

SHORT COMMUNICATIONS

Autosensitization Due to Pediculosis Capitis (A “Pediculid”) in a 16-Year Old Female: A Case Report

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A 16-year-old Indian female presented for a generalized, pruritic eruption of two weeks duration. Hydrocortisone cream, oral antihistamines, and permethrin provided minimal relief. She denied recent travel or new medications. Physical examination revealed generalized, excoriated erythematous papules and plaques with associated periorbital edema (Figure 1 & 2). No mucosal changes or lymphadenopathy were noted.

Lesional and perilesional biopsies revealed a mixed perivascular lymphocyte-rich infiltrate with conspicuous eosinophils and negative direct immunofluorescence. Labs revealed peripheral eosinophilia and negative celiac markers; therefore, a tentative diagnosis of hypersensitivity reaction was reached. The patient was started on prednisone 40mg daily, triamcinolone cream BID, and hydroxyzine 50 mg QHS.

At one-week follow-up, the patient had minimal improvement and recalled having a slumber party with a contact with visible nits of her scalp. Upon reexamination, numerous nits were observed on the hair shafts 1-4 cm from the scalp (Figure 3). Subsequently, the diagnosis of pediculosis

capitis with an associated pediculid reaction was made. She was given two doses of oral ivermectin 10-days apart and a 3-week prednisone taper. The decision to treat with ivermectin was made because the patient refused to cut her hair and believed that current topical treatments were “toxic”. Immediate family members were treated with topical ivermectin.

Pediculosis capitis is an infestation of the scalp by the head louse *Pediculus humanus capitis*. The development of disseminated eczematous lesions as a result of the primary localized rash is known as an “Id” reaction; commonly reported in association with stasis dermatitis, tinea, or scabies.¹ Autosensitization secondary to pediculosis capitis was first reported in 1946, and to our knowledge, this is just the fourth report of a pediculid case.^{2,3} Histopathologic examination of Id lesions shows acute spongiotic dermatitis with lymphocytic inflammation and eosinophils, as was seen in our patient.¹ Treatment of Id reactions are twofold: suppressing inflammation with systemic corticosteroids, but most importantly, treating the underlying suspected source of hypersensitivity. In our case, the patient’s symptoms responded

minimally to prednisone until adequate anti-pediculosis treatment was instituted.

Figure 1. Mildly erythematous, scaly papules coalescing on forehead, temple and cheek with periorbital edema.



The prevalence of louse infections has increased over recent decades and is not limited to lower socioeconomic groups.⁴ Interestingly, increased resistance to topical permethrin has been found in higher socioeconomic communities.⁴ The treatment of pediculosis capitis is often challenging due to increasing louse resistance, patient refusal to cut the affected hair, and increasing cost of treatment. There has been an emergence of lice removal centers that utilize machines to deliver intense heat

to lice and nits; however, these treatments are costly to patients and are supported by small studies.⁵ There is also a trend towards refusal of topical treatment by patients due to fear of “chemicals” and preference towards treatments considered more natural (e.g. essential oils). Modern-day practices of taking “selfies” with friends and the taboos of disclosing personal infestation, which both played a role in acquiring the infestation in our patient, will pose challenges in decreasing the incidence. Practitioners should be aware of these current trends, evolving treatments, and the presence of id reactions related to pediculosis.

Figure 2. Numerous excoriation macules and papules of bilateral upper and lower extremities with sparing of the palms.



Figure 3. Numerous nits identified on scalp hair view under dermoscopy (inset: nit on hair shaft submerged in mineral oil, observed at 10x magnification)



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