Allergy to Prednisolone: A Single Case of Anaphylaxis in a Teenager

Carlos Alberto Sánchez Salguero1* and Patricia Ceballos Chacón2

1Department of Pediatric Allergy Unit, University Hospital Puerto Real, Cádiz, Spain
2Department of Physiotherapy, University Hospital Puerto Real, Cádiz, Spain

Abstract

The use of corticosteroids is frequent in diverse diseases, like asthma, dermatological process and allergic diseases. Allergic reactions caused by corticosteroids are unusual but in some cases have been reported although are more frequent as secondary effects like reactions in the application of corticosteroids ointments. Herein, we report the case of a 14-year old girl with prednisolone-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.

Keywords: Anaphylaxis; Child; Prednisolone

Introduction

The properties of corticosteroids as immunosuppressive, anti-proliferative, anti-inflammatory, and anti-allergic effects are used in the management of many diseases, including malignancy, transplantation, autoimmune and allergic disease [1]. Hypersensitivity reactions to systemic corticosteroids, although are very uncommon, 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 14-year-old with anaphylaxis caused by oral prednisolone.

Case Presentation

A 14-year-old girl presented to the emergency department with complains of urticaria. She also had a 5 year history of allergic asthma and rhinoconjunctivitis. She used intermittent nasal corticosteroid spray (fluticasone furoate). Skin prick test was positive to Dermatophagoides pteronyssinus, D. farinae, Lepidoglyphus, Alternaria alternata, Olive, Lolium and Phleum pollen. In her past clinical history she had never had a reaction to drugs, but in some cases she presented reactions with some foods like fruits, Oral Allergic Syndrome with Apple and Watermelon. The patient had typically used an oral steroid for relief of idiopathic generalized urticaria (<2 times/year). Four months back, she had been treated for generalized urticaria for 2 days with Estilsona® (commercial Spanish name) drops 7 mg/ml, and there had been no adverse reaction. Her mother had a history of allergic rhinitis. She developed generalized urticaria for 2 days without respiratory or gastrointestinal symptoms. She took anti-histamine for 1 day, but cutaneous symptoms did not improve.

In the hospital, approximately 10 min after ingesting Estilsona® 21 mg, she complained of worsening urticaria with itching sensation, tightness of the chest, abdominal pain, nausea and dizziness, and she finally collapsed. Systolic blood pressure was 50 mmHg. A diagnosis of anaphylaxis was made. She was treated with epinephrine. All symptoms resolved within a few hours.

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 Prednisolone 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 Prick-Prick response was observed for Prednisolone sodium succinate (Figure 1), but not hydrocortisone sodium succinate, dexamethasone disodium phosphate, prednisone sodium succinate, or triamcinolone acetonide. To obtain a definitive diagnosis, oral Prednisolone provocation test was carried out. After each drug dose (3 mg, 6 mg, 12 mg, 21 mg) the patient’s response was observed every 30 min. Ten minutes
after taking 12 mg oral Estilsona®, 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 and deflazacort were negative. Provocation test with nasal corticosteroid spray (fluticasone furoate) was also negative. Finally, we diagnosed the patient with Prednisolone hypersensitivity, and recommended avoidance of this drug. During the next 2 years, she did not experience recurrence of anaphylaxis. She has been treated with other corticoids with no reactions.

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 hyper-sensitivity 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 follow-ing 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 Prednisolone. Allergic skin test and oral provocation test were positive. This suggests that the pathogenic mechanism is an IgE-mediated reaction. The allergic 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 allergic 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 Prednisolone. We suggest that the sensitization route in the present case may have been through oral treatment, and even a low dose of oral Prednisolone could induce an immediate allergic reaction in exposed patients. Some papers noted that the incidence of hydrocortisone and Prednisolone 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, Prednisolone, 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

Clinicians should be aware of hypersensitivity reaction to systemic steroids and take it into consideration in the differential diagnosis of patients who receive systemic corticosteroid.

References