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A Rare Case Report on Jaundice, Alcoholic Liver Disease with a History of Herbal Medicine Induced Liver Injury

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Abstract

Background: Alcoholic liver disease (ALD) is a complex condition caused by excessive alcohol consumption, affecting fatty liver, alcoholic hepatitis, and cirrhosis. Risk factors include alcohol intake, genetic predisposition, and co-existing liver conditions. Alcoholic fatty liver disease (AFLD) accumulates excessive fat in the liver, leading to steatosis and fibrosis. ALD complications include portal hypertension, liver failure, and hepatocellular carcinoma. The use of herbal medicines, such as tamarind and neem extracts, is also concerning due to potential liver damage. Increased awareness and caution are needed from patients and healthcare providers.

Case report: A 33-year-old male patient presented with symptoms including right hypochondrium pain, yellowish discoloration of the sclera, fever, decreased urine output, light yellow tongue, and generalized weakness. He was a chronic alcoholic, consuming various alcoholic beverages for 15 years. His dietary routine was mixed, but he preferred non-vegetarian foods. His family suggested he consume traditional herbal medicine to treat jaundice. After collecting his history, lab tests were conducted to confirm his diagnosis of alcoholic liver disease (ALD) with herbal medicine-induced liver (DILI). LFT tests were alarming, and he was diagnosed with alcoholic liver disease with jaundice and DILI due to herbal medicine. To improve his symptoms and prevent liver failure complications, he was prescribed a list of medications to improve his symptoms and to prevent complications of liver failure.

Discussion: A holistic treatment approach is needed for patients with alcoholic liver disease and herbal medicine-induced liver injury, focusing on symptom relief, liver function enhancement, and complication prevention. This includes lifestyle changes, nutritional support, and managing potential issues. Regular monitoring is crucial for adjustments.

Conclusion: This case report highlights the complex relationship between alcoholic liver disease and herbal medicine-induced liver injury, especially in jaundice cases. It emphasizes the need for thorough patient histories, increased awareness of hepatotoxic potential, and educating patients about alcohol and herbal supplement risks. It calls for multidisciplinary collaboration, enhanced regulation, and safety evaluations of herbal medicines to protect public health. It also underscores the importance of continuous monitoring and monitoring patients with liver injury histories.

Keywords: ALD, Herbal medicine, DILI, Liver failure, Patient care

Introduction

Alcoholic liver disease (ALD) represents a spectrum of liver injuries resulting from excessive alcohol consumption, encompassing conditions ranging from fatty liver to alcoholic hepatitis and cirrhosis **(1).** With the increasing prevalence of alcohol use disorders globally, understanding ALD has become imperative for both clinical and public health perspectives. The pathophysiology of ALD is complex, involving metabolic, inflammatory, and fibrotic processes that ultimately lead to liver dysfunction. Risk factors such as the quantity and duration of alcohol intake, genetic predisposition, and coexisting liver conditions contribute significantly to the disease's onset and progression **(2).**

The alcohol content, concentration, and caloric value of alcoholic drinks vary considerably. Beer typically contains 4-7% alcohol by volume (ABV), with light beers around 4% and craft or specialty beers reaching up to 12% or more **(3).** The calorie content in a 12-ounce serving of beer ranges from about 95-200, with lighter beers at the lower end and stronger, heavier beers at the higher end. Wine generally has a higher ABV than beer, usually between 11-14% for most table wines **(4).** A standard 5-ounce wine serving typically contains 120-130 calories, though this can fluctuate based on sweetness and alcohol content. Spirits and distilled liquors have the highest alcohol concentration, usually 40% ABV (80 proof) or above, including beverages like vodka, gin, rum, and whisky. Despite their high alcohol content, pure spirits are relatively low in calories, with approximately 97 calories per 1.5-ounce serving. However, mixing them with sugary drinks can significantly increase the calorie count **(5).** Liqueurs, which are flavored and sweetened spirits, typically have 15-55% ABV and tend to be more caloric due to their sugar content, often containing 150-200 calories per 1.5-ounce serving. Ready-to-drink cocktails generally have a lower ABV, usually 4-7%, comparable to beer. However, their calorie content can be high due to added sugars, frequently ranging from 150-300 calories per serving **(6).**

Chronic alcohol use causes excessive fat to accumulate in the liver, a condition known as alcoholic fatty liver disease (AFLD). The illness can advance through multiple stages, including fibrosis and steatosis, which are mainly caused by consuming too many calories, especially from alcohol **(7).** The first stage of AFLD is steatosis, also known as fatty liver. It happens when the liver produces more fat and breaks down less fat due to the additional calories from drinking alcohol. Triglycerides make up the majority of the lipid droplets that fill the hepatocytes, or liver cells. The accumulation results from impaired fatty acid oxidation, decreased hepatic lipid disposition, and increased fatty acid synthesis brought on by alcohol metabolism **(8).** Even though steatosis can usually be reversed with lifestyle modifications and alcohol abstinence, prolonged excessive calorie and alcohol intake might cause more serious liver damage. Fibrosis Steatosis might develop into fibrosis if alcohol misuse continues **(9).** The accumulation of extracellular matrix proteins, mostly collagen, in the liver tissue is a hallmark of fibrosis. This process is driven by oxidative stress from alcohol metabolism, excessive calorie consumption, activation of hepatic stellate cells, which make excessive amounts of collagen, and chronic inflammation brought on by alcohol-induced liver injury. Liver impairment and architectural distortion can result from fibrosis. In contrast to steatosis, fibrosis is frequently permanent and can develop into cirrhosis if excessive calorie intake and alcohol use persist **(10).**

