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### **Localized Immediate Type Hypersensitivity Reaction Following Intralesional Triamcinolone Acetonide into a Keloid**

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#### **Abstract**

Corticosteroids are rare causes of immediate type hypersensitivity reactions. We report a rare case of immediate type hypersensitivity reaction following an intralesional injection of triamcinolone acetonide into a presternal keloid. He had itching in and around the keloid along with an erythematous rash around the keloid, which had started 4 hours after the injection and had lasted for the next 18 hours. He did not have any systemic symptoms of anaphylaxis. Intra-dermal test with 1:100 (concentration of 0.4 mg/ml) dilution of the same commercial preparation of triamcinolone acetonide (Tricort) showed a positive intra-dermal test reaction to Tricort (Tricort - wheal size 12 mm, saline - wheal size 4 mm).

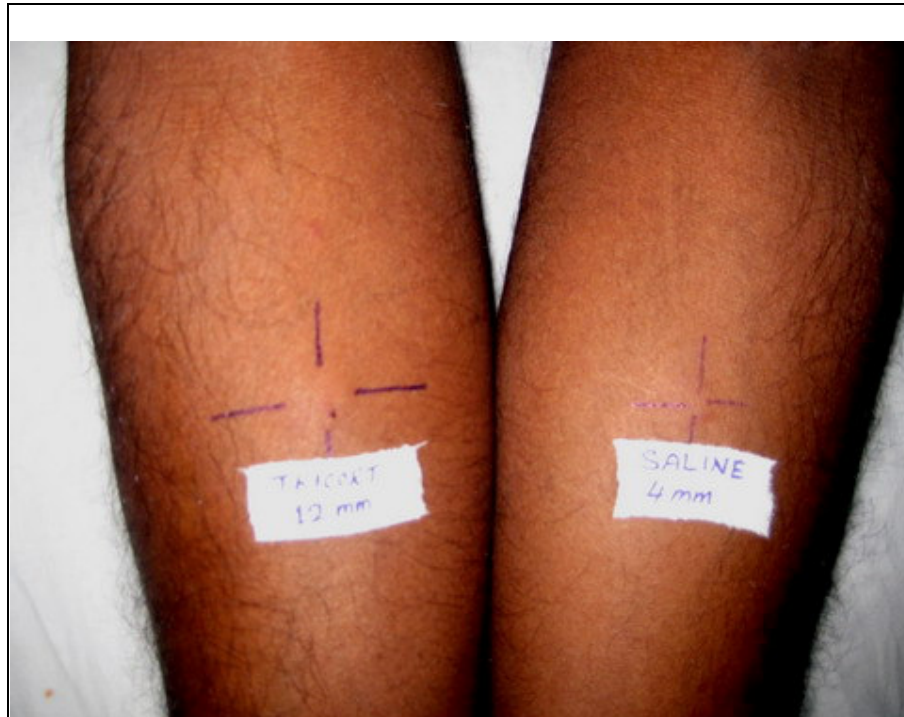
#### **Introduction**

Corticosteroids are life saving in severe immediate type hypersensitivity reactions. The same molecules are, however, capable of inducing such reactions, albeit rarely. What is important for the physician is to be able to identify such an eventuality at the right time and help prevent serious (sometimes fatal) consequences. Delayed and immediate type hypersensitivity reactions (including the most severe form, anaphylaxis) have been described due to topical and systemic

corticosteroids [1]. Here, we highlight a rare case of immediate type hypersensitivity reaction following an intra-lesional injection of triamcinolone acetonide into a presternal keloid.

### Case History

A 32-year-old male patient presented to our Dermatology outpatient department with a solitary, untreated, asymptomatic, 5X2 cm sized presternal keloid, which had began spontaneously 6 months earlier. He did not suffer from any other dermatological or systemic illnesses including asthma or other allergic disorders. He was treated with intra-lesional triamcinolone acetonide (40 mg/ml) and advised to come for a review after 3 weeks. During his next visit the patient complained that he had itching in and around the keloid along with an erythematous rash around the keloid, which had started 4 hours after the injection and had lasted for the next 18 hours (and it subsided after taking some unknown, over the counter medicines). There was no history of urticarial wheals, angioedema, breathlessness or fainting attacks during the episode. We withheld his next intra-lesional triamcinolone acetonide injection and performed an intra-dermal test with 1:100 (concentration of 0.4 mg/ml) dilution of the same commercial preparation of triamcinolone acetonide (Tricort) keeping saline as the control. Reading the test site at 20 minutes showed a positive intra-dermal test reaction to Tricort (Tricort - wheal size 12 mm, saline - wheal size 4 mm) [fig. 1]. We, therefore, concluded that the patient had immediate type hypersensitivity reaction to the triamcinolone acetonide preparation (Tricort) and stopped the intra-lesional triamcinolone therapy for him. Later, his keloid was managed with an alternative therapeutic agent.



