Naturally Occurring Histoplasmosis in the Chinchilla (Chinchilla laniger)

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Received for publication 14 February 1975

Histoplasmosis was diagnosed histopathologically in a female chinchilla. This animal had originated from a commercial chinchilla ranch in central Missouri. Seventeen of 130 animals in the colony had died within a month’s period with a respiratory illness. This animal had a history of fur chewing, but this was not true of all the other animals that had died. Histoplasma capsulatum was cultured from timothy hay used for food.

Histoplasmosis is a relatively common disease of man, the canine, and several other animal species within a rather well defined endemic areas (4, 8): North America, especially in the Mississippi River Valley and its tributaries (4). The organism exists in a hyphal form in unused bird roosts and chicken houses or other suitable environments with high moisture and organic content. Once the microorganisms gain entrance to a susceptible host through inhalation of airborne spores, it begins to invade tissue via a single budding yeast phase.

The occurrence of Histoplasma capsulatum in the chinchilla has previously been reported in Europe (1). However, this is the first report of spontaneously occurring histoplasmosis in chinchillas in the United States.

Methods. Six adult chinchillas approximately 1 year of age were presented to the Veterinary Medical Diagnostic Laboratory at the University of Missouri-Columbia, for examination on 31 October 1968. One animal was alive and the others were dead and frozen when received at the laboratory.

A necropsy was performed on all six animals. The animal which was presented alive was euthanized and tissue specimens were removed aseptically from several of the organs and submitted for microbiological examination. At the time the microbiology sample was taken a duplicate sample was taken and placed into 10% buffered Formalin for subsequent histopathological examination.

The tissues submitted for bacteriological culture were inoculated onto Trypticase soy agar enriched with 5% defibrinated sheep blood and MacConkey agar. These two media were utilized for culturing all tissues. In addition Sabouraud and Mycosel agars were utilized for culturing selected organs.

Wet moldy hay samples, used as feed, and wood chips, used as litter, were analyzed for the presence of H. capsulatum by animal inoculation. Ten grams of each sample were mixed with 100 ml of sterile saline and shaken for 60 min. Six Swiss mice were inoculated intraperitoneally with 2 ml of the supernatant from each sample. The mice were sacrificed and necropsied 1 month from time of inoculation. The liver and spleen were removed aseptically from each animal in the respective groups, pooled and homogenized in a sterile ten Broeck grinder, and 0.5-ml aliquots of each suspension were inoculated onto duplicate Sabouraud dextrose agar plates. Additionally two Mycosel plates and two brain heart infusion agar plates with 5% defibrinated sheep blood added for enrichment were also inoculated. All plates were incubated at 25 to 28°C and were examined daily for growth.

History. The chinchillas which were necropsied were from an original group of 130. Seventeen animals had died within a period of 1 month. Clinically all appeared to be suffering from an acute respiratory illness. Prior to this time, no excessive death losses or illnesses occurred in the group. Some of the animals which died were fur chewers, but no other problems were noted.

The original stock was housed in the basement of a private home in Columbia, (Boone County) Mo., from March 1966 to March 1968. They were then relocated to Rolla, (Phelps County) Mo. where they remained until illness and death losses began to occur in October 1968.
No new additions had been made to the group within the previous year. Similar husbandry practices were followed at both locations. The animals were housed in cages provided with walnut or pine wood chip bedding which was changed weekly. Their diet included a commercial pelleted chinchilla chow, a multiple vitamin supplement, timothy hay, and water. A mixture of captan (Imperial Chemical Industries) and fullers earth was occasionally used as a dust bath to prevent mycotic skin infections and enhance fur quality.

The clinical signs observed in the affected animals were anorexia followed by weight loss, constipation, and an increased respiratory rate. No ocular or nasal discharge was noted. Once respiratory signs were seen, death usually occurred within 2 to 4 days. None of the animals were treated during the course of the disease.

**Necropsy findings.** Gross necropsy examination of the animal that had been received alive revealed emphysematous lungs with numerous foci of hemorrhage and bronchopneumonia. The spleen was enlarged and had prominent white nodules throughout the parenchyma. The liver had extensive areas of focal necrosis. The stomach and both small and large intestines were empty. No other gross lesions were seen.