Complications of ALD, including portal hypertension, liver failure, and hepatocellular carcinoma, pose significant challenges to patient management and require a multidisciplinary approach for effective intervention **(11).** Clinical manifestations of ALD can vary widely, ranging from asymptomatic presentations to severe symptoms indicative of advanced liver disease.

Laboratory parameters, including liver function tests, serum markers of inflammation, and imaging studies, play a crucial role in the diagnosis, monitoring, and treatment strategies for individuals affected by this condition. This comprehensive overview seeks to provide a foundation for further exploration and understanding of alcoholic liver disease, emphasizing the need for timely intervention and prevention strategies in at-risk populations **(12).**

The use of herbal medicines is on the rise, but their potential to cause liver injury is concerning and not well understood. Certain herbal remedies, such as tamarind and neem extracts, which are widely used in rural India, have been linked to cases of acute hepatitis **(13).** The fact that the hepatotoxic (liver-damaging) effects of many herbal preparations are not fully characterized or well-documented makes it challenging for healthcare providers to accurately predict and monitor the safety of these products. Additionally, factors like adulteration, improper storage, and mislabeling of herbal medicines can further compromise their safety and increase the risk of liver damage. The concurrent use of alcohol and herbal medicines can have a synergistic effect that leads to liver injury, potentially resulting in liver failure in patients. This is a concerning scenario that warrants further examination. Alcohol and herbal remedies can exacerbate liver damage, potentially leading to life-threatening failure if not addressed promptly. Patients should be aware of risks and consult healthcare providers **(14).**

In summary, the combined hepatotoxicity of alcohol and herbal medicines is a significant health concern that demands increased awareness and caution from both patients and healthcare providers **(15).** It is crucial for healthcare professionals to educate patients about the risks associated with using these substances together, as they can lead to severe and potentially fatal liver damage. Patients should also be proactive in sharing information about all the medications they are taking, including herbal supplements, to ensure they receive appropriate guidance and monitoring. Addressing this issue effectively requires a joint effort between patients and the healthcare community to improve outcomes and prevent liver failure **(16).**

Case Report:

A 33-year-old male patient came to the general medicine outpatient department with the complaints of right hypochondrium pain for 20 days, yellowish discoloration of sclera for 8 days; fever with on and off symptoms for 4 days, decreased urine output for 15 days, a light yellow-colored tongue, a history of yellow-colored urine, and generalized weakness. Upon physical examination, tenderness was noted in the right hypochondrium region, and no other comorbodities were known upon speaking with the patient. As a routine preliminary screening, we noted his blood pressure was 120/70 mmHg, pulse rate 110 beats per minute, and random blood sugar level is 98 mg/dL.

Upon asking the patient about his personal history, we came to know he is a chronic alcoholic, consuming since 15 years his last drink was last week, various alcoholic beverages like beer and hard liquor with unspecified quantity, but on average he said he will consume 2 beers or 120 mL of hard liquor like gin or whiskey. His dietary routine is mixed, but most of the time he prefers to eat non-vegetarian foods. He has no history of smoking or any other habits noted. His family history was not significant. During the next day's ward round and patient interview, he said he had similar complaints in the past, and he consumed some traditional herbal medicine to treat jaundice, which is locally available to his hometown, so his family members suggested he consume the same herbal medicine now to get relief from the current symptoms, so he consumed 3 doses of herbal medicine to treat the jaundice-like symptoms.

After collecting the patient history, we had suspected alcoholic liver disease (ALD) with herbal medicine-induced liver (DILI). To get confirmation, we advised some lab tests, which include USG abdomen & pelvis, CT plain

abdomen & pelvis, liver functioning tests (LFT), renal functioning test (RFT), upper GI endoscopy, HBsAg, HCV I & II, 2DEcho, complete urine examination (CUE), dengue check, smear for malaria parasite, complete blood picture, and coagulation profile.

His LFTs were alarming, with serum bilirubin total: 24.3 mg/dl, direct bilirubin: 18.0 mg/dl, albumin: 2.0 gm/dl, A/G ratio: 0.4, SGOT: 152 IU/L, SGPT: 25 IU/L, and CBP showing leukocytosis with 15,000 cells/cmm and a marked rise in the neutrophils. Hemoglobin: 6.4 gm%, RBC: 3.80 mil/cmm, PCV: 26.8%, RFT parameters normal, CT scan reports moderate hepatosplenomegaly, atrophic pancreas, PT: 17 sec, INR: 1.7, APTT: 58 sec, HCV, HBsAg, dengue, and malaria were negative. USG indicates Grade II fatty liver, moderate hepatosplenomegaly.

