Epidemiology

Histoplasmosis is a fungal disease, caused by the spores of Histoplasma capsulatum, which can present in five clinical varieties:

• acute benign pulmonary – not uncommon
• acute disseminated – rare
• chronic pulmonary – rare
• chronic localised – rare but increasing
• chronic disseminated – rare but increasing.

It is widely distributed in tropical and sub-tropical parts of the world, the spores being endemic in bird and bat droppings and in soil in the USA. However, in the rest of the world it appears to be confined to bat guano in caves and abandoned mines.

Clinical features of acute benign pulmonary histoplasmosis

Most people who inhale the spores develop a subclinical infection, stimulating the development of antibodies which protect against further infections. Clinical symptoms are fever, non-productive cough, dyspnoea, chest pains and malaise occurring 1 – 4 weeks after exposure, and depend on:

• the dose inhaled and virulence of the fungus
• previous exposure
• immune status – lowered by previous HIV infection, malnutrition, immunosuppressive therapy.

The diagnosis is suggested if several people simultaneously exposed become symptomatic simultaneously, and is confirmed by a snow-storm appearance on chest X-ray. The radiological changes normalise soon after recovery, but may eventually develop asymptomatic microcalcification after a few years. No other investigation is necessary.

Treatment

If confined to the lungs and immunocompetent, treatment must be symptomatic only.1

If immunosuppressed and/or present beyond the lungs, the following treatment must be given:

• amphotericin-B maximum dose 1.5 mg/kg/day; when improving change to/OR
• ketoconazole up to 400 mg 12 hourly OR
• itraconazole 200 mg 12 hourly for at least a week, or much longer if chronic, after becoming asymptomatic.

Experience in southern Africa

Acute, benign pulmonary histoplasmosis is well known in Zambia,2 Zimbabwe3 and the former Transvaal,4 but was thought not to occur in the southern Cape.5 However, H. capsulatum is now known to exist in guano caves in the Swartberg foothills north of Oudtshoorn,6 and in the De Hoop guano cave east of Bredasdorp.

1943: The cave was mined for guano,7 with no report of chest disease in the town.

1977: Ten members of a rambling club entered the cave and developed acute histoplasmosis. There was no correlation between the clinical and radiological features and the antibody studies. The outbreak was believed...
to have been caused by spores introduced into the
cave from the northern Cape by bat researchers. We
further believed that conditions inside the cave were
unfavourable for the survival of the spores, which
would eventually die. The cave was immediately
closed to all except researchers, all of whom
remained well.9

1987: The cave was reopened and visited by 12 school chil-
dren and 3 speleologists who remained well, and by
5 locals who became infected.10

2004: Three more locals entered the cave and became
infected, thereby disproving the 1977 theory. The
accompanying speleologists remained well.

Comments and questions
1. What are the optimal conditions for survival of H. capsula-
tum spores, viz. humidity, temperature, pCO2 and pNH3
etc. in the cave, and the insectivorous bats’ diet?

2. Why should there be no correlation between the clinical
and radiological features and the antibody studies, for
which the kits are imported from north America? Bats do
not migrate across the Atlantic ocean. Therefore H. capsu-
latum in north America may have evolved independently of
that in South Africa. Morphologically they are identical, but
antigenically they appear to be different.

3. Why did the children, who had no previous exposure to H.
capsulatum, remain well while the five local adults became
ill? The children can be expected to have raised more dust
than the adults, and to have had their mouths and noses
nearer to the guano. Therefore they would have received a
larger dose of spores than the adults.

4. In Cape Town we are now seeing HIV-positive patients with
chronic and disseminated histoplasmosis who give no histo-
ry of exposure to bat, chicken, pigeon and other faeces.11
From where have they acquired the infection? Perhaps we
should be looking more closely at urban soil samples which
may be contaminated with the faeces of pigeons and other
birds?

References available on request.

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