Acute-Onset Alopecia

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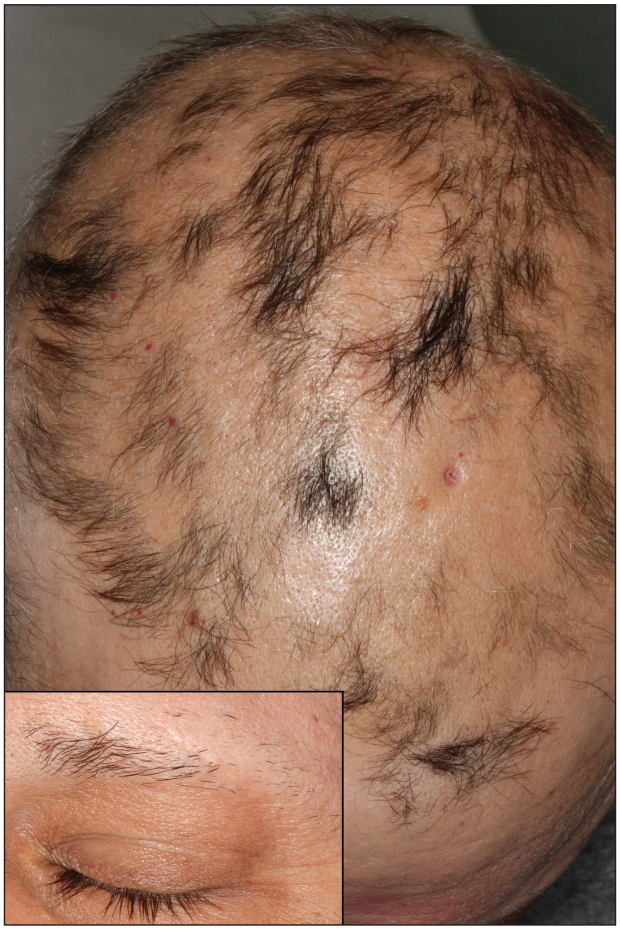

A previously healthy 45-year-old man presented to the dermatology department with abrupt onset of patchy, progressively worsening alopecia of the scalp as well as nausea with emesis and blurry vision of a few weeks' duration. All symptoms were temporally associated with a new demolition job the patient had started at an industrial site. He reported 10 other contractors were similarly affected. The patient denied paresthesia or other skin changes. On physical examination, large patches of smooth alopecia without erythema, scale, scarring, tenderness, or edema that coalesced to involve the majority of the scalp, eyebrows, and eyelashes (inset) were noted.

WHAT'S THE **DIAGNOSIS?**

a. alopecia areata
b. dioxin-induced alopecia
c. phosgene-induced alopecia
d. syphilitic alopecia
e. thallium-induced alopecia

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THE **DIAGNOSIS:** Thallium-Induced Alopecia

At the time of presentation, a punch biopsy specimen of the scalp revealed nonscarring alopecia with increased catagen hairs; follicular miniaturization; peribulbar lymphoid infiltrates; and fibrous tract remnants containing melanin, lymphocytes, and occasional mast cells (Figure 1). The differential diagnosis included alopecia areata, syphilis, and toxin-mediated anagen effluvium (AE). Given the abrupt onset affecting multiple individuals in an industrial environment, heavy metal poisoning was suspected. Blood and urine testing was negative, but a few months had elapsed since exposure. Several months after his initial presentation, the patient reported problems with his teeth, thin brittle nails, and resolution of the visual changes. Photographs sent by the patient revealed darkening and degeneration of the gingival margin (Figure 2).

Environmental review revealed the patient was working on a demolition site of a 150-year-old electrical plant near a river. Inundation of rainfall caused a river swell and subsequent flooding of the work site. The patient reported working for more than 2 months in knee-deep muddy water, and he noted that water for consumption and showers was procured on-site from a well-based source that may have been contaminated by the floodwaters.

Acute nonscarring alopecia can be an AE or telogen effluvium (TE), also known as telogen defluvium. The key distinguishing factor is the mode of injury.[1] In TE, medications, stress, hormonal shifts, or inflammation induce a synchronized and abrupt transition of hairs from anagen phase to catagen phase, a committed step that then must fully cycle through the telogen phase, culminating in the simultaneous shedding of numerous telogen hairs approximately 3 to 4 months later. Conversely, AE is caused by a sudden insult to the metabolic machinery of the hair matrix. Affected follicles rapidly produce thinner weaker shafts yielding Pohl-Pinkus constrictions or pencil point–shaped fractures that shed approximately 1 to 2 months after injury. The 10% of scalp hairs in the resting telogen phase have no matrix and thus are unaffected. Some etiologies can cause either AE or TE, depending on the dose and intensity of the insult. Common causes of AE include alopecia areata and syphilis, both consisting of abrupt severe bulbar inflammation.[1] Other causes include chemotherapy, particularly antimetabolites, alkylating agents, and mitotic inhibitors; radiation; medications (eg, isoniazid); severe protein malnutrition; toxic chemicals (eg, boron/boric acid); and heavy metals (eg, thallium, mercury).

