White Concretions on the Hair Shaft

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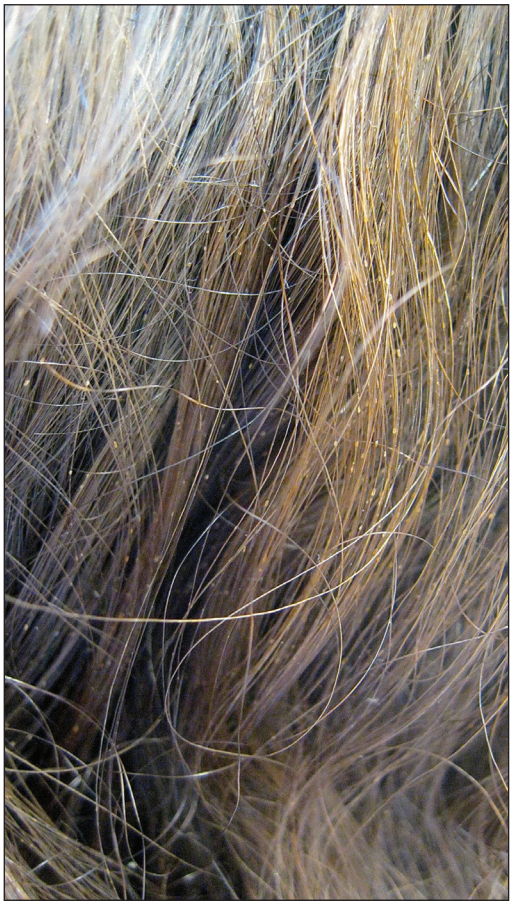

A 35-year-old woman presented with possible nits on the hair of 1 year's duration. She was previously evaluated by several outside medical providers and was unsuccessfully treated with topical and systemic medications for pediculosis. She reported sporadic scalp pruritus but denied hair loss, breakage, close contacts with similar symptoms, or recent travel outside the United States. She was otherwise healthy and was not taking any medications. Physical examination revealed small 1- to 2-mm, generalized, somewhat detachable, white concretions randomly distributed on the hair shafts. No broken hairs were observed. The eyebrows, eyelash hairs, and surrounding skin were normal. Potassium hydroxide mount was equivocal for nits.

WHAT'S THE **DIAGNOSIS?**

a. hair cast
b. resistant pediculosis
c. tinea capitis
d. trichobacteriosis
e. white piedra

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THE **DIAGNOSIS:** White Piedra

A fungal culture demonstrated a filamentous fungus that was identified as *Trichosporon inkin* via DNA sequencing, which confirmed the diagnosis of white piedra (WP).

Piedra refers to a group of fungal infections presenting as gritty nodules adherent to the hair shaft.[1] It is further categorized into black piedra, which occurs more commonly in tropical climates and is caused by *Piedraia hortae*, and WP, which occurs in tropical and temperate climates and is caused by the *Trichosporon* genus.[1-3] Among the *Trichosporon* genus, clinical manifestations have varied based on species; for example, *T inkin* commonly causes genital WP, *Trichosporon ovoides* commonly causes scalp WP, and *Trichosporon asahii* and *Trichosporon mucoides* have been described to cause systemic fungal infections in immunocompromised hosts.[1,4] Scalp WP most commonly occurs in children and young adults, and females are at greater risk than males.[1,2,5,6]

Clinically, WP presents with pale irregular nodules along the hair shaft that are not fluorescent on Wood lamp examination.[1,6,7] Nodules are soft and easily detached from the hair shaft, unlike the hard, tightly adherent nodules seen in black piedra.[1,7] White piedra affects hair in a variety of areas including the scalp, beard, eyebrows, eyelashes, axillae, and genitals.[1,7] Affected hair may become brittle and break at points of invasion.[1] Alternatively, WP may resemble tinea capitis with scalp hyperkeratosis and alopecia, though tinea typically affects the base of the hair shaft.[1] Immunocompromised patients can develop disseminated WP, and cases of progressive pneumonia, lung abscess, peritonitis, vascular access infection, and endocarditis have been reported.[2]

Diagnosis of WP is made through a combination of clinical findings and culture of infected hair. Potassium hydroxide preparation demonstrates sleevelike concretions formed of masses of septate hyphae with dense zones of arthrospores and blastospores.[1,2] Culture on Sabouraud agar demonstrates creamy colonies that develop a dull, gray, wrinkled surface.[1,2] Differential diagnosis includes pediculosis; however, the concretions of WP are circumferential around the hair shaft on microscopy.[1] Notably, a case of concomitant WP and pediculosis has been reported.[8] In cases of potential pediculosis resistant to therapy, consider hair casts, which are asymptomatic, white, cylindrical concretions that encircle the hair without adherence and can therefore be differentiated from pediculosis via dermoscopy.[9] Because this phenomenon is more commonly observed in preadolescent girls, it is hypothesized that scalp inflammation due to traction from hairstyles or atopic dermatitis contributes to the development of hair casts.[9,10] Thus, when a potassium hydroxide mount is equivocal for nits and dermoscopy demonstrates concretions that completely encircle the hair shaft, it is important to perform a microbiologic culture to rule out piedra of the hair or scalp. Other differential diagnoses include tinea capitis, black piedra, trichobacteriosis, and hair shaft abnormalities.

Transmission of WP is thought to result from a combination of poor hygiene; humidity due to climate; personal care practices such as habitually tying wet hair, applying hair oils and conditioners, or covering hair according to social customs; and close contact with an infected individual.[1,3,6] Long scalp hair potentially correlates with increased risk.[1,6] Finally, WP has been described in animals and has been isolated from soil, vegetable matter, and water.[3,10]

Treatment of WP generally involves removal of infected hair, antifungal agents, and improved hygienic habits to avoid relapses. The American Academy of Dermatology's Guidelines/Outcomes Committee recommends complete removal of infected hair; however, patients may desire hair-preserving treatments.[11] Kiken et al[1] reported success with the combination of an oral azole antifungal agent for 3 weeks to 1 month and an antifungal shampoo for 2 to 3 months. The authors proposed that oral medication eliminates scalp carriage while antifungal shampoo eliminates hair shaft concretions.[1]

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