

Effect of corticosteroids on seasonal increases in IgE antibody

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We analyzed seasonal changes in IgE antibodies to ragweed antigens in 28 patients with ragweed hay fever during the 1972 pollination season. Thirteen patients were treated with an injection of 60 mg. of triamcinolone acetonide suspension and 16 mg. of methylprednisolone orally on 3 successive days shortly after the onset of their symptoms in late August. Fifteen patients were untreated and served as controls. Serum samples were obtained in July (control group) or just prior to steroid treatment in August, and from both groups in mid-September and finally in mid-October. Initially, IgE antibody levels did not differ between the groups. IgE antibody increased after pollen exposure in both groups, but there was no significant difference in the increases. In contrast, symptom scores were significantly less in patients treated with steroids ($P < 0.01$). The results indicate that corticosteroid treatment does not prevent the seasonal increase in IgE antibody after pollen exposure but does significantly reduce the severity of hay fever symptoms.

Since the original reports in 1950,^{1, 2} experience has proved that corticosteroids have a favorable effect on the symptoms of allergic rhinitis.^{3, 4} The mechanism of action, however, remains elusive.⁵ Several laboratories have shown that IgE antibody to ragweed increases during the pollination season,⁶⁻¹⁰ but we are not aware of any report concerning the effect of steroids on this phenomenon. During the 1972 ragweed pollen season, we attempted to determine whether steroids, when administered in doses that relieve symptoms, might exert an immunosuppressant effect, altering the formation of the IgE antibody.

MATERIALS AND METHODS

Patients

All patients in the study had had ragweed pollinosis for several years. The 15 controls were patients who preferred not to take steroids or who had not asked for treatment other than antihistamines. The 13 patients who were treated with steroids were patients who complained that they needed more relief than the antihistamines had afforded and were willing to enter the study and to take steroids after having been apprised of the potential hazards. The characteristics of the two groups were similar in age range, sex ratio, and duration of the hay fever (Table I). All had unequivocally positive reactions to skin tests with ragweed pollen extract. None had received immunotherapy during the 2 years before the study, and

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TABLE I. Clinical data on patients with ragweed pollinosis

	Control	Steroid-treated
Number of patients	15	13
Age (years)		
Range	18-49	17-38
Mean	33	27
Sex, male/female	11/4	9/4
Duration of hay fever (years)		
Range	5-35	4-25
Mean	19	12

21 had never received immunotherapy. The estimated degree of exposure was comparable for both groups. Each patient was asked to maintain a diary concerning symptoms and medication, and this was checked weekly. Two patients from the control group and 2 from the steroid-treated group failed to keep satisfactory records, so that data on the effect of corticosteroids pertain to 13 and 11 patients, respectively.

Treatment

At the time of first interview, each patient in the steroid-treated group was given 60 mg. of triamcinolone acetonide suspension (Kenalog) intramuscularly and 16 mg. of methylprednisolone (Medrol) orally. He was directed to take two more daily doses of 16 mg. each of methylprednisolone orally on succeeding days. Both steroid-treated and control patients were allowed to use antihistamines as needed, keeping a record of each dose.

Symptom scoring

This was patterned after the method advised by Norman, Rhyne, and Mellits¹¹ in which the severity of symptoms (sneezing, coughing, stuffy or runny nose, red itchy eyes, and asthma) during the 12 hour periods from midnight to noon and noon to midnight each day was scored on the basis of 0 to 3. A score of 1 was assigned for each 4 mg. unit of chlorpheniramine or equivalent that was used during the 12 hour period. An average score was determined for each patient over the entire season.

Blood samples for IgE antiragweed determination

The initial samples were obtained from the control group shortly prior to the pollen season, while those from the treated group were obtained at the time of their first visit, from August 10 to August 28, the mean being August 21. The second sample was obtained from each group in mid-September, and the final sample in mid-October. All specimens were stored at -20° C and analyzed in the same assay.

