

Hypersensitivity pneumonitis related to a home furnace humidifier

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A thermophilic actinomyceete was isolated from the home furnace humidifier of a patient with biopsy-proved interstitial granulomatous pneumonitis. Extracts of this organism produced a positive Arthus-like reaction and a positive inhalation challenge in the patient and gave a weak precipitin line in agar gel diffusion against the serum. Subsequent operation of the furnace in the patient's home regularly induced symptoms before but not after removal of the humidifier. This common household appliance may increasingly be a source of exposure to thermophilic actinomyceetes.

Increasing interest in the phenomenon of hypersensitivity pneumonitis has led to recognition of an ever-increasing number of different organic dusts which may be responsible for this condition. The resulting nomenclature bears witness to the association of this disorder with a particular occupation (farmer's lung,¹ mushroom worker's lung,² malt worker's lung,³ etc.), a hobby (pigeon breeder's disease⁴), or a disease (pituitary snuff taker's lung⁵). While the fancier of a single parakeet may have sufficient exposure to develop hypersensitivity pneumonitis,⁶ a carefully taken history will more often reveal evidence of extensive contact with a dust which can be considered suspect. Of special interest is the recent report of Banaszak, Thiede and Fink⁷ concerning an "epidemic" of hypersensitivity pneumonitis resulting from contamination of an office air conditioning system with thermophilic actinomyceetes. The occurrence of more than one case among the office employees in the absence of any apparent "dust" focused attention upon a common source of exposure. An equally intriguing situation is presented here in which the source of thermophilic actinomyceetes proved to be a common household appliance—the furnace humidifier. Fink and associates⁸ have also reported a similar case.

CASE REPORT

P. K., a 53-year-old Caucasian housewife, was admitted to the Henry Ford Hospital for the first time on Jan. 16, 1970. She had moved into a 6-year-old suburban Saginaw, Michigan, home in March, 1968. In September 1968 she had a sudden onset of chills, fever, cough, and dyspnea. A chest roentgenogram revealed evidence of "pneumonitis." One week's hospitalization resulted in symptomatic improvement. However, several

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Received for publication Feb. 19, 1971.

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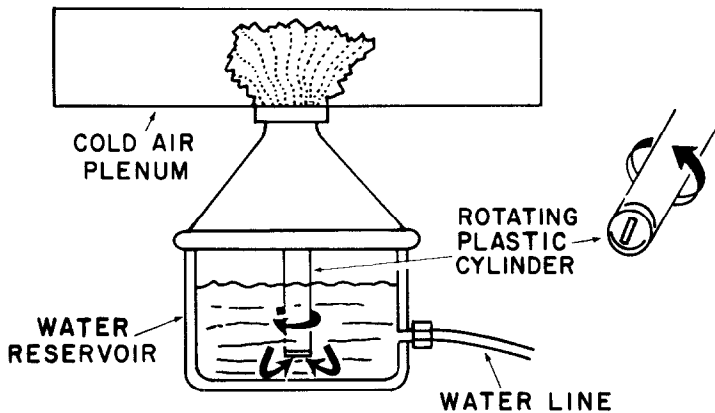


FIG. 1. Schematic diagram illustrating the method of operation of the power humidifying unit. Water vapor emerges from the top of the unit as a visible fog.

hours after returning home, her cough recurred, and over the next 6 months she developed progressive dyspnea on exertion, general malaise, and almost constant cough productive of scanty amounts of yellow to green sputum. In March, 1969, a 2 week course of steroids produced remarkable symptomatic improvement, but within another 2 weeks all symptoms had recurred. A 2 week sojourn in Florida resulted in only slight amelioration of symptoms; however, there was clear-cut worsening on her return home. A second similar course of steroids produced less dramatic improvement. A slight spontaneous regression of symptoms appeared during the warm summer months, but during the fall of 1969 symptoms again increased. The patient was unable to associate this change with certainty to the onset of the heating season. A roentgenogram in November, 1969, was again said to reveal "pneumonia," and a second hospitalization for one week resulted in improvement. A third episode of "pneumonia" occurred in December, 1969.

