

Anaphylaxis induced by oral methylprednisolone in a 10-year-old boy

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Abstract Corticosteroids are potent anti-inflammatory and anti-allergic agents used in the treatment of various inflammatory diseases, including allergic diseases. Allergic reactions caused by corticosteroids, however, have been reported. Among these, delayed reactions to topical steroids are more common, whereas immediate reactions to systemic steroids are rare. Herein, we report the case of a 10-year old boy with methylprednisolone-induced anaphylaxis, in which the patient had a positive oral challenge test result. Physicians should be aware of the possibility of anaphylaxis or other allergic hypersensitivity in response to corticosteroids.

Key words anaphylaxis, child, methylprednisolone.

Corticosteroids have immunosuppressive, anti-proliferative, anti-inflammatory, and anti-allergic effects, and are used in the management of numerous conditions, including malignancy, transplantation, autoimmune and allergic disease.¹ Hypersensitivity reactions to systemic corticosteroids, however, have been reported.^{1,2} Immediate reactions to corticosteroids are very rare, but could potentially be life-threatening conditions. Herein we report the case of a 10-year-old boy with anaphylaxis caused by oral methylprednisolone (MP).

Case report

A 10-year-old boy presented to the emergency department for treatment for generalized urticaria. He also had a 3 year history of allergic rhinitis. He used intermittent nasal corticosteroid spray (fluticasone furoate). Skin prick test was positive to *Dermatophagoides pteronyssinus* and *D. farinae*. He had no history of food or drug allergy. The patient had typically used a systemic steroid for relief of idiopathic generalized urticaria (<2 times/year). Four months previously, he had been treated for generalized urticaria for 2 days with methylon® tablets 4 mg (Kunwha Pharmaceutical, Seoul, Korea; MP 4 mg, lactose), and there had been no adverse reaction. His mother had a history of allergic rhinitis. He developed generalized urticaria for 2 days without respiratory or gastrointestinal symptoms. He took anti-histamine for 1 day, but cutaneous symptoms did not improve.

In the hospital, approximately 10 min after ingesting methylon® 4 mg, he complained of worsening urticaria with itching sensation, tightness of the chest, abdominal pain, nausea and dizziness, and he finally collapsed. Systolic blood pressure was 50 mmHg. A diagnosis of anaphylaxis was made. He was treated with epinephrine. All symptoms resolved within a few hours.

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To confirm the causal relationship between the symptoms and corticosteroid, 6 weeks after anaphylactic reaction, allergic skin test and oral provocation test was performed. We performed skin prick and intradermal tests (1/100, 1/10, 1) for MP sodium succinate (preparation: anhydrous disodium hydrogen phosphate, sodium phosphate, sodium hydroxide, dilute hydrochloric acid), hydrocortisone sodium succinate, dexamethasone disodium phosphate, prednisone sodium succinate, and triamcinolone acetonide. Positive intradermal response (1, wheal; 8 × 10 mm; flare, 30 × 27 mm) was observed for MP sodium succinate (Fig. 1), but not hydrocortisone sodium succinate, dexamethasone disodium phosphate, prednisone sodium succinate, or triamcinolone acetonide. To obtain a definitive diagnosis, oral MP provocation test was carried out. After each drug dose (0.5 mg, 1.0 mg, 1.5 mg, 2 mg, 4 mg) the patient's response was observed every 30 min. Five minutes after taking 2 mg oral methylon® (Kunwha Pharmaceutical), tiny reddish papules appeared on the patient's neck and trunk, and hoarseness and stridor with throat itching sensation developed. Oral provocation tests with dexamethasone® (Yuhanmedica, Seoul, Korea; dexamethasone, lactose) and solondo® (Yuhanmedica; prednisone, lactose) were negative. Provocation test with nasal corticosteroid spray (fluticasone furoate) was also negative.

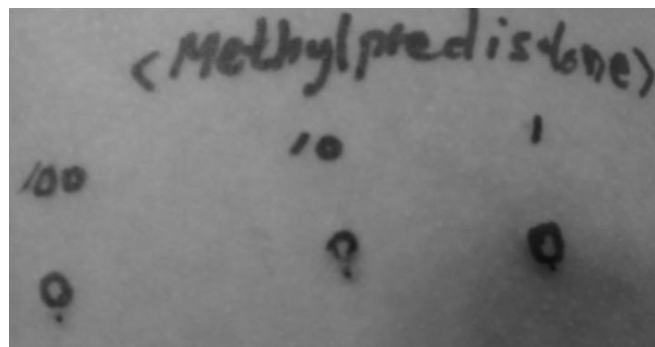


Fig. 1 Positive response to intradermal test with methylprednisolone sodium succinate (1:1).

Finally, we diagnosed the patient with MP hypersensitivity, and recommended avoidance of this drug. During the next 2 years, he did not experience recurrence of anaphylaxis.

Discussion

Although corticosteroids are widely used to treat immunological disorders, sensitization to these compounds can occur, ranging in frequency from 0.5% to 5%. Delayed allergic reactions to topical corticosteroids are frequently observed (2.9%). In contrast, immediate hypersensitivity reactions to systemic corticosteroids are very rare (0.3%).^{1,2} Signs and symptoms of immediate hypersensitivity to glucocorticoids include various manifestations, such as pruritus, rash, angioedema, sneezing, dyspnea, throat tightness, wheezing, nausea, vomiting, hypotension, and anaphylactic shock.¹⁻³ Immediate reactions are generally found following use of i.v. corticosteroid, but they have also been reported following respiratory (nasal or oral inhalation), i.m., intra-articular, and oral use.¹⁻⁴

In the present case, anaphylaxis was induced by oral MP. Allergic skin test and oral provocation test were positive. This suggests that the pathogenic mechanism is an IgE-mediated reaction. The allergenic moiety in steroids that is responsible for immediate reactions has not been determined. It could be part of the native molecule or a metabolite that acts as a hapten and binds to serum proteins, creating an allergenic complex.³ Data on risk for development of hypersensitivity reactions to corticosteroids are still lacking. Some reports, however, have suggested that renal transplant or asthmatic patients, especially those who are aspirin sensitive, may be at higher risk,⁵⁻⁷ but this could be due to the frequent use of these medications and not to the disease itself.⁸ The present patient had a history of idiopathic generalized urticaria and had been occasionally exposed to low-dose oral MP. We suggest that the sensitization route in the present case may have been through oral treatment, and even a low dose of oral MP could induce an immediate allergic reaction in exposed patients.

Some papers noted that the incidence of hydrocortisone and MP allergy seemed higher than allergy to other glucocorticoids.^{8,9} This could be related to the frequency with which it is used.

Possible cross-reactivity among hydrocortisone, MP, and prednisone has been suggested.¹⁰ The present patient had no cross-reaction to the other steroids, which was confirmed on oral provocation and allergic skin test.

Conclusion

We report a unique case of MP-induced anaphylaxis, in which the patient had a positive oral challenge test result. Clinicians should be aware of this type of hypersensitivity reaction and take it into consideration in the differential diagnosis of patients who receive systemic corticosteroid.

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Parotid lymphangioma associated with facial nerve paralysis

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Abstract Parotid lymphangioma is a relatively rare disease that is usually detected in infancy or early childhood, and which has typical features. Clinical reports of facial nerve paralysis caused by lymphangioma, however, are very rare. Usually, facial nerve paralysis in a child suggests malignancy. Here we report a very rare case of parotid lymphangioma associated

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