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Delayed hypersensitivity reaction to intralesional triamcinolone acetonide following treatment for alopecia areata. Intradermal testing.

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Abstract

Background: Hypersensitivity reactions to intralesional corticosteroids are very rare and have been infrequently reported. Patch testing is considered the gold standard for diagnosing contact allergic dermatitis. However, intradermal testing is thought to be more accurate and sensitive in selected cases.

Main observation: We describe a case of a delayed hypersensitivity reaction to intralesional triamcinolone acetonide following the treatment of alopecia areata. Patch testing to triamcinolone was negative but intradermal testing with a small volume of the same reagent elicited a strong reaction.

Conclusions: Patch testing may be unsuccessful in detecting delayed reactions to intralesional corticosteroids. In such cases intradermal testing at a non-cosmetically sensitive site may be a useful diagnostic tool. (*J Dermatol Case Rep.* 2015; 9(4): 107-109)

Introduction

Corticosteroids are widely prescribed by dermatologists for their potent antiinflammatory properties. Despite their use in the treatment of various allergic and inflammatory diseases, they are capable of inducing hypersensitivity reactions. Allergic contact dermatitis to topical corticosteroids is common and well established with a prevalence of 0.2% to 5%. However, cutaneous sensitisation to systemic or intralesional corticosteroids have rarely been reported. Patch testing is usually the method of choice in detecting contact allergy to corticosteroids, but in selected cases with a high index of suspicion intradermal testing can be a useful adjunctive tool.

Case Report

A 35-year-old woman presented to our dermatology department in January 2014 with a recurrence of alopecia areata at the occipital scalp. She had a history of recurrent alo-

pecia areata since childhood, treated successfully with multiple courses of intralesional traimcinolone acetonide 10 mg/ml (Kenalog10) delivered by dermojet. She had received courses of intralesional triamcinolone acetonide in 1993, 1996, 2004 and 2007. Each course consisted of a total of 6 treatments given fortnightly. The same brand, strength and dose of triamcinolone acetonide was used in all courses. Initial treatment with topical clobetasone propionate 0.05% (Dermovate) been unsuccessful. She was otherwise well with no history of contact or atopic eczema.

Three days after receiving the first 3 ml of triamcinolone acetonide 10 mg/ml (Kenalog10), she developed tender itchy scaly nodules at all of the treated sites (Fig. 1). Treatment with clobetasone dipropionate 0.05% led to a slow resolution over 7 days. Partial hair growth was noticed three months following this inflammatory reaction.

Patch testing was performed to the european standard battery, medicaments, corticosteroids and cosmetic battery. A weak positive reaction 1+ to balsam of peru and a 1+ to budesonide 0.1% in petrolatum was observed at 96 hours. The remainder of the patch tests was negative, including

triamcinolone 1.0% in petrolatum, tixocortol pivalate 1.0% in petrolatum, and benzyl alcohol 1.0% in petrolatum, the preservative in the suspension. Due to a high suspicion of triamcinolone allergy we proceeded with an intradermal test using 0.25 ml of the same brand of triamcinolone (Kenalog10), "as is", injected intradermally at the inner aspect of the left medial upper arm. This elicited a strong positive re-

action at 72 hours with erythema, oedema, and itching. Wheal reaction measured more than 15 mm (Fig. 2). Intradermal testing methods sometimes include a control injection. The value of a normal saline injection is debatable. If available, an injection of the steroid injection vehicle without the active drug can be administered to control for allergy to preservatives and other components. However, this is usually unavailable.



Figure 1Delayed hypersensitivity reaction to intralesional triamcinolone acetonide following treatment for alopecia areata.



Figure 2

Strong positive reaction to the intradermal test (triamcinolone injection) in the upper arm.

Discussion

Systemic administration of corticosteroids (orally, parenterally and intralesionally) are an infrequent cause of either immediate or delayed hypersensitivity reactions. Immediate reactions can be manifested clinically as either urticaria or rarely as anaphylactic reactions.² Delayed reactions are commoner and present mainly as maculopapular exanthems.^{3,4}

Our patient developed a delayed hypersensitivity reaction to intralesional triamcinolone acetonide. The patch test was positive to budesonide 0.1% in petrolatum, a marker of corticosteroid allergy to triamcinolone acetonide. The patch test was negative to triamcinolone 1.0% in petrolatum, which could be a result of poor percutaneous penetration or related to the vehicle used. Tixocortol pivalate, a marker for hydrocortisone allergy, was negative on patch testing. Benzyl alcohol, a preservative found in corticosteroid preparations and in triamcinolone suspension, was negative on patch testing. Intradermal testing with triamcinolone acetonide, "as is", was strongly positive confirming the allergic reaction. We believe our patient became sensitised to triamcinolone acetonide following repetitive exposure.

Patch testing is considered effective and safe to screen for delayed hypersensitivity reaction to topical and systemic corticosteroids. Tixocortol pivalate is considered a marker for hydrocortisone type corticosteroid, while budesonide is a marker for triamcinolone type. Together, these markers can detect up to 90% of all corticosteroid hypersensitivities.⁵ The concentration and the vehicle used in patch testing have been controversial. Most studies have recommended testing corticosteroid at a concentration of 1.0% and petrolatum or ethanol as the vehicle.⁶ However, ethanol has been shown to be a better vehicle to detect allergy to corticosteroids than petrolatum.^{7,8}

The antiinflammatory properties of the corticosteroids, the concentration used and inadequate cutaneous penetration can lead to false negative results in patch testing. Various studies in the literature showed that hypersensitivity to corticosteroids might be missed by patch testing when used as the lone investigating test and that intradermal testing was necessary to complement the patch test. In 1993, Herbst *et al.* reviewed the literature for studies employing both patch and intradermal test for evaluation of contact hypersensitivity to corticosteroids. They found that intradermal testing was more sensitive than patch testing. In 1997, Seurkeran *et al.* found that 30% of hydrocortisone butyrate reactions were missed by patch testing and intradermal testing was able to detect further allergies to different corticosteroids of which triamcinolone acetonide was one of them. In 2011, Soria *et al.*

compared test results obtained with patch, prick and intradermal testing. Intradermal testing was able to detect few additional allergies.⁸

Brancaccio and Zappi reported a case of localised delayed reaction following intralesional injection into a keloid scar. The patient had positive patch test to budesonide 0.1% in petrolatum but a negative test to triamcinolone acetonide 1.0% in petrolatum. The intradermal testing was strongly positive.¹¹

Atrophy, provoking a significant allergic reaction, and rarely anaphylaxis are all potential risks of intradermal testing. However, it might be necessary in particular cases to confirm the allergic reaction especially when the offending corticosteroid has been of benefit to the patient such as in our case.

Conclusions

Patch test may not be always successful in detecting hypersensitivity reactions to intralesional corticosteroids. Intradermal testing is less safe but more sensitive and can detect further allergies. Although there are no standardised protocols for intradermal testing in the literature, it is sometimes necessary to proceed in selected cases. In these cases a small volume of the test reagent should be used at a noncosmetically sensitive site.

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