Past history revealed 29 years of classic ragweed hay fever which had been treated with hyposensitization from 1961 to 1964 with excellent symptomatic relief. No evidence of asthma was ever manifested. Hyposensitization therapy (dust, trees, grass, weeds, molds) reinstituted from April until November, 1969, failed to influence the course of chest symptoms.

The patient had no exposure to birds or, apparently, to any of the known sources of antigens responsible for hypersensitivity pneumonitis. Her home heating system was oil-fired forced air. A power humidifier designated as a "true centrifugal atomizer" by the manufacturer was mounted in the cold air return duct of the furnace. As shown in Fig. 1, a motor-driven rapidly rotating plastic cylinder fitted with a slotted disk at its end draws water from the reservoir into the domed top of the humidifier. The water emerges as a visible mist directly into the cold air duct. Although not directly connected to the hot air portion of the furnace, the humidifier was installed directly in front of the hot air plenum and directly above a vent over the furnace burner (Fig. 2). In addition, a portable humidifier had been used in the patient's bedroom.

Physical examination on admission was unremarkable except for the presence of crepitant inspiratory rales at both lung bases. The patient was afebrile and in no respiratory distress at rest.

The hemoglobin was 12.4 Gm., and white blood cell count was 7,600 with 56 neutrophils, 33 lymphocytes, 6 monocytes, 4 eosinophils, and 1 basophil. Urinalysis was normal and serology nonreactive. Fasting blood sugar, creatinine, electrolytes, alkaline phosphatase, bilirubin, serum glutamic oxalacetic transaminase (SGOT), calcium, and phosphorus were normal. Three lupus erythematosus cell preparations and antinuclear factor were negative.

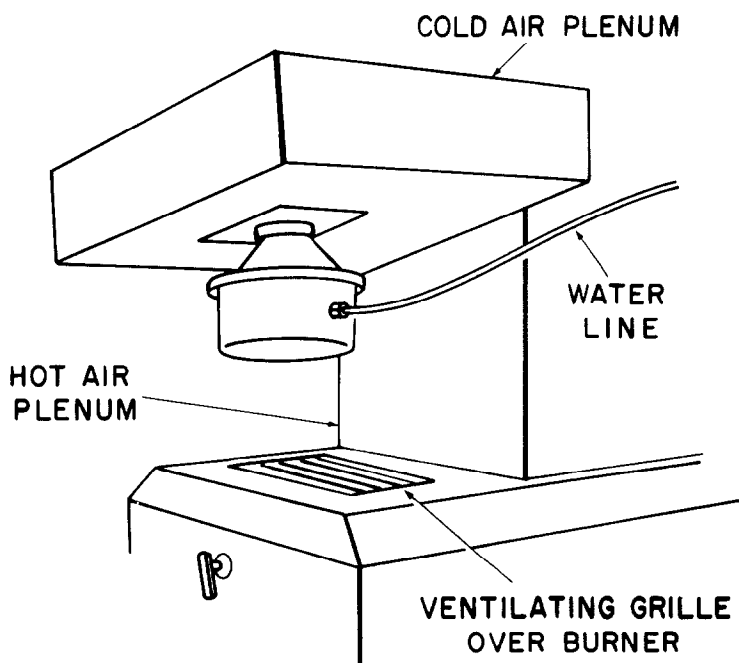


FIG. 2. Illustration of the mode of installation of the humidifier on the cold air-return duct. Note the close approximation of the water reservoir to the hot air plenum and the vent over the furnace burner.

