Chronic Pulmonary Histoplasmosis Following the Excavation of a Bird Roost

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The clinical and epidemiologic characteristics of chronic pulmonary histoplasmosis, as compared with the more familiar acute pulmonary histoplasmosis, are relatively unknown. Opinions vary as to the pathogenesis, and only the severe forms of the disease are readily recognized.

Over a 22 month period following the excavation of a blackbird roost, an unusual outbreak of chronic pulmonary histoplasmosis occurred in a town in southern Kentucky. Thirteen of the cases developed over a span of only four months. An associated outbreak of acute pulmonary histoplasmosis did not occur.

During the course of the ensuing investigation, the residential addresses of the affected persons were noted to be clustered about the previously excavated blackbird roost. A case-control study was initiated; the median distance of the residential addresses of the affected persons was found to be 1.0 miles from the roost, compared with 3.2 miles for the control subjects (P <0.001).

It was concluded that (1) the excavated blackbird roost had served as the common source of the epidemic; (2) the inhalation of exogenous spores accounted for the infections; (3) the spectrum of clinical illness ranged from asymptomatic and mild illness to cavitary disease with considerable morbidity; and (4) following excavation, blackbird roosts may remain an infection hazard for an indefinite period of time.

Chronic pulmonary histoplasmosis differs from the more familiar acute pulmonary histoplasmosis in several respects. Acute symptomatic pulmonary histoplasmosis is almost always a self-limited febrile illness of several days' duration; the etiology of this form of the disease is well described and is linked to the inhalation of large numbers of Histoplasma capsulatum spores by otherwise healthy persons [1]. In contrast, chronic pulmonary histoplasmosis often results in considerable morbidity, characterized by months or years of progressive cavitary disease and functional debility [2,3]. A less severe and self-limited form of chronic pulmonary histoplasmosis also has been described [4]. However, the signs and symptoms of this form of disease are not easily recognized and diagnosis is difficult [2].

Although it is known that chronic pulmonary histoplasmosis usually develops in middle-aged Caucasian men with underlying chronic obstructive pulmonary disease, the route of infection is uncertain [5,6]. The reactivation of a dormant endogenous focus of infection, as occurs in postprimary tuberculosis, was once thought to best explain the pathogenesis of chronic pulmonary histoplasmosis [7]. However, reinfection from exogenously inhaled spores recently has been offered, as an explanation of the initiating events of this disease [8–10].
In this report we describe the epidemiologic investigation of a rarely recognized event: an outbreak of chronic pulmonary histoplasmosis. Evidence is provided supporting the importance of the exogenous route of infection. Several patients with asymptomatic or mild disease were identified during the investigation, providing unique insight into the wide range in the severity of clinical disease.

THE EPIDEMIC

During the early 1970's, a residential area located 3 miles south of downtown Hopkinsville, Kentucky (population, 26,000) became the winter roost of thousands of blackbirds. The birds left en masse each morning and returned in the evening (Figure 1), at which times the noise of the birds made normal speech impossible; the bird droppings made unsheltered existence hazardous. In November 1975, local townspeople bulldozed the foliage of the 28 acre roost into large brush piles; insufficient funds precluded disinfection procedures. Although none of 16 soil samples taken from the roost prior to the excavation had been positive for H. capsulatum on culture, soil samples obtained at two, seven and 18 months following the excavation were positive for the fungus. In one bulldozer operator an illness developed compatible with acute primary pulmonary histoplasmosis two weeks after the excavation (Figure 2). Subsequently progressive pulmonary insufficiency developed, and culture-positive chronic pulmonary histoplasmosis was diagnosed two months after the excavation.

Above average rainfall occurred following the excavation and persisted during the first two quarters of 1976. However, the least rainfall in three years was experienced during the fall and winter of 1976.

During the extremely dry and dusty conditions which existed 10 months after the excavation of the roost, four patients from this residential area were hospitalized with severe pulmonary disease. Open lung biopsy specimens compatible with chronic pulmonary histoplasmosis were obtained in all four patients. An investigation of this unusual cluster of cases led to the identification of nine additional patients in whom infection developed during the last four months of 1976 (Figure 2). In a final patient chronic pulmonary histoplasmosis developed three months after the roost had been re-excavated in preparation for disinfection with formalin.

METHODS

Case Detection. Chest roentgenograms were reviewed of all patients discharged from the local hospital with an undiagnosed pulmonary infiltrate from July 1976 through January 1977.

Charts were reviewed of those patients whose chest roentgenograms exhibited interstitial infiltrates in the upper lobes. Patients suspected or proved to have a tuberculous, viral or bacterial infection were excluded. The remaining patients were interviewed for additional information regarding their exposure history and symptoms.

All local internists were interviewed and hospital records evaluated for additional patients seen during the previous four
years with unexplained interstitial infiltrates of the upper lobes or a diagnosis of histoplasmosis. One further case was identified by these methods. In this patient culture-positive pulmonary histoplasmosis had developed in 1973, and was considered to be a part of the endemic occurrence of disease.

**Control Population.** A control group was selected from patients admitted to the local hospital during the period of time when the cluster of 13 cases had occurred. Preexisting chronic obstructive pulmonary disease was common in the epidemic population. Therefore, the control group consisted of every other patient admitted between September 1, 1976, and January 31, 1977, with a discharge diagnosis compatible with chronic obstructive pulmonary disease. Control patients were comparable in age, sex and race to the epidemic group.

