

Case Report

At First Glance: Psoriatic Response to Transdermal Nicotine Patch Application

Joseph M. LaRochelle^{a, b} Victoria R. Dimitriades^c^aCollege of Pharmacy, Xavier University of Louisiana, New Orleans, LA, USA; ^bSchool of Medicine, Louisiana State University Health Sciences Center, New Orleans, LA, USA;^cDivision of Pediatric Allergy, Immunology and Rheumatology, University of California Davis Health, Sacramento, CA, USA**Keywords**

Contact dermatitis · Psoriasis · Rash · Nicotine

Abstract

Contact dermatitis and psoriasis are common skin disorders which represent two distinct pathologies. Skin disorders heavily rely on corresponding history for diagnosis; this case demonstrates the challenges of relying on history alone for final identification. A patient presented to clinic for evaluation of a new rash on his abdomen. Past medical history was notable for recent initiation of a smoking cessation program utilizing nicotine patches as well as a pharyngitis treated with antibiotics 1 week prior. Despite use of topical steroid and cessation of the patches, the well-demarcated rash became more generalized. Patch testing for the nicotine patch and chemical sensitizers was negative and an eventual biopsy was consistent with guttate psoriasis. Contact dermatitis relies heavily on clinical history for diagnosis. This case demonstrates that history can be misleading. In retrospect, it is likely that a preceding presumed streptococcal infection was the primary inducer of guttate psoriasis.

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Contact dermatitis (CD) and psoriasis are skin disorders which represent two distinct pathologies. Although both are initiated by keratinocytes and T cells, the former is mediated by a delayed-type IV hypersensitivity reaction and the latter is mediated predominantly by proinflammatory cytokines released by Th1 and Th17 cells [1]. Skin disorders rely on corre-

Joseph M. LaRochelle, PharmD, BCPPS, FCCP
Division of Clinical and Administrative Sciences College of Pharmacy
Louisiana State University Health Sciences Center School of Medicine
Xavier University of Louisiana, 1 Drexel Drive, New Orleans, LA 70125 (USA)
E-Mail jlaroche@xula.edu



Fig. 1. Application point of transdermal nicotine patch.



Fig. 2. Psoriasis lesions distant from patch application point.

sponding history for diagnosis; this case demonstrates the challenges of relying on history alone for final identification.

A 35-year-old Caucasian male presented to Allergy clinic for evaluation of a new rash on his flank. Four days prior to the development of the rash, the patient began a smoking cessation program utilizing transdermal nicotine patches. After 2 days, a rash was noted and they were discontinued. The rash at presentation was a well-demarcated, erythematous, papular patch measuring approximately 20 cm × 10 cm (Fig. 1). He also reported that he was being treated for a presumed Group A *Streptococcus* throat infection diagnosed 1 week prior.

Initially felt to be an allergic CD to the nicotine patches, he was prescribed a medium-potency topical steroid. Despite this, the rash worsened and began to involve other areas of skin as well (Fig. 2). Patch testing on unaffected skin was negative to three standard panels of epicutaneous patch testing and a new nicotine patch. Additionally, biopsies of the original rash and one distal from the original eruption showed “psoriasiform and superficial perivascular dermatitis with mounds of parakeratosis and neutrophils,” consistent with guttate

psoriasis (data not shown). The patient was treated with UV light and high-potency topical steroids with improvement, though he continues to have flares occasionally.

Psoriasis is a chronic immune-mediated skin condition resulting in well-demarcated erythematous plaque formation. Multiple presentations of psoriasis exist, including plaque, guttate, pustular, and erythrodermic forms. Along with genetic risk factors, environmental factors are known to trigger or exacerbate psoriasis, including trauma, infections, and medications [1]. Specifically, streptococcal infections are known to be inducers of guttate psoriasis. The mechanism for the induction of psoriasis by infection is hypothesized to be due to superantigen-induced T cells which produce increased amounts of interferon- γ locally [2].

Transdermal nicotine patches have been implicated in causing local irritation [3] and CD [4]. Nicotine's potent vasodilatory properties have been implicated in the etiology of skin irritation; methacrylates, a nicotine patch component, can induce CD as well [5, 6].

Early reports hypothesized that CD and psoriasis could not occur simultaneously due to their seemingly contradictory mechanisms of action: CD causes increased cell apoptosis, in stark contrast to the poor cell turnover seen in psoriasis [7, 8]. Newer studies have shown that CD and psoriasis do exist together and CD may induce psoriasis through an isomorphic response as well as through the contact allergen itself [9, 10]. Other provoking factors include physical trauma, burns, friction, insect bites, surgical incision, radiation exposure, medications, needle acupuncture, and tattoos [11]. Finally, the risk of CD in patients with psoriasis may be allergen-specific and often will appear later due to the delay of migration of antigen-presenting cells in psoriasis patients [8, 12].

CD relies heavily on clinical history for diagnosis; this case demonstrates that history can be misleading. The time of onset and the distribution of the rash was highly concerning for CD. However, after poor response to traditional CD therapies, and without confirmatory findings on patch testing, the biopsy ultimately confirmed the diagnosis of psoriasis. In retrospect, it is likely that the preceding presumed Streptococcal infection was the primary inducer of guttate psoriasis and the concomitant friction irritation of the transdermal patch was a secondary point of inflammatory response.

Statement of Ethics

Patient consent was obtained and granted. This report does not include any animal experiments.

Disclosure Statement

The authors have no conflicts of interests to declare.

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Author Contributions

Both authors were extensively involved in data collection, research, and writing of the manuscript.

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