Upon clinical, physical examination, lab data, and patient history, we have diagnosed him with alcoholic liver diseases with jaundice and DILI due to herbal medicine. To improve the patient symptoms and prevent the liver failure complications, he was advised with a list of medicines that includes Tab Pantoprazole 40 mg, Cefoperazone and Sulbactam 1.5 gm 12th hourly, paracetamol 650 SOS, injection benfothiamine 100 mg IV/12th hourly, tab spiranolactone 25 mg/BD, tablet rifaximin 550 mg/BD, tab Ursodeoxycholic Acid 300 mg/BD, and syrup Lactulose 10 ml/TID.

Disscusion:

For patients diagnosed with alcoholic liver disease and herbal medicine-induced liver injury (DILI), a holistic treatment approach is necessary. This approach should focus on symptom relief, liver function enhancement, and complication prevention **(17).** The treatment strategy must incorporate lifestyle changes, including complete alcohol cessation and avoidance of liver-toxic substances, particularly the herbal medicine responsible for the DILI. Attention should also be given to nutritional support and managing potential issues such as ascites, encephalopathy, and portal hypertension. The patient's progress should be closely monitored through regular liver function tests and follow-up appointments, allowing for necessary adjustments to the treatment plan. This comprehensive approach aims to address the patient's current condition while preventing further liver damage and promoting recovery. This regimen addresses various complications of liver disease, including infection prevention, fluid management, and hepatic encephalopathy prevention. However, close monitoring of liver function and potential drug interactions is crucial **(18)**.

The patient was given a combination of medications to manage various aspects of their liver condition. Rifaximin and lactulose were prescribed to control ammonia levels, preventing hepatic encephalopathy. Ursodeoxycholic acid was administered as a liver protectant to enhance bile flow, reducing further damage and alleviating symptoms. To address potential thiamine deficiency, common in alcoholics, an injection of benfothiamine, a vitamin B1 derivative, was provided. Spironolactone, a diuretic, was used to manage fluid retention and ascites associated with liver disease, crucial for preventing volume overload and treating edema. Lastly, a potent antibiotic combination of cefoperazone and sulbactam was prescribed to prevent life-threatening infections.

The patient presented with elevated bilirubin levels and liver function abnormalities, prompting an investigation into the potential causes of their jaundice and acute liver dysfunction. The clinical evaluation revealed a complex interplay between the patient's history of herbal medicine use and the development of drug-induced liver injury (DILI), necessitating a thorough assessment of the specific herbal products consumed. This case highlights the importance of considering alternative medicine practices in patients presenting with liver dysfunction, as certain herbal compounds can have hepatotoxic effects that may not be widely recognized. Understanding the potential risks

associated with herbal medicine is crucial for healthcare providers, as they must balance the benefits of these treatments against their possible adverse effects on liver health. In light of this, further research is needed to establish clear guidelines for the safe use of herbal remedies, particularly in patients with pre-existing liver conditions or those taking concurrent medications that may exacerbate the risk of liver injury. Establishing a comprehensive framework for evaluating the safety and efficacy of herbal products will not only enhance patient care but also foster informed decision-making among healthcare providers and patients alike. This framework should include rigorous clinical trials, standardized dosing recommendations, and thorough assessments of potential herb-drug interactions to ensure the safe integration of herbal medicine into conventional treatment plans. Such measures will ultimately contribute to a more holistic approach to patient care, bridging the gap between traditional practices and modern medical standards.

Conclusion:

In conclusion, this case report elucidates the complex interplay between alcoholic liver disease and liver injury induced by herbal medicine, particularly in the presentation of jaundice. It accentuates the imperative for healthcare practitioners to perform thorough patient histories that include both alcohol intake and the consumption of herbal supplements. The difficulties in differentiating among various etiologies of liver injury in patients presenting with multiple risk factors are considerable and demand an increased awareness of the hepatotoxic potential associated with herbal remedies, particularly in individuals who have pre-existing liver conditions. Moreover, educating patients about the risks linked to alcohol and unregulated herbal supplements are of utmost importance. A multidisciplinary framework is vital for the effective management of intricate cases of liver disease, and this case serves as a stimulus for forthcoming research aimed at clarifying the interactions between these two categories of liver injury. Additionally, it underscores the pressing necessity for enhanced regulation and safety evaluations of herbal medicines to safeguard public health. Continuous follow-up and monitoring of patients with a history of liver injury from diverse sources are essential, as are the implications for public health policy regarding alcohol consumption and the regulation of herbal supplements. Overall, this case report provides significant insights that can inform clinical practice and policy-making within the domain of liver health. Such findings highlight the critical importance of interdisciplinary collaboration among healthcare professionals, researchers, and policymakers to formulate comprehensive strategies that address the complex challenges presented by liver diseases.

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