

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.

R. POLOSA*, G. PROSPERINI, L. PINTALDI, J.-P. REY, R. COLOMBRITA

*University Medicine

Southampton General Hospital

Southampton SO16 6PD

UK

Tel. 0044 1703 796960

Fax: 0044 1703 701771

E-mail: r.polosa@soton.ac.uk

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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

Table 1. Characteristics of eosinopenic patients

Patient no. Age (years) sex (Reference)	Eosinophils	Basophils	Immunoglobulins	Associated disorder(s)
1 28 F (2)	PS=0 BM=0		G Glob=25%	Urticaria Drug fever Hyperthyroidism Cholecystitis Asthma
2 74 F (3)	PS=0 BM=0	PS=0	G Glob=1.36 g/l	
3 52 M (4)	PS=0 TEC=0 BM=0 SP=0	PS=0 TBC=0 BM=0 SP=0	IgA=0.03–0.05 g/l IgE < 1 U/ml	Asthma Acquired hemolytic anemia Alopecia totalis Multiple infections
4 45 F (5)	PS=0 BM=0 NS=0	PS=0.4%	IgA=normal IgE=45–78 IU/ml (normal 5–237)	Goiter Urticaria Rhinitis
5 59 F (6)	PS=0 BM=0	PS=normal	IgG=normal IgA=normal IgM=normal IgE=normal	Granulocytopenic after Nembutal Goiter Toxic hepatitis Asthma
6 52 F (7)	PS=0 BC=0	BC=normal		Type II diabetes Seasonal rhinitis Penicillin allergy Urticaria Vitiligo
7 71 F (8)	PS=0 BM=0	PS=0 BM=0	IgE < 1 IU/ml	Congenital left kidney Thyroid adenoma ASA sensitivity
8 31/F (present report)	PS=0	PS=0	IgA < 6.7 mg/dl (normal 70–312) Salivary IgA=1.0 µg/ml (normal 10–78)	Urticaria Angioedema Urinary tract infections Sulfa allergy

BC: buffy coat; BM: bone marrow; NS: nasal smear; PS: peripheral blood smear; SP: sputum; TBC: total basophil count; TEC: total eosinophil count.

patient's parents were reported to have normal serum IgA levels, while her 11-year-old son had a low serum IgA at 7 mg/ml and a normal IgE a 8 IU/ml.

In summary, the patient was a 31-year-old woman with eosinopenia, basopenia, urticaria, and angioedema, one episode of bronchospasm, sulfa allergy, circulating immune complexes, IgA deficiency, and anti-IgA autoantibodies of both the IgG and IgE classes.

This review includes seven cases from the literature plus the case reported here (Table 1). Seven of eight cases were those of women. All eight patients lacked eosinophils on the peripheral blood smear. The bone marrow also was devoid of these cells in all six examined (nos. 1–5, no. 7). In three of these cases (nos. 2, 4, 5), no recognizable eosinophil precursors were observed in the bone marrow. Basophils were absent in four cases (nos. 2, 3, 7, 8). Complement was found to be normal in all five cases in which it was assayed, while immunoglobulins were deficient in three of the six patients examined (nos. 3, 7, 8).

The corticosteroid treatment history of these patients is relevant because of the known eosinopenic effect of these hormones (1). Seven of the eight patients were not recorded as receiving systemic corticosteroids when eosinophils were sought, while the treatment status of one is uncertain.

The mechanism of eosinopenia was explored in five of the eight cases. Serum antieosinophil activity was found in three cases (nos. 3, 4, 6), but not in two (nos. 2, 5). This activity required complement (nos. 4, 6) and was blocked by normal serum (no. 3). The activity was absorbed by rabbit anti-human IgG, but not anti-IgM antibody. Additionally, eosinophil colonies could be cultured *in vitro* from the peripheral blood and bone marrow of one patient (no. 6) after the cells were washed free of homologous serum.

The clinical associates of eosinopenia in the eight cases summarized here (Table 1) include allergy, asthma, drug allergy, urticaria, immunoglobulin deficiencies, and autoimmune phenomena. The last

occurred in two cases; autoimmune hemolytic anemia plus alopecia totalis in one case (no. 3) and IgG and IgE anti-IgA autoantibodies in the other (no. 8). In addition, thyroid disease occurred in three cases (nos. 4, 5, 7), and urticaria in four cases (nos. 1, 4, 7, 8). However, these last two syndromes may be either immunologic or nonimmunologic in origin. Relevant also is a paper describing eosinopenia occurring in five patients with a combination of thymoma and hypogammaglobulinemia, but not in other patients with either condition alone (9).

There are, as yet, insufficient data to establish a significant association between eosinopenia and the various syndromes described in the cases summarized here. Instead, this report and review are presented to stimulate additional studies of the clinical associates of eosinopenia.

Key words: autoimmune diseases; basophils; eosinopenia; IgA deficiency.

G. L. FREEMAN
Department of Internal Medicine
Allergy Section
Cigna Healthcare of Arizona
755 East McDowell Road
Phoenix, Arizona 85006
USA
Tel. (602) 271-5235
Fax: (602) 271-5296

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Conjunctivitis to thimerosal mistaken as hay fever

IN patients whose symptoms disappear during their vacation, even experienced allergologists would first think of an occupational origin. In the following case, we show that even with a clear personal history there might be pitfalls in diagnosis we have to be aware of.

■ A 26-year-old man presented with a 2-year history of repeated episodes of dermatitis, swelling of his eyelids, and "burning eyes". He had been treated with various antibiotic-containing eye-drops. When his ophthalmologist diagnosed epidemic conjunctivitis 18 months before, he received i.v. treatment with rocephine for 2 weeks. After that therapy, the symptoms completely resolved. Six months later, he suffered from the same symptoms and was treated again with i.v. rocephine and local thimerosal-containing eye-drops. The symptoms only partly resolved. At that time, the patient stopped working at a milling cutter, and his symptoms completely disappeared. Therefore, he thought he had an occupationally related illness. Nevertheless, 4 weeks later, after starting a different job at the same company, he suffered redness of the eyes and conjunctivitis within days. Finally, this led to an allergologic checkup, as suggested by his insurance company, on suspicion of an occupational allergy.

■ Skin prick tests (SPT) with routine inhalants were all negative. Patch tests were performed with

An apparent case of occupational allergy turned out to be one of contact allergy to thimerosal.

the European Standard Series, a disinfection and preservative series, a base preparation and emollient series, a drug series, a mechanical workers' series, and different thimerosal-containing eye-drops, using Finn chambers on

Scanpore fixed to the back for 48 h. Test sites were examined after 48, 72, and 96 h. Delayed-type hypersensitivities could be found for *p*-phenylenediamine (PPD), thimerosal, phenylmercuric borate, benzoyl peroxide, and thimerosal-containing eye-drops, which the patient used most. Therefore, this delayed-type contact allergy was the cause of the conjunctivitis and the eyelid eczema, as the eye

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