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Churg-Strauss syndrome is a form of serum sickness

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To the editor:

It is an interesting coincidence that the article on Churg-Strauss syndrome by Lilly et al¹ appeared in the same issue as the article on Rackemann and Colmes and the clinical characteristics of hypersensitivity (edited by Dworetzky and Cohen²), because I believe that the former³ could learn from the latter. The older immunologists were quite aware of von Pirquet's³ work on serum sickness, and they included these reactions in their "hypersensitivities." In contrast, modern thinking by allergists all but ignores immune complexes or grants them a very minor role in the production of morbidity—an unsupportable assumption fueled by the current obsession with IgE.

Churg-Strauss syndrome is probably a special form of serum sickness involving immune complexes of a special solubility, surface charge, and biological activity to give them a predilection for deposition in small vessels, producing vasculitis. Although various autoimmune antibodies might appear, they are likely to be a consequence of the primary inflammatory processes triggered by deposition of immune complexes that are composed of antibody (most likely IgG) and a foreign peptide.

The importance of recognizing the presence of a foreign peptide in the syndrome is obvious; one can search for the protein in the patient's environment and, once it is found, remove it. The likely sources of the antigen are high-exposure proteins such as foods, molds, and occupational antigens. These can be looked for by testing for high IgG antibody titers to food and mold antigens through use of ELISA. As in all hypersensitivity illnesses, the best therapy is avoidance.

To carry the speculation further: in a recent report, Ponikau et al,⁴ studying chronic sinusitis, have suggested that local eosinophilia is common in the disease, that IgE mechanisms are not involved, and that fungal elements might be causal. Given that chronic sinusitis and asthma are often comorbid conditions, it could be argued that Churg-Strauss syndrome is a variant of eosinophilic fungal sinusitis and that non-IgE mechanisms (eg, immune complexes) are involved.

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References

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1. Lilly CM, Churg A, Lazarovich M, Pauwels R, Hendeles L, Rosenwasser LJ. Asthma therapies and Churg-Strauss syndrome. *J Allergy Clin Immunol* 2002;109:S1-S20.

MEDLINE

CROSSREF

2. Dworetzky M, Cohen S, editors. *Pioneers and milestones*. Francis M. Rackemann, MD. *J Allergy Clin Immunol* 2002;109:179-84.

MEDLINE

CROSSREF

FULL TEXT

3. von Pirquet DP, Schick B. *Serum sickness*. Leipzig und Wien: Franz Deuticke; 1908.
4. Ponikau JU, Sherris DA, Kern EB, Homburger HA, Frigas E, Gaffey TA, et al. The diagnosis and incidence of allergic fungal sinusitis. *Mayo Clinic Proc* 1999;74:877-84.

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