Thallium is one of the most common causes of heavy metal poisoning and is particularly dangerous due to its colorless, tasteless, and odorless characteristics. Although its common use as a rodenticide has dramatically decreased in the United States after it was banned in 1965, it is still used in this fashion in other countries and has a notable industrial presence, particularly in electronics, superconductors, and low-temperature thermometers. Accidental poisoning of a graduate chemistry student during copper research has been reported,[2] highlighting that thallium can be inhaled, ingested, or absorbed through the skin. Thallium is even present in mycoplasma agar plates, the ingestion of which has resulted in poisoning.[3]

Systemic symptoms of thallium poisoning include somnolence, weakness, nausea, vomiting, stomatitis, abdominal pain, diarrhea, tachycardia, hypertension, and polyneuropathy.[4-7] Neuropathy often manifests as painful acral dysesthesia and paresthesia, perioral numbness, optic neuropathy causing visual changes, and encephalopathy. Cutaneous findings include diffuse alopecia of the scalp and eyebrows, perioral dermatitis, glossitis, diffuse hyperpigmentation, oral hyperpigmentation (often as a stippled lead line along the gingival margin with

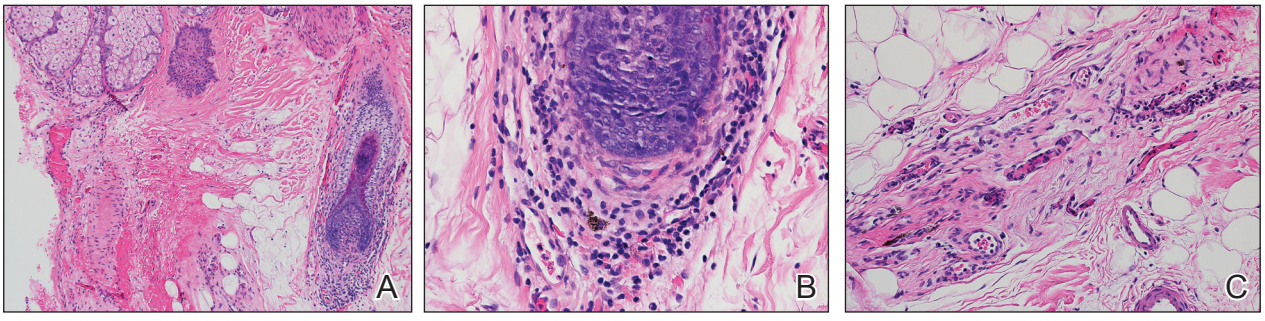

FIGURE 1. A, Peribulbar lymphoid infiltrate with a nearby catagen hair (H&E, original magnification ×10). B, Peribulbar lymphoid infiltrate with melanin deposition (H&E, original magnification ×40). C, Fibrous tract remnant with lymphoid infiltrate, occasional mast cells, and melanin deposition (H&E, original magnification ×20).

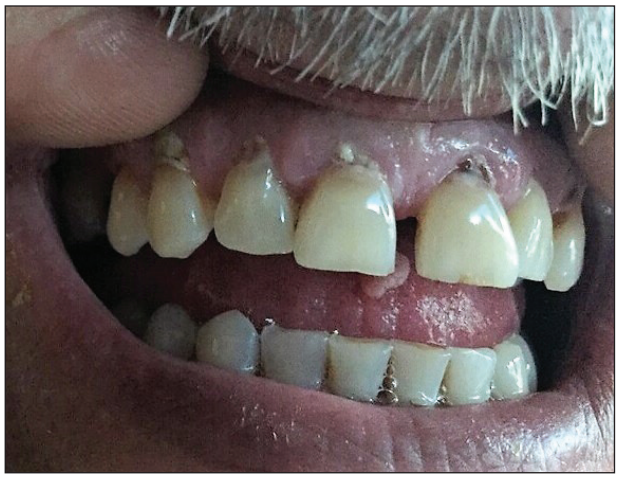

FIGURE 2. Darkening and degeneration of the gingival margin.

subsequent alveolar damage and resorption), melanonychia, palmoplantar keratoderma, acneform or pustular eruption, and nail changes including Mees lines.[2,4,5,7-9] Rarely, major organ failure and death may result.[10]

Toxin panels may not include thallium, and urine and serum tests may be negative if too much time has transpired since the acute exposure. Hair or nail analysis has proved useful in subacute cases[11]; however, most laboratories require a pencil-thick segment of hair cut at the roots and bundled, weighing at least 500 mg. Thallium poisoning is treated with activated charcoal, Prussian blue, and blood purification therapies (eg, hemodialysis, hemoperfusion, hemofiltration).[4,7] Cutaneous findings typically resolve, but neuropathic changes may persist.

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