholestasis (arrowhead). (B) Low-power view of a portal area with mild chronic inflammation. Note absence of bile pigment in periportal area. (C and D) High-power view of two central veins (arrows) surrounded by pericentral cholestasis (arrowheads, yellow pigment).

for liver injury secondary to herbal supplements as outlined by Navarro VJ et al, except for hepatotoxicity after toxin re-exposure.<sup>2</sup> The patient's liver function tests improved and he was discharged home. He showed continued improvement in liver function tests 2 weeks later.

## Discussion

Rooibos and buchu herbal tea is a common South African beverage. Rooibos is derived from the dried needles of *Aspalathus linearis*, which is native to South Africa. Commonly referred to as red tea, it is used as a traditional remedy for infantile colic and dermatologic conditions.<sup>3</sup> Rooibos tea consumption has reported antioxidant activity, with one study showing hepatoprotective effects in rats exposed to CCl<sub>4</sub>; however, hepatotoxicity has also been reported.<sup>4,5</sup> Buchu tea is derived from *Agathosma betulina* or *Agathosma crenulata* and has diuretic and antimicrobial effects.<sup>6</sup> Buchu tea hepatotoxicity has never been reported, but one of its traditional components, pennyroyal oil, is hepatotoxic.<sup>7,8</sup> Consultation with a South African hepatologist revealed anecdotal cases

of DILI attributed to rooibos and buchu herbal tea, but the exact mechanism has not been elucidated. The rarity of rooibos and buchu tea DILI may be due to small-batch production, which allows for variability in the exact components of each tea after processing.<sup>9</sup> Other teas with reported hepatotoxicity include chaparral, kava, germander, and *Camellia sinensis* (green tea).<sup>10</sup>

Published reviews showed herbs to be a causative or contributing agent in 9–11% of DILI cases, but diagnosis is often difficult when patients do not report herbal consumption.<sup>11–13</sup> This may be due to lack of clinician understanding of the adverse effects or medical interactions of herbs. In our case, tea consumption was elicited only after admission to the hospital, which delayed diagnosis. Our report highlights the importance of education and review of herbal drugs and supplements in cases of hepatotoxicity.

## Disclosures

Author contributions: M. Engels, C. Wang, and E. Maidan wrote the manuscript; A. Matoso created the slides. C. Wang is the author guarantor. C. Wang and M. Engels share first authorship of this article.

Financial disclosure: There is no financial support, financial conflicts, or conflicts of interest to disclose.

Received: July 14, 2013; Accepted: September 25, 2013

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