**Fig 1:** A positive intra-dermal test reaction to Tricort.

## Discussion

Delayed type hypersensitivity reactions to topically applied corticosteroids are well known (2 and 5% of patients attending a patch test clinic) and, among topical corticosteroids, hydrocortisone tops the list [2]. Immediate type hypersensitivity reactions and anaphylaxis due to systemic corticosteroids are relatively uncommon, and, in limited reports, several systemic corticosteroids have been responsible [2]. However, there are only a handful of reports of immediate type hypersensitivity reactions due to triamcinolone, especially the intra-lesional form.

Laing et al [3] reported a case of alopecia areata developing anaphylactic reaction to intra-lesional triamcinolone acetonide due to the presence of carboxymethylcellulose used as a dispersant in corticosteroid preparations. Carboxymethylcellulose is a hydrophilic derivative of cellulose found in many food products, pharmaceuticals, cosmetics and is an active ingredient in hydrocolloid dressings [3,4]. It is responsible for anaphylactic reactions to radio-contrast media also [3]. Patterson et al [5] described a case of anaphylaxis after triamcinolone acetonide injection which was due to the carboxy-methylcellulose component of the Kenalog preparation.

Downs et al [1] reported a case of anaphylaxis after triamcinolone acetonide injection for alopecia areata and confirmed it by a re-challenge

test. Karsh et al [6] reported a case of anaphylactic reaction following intra-articular triamcinolone. Anaphylactic shock after triamcinolone acetone injections has also been described by Gonzalo et al [7] and Larsson et al [8]. De Souza and Bantick [4] reported a case of anaphylaxis reaction to intra-lesional triamcinolone acetone following injection into a presternal keloid where the patient developed the manifestations of the reaction immediately. In our case, the manifestations of the immediate hypersensitivity were in the form of itching and erythema around the keloid, which occurred only after a period of 4 hours. The high collagen content of the keloid probably did not allow enough triamcinolone to leak into the nearby dermis to elicit a stronger hypersensitivity reaction or an anaphylaxis.

It is ironic that the very molecules that are used for the treatment of hypersensitivity reactions are also capable of inducing these reactions. Reports of urticaria, angioedema, bronchospasm, worsening of asthma and anaphylaxis have been described due to oral or parenteral corticosteroids [6]. Although, the vehicle was blamed most of the time, corticosteroid molecules also have been held responsible as proven by skin prick tests, intra-dermal skin tests or a re-challenge test in some instances supplemented by RAST or a positive passive cutaneous anaphylaxis test (transfer by serum of positive reactivity to a control). Both immunologic and non-immunologic mechanisms, for example direct triggering of mast cells, have been proposed to explain the immediate-type hypersensitivity reactions to corticosteroids, but nothing is conclusively proven. It is also possible that corticosteroid molecules act as haptens, and the protein steroid complex that is most likely unknown mediates the reactions [6].

Having experienced a case of immediate hypersensitivity reaction to intra-lesional triamcinolone acetone and after reviewing the relevant literature, we have made the following observations:

- A proposed line of evaluation of patients who have experienced immediate hypersensitivity reactions to corticosteroids could be at first evaluation by the more specific but less sensitive prick test followed by intra-dermal testing if the prick tests are negative [6].
- Intra-dermal tests have a higher rate of nonspecific (false-positive) reactions, especially when higher concentrations are used, and they are more likely to induce anaphylactic-like reactions [6].
- However, with appropriate dilutions (a range of 1:10 to 1:10000 dilutions have been used [6]), intra-dermal tests are best for identifying immediate hypersensitivity reactions to corticosteroids.
- Patients should be tested with other steroid preparations to identify a safe steroid molecule for the patient.
- The patient could be sensitive to the corticosteroid molecule or the preservatives or other constituents of the liquid preparation. Testing should

be done with the steroid molecule as well as with individual constituents of the corticosteroid preparation.

- Cross-reactions may exist among different corticosteroids molecules [2].

- Resuscitation measures should be kept ready before injecting triamcinolone acetonide. An intra-dermal test with suitable dilution of triamcinolone will be helpful in identifying a sensitive subject.

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