Title

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CLINICAL VIGNETTE

Acute Hepatotoxicity: A Complication of Chaparral Ingestion

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Case Presentation

A 65-year-old Caucasian man with no significant past medical history presented to the Emergency Department with a one week history of chills and fevers up to 39°C. He reported myalgias, nausea, anorexia, night sweats, and fatigue. He denied abdominal pain, diarrhea, melena, weight loss, or recent travel. The patient was not taking any medications, supplements, or herbals. He was seeing an acupuncturist regularly. Physical exam was notable for a temperature of 38.1°C, blood pressure of 78/58, heart rate of 130, a jaundiced general appearance, scleral icterus, dry mucous membranes, and hepatomegaly. The patient was given IV fluids with improvement in blood pressure to 116/73 and heart rate to 113.

Laboratory work-up revealed elevated liver tests with an AST of 178 U/L, ALT of 342 U/L, Total Bilirubin of 4.3 mg/dL, and Alk Phos of 257 U/L. CBC, basic metabolic panel, and INR were within the normal range. An abdominal ultrasound revealed an enlarged liver with mild perihepatic ascites and marked gallbladder wall thickening with no evidence of cholecystitis. A CT abdomen and pelvis with contrast confirmed these findings. There was no evidence of drainable fluid collections.

IV fluid resuscitation was continued. Empiric antibiotics were not given due to a lack of an infectious source, normal WBC, and hemodynamic stability following fluid resuscitation. Hepatitis A, Hepatitis B, and Hepatitis C serologies were negative. Serum IgG 4 and smooth muscle antibody were both negative, effectively ruling out autoimmune hepatitis as an etiology. Acetaminophen level was normal ruling out acetaminophen toxicity. An abdominal ultrasound with doppler was negative for vascular occlusion.

Over the next 48 hours, the patient had complete resolution of his symptoms without additional intervention. Bacterial cultures returned negative, and his liver tests were downtrending. A call to the patient's acupuncturist revealed that the patient had been taking Chaparral to promote liver health.

Discussion

The prevalence of herbal product use and incidence of acute herbal hepatotoxicity are unknown. Herbal products are implicated as a cause of hepatotoxicity in up to 10% of drug-induced liver injury and acute liver failure cases.[1] Chaparral-induced hepatotoxicity typically manifests with symptoms that include fatigue, abdominal pain, dark urine, light stools, nausea, and diarrhea. Most patients have marked jaundice and elevated liver tests.[2] Although most cases of hepatotoxicity resolve following cessation of Chaparral ingestion, some evolve into cirrhosis or acute liver failure.

Chaparral is made from the leaves of the Creosote bush or Greasewood bush endemic to California and the Northern Baja Peninsula of Mexico.[3] It has been used for its claimed antioxidant and anti-aging properties as well as for treatment of various conditions such as cancer and AIDS. The mechanism of Chaparral-induced hepatotoxicity is not known but may be related to inhibition of lipoxygenase and cyclooxygenase pathways, cytochrome P450 inhibition, or the estrogen activity of chaparral metabolites.[4]

In this case, the patient presented with acute hepatotoxicity with marked hypotension. This may have led to acute liver failure had prompt IV fluid resuscitation not been employed. Awareness that the ingestion of alternative medicines can lead to severe hepatotoxicity is essential in cases of unclear liver disease. This case also reminds us that inquiring about the use of alternative medicines is an important aspect of taking a thorough medication history.

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