Assay for antiragweed-specific IgE

The content of IgE antibodies to ragweed antigen was measured by the radioallergosorbent test (RAST), performed as described elsewhere,⁶ except that crude, undialyzed extract of short ragweed obtained from Center Laboratories was coupled to cellulose. We have expressed RAST results as the ratio of counts bound divided by total counts added. These ratios, expressed as percentages, are related to the quantity of IgE antibody in the serum, but they are not linearly proportional to the amount of serum added. Because our results have been analyzed by nonparametric statistical methods, expression of the result in terms of percentage of total counts bound should not influence the comparison between the groups.

Pollen counts

These were recorded as granules per square centimeter of a slide exposed for 24 hours in a Durham sampler.

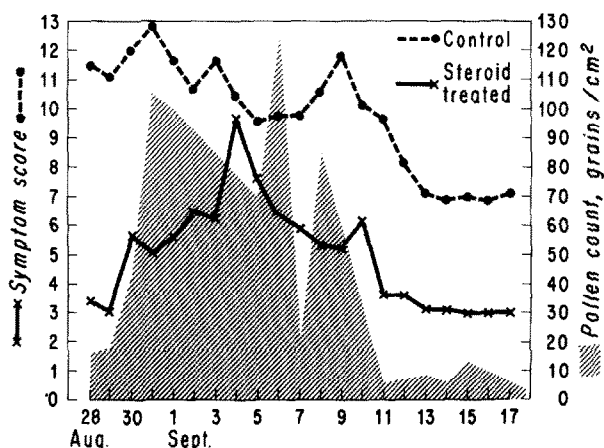


FIG. 1. Comparison of pollen counts and symptom scores for patients with ragweed pollinosis: 13 control patients; 11 steroid-treated patients.

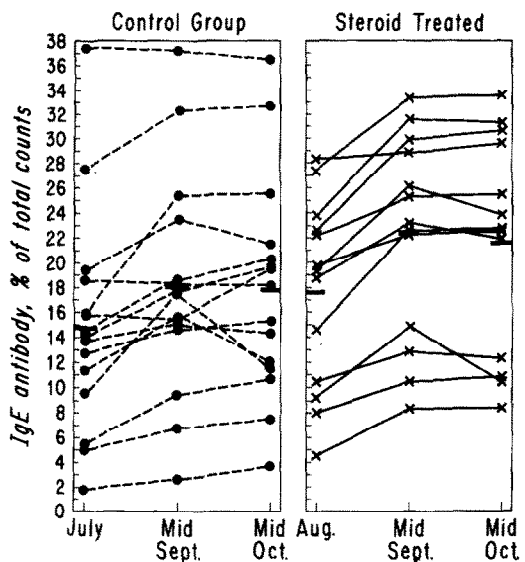


FIG. 2. IgE values in each patient (counts bound, divided by counts added, multiplied by 100). Short horizontal bars designate means. Serum from a nonallergic subject yielded value of 0.48 per cent in assay.

RESULTS

The ragweed pollen counts and the mean symptom scores for the 13 control and 11 steroid-treated patients who maintained satisfactory diaries are depicted in Fig. 1. Although the ragweed pollen season in our area begins about August 10, the graph concerns only the time after August 28 because 3 patients of the steroid group were not seen for treatment until that date. As previously mentioned, these patients were given steroids because of the severity of their symptoms, but the relief that the earlier treated patients experienced resulted in a

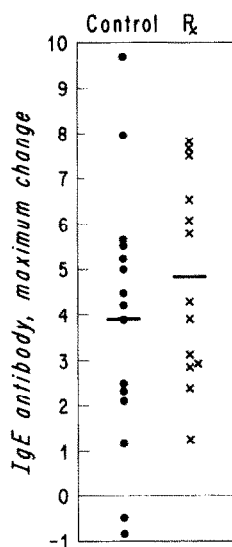


FIG. 3. Maximum changes of IgE value in each patient. These are the absolute changes in the IgE values (counts bound, divided by counts added, multiplied by 100). ●, Control patient; x, steroid-treated patient.

