The Egyptian Journal of Internal Medicine

CASE REPORT Open Access

Multiple corticosteroids allergy in a patient with asthma: a case report

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Abstract

Background: Allergy towards systemic corticosteroid is rare. This case report discusses the investigations to confirm diagnosis and alternative treatments.

Case presentation: A 51-year-old asthmatic woman developed severe anaphylactic reaction following administration of systemic hydrocortisone. Skin prick, intradermal, and intravenous provocation tests confirmed allergy to triamcinolone, hydrocortisone, and dexamethasone. Skin prick tests (SPTs) were negative to all the aforementioned drugs. Intradermal test (IDT) with triamcinolone 1:10 concentration resulted in a 2-mm wheal associated with rhonchi. IDT with hydrocortisone 1:10 concentration showed an 8-mm wheal with rhonchi. IDTs to dexamethasone and carboxymethylcellulose were negative. Generalized rhonchi were observed with intravenous dexamethasone full concentration challenge.

Conclusions: Corticosteroid allergy should be suspected in asthma patients with worsening bronchospasm after its administration. Due to its rarity, such diagnosis can easily be missed, resulting in increased morbidity and mortality in patients.

Keywords: Glucocorticoid, Drug hypersensitivity, Skin tests, Asthma

Key messages

Corticosteroid allergy is rare. It should be suspected in asthmatic patients with worsening bronchospasm after steroid administration. Allergic skin tests are useful to identify the allergen and suitable alternatives.

Background

Systemic corticosteroid allergy is rare with 0.1% prevalence rate [1]. The most common offending agents are hydrocortisone and methylprednisolone [2, 3]. Clinical manifestation includes urticaria, pruritus, flushing, rash, angioedema, dyspnoea, bronchospasm, stridor, hypoxemia, nausea, vomiting, abdominal pain, and hypotension. Corticosteroid allergy is difficult to recognize clinically especially if it is used to treat an allergic reaction or a condition with symptoms similar to allergy and anaphylaxis. Corticosteroid allergy occurs with various administration routes including intravenous, oral, inhaled, intramuscular, intra-articular, ocular, intralesional, and topical applications [4]. Type IV hypersensitivity reaction in the form of allergic contact dermatitis is observed mainly with topical preparations. Systemic corticosteroid allergy frequently manifests as type I hypersensitivity reactions [4]. The presence of respiratory symptoms is more indicative of true steroid allergy [5]. Risk factors for corticosteroid allergy are aspirin-sensitive patients with asthma and renal transplant [3, 6].

We report a 51-year-old woman with asthma and multiple drug allergies including diclofenac, esomeprazole, and corticosteroids. This case highlights the importance to consider corticosteroid allergy in a patient with asthma whose bronchospasm worsens after initiation of therapy.

Case presentation

A 51-year-old woman with bronchial asthma presented with an acute exacerbation secondary to stressful event.

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Her asthma has been poorly controlled on inhaled beclomethasone dipropionate/formoterol fumarate dihydrate (Foster®) and inhaled salbutamol.

She was given intravenous hydrocortisone and nebulized salbutamol. She developed generalized pruritus and worsening breathlessness 15 min later. Blood pressure was 122/65 pulse rate 100 beats per minute with oxygen saturation of 85% on high flow oxygen 15 l/min. The chest was silent on auscultation. She developed severe hypoxia leading to seizures and respiratory arrest requiring intubation and mechanical ventilation. She was treated with intravenous aminophylline, magnesium sulphate, and nebulized ipratropium without systemic corticosteroids as corticosteroid hypersensitivity was suspected. Her condition subsequently improved, and she was extubated after 3 days with no further complications. She was able to tolerate inhaled beclomethasone dipropionate/formoterol fumarate dihydrate (Foster®), inhaled fluticasone propionate/salmeterol (Seretide Accuhaler™), inhaled salbutamol, inhaled tiotropium bromide (Spiriva®), theophylline, and montelukast.

She has received multiple doses of intravenous hydrocortisone that relieved symptoms of bronchospasm in the past. However of late, she experienced pruritus around hydrocortisone intravenous access site during her asthma exacerbations. Previous exposure to diclofenac caused anaphylactic shock. She developed generalized pruritis with esomeprazole and prednisolone. All suspected drug allergies were diagnosed clinically. There was no history of food allergy or other atopy.

