

MELIOIDOSIS IN A SULPHUR-CRESTED COCKATOO (*CACATUA GALERITA*)

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SUMMARY: A case of melioidosis in a sulphur-crested cockatoo (*Cacatua galerita*) kept as a pet in Townsville is described. This case was characterised by liver necrosis with both a heterophil and mononuclear cell inflammatory response associated with giant cells. *Pseudomonas pseudomallei* was isolated by blood plate culture and guinea pig inoculation. This is the first case of melioidosis reported in a native bird in Australia.

Introduction

Pseudomonas pseudomallei was first isolated in Australia from sheep by Cottew (1950). Since then it has been recorded in man (Rimington 1962), a range of domestic animals (Laws and Hall 1963) and in zoological animals (Tammernagi and Johnston 1963; Anon. 1965).

The present paper reports a case of melioidosis in a sulphur-crested cockatoo (*Cacatua galerita*) kept as a pet in Townsville. This is the first report of a natural melioidosis infection in a bird in Australia.

History and Clinical Signs

An adult, male sulphur-crested cockatoo had been kept in captivity in one of the lowlying suburbs of Townsville. During the latter stages of its life, it was allowed to wander freely in the backyard. The bird was in good condition when the owner died and the cockatoo was then sent to an animal refuge where it was permanently caged. Five weeks later, the bird contracted diarrhoea, which was more pronounced after 24 hours. This was followed 24 hours later by recumbency and death.

Autopsy Findings

The liver was greatly enlarged and focal white areas were present in large numbers on the surface and throughout the parenchyma. The size of these foci ranged from being just visible to 4 mm in diameter. The intestinal mucosa was inflamed and the intestinal contents were thin and watery. No other abnormality was observed.

Histopathology

Multiple foci of inflammation of variable size were observed throughout the parenchyma of the liver. The smaller foci consisted of clumps of

hepatic cells showing pyknosis and karyorrhexis surrounded by inflammatory cells. The latter consisted of approximately equal numbers of heterophils and mononuclear cells (lymphocytes, plasma cells and macrophages). A few giant cells were present at the edge of the lesions. Clumps of long, slender, Gram negative rods were also observed on the edge of the lesion. These clumps of bacteria were more readily observed when Giemsa stain was used. No bipolarity was observed in either stain. The centres of the larger lesions were filled with caseated material without mineralisation. Large areas of hepatic and inflammatory cells showing pyknosis and karyorrhexis were present around the caseous centres. These in turn were surrounded by a wide zone of inflammatory cells (heterophils and mononuclear cells) and many palisading macrophages. Giant cells in greater numbers were present in the centres of these larger lesions. There was no fibrous reaction.

Microbiology

Ps. pseudomallei was isolated in pure culture on sheep blood agar and MacConkey agar from the lesions in the liver. Bipolar staining in both Gram and Giemsa stains was present. The morphological, cultural and biochemical characteristics of the organism agreed with those set down by Cottew (1950) and Laws (1964). It was agglutinated by a positive rabbit antiserum for *Ps. pseudomallei*.

Organisms isolated from the small intestine were *Escherichia coli* and a *Proteus* species. No *Salmonellae* were isolated.

Tissue from the liver was macerated in nutrient broth and 1 ml was inoculated into a guinea pig by the intra-peritoneal route. Death occurred within 24 hours and *Ps. pseudomallei* was recovered from the spleen and liver (where numerous small white foci were in evidence) and also from the omentum which had adhered to the stomach.

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Discussion

Bipolar staining of *Ps. pseudomallei* in sections stained with Giemsa was observed by Laws and Mahoney (1964) in bovine melioidosis. Bipolarity was not observed by us in Giemsa stained liver sections where long, slender cells were seen. Similarly stained culture smears showed short, fat, bipolar-staining organisms characteristic of the species. *Ps. pseudomallei* was isolated in pure culture from the liver and it is probable that tissue fixation and staining causes the organisms to look longer than they are. Also, bipolar staining tends to make an organism appear wider than normal. Birds are regarded as relatively resistant to infection with *Ps. pseudomallei* (Laws and Hall 1964). In a serological survey in Australia, Cook (1962) tested sera from four species of birds but found no antibodies present. Laws and Hall (1964) noted the presence of 2 species of birds in a paddock where sheep developed lesions of melioidosis. No attempt was made to infect these birds experimentally.

Ps. pseudomallei is a soil organism occurring mainly in tropical and sub-tropical climates (Chambon 1955; Fournier 1965; Strauss *et al* 1969; Jayanetra *et al* 1974) and is an opportunistic pathogen of animals. It has been isolated from muddy water (Laws and Hall 1964), soil (Thomas 1977) and naturally infected animals (Laws and Hall 1963) in the Townsville area.

In the present case, the origin of infection is not known. The cockatoo was allowed to roam within the confines of the household plot and thus may have acquired *Ps. pseudomallei* by ingestion or through injury. Although uncaged, the bird made no attempt to fly off so that the source of infection would appear to be restricted to the muddy soil and water that accumulates in this suburb in the wet season. It seems unlikely that the bird con-

tracted the disease at the animal refuge as it did not come in contact with the soil.

Subclinical infections are known to be triggered off by other debilitating diseases, burns, surgery or traumatic conditions (Howe *et al* 1971). The large number of giant cells seen is not generally consistent with an acute bacterial disease although giant cells have been reported in lesions of bovine melioidosis (Ketterer *et al* 1975). It is possible that they were present in response to a parasitic migration through the liver which triggered a subclinical melioidosis infection. Alternatively, the disease may have been brought on by the stress of being removed from its owner's care and placed permanently in a cage.

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