

Anaphylaxis after prednisone

ANAPHYLACTIC reactions to corticosteroids are relatively uncommon (1, 2). These reactions may be immunologic or nonimmunologic in nature, and the clinical signs of anaphylaxis may be seen with both types of reaction. It is somewhat paradoxical that on rare occasions steroids themselves can induce systemic adverse reactions. We describe here the case of a girl with asthma and aspirin intolerance who developed severe systemic symptoms and loss of consciousness after a single dose of 25 mg prednisone.

■ A 17-year-old girl with intrinsic asthma and aspirin intolerance was seen in the outpatient clinic with acute exacerbation of her symptoms for which she was taking inhaled salbutamol p.r.n. and beclomethasone dipropionate 500 µg b.i.d. For the last 2 years, she had had only occasional exacerbation of her asthma, which resolved with standard nebulized therapy. She had received oral and i.v. steroids on different occasions. On auscultation of her chest, wheezing was noted. She was prescribed a short course of oral prednisone (25 mg/day) for better control of her symptoms. Forty minutes after prednisone, she experienced an anaphylactic episode with generalized flushing and hives. This was accompanied by hypogastric pain and abdominal cramps and was followed by vomiting and diarrhea. The patient lost consciousness and arterial hypotension occurred. On her admission to hospital, symptoms were promptly reversed with standard shock therapy and i.v. administration of diphenhydramine and hydrocortisone. She was advised to avoid systemic steroids and instructed in the use of autoinjectors of epinephrine.

■ Complete blood count, serum IgE level, and C4 and CH50 values were normal. Intradermal skin testing with prednisone (from 0.5 µg to 50 mg) yielded a 3+ reaction at 500 µg. Three control subjects did not show any reaction to intradermal prednisone. The results of intradermal skin testing with methylprednisolone and hydrocortisone were negative. Oral challenges with increasing doses of prednisone were performed. Twenty minutes after ingestion of 10 mg prednisone, the patient experienced flushing, nausea, dizziness, tachycardia, and hypotension. She recovered rapidly after i.v. administration of diphenhydramine and hydrocortisone. Additional challenges were performed with increasing doses of methylprednisolone and hydrocortisone administered i.v. No reactions were

observed. She was advised to take methylprednisolone for acute exacerbation of her asthma.

■ This case emphasizes the dangers of steroid administration, especially in patients with a history of drug allergy. It is known that i.v. therapy with hydrocortisone and methylprednisolone is sometimes associated with adverse reactions, whereas prednisone, betamethasone, and dexamethasone are reported less frequently as causative agents (1-3). A complete search of the literature in English by MEDLINE did not locate any other reports of acute anaphylaxis induced by oral prednisone. Considering that bronchial asthma is a rather common illness and that oral prednisone is widely prescribed for acute exacerbation of asthma, it is surprising that allergic reactions to prednisone have not been more frequently reported in the literature. A possible explanation is that either the treating physicians have not been aware of the worsening of asthma (which may be mild in most cases) in their patients on oral steroids, or the adverse reaction described is a very rare one indeed. However, reactions to prednisone probably occur more often than we realize. In

*A rare case of
bronchial asthma in a
patient who developed
an anaphylactic
reaction to oral
prednisone.*

these circumstances, suspension of the offending drug and replacement with an alternative one is the logical approach. Since the patient needed steroid therapy for the management of the acute exacerbation of her asthma, it was very important to determine which cortico-

steroid could be tolerated by her. It is not advisable simply to rely on the results of intradermal skin testing in order to exclude sensitivity to corticosteroids, since positive wheal and flare responses may not always be present in susceptible patients (1, 2). Therefore, we also performed challenges with i.v. methylprednisolone and hydrocortisone. This case indicates that systemic corticosteroids should be used with caution in asthmatics with a history of drug intolerance. Indeed, corticosteroid-induced adverse effects are more common in patients with aspirin intolerance (4, 5).

Our patient had a significant skin reaction to intradermal prednisone, whereas methylprednisolone and hydrocortisone were negative, and the specificity of this reaction was confirmed by the results of the oral and i.v. challenges with prednisone, methylprednisolone, and hydrocortisone. It

is possible that IgE sensitization to oral prednisone might have occurred in this girl. Although we did not measure IgE antibody specific to prednisone, the positive results of intradermal skin testing suggest that this case of acute anaphylaxis may be the result of either an IgE-mediated response or direct release of vasoactive mediators from mast cells or circulating basophils.

Key words: anaphylaxis; prednisone.

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Accepted for publication 8 October 1997

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ALLERGY 1998;53:330-331

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Syndromes associated with eosinopenia

EOSINOPHILIA is a relatively common finding that is associated with allergy, helminth infections, neoplasia, and the hypereosinophilic syndromes (1). In contrast, eosinopenia is held to be an uncommon abnormality that is associated with certain bacterial and viral infections, with corticosteroids and adrenergic agents, and with stress. Herein is described a patient with eosinopenia, urticaria, drug allergy, IgA deficiency, and anti-IgA autoantibodies in both the IgG and IgE classes. This patient and cases from the literature (MEDLINE through 1996) suggest that, in addition to the conditions noted above, eosinopenia may also be associated with asthma, allergic reactions, urticaria, immunoglobulin deficiencies, and autoimmune phenomena.

The patient was a 31-year-old woman with a 9-month history of diffuse urticaria, perioral and

periorbital angioedema, pruritus, and an episode of bronchospasm. She had received several courses of prednisone, hydroxyzine, and cimetidine in the past. Prior laboratory studies included normal thyroid profile and RPR, positive streptozyme test (1:100), negative throat culture, and leukocytosis of 22 000/mm³ with neutrophilia, possibly during corticosteroid therapy. Past history

In the study of a patient with urticaria and drug allergy, a literature review revealed that eosinopenia may be associated with asthma, allergic reactions, urticaria, immunoglobulin deficiencies, and autoimmunity.

included silicone breast implants, a cesarean section, an abnormal Pap smear, recurrent urinary tract infections with vaginitis, and allergy to sulfa characterized by rash and nausea. A complete blood count off prednisone for 2 weeks showed no peripheral eosinophils. The sedimentation rate, ANA, rheumatoid factor, and total complement were within normal limits. Tuberculin and coccidioidin skin tests were negative, as

was a self-requested HIV antibody test.

Two years later, she returned, still complaining of hives. She had been treated with diphenhydramine and one course of prednisone, which, upon withdrawal, resulted in transient arthralgias and myalgia. White blood cell counts while the patient was off steroids were 11 380/mm³ and 10 530/mm³, and the differential counts showed neither eosinophils nor basophils. Serum protein electrophoresis and quantitative IgG subclasses were normal. The total serum IgE was 2 IU/ml (normal=>0.5 IU/ml). Serum IgA was <6.7 mg/dl (normal=70-312 mg/dl) and, repeated at another laboratory, was <30 mg/dl (normal=40-350 mg/dl). Anti-IgA antibodies of the IgG class were elevated at 4.8 standard deviations above the norm (normal=<2.0 SD), and those of the IgE class were elevated at 3.49 ng/ml (normal=<1.0 ng/ml). IgM anti-IgA antibodies were not elevated at <2.0 SD.

At follow-up 1 year later, both the serum and salivary IgA levels were very low and the serum IgA1 and IgA2 subclasses were undetectable. The serum C3, C4, and factor B were normal. The Raji cell test for circulating immune complexes was 122 µg Eq/ml (normal=10-28 µg Eq/ml). The

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