Delayed Type Hypersensitivity to Intralesional Triamcinolone Acetonide

Ronald R. Brancaccio, MD, New York, New York
Eugene G. Zappi, MD, New York, New York

Corticosteroids are the most widely used class of drugs in dermatology. In the past, allergic contact dermatitis to topical corticosteriods was rarely reported. In this article, we present a case of delayed type hypersensitivity to triamcinolone acetonide.

Corticosteroids are the most widely used class of drugs in dermatology. Although allergic contact dermatitis had seldom been reported in the past, recent studies in this decade have demonstrated that contact allergy to topically applied corticosteroids is common.[1,2] However, sensitization to corticosteroids administered orally, parenterally, or intralesionally has been reported infrequently.[3] Allergic reactions to intralesional triamcinolone acetonide suspension have rarely been reported.[4] We present a case of delayed type hypersensitivity to triamcinolone acetonide used intralesionally for the treatment of a scar.

Case Report:

An 85-year-old woman developed a painful hypertrophic scar on her right index finger following excisional surgery of a squamous cell carcinoma. Three days after an intralesional injection of triamcinolone acetonide 10 mg/ml (Kenalog-10 injection, Apothecon®, Bristol Meyers Squibb, Princeton, NJ) a tender, firm, indurated, erythematous plaque developed at the injection site. A diagnosis of cellulitis was made and treatment was initiated with oral antibiotics. One month later, Kenalog-10 was again injected into the scar, and her finger became erythematous and edematous in 24 hours (Figure 1). Oral antibiotics were reinstituted, but at this point, an allergic reaction to the injection material was considered.

The patient's past medical history is important for lack of any other significant dermatologic disorders or use of topical or systemic corticosteroids.

From the Ronald O. Perelman Department of Dermatology, New York University School of Medicine, New York, New York. REPRINT REQUESTS to 7901 Fourth Avenue, Brooklyn, New York 11209 (Dr. Brancaccio).

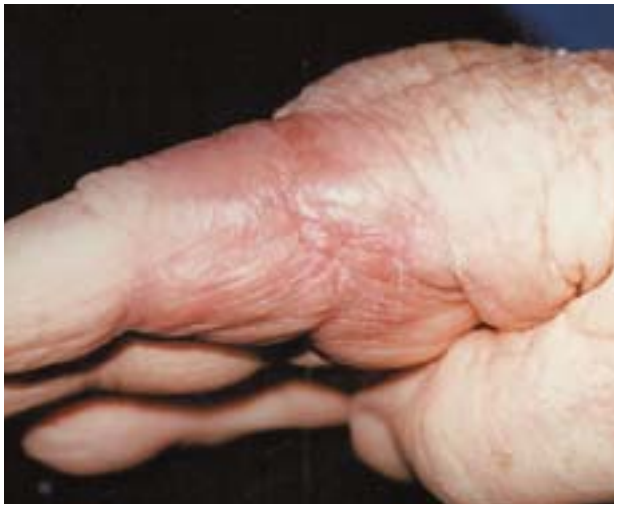

FIGURE 1. Appearance of injection site 24 hours after intralesional triamcinolone acetonide.

Patch testing was performed to our standard screening series of 43 allergens and selected corticosteroids. Strongly positive 3+ spreading reactions were noted at 48 hours and 1 week to budesonide 0.1% in petrolatum and amcinonide 0.1% cream, and a weak 1+ positive reaction was observed to Kenalog-10, as is. The remainder of the tests were negative, including triamcinolone acetonide 1.0% in petrolatum, tixocortol pivolate 1.0% in petrolatum, and benzyl alcohol 1.0% in petrolatum, the preservative in the Kenalog-10 injection. An intradermal test to Kenalog-10, as is, was strongly positive at 72 hours (Figure 2). Scratch tests to Kenalog-10, as is, and pure triamcinolone ointment 1%, were weakly positive. A scratch test to benzyl alcohol was negative.

