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CASE REPORT

Drug-Induced Hepatitis Following Albendazole Use: A Case Report and Literature Review

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Abstract

Albendazole is a broad-spectrum anthelmintic drug generally regarded as safe. Rarely, it may cause acute toxic hepatitis. We report a case of severe hepatotoxicity following albendazole use in a pediatric patient. Other causes of acute hepatitis were excluded, and the Roussel Uclaf Causality Assessment Method (RUCAM) score was 8, indicating a highly probable drug-induced liver injury.

Keywords: Albendazole; Drug-induced hepatitis; Hepatotoxicity; RUCAM; Anthelmintics.

1. Introduction

Albendazole is a well-known anthelmintic medication that is effective against a variety of parasitic infections. While albendazole is generally considered safe and well-tolerated, there have been reports of hepatotoxicity associated with its use [2]. The precise mechanism by which albendazole causes hepatitis remains unknown. It is thought to be related to the drug's metabolism in the liver, which results in the formation of toxic metabolites. Pre-existing liver disease, concurrent use of other hepatotoxic drugs, or individual susceptibility may all increase the risk of developing albendazole-induced hepatitis [3]. While albendazole is an effective treatment for parasitic infections, healthcare providers should be aware of the possibility of albendazole-induced hepatitis. Vigilant monitoring and careful consideration of risk factors are required to ensure the safe use of this medication, and if signs of liver dysfunction appear during treatment, prompt action should be taken.

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2. Case Report

A 13-year-old girl presented to the emergency department complaining of 2 days of generalized abdominal pain, jaundice, nausea, vomiting, and diarrhea. There had been no history of outside eating, recent travel, fever, joint pains, or rashes. None of the other family members had similar complaints. Except for two doses of the antiparasitic drug albendazole administered for deworming, she had no history of medication use prior to the onset of symptoms. The first dose was given 15 days before the onset of symptoms, and the second dose was given three days before. There was no prior use of herbal medications. She was taking an iron supplement orally. There was no history of such illness in the family. The initial vital signs were 97/55 blood pressure, 88 heart rate, 18 respiratory rate, and 36.5 °C body temperature (oral). Physical examination revealed tenderness on the left side of the epigastrium, no palpable liver, normal liver percussion span, and no other notable findings at admission. Laboratory investigations revealed a white blood cell count of 6630 /µL, hemoglobin 13.5 g/dL, and platelets were 194,000/µL. The prothrombin time 20.60 sec (INR 1.86), Aspartate aminotransferase 2067 IU/L, Alanine aminotransferase 2931 IU/L, Alkaline phosphatase 135.58 IU/L, total bilirubin 51.3 mg/dL, total protein 77 g/L, albumin 39.2 g/L and serum glucose 4.3 mmol/L. Hepatitis A (IgM anti-HAV), Hepatitis B (HBSAg, HBCAg, HBC IgM, HBEAg, HBEAb), Hepatitis E (HEV IGM), and Hepatitis C (anti-HCV) viral markers were all negative. Anti-CMV, anti-HSV, and HIV serology were also negative. Autoimmune hepatitis markers (anti-SMA, anti AMA) were negative. The level of ceruloplasmin in the blood was normal. Thyroid function tests were normal, and the serum toxicology for acetaminophen level was not high. Urinalysis results were normal. Initial imaging with computerized tomography (CT) of the abdomen without contrast ruled out other abdominal pathology. Abdominal ultrasound showed a normal liver with no focal lesions or biliary dilatation. After excluding all possible causes, drug-induced hepatitis secondary to albendazole was suspected. The patient was admitted in a high dependency unit, where she stabilized and progressively improved. She recovered with supportive treatment and nutrition, with her liver function tests gradually improving to normal. The patient was subsequently discharged and followed up without significant symptoms (Figs. 1 and 2).

