

DISCUSSION

The most common hypersensitivity pneumonitis occurring in the farm environment is farmer's lung, which is classically caused by exposure to various thermophilic actinomycetes that grow in hay, compost, and silage.⁵ *Rhizopus* species have been recovered from site samples taken from farm barns in Pennsylvania, but it is a subdominant species in the farm environment.⁶ To our knowledge, *Rhizopus* species have not been previously reported as a cause of occupational lung disease in the farm environment. In this patient, we propose that the tractor air-conditioning system became contaminated with *Rhizopus* species from the surrounding environment. Once contaminated, the enclosed-cab tractor provided an ideal closed system for circulation of high concentrations of airborne fungi, similar to the environment encountered by Swedish sawmill workers.

This case illustrates the value of a site visit in the diagnosis of occupational lung disease. Contami-

nated ventilating systems in enclosed-cab farm equipment should be considered as sources of unusual antigens in cases of farmer's lung.

REFERENCES

1. Belin KWL. Recognition of allergic alveolitis in the trimming department of a Swedish sawmill. *Eur J Respir Dis* 1980;61(Suppl):163-7.
2. Belin L. Clinical and immunologic data on "Wood-trimmer's disease" in Sweden. *Eur J Respir Dis* 1980;61(Suppl):169-76.
3. Alexandersson R. Exposure, lung function and *Rhizopus* antibodies in wood trimmers. A longitudinal study. *Arh Hig Rada Toksikol* 1988;39:405-9.
4. Bruce RA. Exercise testing of patients with coronary artery disease. *Ann Clin Res* 1971;3:323-32.
5. Fink JN. Hypersensitivity pneumonitis. In: Middleton E Jr, Reed CE, Ellis EF, Adkinson NF Jr, Yunginger JW, Busse WW, eds. *Allergy: principles and practice*. 4th ed. St. Louis: Mosby 1993:1415-31.
6. Halliwell RE, Fleischman JB, Mackay-Smith M. The role of allergy in chronic pulmonary disease of horses. *J Am Vet Med Assoc* 1979;174:277-81.

Vaginal itching as a manifestation of seasonal allergic disease

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Vaginal pruritus as a manifestation of IgE mast cell-mediated disease has rarely been reported in women^{1,2} and not in children. We herein report the first case of putative ragweed-induced vaginitis in a child.

CASE REPORT

The patient is now 10 years old and was first seen in the Allergy-Immunology Clinic of the Medical College of Wisconsin in April 1989. She was then seen with classical allergic rhinitis, which occurred during the

previous year from June through October and after exposure to dogs. There was a strong family history of atopy. She had physical findings of atopic respiratory disease and marked immediate wheal-and-flare skin reactivity to common inhalant antigens, including grass and ragweed. She began receiving antihistamines and had a good response. In August 1989 she began to wheeze and began receiving bronchodilator syrup intermittently. Observation and pharmacologic management were chosen as therapy, and her condition was under good control.

In August 1991 she had nasal and eye symptoms and, for the first time, vaginal pruritus, which led to excoriations and emotional upsets. Maximum use of antihistamines did not provide relief of the pruritus, which became more severe. Daily use of prednisone with tapering to 10 mg every other day through the remainder of the ragweed pollination season resulted in total relief of her vaginal symptoms.

She then did well with intermittent administration of antihistamines and decongestants until August of 1992, when the intense vaginal pruritus began again, along

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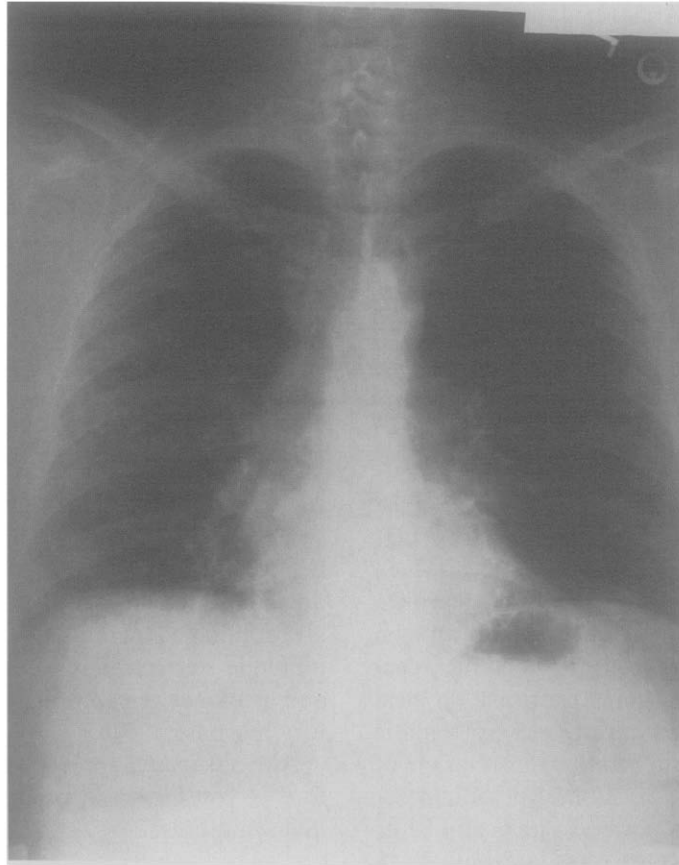


FIG. 1. Frontal chest radiograph demonstrating increased bilateral perihilar markings extending to the periphery.