Total serum complement as determined by hemolytic assay was normal while the patient was symptomatic and before institution of steroid therapy. Serum protein electrophoresis revealed total 9.2 Gm. (normal 6.5 to 8.3), albumin 2.93 (4.1 to 5.6), alpha-1 globulin 0.25 (0.17 to 0.37), alpha-2 globulin 1.19 (0.34 to 0.76), beta globulin 1.04 (0.52 to 1.01), and gamma globulin 3.78 (0.61 to 1.39). Quantitative immunoglobulins revealed IgG 1,699 mg. per cent (600 to 1400), IgA 253 (30 to 135), and IgM 147 (30 to 120). Closer inspection of the admission chest roentgenogram revealed a finely nodular diffuse infiltrate of both lower lung fields (Fig. 3). A left-sided bronchogram was normal. Pulmonary function studies were consistent with a restrictive respiratory deficit and a mild "alveolocapillary block syndrome." Total vital capacity was 1,300 c.c. (49 per cent of predicted), one-second forced expiratory volume (FEV_1) 1,190 (92 per cent of total forced vital capacity [FVC]), maximal voluntary ventilation (MVV) 45 L. per minute (56 per cent of predicted), maximal midexpiratory flow (MMEF) 2.6 L. per second (100 per cent of predicted). The carbon monoxide-diffusing capacity was reduced at 13.2 ml. per millimeter per minute (predicted 19.9). Arterial blood gases at rest revealed PO_2 68, SO_2 94 per cent, PCO_2 30, and pH 7.47. Purified protein derivative, mumps, coccidioidin, and histoplasmin skin tests were negative. *Candida albicans* skin test was positive. Electrocardiogram was within normal limits. Allergy skin tests revealed positive prick test responses to tree pollens, grass pollens, and weed pollens. Intradermal tests to dog, cat, and house dust were positive, while those to common contaminant molds were negative. The mold skin tests were performed utilizing the usual standard procedure for immediate hypersensitivity testing—intradermal injection of 0.02 c.c. of 1:1,000 or 1:10,000 extract. The molds used included six species of *Aspergillus*, six species of *Penicillium*, *Alternaria*, *Curvularia*, *Spondylocladium*, *Helminthosporium*, *Hormodendrum*, *Stemphylium*, *Nigrospora*, *Pullularia*, *Mucor*, *Rhizopus*, *Botrytis*, *Monilia sitophila*, *Mycogone*, *Paecilomyces*, *Fusarium*, *Chaetomium*, *Trichoderma*, *Gliocladium*, *Phoma*, *Rhodotorula*, and *Saccharomyces*. No delayed reactions were noted at any of these skin test sites.



FIG. 3. Admission chest roentgenogram showing bilateral linear and patchy infiltrates, predominantly in the lower half of the lung fields (January 1, 1970).

At open-lung biopsy the lung appeared grossly normal. Microscopic sections showed typical interstitial granulomatous pneumonitis (Fig. 4). In addition to a heavy plasma cell infiltration, numerous large lipid-laden cells, including some giant cells, were present (Fig. 5). X-ray diffraction failed to reveal significant quantities of crystalline material. Routine bacterial and fungal cultures were negative. No special attempt was made to isolate thermophilic organisms.

The patient was started on oral prednisone and discharged from the hospital on January 31, 1970. Over the next several weeks there was striking clinical improvement. A chest roentgenogram obtained on March 24, 1970, was normal (Fig. 6), and pulmonary function studies had returned to normal values by May 5, 1970. By May, prednisone had been tapered to 10 mg. every other day. During the next 3 months the patient turned on the furnace on three occasions at approximately 7:00 A.M. On each occasion she became ill at 3:00 P.M. with nausea, malaise, shaking chills, temperature up to 102°, but no apparent cough or dyspnea. The patient was advised to leave the furnace turned off; prednisone was tapered further and discontinued on August 11, 1970, without incident.

On March 24, 1970, while the patient was taking 20 mg. of prednisone daily, an intradermal skin test with *Thermoactinomyces vulgaris* extract (American type culture)* failed to reveal convincing evidence of immediate, 6 hour, or delayed reaction. Gel diffusion in Hyland ImmunoPlates with this same extract and the serum (obtained during the initial hospitalization) failed to reveal detectable precipitin lines.