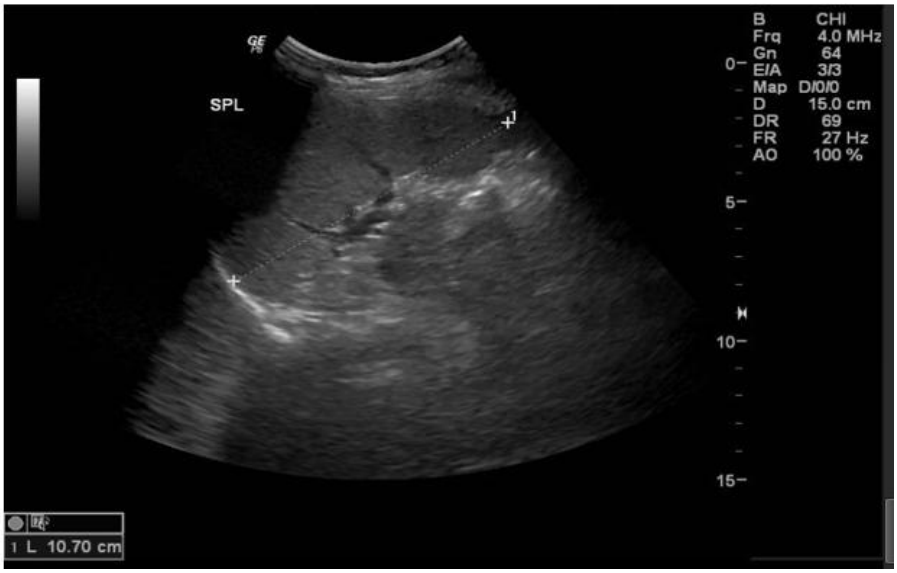

Fig. 1. Abdominal ultrasound shows normal spleen.

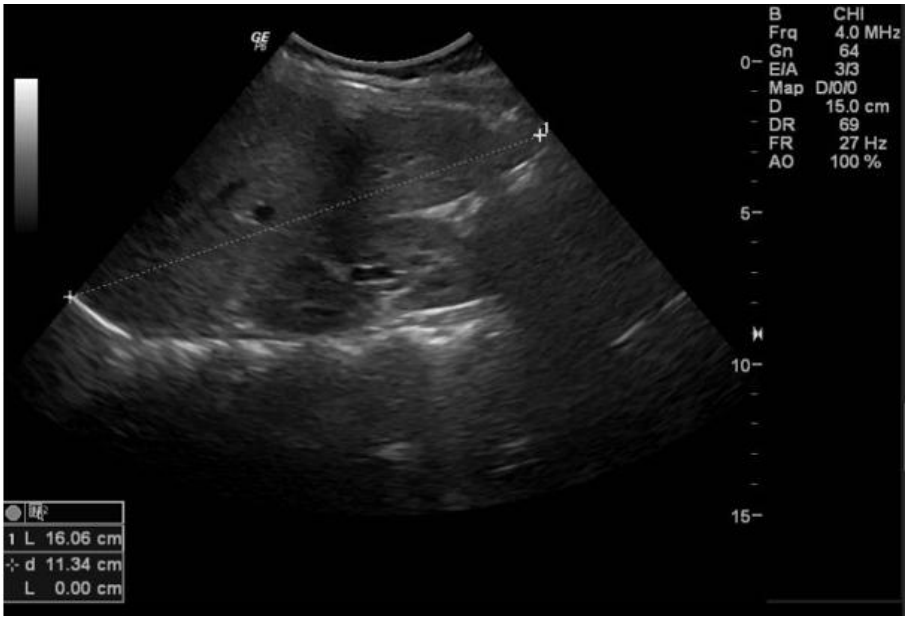

Fig. 2. Abdominal ultrasound shows normal liver.

3. Discussion

Drug-induced liver injury (DILI) is a component of the differential diagnosis for liver conditions, with an estimated annual incidence of 1 in 10,000-100,000 individuals exposed to prescribed medications. Its consideration becomes paramount when other potential etiological factors linked to clinical history have been ruled out. Neglecting the timely diagnosis and management of DILI could result in liver failure and even mortality. Liver injuries are categorized into three primary types: hepatocellular injury, cholestatic and combined. In our case, the elevated ALT (Alanine transaminase) is classified as a hepatocellular injury. A rechallenge test with albendazole was not performed due to the potential risk of severe liver injury.

