To the Editor:

Albendazole is a commonly prescribed anthelmintic that typically is well tolerated. Its broadest application is in developing countries that have a high rate of endemic nematode infection. Albendazole belongs to the benzimidazole class of anthelmintic chemotherapeutic agents that function by inhibiting microtubule dynamics, resulting in cytotoxic antimitotic effects. Benzimidazoles (eg, albendazole, mebendazole) have a binding affinity for helminthic β-tubulin that is 25- to 400-times greater than their binding affinity for the mammalian counterpart. Consequently, benzimidazoles generally are afforded a very broad therapeutic index for helminthic infection.

A 53-year-old man presented to the emergency department (ED) after an episode of syncope and sudden hair loss. At presentation he had a fever (temperature, 103°F [39.4°C]), a heart rate of 120 bpm, and pancytopenia (white blood cell count, 0.4×10^3/μL [reference range, 4.0–10.0×10^3/μL]; hemoglobin, 7.0 g/dL [reference range, 11.2–15.7 g/dL]; platelet count, 100×10^3/μL [reference range, 150–400×10^3/μL]). A toxicology screen was positive for cocaine, opiates, and benzodiazepines. The blood alcohol concentration was 126 mg/dL.

The patient reported severe gastrointestinal (GI) distress and diarrhea for the last year as well as a 25-lb weight loss. He discussed his belief that his GI symptoms were due to a parasite he had acquired the year prior; however, he reported that an exhaustive outpatient GI workup had been negative. Two weeks before presentation to our ED, the patient presented to another ED with stomach upset and was given a dose of albendazole. Perceiving alleviation of his symptoms, he purchased 2 bottles of veterinary albendazole online and consumed 113,000 mg—approximately 300 times the standard dose of 400 mg.

A dermatologic examination in our ED demonstrated reticulated violaceous patches on the face and severe alopecia with preferential sparing of the occipital scalp (Figure 1). Photographs taken by the patient on his phone from a week prior to presentation showed no facial dyschromia or signs of hair loss. A punch biopsy of the chin demonstrated perivascular and perifollicular dermatitis with eosinophils, most consistent with a drug reaction. The patient received broad-spectrum antibiotics and supportive care. Blood count parameters normalized, and his hair began to regrow within 2 weeks after albendazole discontinuation (Figure 2).

Acute Alopecia Associated With Albendazole Toxicosis

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PRACTICE POINTS

- Albendazole functions by inhibiting microtubule dynamics and has a remarkably greater binding affinity for helminthic β-tubulin than for its mammalian counterpart.
- An uncommon adverse effect of albendazole at therapeutic dosing is a dose-dependent telogen effluvium in susceptible persons, likely caused by the inherent susceptibility of follicular matrix keratinocytes to anti-microtubule agents.
- Massive albendazole overdose can cause anagen effluvium and myelosuppression similar to the effects of conventional chemotherapy.

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Our patient exhibited symptoms of tachycardia, pancytopenia, and acute massive hair loss with preferential sparing of the occipital and posterior hair line; this pattern of hair loss is classic in men with chemotherapy-induced anagen effluvium. Conventional chemotherapeutics include taxanes and Vinca alkaloids, both of which bind mammalian $\beta$-tubulin and commonly induce anagen effluvium.

Our patient’s toxicosis syndrome was strikingly similar to common adverse effects in patients treated with conventional chemotherapeutics, including aplastic anemia with severe neutropenia and anagen effluvium. This adverse effect profile suggests that albendazole exerts an effect on mammalian $\beta$-tubulin that is similar to conventional chemotherapy when albendazole is ingested in a massive quantity.

Other reports of albendazole-induced alopecia describe an idiosyncratic, dose-dependent telogen effluvium. Conventional chemotherapy uncommonly might induce telogen effluvium when given below a threshold necessary to induce anagen effluvium. In those cases, follicular matrix keratinocytes are disrupted without complete follicular fracture and attempt to repair the damaged elongating follicle before entering the telogen phase. This observed phenomenon and the inherent susceptibility of matrix keratinocytes to antimicrotubule agents might explain why a therapeutic dose of albendazole has been associated with telogen effluvium in certain individuals.

Our case of albendazole-related toxicosis of this magnitude is unique. Ghias et al reported a case of abendazole-induced anagen effluvium. Future reports might clarify whether this toxicosis syndrome is typical or atypical in massive albendazole overdose.

REFERENCES