Comments

Systemically administered corticosteroids are an infrequent cause of either immediate or delayed allergic reactions, but do occur.[5] Our patient exhibited a delayed type hypersensitivity reaction to the intralesional injection of triamcinolone acetonide suspen-

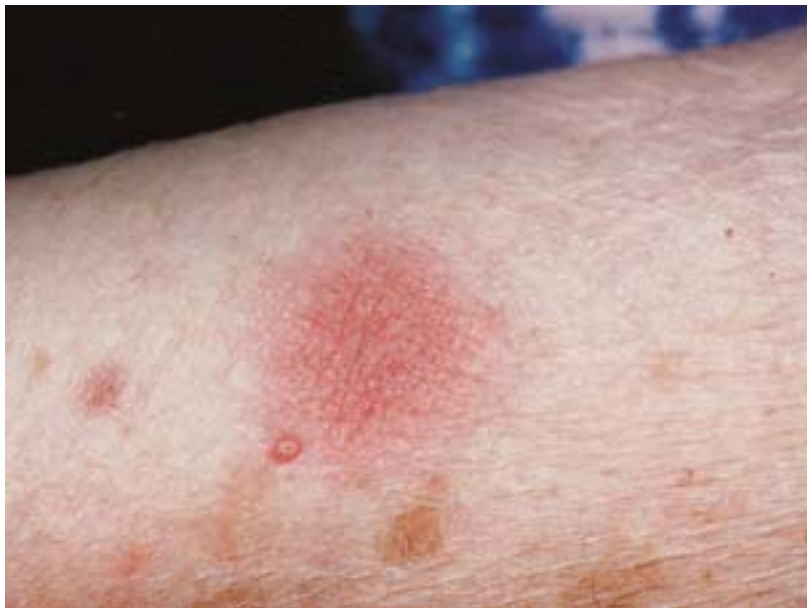

FIGURE 2. Positive intradermal test to triamcinolone acetonide at 72 hours.

sion (Kenalog-10) on two separate occasions. Intradermal testing to Kenalog-10 was strongly positive, as were scratch tests to Kenalog-10 and triamcinolone ointment 1.0%. Our patient was patch test-positive to Kenalog-10, as is, but negative to triamcinolone ointment 1.0%, which is probably due to differences in percutaneous absorption. A strong reaction to budesonide, a marker for corticosteroid allergy, suggests allergy to Class B corticosteroids of Coopman *et al.*,[6] of which triamcinolone acetonide and amcinonide are included. Tixocortal pivolate, a marker for hydrocortisone sensitivity, was negative on patch testing. The patient denied the use of topical corticosteroids, and her only known exposures to steroids were the intralesional injections of Kenalog-10.

Overall, allergic reactions to triamcinolone acetonide are uncommon. Topical sensitization occurs less frequently to this agent than to others.[7] Only 3.6% of 83 patients who were allergic to topical hydrocortisone cross-reacted with triamcinolone acetonide.[8]

A 1995 review of delayed systemic allergic reactions to corticosteroids noted that 24 cases had been published in the literature.[3] In two-thirds of these cases, the diagnoses were supported by positive patch or intradermal testing. The majority reacted to prednisone, prednisolone, or dexamethasone, but one patient developed a generalized papulovesicular reaction to oral triamcinolone acetonide.

There have been several reports of local reactions to the intralesional injections of triamcinolone acetonide.[9] One case that also occurred during scar treatment had positive intradermal tests to triamcinolone acetonide powder 1.0% in saline as well as to Kenalog-10, as is.[4] This patient also had negative patch tests to triamcinolone acetonide 1.0% in petrolatum, but had a positive reaction to this compound in alcohol. Positive patch-test reactions were present to tixocortol pivolate and budesonide, but not to the components of Kenalog-10. Fisher[10] reported a patient with an allergic hypersensitivity to intralesional steroids. Erythema multiforme-like lesions have been described following an intra-articular injection of triamcinolone acetonide into an arthritic knee,[11] and after oral triamcinolone for treatment of a dermatitis.[12]

Anaphylaxis has been reported with triamcinolone acetonide used intradermally for treatment of alopecia areata.[13] In this case, the diagnosis was only able to be confirmed by large-dose rechallenge, since patch tests and intradermal tests were negative. Anaphylaxis has also been reported immediately after an intramuscular[14] and intra-articular injection.[15]

A case of generalized urticaria has been reported with benzyl alcohol, present as a preservative in a corticosteroid preparation.[16] In this instance, patch and prick tests with benzyl alcohol were negative, as they were in our patient, but an intradermal test was positive. Although benzyl alcohol is a rare sensitizer, it must be considered when systemic reactions to medications occur.