Albendazole-induced hepatotoxicity has been identified as a mixed type in previous research. In the most extensive documented series focusing on prolonged albendazole use for hydatid disease, a 5% incidence of hepatotoxicity was observed with a dosage of 400 mg administered twice daily for 28 days. In previously reported cases of albendazole-induced liver injury, liver biopsy has demonstrated periportal inflammation, hepatocellular necrosis, and varying degrees of steatosis, supporting the diagnosis of drug-induced liver injury [4]. However, in the present case, a liver biopsy was not performed, as the patient showed progressive clinical and biochemical improvement following discontinuation of albendazole and supportive management. Given the favorable clinical course and the clear exclusion of alternative etiologies, invasive diagnostic procedures were not pursued. The precise mechanism behind albendazole-induced hepatitis remains unknown. Considering that our patient experienced symptoms within 15 days of drug ingestion, there is a possibility of immune-mediated injury. While albendazole is primarily metabolized in the liver, instances of liver enzyme elevations are rarely reported. In the majority of documented cases, these elevations have been mild to moderate, occurring after prolonged administration and improving upon discontinuation of treatment [5]. A meta-analysis encompassing patients treated with benzimidazoles (albendazole and mebendazole) revealed that only 13 out of 1681 patients (0.7%) developed mild and transient elevated liver enzyme levels. Notably, severe liver toxicity induced by albendazole is exceedingly rare [6].

Additionally, albendazole disrupts glucose uptake by helminthic cells. Albendazole contains numerous inactive components that can trigger allergic reactions [7]. These adverse effects encompass difficulties in breathing, feelings of nausea, bouts of vomiting, abdominal discomfort, headaches, temporary hair loss, jaundice, fatigue, darkened urine, constipation, increased thirst, and pruritus [8]. Serum analysis yielded negative results for hepatitis A, B, and C. Additionally, conditions such as hemoglobinopathy or Wilson's disease were excluded. A liver biopsy was not performed as the patient exhibited progressive improvement and made a complete recovery with discontinuation of the drug and application of supportive treatment.

4. Literature Review

These reports suggest that while the overall risk is low, clinicians must remain vigilant, especially in pediatric populations or patients with comorbidities or polypharmacy. Tajiri and Shimizu's guidelines emphasize the use of the RUCAM scale in identifying DILI, which is a structured, validated tool for assessing causality [4]. Pediatric cases, such as the one reported by Amoruso et al., further illustrate the rare but significant hepatotoxic potential of albendazole, even with short courses of therapy [5]. Garcia-Cortes et al. and Chalasani et al. have also discussed DILI pathophysiology and epidemiology in greater detail, underlining the idiosyncratic nature of such reactions [6], [7].

Albendazole, a benzimidazole derivative, is widely used in the treatment of helminthic infections due to its broad-spectrum efficacy and general safety profile [1]. However, cases of drug-induced liver injury (DILI) have been reported. Choi et al. described acute hepatitis linked to albendazole in an adult patient, highlighting the importance of early detection and discontinuation of the drug [2]. In another case, Ríos and Restrepo documented hepatotoxicity in a young patient, demonstrating periportal necrosis and inflammation on biopsy [3] (Table 1).

Table 1: RUCAM Score for Hepatocellular Injury.

RUCAM Criterion	Score	Explanation
Time to onset from drug intake (5–90 days)	+2	Symptoms began 3 days after 2nd dose (15 days after 1st dose)
Course after cessation (≥50% decrease in ALT within 8d)	+3	Significant improvement observed during hospitalization
Risk factors (age, alcohol, pregnancy)	0	None present
Concomitant drugs	0	None known to be hepatotoxic
Non-drug causes ruled out	+2	Viral, autoimmune, metabolic causes excluded
Previous hepatotoxicity of the drug	+1	Known rare reports exist
Rechallenge	0	Rechallenge was not performed due to safety concerns
Total Score	8	Highly probable

5. Conclusion

As demonstrated in our case, albendazole can lead to severe liver injury. Given that antiparasitic medications are commonly taken, often on an empirical basis, physicians should be aware of their potential adverse effects despite their rarity. The use of these readily available antiparasitic drugs for prophylactic purposes should be strongly discouraged, and consultation with a physician is essential to determine their appropriateness. Based on this case, we deduce that in the medical history assessment of a patient with hepatitis, inquiries about the possible ingestion of antiparasitic drugs should be included [10]. Additionally, there is a need for further studies and reports on albendazole-induced liver injuries.

5. Conflict of Interest

The authors declare no competing interests.

7. References

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