Histological sections were prepared of the lung, liver, kidney, lymph node, and spleen and were stained with both hematoxylin and eosin. Similar sections were stained with the periodic acid-Schiff methods. Examination of these sections revealed that the lung had extensive proliferation of sepal cells with focal areas of consolidation. *H. capsulatum* organisms were found in numerous multinucleated cells, single free alveolar macrophages, and in endothelial cells lining vessels. There was fibrin attached to the pleural surface of the lung suggesting the presence of fibrinous pleuritis. The liver contained multiple focal granulomas characterized by the presence of numerous epithelioid cells and mixed inflammatory cell accumulation in the area of the portal triads. Epithelioid cells within venous channels as well as scattered individual hepatocytes contained *H. capsulatum*. The spleen had focal necrosis, small abscesses, multiple focal granulomas, and diffusely scattered epithelioid cells containing *H. capsulatum* organisms. The kidney had multiple focal areas of glomerular nephritis characterized by an accumulation of polymorphonuclear cells in the proximal tubules. Aggregates of *H. capsulatum* yeast cells were present in glomerular epithelioid cells. Sections of a lymph node showed markedly distended sinuses containing many large epithelioid cells which were engorged with *H. capsulatum* (Fig. 1).

Examination of the other five animals revealed three to have central lobular degeneration in the liver and slight to moderate congestion in the lungs. The remaining two animals showed no gross lesions. Histopathological and cultural procedures failed to reveal *H. capsulatum* in the five frozen animals. *Pasteurella multocida* was recovered from the lung of the animal with histoplasmosis and one of the other animals. Histopathological changes observed in three of the animals were characteristic of bacterial pneumonia rather than histoplasmosis which was observed in the first animal necropsied.

**Discussion.** This report constitutes the first reported case of histoplasmosis in the chinchilla (*Chinchilla laniger*) in the United States, although histoplasmosis has been reported from a chinchilla in Switzerland which had been imported from the United States (1).

As histoplasmosis was diagnosed in only one animal from this colony in central Missouri, it would appear that it was an incidental finding in this group of chinchillas and not the primary problem. However, it was not possible to obtain diagnostic information on the first seventeen animals which died. Recovery of *H. capsulatum* from the hay samples indicates the microorganism was present in the environment from which the hay came, or that the hay had been contaminated where it was stored. The basement in which the animals and hay were kept was in a house which had recently been built. The area in which the house was located was heavily wooded and apparently had been a roosting area for many birds. It is possible that the fungus had been stirred up when the house was being built, contaminating the basement. However, since the hay had originated from an area of central Missouri known to be endemic for histoplasmosis (7), it would seem possible that the organism was present in the hay when brought into the house.

The finding of multiple foci of histoplasmal infection in the animal presented alive indicates a disseminated form of the disease. It was not determined if other animals in the colony exhibited a skin test reaction to histoplasmosis. However, none of the other five animals presented to the laboratory had histological lesions of histoplasmosis.

Recent research has shown that the chinchilla responds poorly to certain antigens: serum albumin, *Salmonella typhosa* "O" cells, and sheep erythrocytes. It appears that "strong"
antigens elicit good antibody response whereas "weak" antigens, in which the major antigenic constituents are polysaccharides, elicit little or no detectable antibody (3). The cell wall of H. capsulatum is composed of a high percentage of polysaccharides (6). This may indicate that the chinchilla is unable to produce immunity to H. capsulatum via a B cell antibody production system. On the contrary, production of humoral antibody may not be necessary since immunity induced in some animals by H. capsulatum is cellular rather than humoral and depends upon a T cell lymphocyte response (2, 5). In fact, in humans, infection appears to be related to an impaired T lymphocytic response (5). The disseminated type of infection found in the chinchilla could have resulted from a lack of lymphocytic response or from a high number of spores overwhelming the animal with or without an impaired lymphocytic system. Additional studies are in progress to determine the nature of the chinchilla's susceptibility to H. capsulatum.

Although this finding may have been incidental, this case confirms that a new host for H. capsulatum has been found and documents the occurrence of the disease in chinchillas in the United States.

We thank Robert T. Haberman, Food and Drug Administration, Washington, D.C., for definitive identification of Histoplasma capsulatum.

This research was supported in part by Public Health Service grant RR00471.

LITERATURE CITED