CASE REPORT

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Skin Reaction to an Asthmatic Patient on Hydrocortisone

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ABSTRACT

Steroids constitute the basic of treatment of asthma by interrupting the development of inflammation. Although being prescribed to subjects for allergies, they can also be the culprit behind allergic reactions. We report a case of a 38-year-old woman, who was prescribed hydrocortisone for severe acute asthma, and presented with an allergy to hydrocortisone in the form of generalized pruritus, cutaneous mucosal lesions in the form of purpura and ecchymoses on the abdomen and limbs. The substitution of hydrocortisone led to recovery.

KEYWORDS: Pruritus, Hydrocortisone, Skin Reaction

INTRODUCTION

The inflammatory component is an essential part of asthma, whether or not it is allergic in nature. Corticosteroid therapy undoubtedly constitutes the basis of the treatment of asthmatic disease by interrupting the development of this inflammation.[1] Paradoxically, these same corticosteroids can be responsible for allergic reactions, such as anaphylaxis or delayed hypersensitivity, during an asthma attack, and can thus be life-threatening. The existence of allergic reactions to steroids has been a matter of controversy and their physiopathology remains a mystery. Immediate withdrawal of the causative agent can help in such scenarios.

CASE REPORT

A 38-year-old woman, was known to have been asthmatic for six years, suffering from severe persistent asthma and allergy to dust mites. She was hospitalized for severe acute asthma. On physical examination, there was polypnea of 28 breaths/min with diffuse wheezing in both lung fields and a peak flow of 180 L/min. The chest X-ray showed distension of the chest. The complete blood count was normal; the platelet count was 160,000 cells/µL and the eosinophil count was 180 cells/µL. The coagulation panel was normal with a normal bleeding time. Blood gas analysis revealed hypoxemia with a PaO_2 of 74 mmHg and a $PaCO_2$ of 35 mmHg. Upon admission, the patient was started on hydrocortisone and used terbutaline inhalers with no other medication. The clinical course was marked, immediately after injection of the hydrocortisone, by the appearance of generalized pruritus associated with purpura and ecchymoses in the upper and lower limbs and on the front of the abdomen. The diagnosis of allergy to hydrocortisone was suggested. Allergy to food or to another drug was ruled out by the interview. In addition, the patient reported no allergic symptoms after the injection of hydrocortisone during her previous hospitalizations. Vasculitis was ruled out given the normality of the anti-neutrophil cytoplasmic antibody assay, the immunology panel, the IgE assay, the renal panel and the skin lesion biopsies. The number of eosinophils in the complete blood count was normal after the allergic event (310/µL). The patient was started on prednisone with gradually decreasing doses. The clinical course was favorable, with a hospital stay of 29 days. During the year following this episode, she was readmitted to hospital three times for asthma attacks. These attacks progressed well on oral prednisone, with a mean hospital stay of 15 days.

DISCUSSION

Allergic reactions to corticosteroids, which may seem paradoxical, has been a little-known, underestimated and controversial concept for a long time.[2] The often-misleading clinical symptoms can be in the form of a delayed hypersensitivity reaction, appearing as a great variety of skin lesions whose immune-mediated origin is demonstrated by the positivity of patch tests, or in the form of an anaphylactic-type immediate hypersensitivity reaction (IHS).[3] The reactions are usually secondary to the administration of injectable preparations containing hydrocortisone or

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methylprednisolone.[4,5] Dexamethasone and betamethasone are rarely implicated.[6,7] The clinical symptoms, consistent with the classical symptoms of anaphylaxis, are skin reactions (pruritus, urticaria, angioedema), bronchospasm, nausea, vomiting, severe anaphylactic reactions and shock.[2] They can be isolated or combined and are of variable severity.[3] Around ten cases of bronchospasm progressing to respiratory arrest after the injection of hydrocortisone or methylprednisolone have been reported.[8] Cases of sudden death have also been described even after bolus injections of corticosteroids.[9] More rarely, these products have also been implicated when taken by nebulization[10] and inhalation.[11] The most common sign is bronchospasm in the asthmatic. The time to onset is usually short: less than five minutes.[4] The reaction is dose-dependent and is often associated with skin signs such as urticaria[12] even after an inhaler.[13] Its frequency and severity are underestimated insofar as the bronchospasm can be confused with an independent exacerbation of the asthma.[14] It is more common in patients allergic to aspirin with or without nasal polyposis.[15] The pathophysiology of anaphylactic adverse reactions to corticosteroids remains puzzling. The corticosteroid seems to behave as a hapten, binding to a carrier protein to become immunogenic.[16] Hydrocortisone, by inhibiting the release of arachidonic acid from membrane phospholipids by means of lipocortins, seems to prevent the release of prostaglandin E2 and prostacyclins. Dominance of the bronchoconstrictor eicosanoids seems to ensue.[17]

The additives or excipients of the preparation are also implicated, especially sulfites.[18] IHS reactions appear to be sometimes related to these products. This is the case for dexamethasone and betamethasone.[3] The mechanisms of sulfite intolerance are still poorly understood; a IgE-dependent reaction may be the cause. Diagnostic confirmation can be obtained from the medical history, by assaying the released mediators (histamine, methylhistidine or serum tryptase), from the leukotriene C4 release test or basophil activation test, by screening for specific IgEs (difficult to perform), and from somewhat unreliable skin tests (prick or intradermal tests).[12,15] Treatment is based on the immediate discontinuation of the injected product and its elimination, as well as on antihistamines, epinephrine in the event of shock, and symptomatic treatment. Some authors suggest only using oral corticosteroid therapy during an asthma attack, particularly in atopic subjects, as the oral administration of these products is less likely to cause an IHS reaction.

CONCLUSION

Corticosteroids can cause allergic adverse reactions, the mechanisms of which remain controversial. Given the frequency of use of these drugs, these reactions are exceptional, but the possibility of the bronchospasm worsening after the systemic administration of these products should be remembered.

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