**RESULTS**

Fifteen patients were identified during the investigation with infiltrates in the upper lobes that were compatible with chronic pulmonary histoplasmosis (Table I). Thirteen patients were male; all were Caucasian. Their mean age was 50 years. Infiltrates occasionally were noted to coalesce to form central areas of infarction. Multiple luencies suggestive of centlobular emphysema were present within the majority of infiltrates [5]. Infiltrates persisted for a minimum of six weeks in all patients. Cavitary disease eventually developed in four patients.

H. capsulatum infection was confirmed in five patients following open-lung biopsy. A definitive diagnosis was not made in the remaining 10 patients who are classified as having "presumptive chronic histoplasmosis." One of these patients underwent an open lung biopsy; the histopathology was entirely consistent with chronic pulmonary histoplasmosis, although no organisms were seen or cultured. Extensive evaluation short of open lung biopsy revealed no infectious or other etiology in the remaining nine patients. These patients with presumptive cases shared roentgenographic and clinical features similar to those of the patients with proved histoplasmosis (Table I). However, the severity of disease did differ. Three of the 10 patients with presumptive chronic pulmonary histoplasmosis had mild to absent symptoms, and infiltrates resolved within three months in seven of them. However, the five patients with proved infection experienced prolonged hospi-

![Epidemic curve of patients with culture-proved and presumed chronic pulmonary histoplasmosis. The blackbird roost was excavated on November 28, 1975. Two weeks later, acute pulmonary histoplasmosis developed in a bulldozer operator followed by chronic histoplasmosis. Twenty-two months after the excavation of the roost, an additional patient with presumed chronic histoplasmosis was identified. The roost had been re-excavated on May 23, 1977.

<table>
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<tr>
<th>TABLE I</th>
<th>Clinical Characteristics of 15 Patients with Chronic Pulmonary Histoplasmosis</th>
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<tr>
<td>Culture or Presumptive</td>
<td>Proved Chronic</td>
</tr>
<tr>
<td>Biopsy Proved Histoplasmosis (5)</td>
<td>Histoplasmosis (10)</td>
</tr>
<tr>
<td>Chronic illness and infiltrates in upper lobes</td>
<td>5</td>
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<tr>
<td>Smoking history (20 pack-years or greater)</td>
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<td>Malaise, fatigue, fever</td>
<td>5</td>
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<tr>
<td>Underlying lung disease*</td>
<td>5</td>
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<tr>
<td>Infiltrates persisting after three months</td>
<td>5</td>
</tr>
</tbody>
</table>

**NOTE:** Figures in parentheses indicate the number of patients.
* Infiltrates and/or symptoms of at least six weeks' duration.
† One second forced expiratory volume < 80 per cent or chronic productive cough or cystic changes on chest roentgenogram.
A case-control analysis was undertaken in an effort to investigate whether the space-time cluster of 13 cases which occurred during the last four months of 1976 could be related to the previously excavated blackbird roost. The home addresses of these 13 subjects were compared with those of the 62 patients in the control population. The epicenter of the cases was found to be only 0.5 miles from the blackbird roost, whereas the epicenter of the control patients was 3.0 miles away (Figure 3). Additionally, the median distance from the roost was 3.25 miles for the control subjects, whereas the median distance for the 13 subjects was only 1.0 mile ($P = 0.0004$, Wilcoxon Rank-Sum test). These analyses indicate that persons with underlying lung disease residing near the blackbird roost were at an increased risk of acquiring chronic pulmonary histoplasmosis.

**COMMENTS**

The incidence of chronic pulmonary histoplasmosis is estimated at one per 100,000 population per year in highly endemic areas [5]. Thus, even excluding the bulldozer operator and the 10 cases of presumptive disease, the four proved cases occurring within four months in a town of 26,000 population is highly unusual (equivalent to 60 cases/100,000/year). Additionally, an intensive investigation of hospital records, review of...
hospital roentgenograms and personal inquiry with area physicians uncovered only one additional case within the previous three years.

The long interval (10 months) between the excavation of the roost and the onset of the epidemic was unexpected. Conceivably, the cold and rainy weather which followed the excavation may have delayed the maturation and air-borne dissemination of spores. Alternatively, this prolonged interval may represent the in vivo incubation period of chronic pulmonary histoplasmosis. Whatever the reason, it is probable that contaminated roosts may serve as a persistent source of infection. This point is amply demonstrated by the persistence of positive soil cultures 18 months after the excavation of the roost and by the occurrence of a case 22 months after the excavation.

Controversy has existed concerning the importance of endogenous and exogenous routes of infection in initiating this disease [5,8,10]. Clusters of patients with chronic pulmonary histoplasmosis have been rarely recognized; the two previously reported outbreaks were discovered in association with large epidemics of acute pulmonary histoplasmosis [8,11]. The present epidemic is, therefore, important in that it offers persuasive evidence that chronic pulmonary histoplasmosis may develop from a common source of exogenous exposure, unaccompanied by a concurrent outbreak of acute pulmonary histoplasmosis.

The clinical impression that chronic pulmonary histoplasmosis occurs primarily as a sporadic disease also needs to be re-evaluated. The existence of this present outbreak was realized only through the informal sharing of information between patients and physicians in a small community. Formal surveillance of chronic pulmonary histoplasmosis should be initiated by public health agencies in areas of high endemicity for histoplasmosis. Clusters of cases may prove to be more prevalent than previously thought, and additional reservoirs of infection with Histoplasma capsulatum may be identified.

ACKNOWLEDGMENT

We wish to express our gratitude to Faxon Payne, M.D. and Frank Pitzer, M.D. for their help with the investigation, and to Lawrence Grossman, M.D. and John Cotthoff, M.D. for allowing us to include their patients.

REFERENCES