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STREPTOBACILLARY PLEURITIS IN A KOALA (*Phascolarctos cinereus*)

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Abstract: A case of *Streptobacillus moniliformis* pleuritis in a koala (*Phascolarctos cinereus*) is reported. Lesions were granulomatous in nature. *S. moniliformis* was recovered in pure culture, and found by experimental inoculation to be pathogenic for mice but not for a rat.

INTRODUCTION

Australia's unique arboreal marsupial, the koala (*Phascolarctos cinereus*) inhabits areas of the east coast from northern Queensland to southern Victoria. While some studies of the ecology and physiology of the koala have been published, little has been recorded on disease conditions.⁷ This paper reports the pathologic and bacteriologic investigation of a case of pneumonia.

MATERIALS AND METHODS

An adult male koala was found ill on Phillip Island in southern Victoria. It died during the 120 km trip to the laboratory. The animal was examined at necropsy and samples of heart, spleen, kidney, liver, skin and lung were fixed in 10% buffered formal saline for histopathology. Tissues were processed routinely, sectioned, and stained with hematoxylin and eosin. Lung sections were stained by the Brown and Brenn method for bacteria and by Masson's trichrome stain for connective tissue.

Thoracic fluid was cultured at 37 C on horse-blood agar and MacConkey agar aerobically, on horse-blood agar microaerophilically in a candle-jar and anaerobically in a Gas Pak \square apparatus. Biochemical tests were carried out as

described by Cohen *et al.*⁵ Acid production from carbohydrates was assayed in 1% peptone water with Andrade indicator and checked with a portable pH meter. \square To determine the pathogenicity of the organism in laboratory animals, an adult rat was inoculated intraperitoneally with 1.0×10^5 organisms, three mice were inoculated intraperitoneally with 5.0×10^4 organisms and three more mice intravenously with 5.0×10^4 organisms.

RESULTS

Gross Lesions

Lesions were confined to the thorax. There was a granular yellow-brown pleural exudate in the right chest cavity. The right lung was consolidated with severe pleuritis. The left lung appeared normal. The heart was moderately enlarged.

Histopathology

The right lung had extensive atelectasis, with few focal areas of emphysema. The normal architecture of the alveolar septa was difficult to discern, the basement membrane was not visible, and there was an apparent decrease in cell numbers associated with necrosis of single alveolar cells. The

\square Baltimore Biological Laboratory, Baltimore, Maryland.

\square Model 293, Pye Unicam, Cambridge.

atelectatic alveolar spaces, bronchioles and alveolar ducts were filled with eosinophilic material. Numerous macrophages were interspersed throughout the tissue. Bronchi had no visible lesions.

The visceral pleura was extremely thick due to a stratum of loose granulation tissue and was heavily infiltrated with mononuclear and epithelioid cells. Numerous foci of Gram-negative bacteria were present on the surface.

The left lung had only occasional macrophages scattered in the alveolar lumen. The liver was mildly congested and the heart showed superficial myocardial degeneration.

Bacteriology

No growth was observed after 24 h on aerobically incubated plates, but a moderately heavy growth of tiny translucent colonies was noted on the microaerophilically incubated plate. Similar colonies were the only type present on the anaerobic plate after 48 h.

The organism was a small Gram-negative rod with some long filamentous elements. In nutrient broth with added horse serum, culture sediments appeared granular. Unstained wet preparations showed chains of rods and long filaments. The organism was non-motile and was not encapsulated. Metabolism of glucose was fermentative, with acid but not gas produced. Subcultures on horse-blood agar, particularly under CO₂, had satellite colonies. The organism was provisionally identified as *Streptobacillus moniliformis*. This identification was confirmed by further biochemical tests, the results of which compared well with descriptions of other isolates of *S. moniliformis*.^{1,19,21}

The inoculated rat was unaffected by the dose. After 2 weeks it was euthanized and examined at necropsy. Cultures from all organs were negative for *S. moniliformis*. Intravenously inoculated mice died within 5-9 days, with cultures from blood, liver and lungs positive for *S. moniliformis*. Intraperitoneally inoculated mice

died within 11-14 days with blood, but not tissue, cultures positive for *S. moniliformis*.

DISCUSSION

Streptobacillus moniliformis was first described as the cause of streptobacillary rat-bite fever in man by Blake³ and named by Levaditi *et al.*¹³ The natural host of the organism is the wild rat^{9,18} and human infection occurs after rat bites,³ other contact with rats,¹² or via infected milk.¹⁴ It is of world-wide distribution, and also causes cervical abscesses in guinea-pigs,^{1,10,17} polyarthritis in mice,¹¹ and tendon-sheath arthritis in turkeys.^{4,21} Human, guinea-pig and murine field strains are nearly always lethal in experimentally inoculated rats and mice; however, isolates from turkeys have been variably pathogenic⁴ or avirulent.²¹

Bacterial pneumonic conditions have been diagnosed previously in koalas. A *Corynebacterium sp.* similar to *C. equi* has been isolated from an outbreak of rhinitis and pneumonia.¹⁵ All affected koalas recovered, except one which had intense pulmonary oedema and some emphysema at post-mortem. Backhouse and Bolliger² reported pneumonia in 6 of 28 koalas examined at necropsy, but did not do bacteriologic studies. They noted the occurrence of excess pleural fluid in some cases, and lung collapse and empyema in another. Pneumonia was reported to be common in koalas in Queensland and *Bordetella bronchiseptica* was isolated from affected cases.⁸ Wood²⁰ reported the occurrence of acute fibrinous pneumonia in koalas, and also radiographic evidence of chronic lung damage in recovered koalas.

Streptobacillary rat-bite fever in man is usually acute;^{6,16} however, no adequate histological description of the lesions has been noted in the literature. In this case, the pulmonary response to *S. moniliformis* was granulomatous in nature.

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