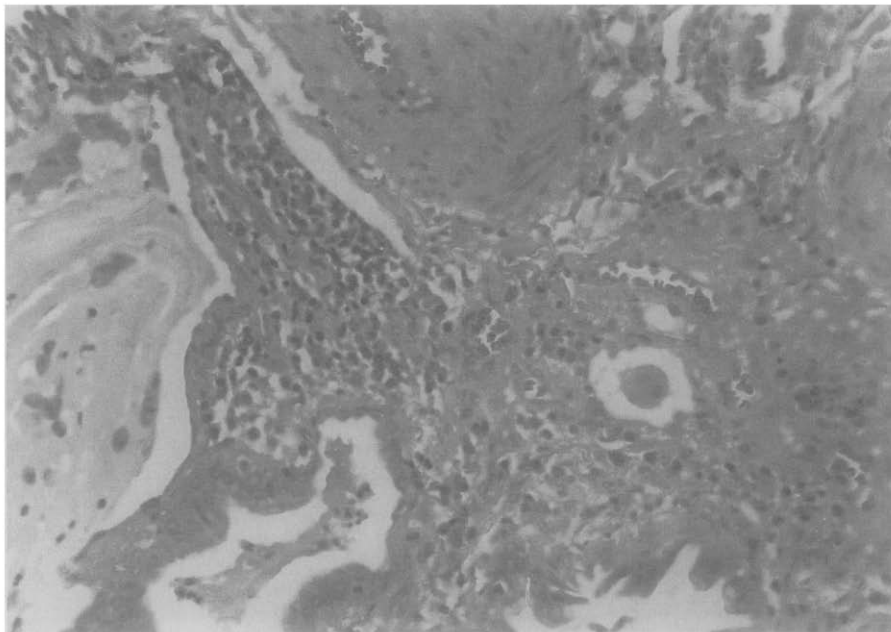


FIG. 2. Lymphocytic infiltration and epithelioid granuloma formation in lung parenchyma (hematoxylin-eosin stain; ×200.)

with her nasal symptoms. Again, antihistamines were of no value for control of the symptoms. Prednisone was provided for the ragweed season, and complete control of symptoms was achieved.

Immunotherapy with ragweed was then begun in an attempt to control the anticipated future vaginal symptoms. The injections were associated with intense emotional distress, and two systemic reactions occurred, each of which was associated with recurrence of the vaginal itching. That treatment was then discontinued. Before the 1993 and 1994 ragweed season the patient began receiving prednisone (tapered from 10 mg/day to 2.5 mg every other day), which was continued during the season, and no respiratory or vaginal symptoms occurred.

DISCUSSION

This child had classic ragweed-induced IgE mast cell-mediated rhinitis. Accompanying these symptoms was intense seasonal vaginal pruritus, which was controlled after the use of systemic steroids. Immunotherapy was begun with ragweed antigen but was discontinued because of systemic reactions, which included vaginal pruritus. Physical examination of the vagina during the symptomatic period was not done. In addition, instillation of topical medications such as cromolyn sodium solution, which has been reported to be useful,^{1,2} or aqueous corticosteroid preparations would have been desirable. However, the age and emotional status of the child and the trauma that may have been induced precluded our carrying out these measures.

Recurrent vaginitis has been reported as a result of *Candida albicans* or bacterial infections,³ local vaginal allergic reactions from semen containing allergens,⁴ or IgE reactivity to vaginal fluids.⁵ None of these causal relationships were clinically apparent in this case. The symptoms were repeatedly

associated with the ragweed pollination season, were controlled with prednisone, and were induced by systemic administration of specific allergen. Furthermore, IgE has been identified in vaginal fluids,² making a ragweed-induced IgE mast cell-mediated reaction most likely in this case.

The route of the ragweed antigen that induced vaginal symptoms was not clear. Inhalation was the only apparent means of introduction of antigen. The child's clothes were washed regularly and dried indoors, never outside. It is, of course, possible that ragweed antigen was not washed away. Vaginal symptoms did not occur during any other season, even though IgE could be detected to antigens present during those seasons. Finally, vaginal symptoms were induced by ragweed antigen given by injection out of the season. Thus we believe that this child's allergic vaginitis was induced by inhaled ragweed pollen with systemic absorption and an unusual affinity for the vagina or a unique susceptibility of the vagina to mediators and cytokines released during the allergic respiratory response.

Antigen-induced allergic vaginitis should therefore be considered in children with recurrent vaginal symptoms.

REFERENCES

1. Dworetzky M. Allergic vaginitis: a case report. *Am J Obstet Gynecol* 1989;161:1752-3.
2. Galland L. Reply. *Am J Obstet Gynecol* 1989;161:1753.
3. Oriel JD. Clinical overview of candidal vaginitis. *Proc R Soc Med* 1977;70(Suppl 4):7-10.
4. Levine BB, Siraganian RP, Shenkein I. Allergy to human seminal plasma. *N Engl J Med* 1973;288:894-6.
5. Witkin SS, Jan Jeremias AB, Ledger WJ. Recurrent vaginitis as a result of sexual transmission of IgE antibodies. *Am J Obstet Gynecol* 1988;159:32-6.