*A.T.C.C. No. 15347, kindly provided by Dr. Rodney Bentz, University of Michigan, Ann Arbor, Mich.

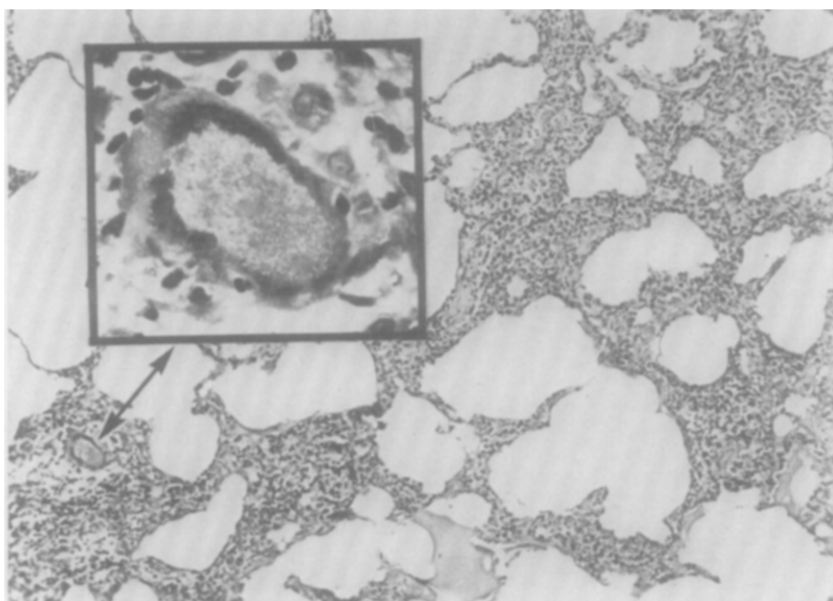


FIG. 4. Photomicrograph of the lung demonstrates diffuse interstitial inflammatory infiltrate composed mainly of lymphocytes and plasma cells. Occasional granulomatous foci with multinucleated giant cells are also present (**insert**). (Hematoxylin and eosin. Original magnification X150; insert original magnification X780.)

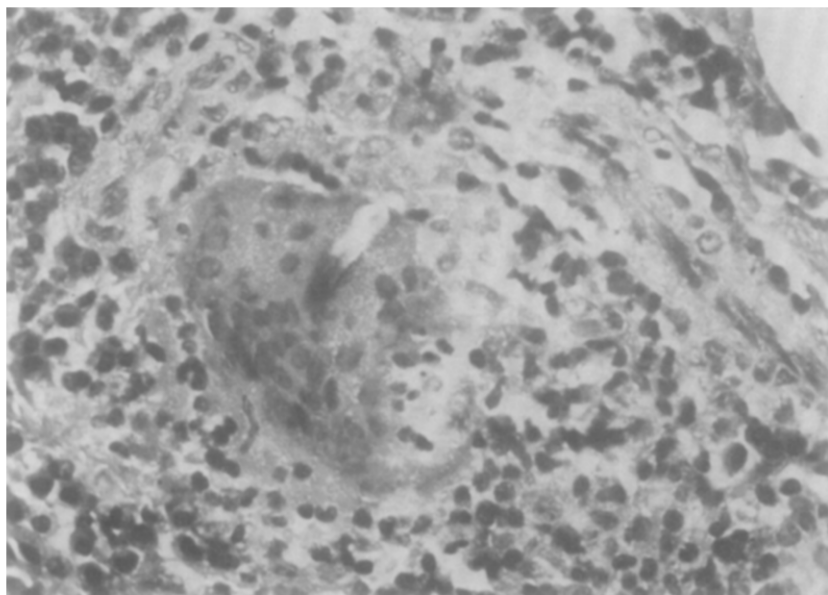


FIG. 5. Detail of an interstitial granuloma with a central multinucleated giant cell containing a needle-like nonrefractile inclusion. (Hematoxylin and eosin. Original magnification X780.)



