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A case study of ayurvedic drug-induced liver injury in middle - aged women

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Abstract

In wealthy nations, 50% of acute liver failure is brought on by drug-induced liver damage. The use of homeo pathic and ayurvedic medications has been associated to liver damage. In this instance, ayurvedic medication was used for a month, resulting in drug-induced liver damage. It may be challenging to diagnose drug-induced liver damage, but using many techniques, such as the ACG algorithms, casual assessment scales, histological results, and imaging, is advised. It's possible that modern imaging techniques like CECT will play a bigger part than has previously been suggested in the literature.

Keywords: Ayurvedic, Drug induced liver injury, Autoimmune hepatitis, Elevated bilirubin

Core tip

Drug-induced liver damage is challenging to identify. Many people take certain ayurvedic drugs without fully understanding all of their side effects. This article illustrates the potential use of cutting-edge imaging techniques in addition to providing a case study of drug-induced liver damage.

Introduction

As early as 2100 BC, ancient China and India embraced complementary and alternative medicine in the form of herbal and homeopathic remedies. Though some herbal medicines have been proven to result in drug-induced liver injury (DILI), there are numerous additional drugs that could also be at fault. In ayurvedic medicine, punarnaya mandur, an extract from the Boerhavia diffusa plant, is frequently used to treat iron deficiency, kidney, and liver disorders. Uterine fibroids and BPH are treated with kanchnar guggulu, a plant extract from the Bauhinia variegate species. Both of these drugs have numerous unlisted uses and characteristics, but more specifically, they have the potential to be anti-inflammatory and hepatoprotective. These two frequently used drugs have never before been linked to DILI. Frequently, DILI is misdiagnosed or not diagnosed.

Case report

A 42-year-old woman with bilateral swelling of the upper and lower limbs, puffiness of the face, decreased urine production, coupled with poor appetite, pale stools, and black urine, presented to the emergency department for treatment. Before presenting to the ED, she had a history of dermatological problems six months earlier. When she started experiencing the aforementioned symptoms, she apparently stopped using the various herbal and homeo pathic remedies she had been using. She denied using

any further medications. She denied ever using para cetamol or having ever had infectious hepatitis.

Her physical examination was exceptional for scleral icterus. Her admission test results showed that her aspartate aminotransferase (AST) level was 1066 U/L, her alanine aminotransferase level was 1136 U/L, her total bilirubin level was 7.0 mg/dL, her direct bilirubin level was 4.2, her INR was 1.8, and her alkaline phos phatase level was normal at 114. An ultrasound was requested, which revealed liver parenchymal disease, splenomegaly, left renal calculus, and minor ascites. Hepatic impairment was found via a HIDA scan. CECT abdomen was performed for additional examination, and the results revealed liver cirrhosis, significant ascites, diffuse abdominal wall edema, prominent para-aortic lymph nodes, and numerous peri-splenic and splenorenal collaterals.

HIV, CMV, hepatitis A, B, C, and E viral serologies were all negative. There was proof of an earlier EBV infection. Antinuclear antibody showed a patchy, slightly positive 1: 40 titer. Antibodies against smooth muscle, liver/ kidney micro somal, and mitochondrial proteins were all negative. Iron saturation was increased to 61% with normal ferritin, TIBC, and ceruloplasmin levels. The HFE gene did not show any mutations.

After one week, the jaundice and test results started to get better. After taking homeopathic and herbal Medi cations, the patient developed DILI. Her homeopathic and herbal remedies (Punarnava Mandur, Kanchnar guggulu, and one unlabeled) were submitted in for toxico logy testing, but the results showed no matches in their toxin database.

Additional liver biopsies were conducted for evaluation. After doing a liver biopsy, it was found that there was grade 3 bridging fibrosis, mild portal chronic inflam mation, and interface activity. The portal tracts contained ceria-rich Kupffer cells. Hepatocytes showed ballooning, which is indicative of damage. Although eosinophils were noticeable, plasma cells were not. Overall, the histo logic pattern was indicative of a medication hypersensitivity reaction coupled with resolving hepatitis.