Skin tests were performed with hydrocortisone, triamcinolone, dexamethasone, and carboxymethylcellulose (excipient of systemic steroid preparation). Skin prick tests (SPTs) were performed using a drop of the drugs at clinical concentrations. Intradermal tests (IDTs) were performed at 1:100 followed by 1:10 concentrations [7]. Saline was used as the negative control and histamine as the positive control. Wheal diameter measuring 3 mm larger than the negative control was considered positive for SPT [7]. Wheal diameter > 8 mm or 3 mm greater than the negative control, or wheal with flare ≥ 2 times the size of the injection wheal was considered positive for IDT [7]. Intravenous challenges with 1:100, 1:10, and full strength concentrations were performed after negative SPT and IDT. SPTs were negative to all tested preparations. IDT with triamcinolone 1:10 resulted in a 2-mm wheal with rhonchi. An 8-mm wheal diameter with rhonchi was observed with hydrocortisone IDT 1:10 concentration. IDTs to dexamethasone and carboxymethylcellulose were negative. Intravenous dexamethasone provocation test was positive at full concentration with development of generalized rhonchi.

Discussion

Skin prick (SPT) and intradermal (IDT) tests are tests for suspected IgE-mediated allergic reaction. SPT is performed by pricking the skin percutaneously with a test needle through the suspected drug solution. If SPT is negative, IDTs are done with serial dilutions of the suspected drug. The dilution starts with 1/100 of the concentration in SPT. If the result is negative, concentration is increased in logarithmic steps (× 10) [7].

Corticosteroids are classified into four groups based on the chemical structure of its cyclopentanoperhydrophenanthrene nucleus. Group A consists of hydrocortisone, prednisolone, and methylprednisolone acetate; group B triamcinolone, desonide, and budesonide; and group C betamethasone, desoximethasone, and dexamethasone; group D is classified into D1—betamethasone-17-valerate, clobetasol propionate, beclomethasone dipropionate, fluticasone propionate, and mometasone furoate—and D2—methylprednisolone aceponate and hydrocortisone valerate [3, 4]. Cross-reaction within the same group is possible due to similarities in chemical structure [4]. Cross-reaction between groups has been observed at a lesser extent. The most significant cross-reaction is between group D2 with group A and budesonide [4].

Once corticosteroid allergy is suspected or confirmed, a safer alternative from another group has to be identified. Groups C and D1 have very few allergic reactions and usually do not cross-react with other groups. Ventura et al. suggested the usage of oral betamethasone sodium phosphate and deflazacort as alternatives in corticosteroid allergy [2]. However, systemic forms of both corticosteroids are not accessible in our country.

Our patient tested positive to agents from groups A, B, and C. She could tolerate inhaled agents from group D1. Agents from group D1 are safer alternatives for her, but systemic preparations are not available; thus, there are no safe corticosteroid options left. If the use of corticosteroid is mandatory for disease treatment, desensitization may be considered. It is a procedure which temporarily alters the clinical sensitivity to the corticosteroid, resulting in short-term tolerance, hence allowing patient to receive the medication safely. To our knowledge, there are only two successful cases of corticosteroid desensitization to date [8, 9]. Acute exacerbations of her asthma should be managed with measures including intravenous aminophylline, magnesium sulphate, nebulized bronchodilators, and inhaled beclomethasone dipropionate or fluticasone propionate. If she develops further episode of anaphylaxis, the first line of treatment is intramuscular adrenaline injection [10].

Conclusion

Corticosteroid hypersensitivity should be considered in asthmatic patients who deteriorate after steroid administration. Allergy skin testing is helpful to identify the culprit drug and to find a safe alternative for the patient. Finding a safe corticosteroid is particularly useful, especially in high-risk patients where steroids are a life-saving treatment.

Abbreviations

IDT: Intradermal test; SPT: Skin prick test

Acknowledgements

Not applicable.

Authors' contributions

MM acquired the patient's data from the clinical information sheet and wrote up the manuscript. WSLWAK performed the allergic skin test, reviewed and revised the manuscript. NMN reviewed and revised the manuscript. AJ supervised the allergic skin test, reviewed and revised the manuscript. All authors read and approved the final manuscript.

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Funding

The authors declare no source of funding for this article.

Availability of data and materials

Not applicable.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for the publication of this case report.

Competing interests

The authors declare that they have no competing interests.

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Received: 9 June 2020 Accepted: 29 June 2020
Published online: 25 August 2020

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