Patch testing is generally considered to be an adequate screening method for corticosteroid contact sensitivity,[17] although this notion has been challenged.[18] Patch testing may fail to identify reactions to intralesional or systemically administered corticosteroids. In such cases, intradermal testing may be necessary, but should only be performed with the realization of the inherent associated risks, such as atrophy, sensitization, prolonged reactions, anaphylaxis, and erythroderma.[19]

REFERENCES

1. Dooms-Goossens A, Morren M: Results of routine patch testing with corticosteroid series in 2073 patients. *Contact Derm* 26: 182-191, 1992.
2. Dooms-Goossens A, Andersen KE, Brandao FM, *et al.*: Corticosteroid contact allergy: an EECDRG multicenter study. *Contact Derm* 35: 40-44, 1996.
3. Whitmore SE: Delayed systemic allergic reactions to corticosteroids. *Contact Derm* 32: 193-198, 1995.
4. Saff DM, Taylor JS, Vidimos AT: Allergic reaction to intralesional triamcinolone acetonide: a case report. *Arch Derm* 131: 742-743, 1995.
5. Rasanen L, Hasan T: Allergy to systemic and intralesional corticosteroids. *Br J Dermatol* 128: 407-411, 1993.
6. Coopman S, De Greef H, Dooms-Goossens A: Identification of cross reaction patterns in allergic contact dermatitis from topical corticosteroids. *Br J Dermatol* 121: 27-34, 1989.
7. Bircher AJ, Levy F, Langauer S, Lepoittevin JP: Contact allergy to topical corticosteroids and systemic contact dermatitis from prednisolone with tolerance of triamcinolone. *Acta Dermato-venereologica* 75: 490-493, 1995.
8. Wilkinson SM, Hollis S, Beck MH: Reactions to other corticosteroids in patients with allergic contact dermatitis from hydrocortisone. *Br J Dermatol* 132: 766-771, 1995.
9. Karck E: Sensitivity to fluorinated steroids presenting as a delayed hypersensitivity. *Contact Derm* 6: 214-216, 1980.
10. Fisher AA. Allergic reactions to intralesional and multiple topical corticosteroids. *Cutis* 23: 708-709, 1979.
11. Valsecchi R, Reseghetti A, Leghissa P: Erythema-multiforme-like lesions from triamcinolone acetonide. *Contact Derm* 38: 362-363, 1997.
12. Stingeni L, Caraffini S, Assalve D, *et al.*: Erythema-multiforme-like contact dermatitis from budesonide. *Contact Derm* 34: 154, 1996.
13. Downs AM, Lear JT: Anaphylaxis to intradermal triamcinolone acetonide. *Arch Dermatol* 134: 1163-1164, 1998.
14. Gonzalo FE, Montugut LB, Vecna ST: Anaphylactic shock caused by triamcinolone acetonide. *Ann Pharmacother* 28: 1310, 1994.
15. Larsson L: Anaphylactic shock after i.a. administration of triamcinolone acetonide in a 35 year old female. *Scand J Rheumatol* 18: 441-442, 1989.
16. Verechen P, Birringer C, Knitelius AS, *et al.*: Sensitization to benzyl alcohol: a possible cause of corticosteroid allergy. *Contact Derm* 38: 106, 1998.
17. Boffa MJ, Wilkinson SM, Bech MH. Screening for corticosteroid contact hypersensitivity. *Contact Derm* 33: 149-151, 1995.
18. Seukeran DC, Wilkinson SM, Beck MH: Patch testing to detect corticosteroid allergy: is it adequate? *Contact Derm* 36: 127-130, 1997.
19. Wilkinson SM, Smith AG, English JSC: Erythroderma following the intradermal injection of the corticosteroid budesonide. *Contact Derm* 27: 121-122, 1992.