FIG. 6. The bilateral infiltrate has largely cleared, leaving only a few linear shadows in the lower halves of the lungs (March 24, 1970).

An attempt to isolate thermophilic organisms from the patient's furnace humidifier was unsuccessful. However, a water specimen from the furnace humidifier submitted to Dr. Jordan Fink, Medical College of Wisconsin, Milwaukee, Wisconsin, produced a thermophilic actinomyceete morphologically similar to the organism isolated in the Banaszak and associates⁷ report. No thermophilic organisms were isolated from the portable humidifier. Specimens were cultured in Trypticase soy broth at 56° C. The patient's serum produced a weak precipitin line in standard Ouchterlony gel diffusion against a 10 per cent w/v saline extract of the organism isolated from the humidifier.

In September, 1970, at which time the patient was asymptomatic, off corticosteroids, and with normal pulmonary function, the following additional special studies were carried out by Dr. Fink: methacholine inhalation (25 mg. per milliliter administered by a power nebulizer) produced no significant change in pulmonary function. Inhalation of the thermophilic actinomyceete extract prepared by saline extraction of the organism produced an immediate (maximum at 30 minutes) drop in FVC, FEV₁, and forced expiratory flow (FEF)_{200-1,200}. The single-breath diffusing capacity decreased from 27.7 to 19.6 ml. per millimeter per minute. Some 6 hours later, the patient experienced symptoms identical to those occurring after her furnace had come on—malaise, chills, and slight fever. Basilar rales were audible at this time. Skin testing with 50 mg. of a purified extract of the organism prepared by Dr. John Salvaggio produced an Arthus-like reaction at 6 hours.⁹

The furnace humidifier was removed from the patient's home in August, 1970. Since that time the furnace has been turned on several times without producing symptoms.

DISCUSSION

While the onset of illness in this patient was somewhat sudden with chills, fever, cough, and dyspnea, the subsequent pattern of the disease was more chronic with progressive fatigue, weight loss, cough, and increasing dyspnea. The only period of significant improvement resulted from administration of corticosteroid drugs. The 2 week vacation in Florida and her somewhat improved condition during the summer of 1969 (probably because the furnace was off) were not significant enough to suggest to the patient that the home heating system might be at fault.

Physical findings at admission, chest roentgenogram, and pulmonary function studies are all consistent with a diagnosis of interstitial pneumonitis. However, hypersensitivity pneumonitis was not suspected until the positive lung biopsy was obtained. The subsequent isolation of thermophilic actinomycetes from the furnace humidifier water, the positive Arthus reaction and inhalation challenge to extracts of this organism, and the subsequent course of the patient with acute attacks precipitated by the operation of the furnace before but not after removal of the humidifier all serve to incriminate the furnace humidifier as the source of difficulty.

The particular mode of installation of the humidifier is felt to be of significance in allowing the growth of thermophilic organisms. Although no temperature recordings were made, the location of the humidifier in close approximation to two heat sources apparently allowed maintenance of adequate temperature conditions for growth of the organism. The water reservoir of the humidifier probably functioned in much the same manner as a kettle on a stove. Location of the humidifier on a different area of the cold air-return duct perhaps would have prevented the problem.

This case re-emphasizes the ability of thermophilic actinomycetes to produce hypersensitivity pneumonitis without the accompaniment of a specific organic dust. In addition, it illustrates again that significant antigenic exposure can occur in the apparent absence of any of the previously described sources of organic dust. The frequent presence of power humidifying units on home forced-air heating systems suggests that this may be increasingly a means of exposure to thermophilic actinomycetes.

The authors wish to thank Dr. Averill Liebow for interpretation of the biopsy and Dr. Jordan Fink for the special studies noted.

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