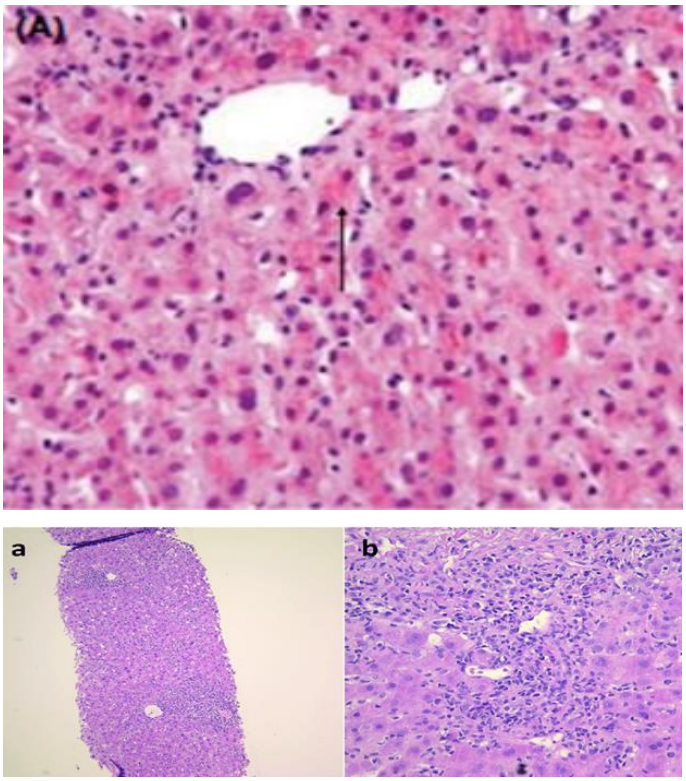

Figure 1:

Discussion

There is no recognized gold standard for assessing suspected idiosyncratic medication caused damage. We suggest combining the Roussel Uclaf Causality Assess ment Method (RUCAM) and the DILI method, which was recently proposed by ACG. DILI is a diagnostic of exclusion, and the ACG algorithm offers a fast, evidence-based diagnosis of DILI brought on by herbal and nutritional supplements. The clinical, biochemical, sero logic, and radiologic indicators of liver injury are given points according to the RUCAM scoring system. This rating provides an overall chance that a medicine is to blame for the hepatotoxicity.

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The algorithm suggests that the diagnostic technique can be adjusted to liver injury patterns (hepatocellular, cholestatic, and mixed) based on the R value after getting a full history and physical. The R value is calculated by dividing serum ALT/ultra-low-normal (ULN) by serum Alk P/ULN [7]. The R value of 26 assigned our patient to the hepatocellular damage (R 5.0) column. After that, the initial testing for hepatitis, autoimmune diseases, and imaging (abdominal ultrasound) was finished. Then, less common serology (HEV, CMV, and EBV) and second-line ceruloplasmin testing are Although the ANA titer in this patient was only 1:40, this characteristic can be present in DILI. A "possible" link with DILI was indicated by the patient's RUCAM score of 5, which was 5. This made it easier to make the diagnosis. The histology was crucial in helping to confirm the diagnosis, along with the use of imaging performed. This was done in conjunction with a thorough evaluation to rule out non-DILI reasons.

Imaging is advised for the assessment of DILI, but the modality will vary depending on clinical manifestations.

The patient's histology contributed to the diagnosis' confirmation. Kupffer cell activation was present, and the interface activity and hepatocyte ballooning were consistent with liver damage. Ceroid accumulation in the Kupffer cells strongly suggests DILI. Over time, the continual active liver damage resulted to bridging fibrosis. Although liver fibrosis and DILI can coexist, several herbal medicines have been suggested as liver fibrosis treatments. Eosinophils are related with DILI and are prevalent. Eosinophil accumulation after hepatic necrosis following specific medicines has been shown in mouse models. The hepatitis started to clear up when the offending substance was eliminated.

These results allowed for the confirmation of the diagnosis of DILI and were together suggestive of a medication reaction.

Withdrawal of the offending substance is part of the primary DILI treatment. Specific treatments, such as N-acetylcysteine for acetaminophen toxicity or L-carnitine for valproic overdoses, may be helpful if certain medicines are the cause. Bile acid sequestrants or antihistamines can be used to treat pruritus. Early detection and subsequent medication discontinuation are crucial for herbal and homeopathic remedies. Serial LFTs should be used to track the patient until normalization. Early transfer to a transplant facility may be necessary for patients who have acute liver failure because this suggests a poor prognosis. The distinctive findings from our case will help in the management of DILI secondary to complementary and alternative medicines in the future and raise awareness of this difficult – to - diag nose condition.

Conclusion

A diagnosis of drug induced liver injury (DILI) is used to rule out a rare adverse drug reaction that can result in jaundice, liver failure, or even death. The Roussel Uclaf Causality Assessment Method is a scoring method that determines the likelihood of medication-induced hepato toxicity by allocating points for clinical, biochemical, serologic, and radiologic results.

All patients with unexplained jaundice should have a thorough medical history taken, and algorithms, causal evaluation scales, histological results, and imaging should be used to diagnose drug-induced liver disease. Early detection of DILI is necessary to avoid acute liver failure.

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