TABLE II. Average increase* of IgE antibody in patients with ragweed pollinosis

Increase	Control (13 patients)		Steroid-treated (11 patients)	
	No.†	%	No.	%
Maximum	3.88	26.1	4.81	27.2
September	3.17	21.4	4.60	26.1
October	3.07	20.7	4.12	23.3

*Above initial level.

†Number refers to increase of IgE antibody in terms of percentage of total counts bound. See legend to Fig. 2.

low symptom score for the group by the time of initial data collection, and this decrease persisted through the rest of the pollen season, compared to scores for the nonsteroid-treated group. A significant difference in symptoms was found between the two groups ($P < 0.01$, rank-sum test).

The values for ragweed-specific IgE were plotted as percentage of total counts (Fig. 2). Initial values for the controls ranged from 1.78 to 37.14, with a mean of 14.8. In general, the values increased by mid-September to a mean of 18, after which some continued to increase and others decreased. Interestingly, 2 control patients showed slight decreases at both mid-September and mid-October. The initial values for the steroid-treated patients ranged from 4.63 to 28.2, with a mean of 17.6. Subsequent changes were similar to those of the control group, with an increase in mid-September to a mean of 22, then further increases in some patients and decreases in others, resulting in a slight decrease in mean value. The initial levels for the control and the steroid-treated groups did not differ significantly (rank-sum test). Fig. 3 depicts the maximum change in ragweed-specific IgE for each patient; this change sometimes occurred in

mid-September and at other times in mid-October. The ranges are comparable, and no difference was found between the increases (rank-sum test). A comparison of the changes in IgE levels as a percentage of the initial values revealed no significant difference between the two groups in the maximum increase or the mid-September or mid-October values (Table II).

DISCUSSION

While we do not recommend the routine use of corticosteroids for treatment of allergic rhinitis, our experience has shown that they are most useful in patients with resistant pollinosis. Some of these are patients who have had poor results with all other therapy including hyposensitization. Others, as in the present group, are persons who were seen for the first time with severe symptoms during the season and who had not been receiving hyposensitization. We have used many different forms and dosages of steroids, and many of the patients have been adequately relieved by appreciably smaller doses than we used in our study. For uniformity in our investigation, however, we chose a substantial dose that, in our experience, had seldom failed to afford relief and would be most likely to have an effect on IgE antibody production.

Apparently, steroids have little effect on the immediate-type skin tests,¹² and Claman¹³ recently stated that in man there are virtually no conclusive data indicating that steroids inhibit the production of circulating antibody. Johansson,¹⁴ in 1967, reported his study of total IgE protein values in 33 asthmatics, noting no difference in the 5 patients who had received steroids. In 1970, Johansson and Juhlin¹⁵ reported no effect on IgE protein levels in patients with atopic dermatitis who received prednisone orally for 2 weeks. However, Kumar and co-workers¹⁶ found, in a study of 116 children with bronchial asthma, that those who had received steroids for from one to 12 years had lower total IgE levels than did nonsteroid-treated patients. In patients whose steroid treatment was discontinued, the levels tended to increase over a period of months. In a subsequent report,¹⁷ they described a patient in whom IgE concentration, after 2 years of systemic steroid treatment, was less than 10 per cent of the value found after the first 2 months of treatment. In another patient, they found essentially no decrease in IgE after 5 months of treatment, but a marked decrease at 8 months and a further decrease at 12 months. Five other patients who were receiving steroids on alternate days showed no change after 5 months. The steroid activity in the dosage that we used probably lasted no longer than 4 to 6 weeks,¹⁸ and its lack of effect on the IgE levels would be consistent with these observations, albeit we were measuring specific, not total, IgE.

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