Caprine vertebral osteomyelitis caused by *Rhodococcus equi*

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ABSTRACT

There have been reports of *Rhodococcus equi* infections in goats in Australia, America and India. In this study, *R. equi* was isolated from an inflamed vertebra in a Boer goat in South Africa. At autopsy, there was a purulent inflammatory reaction in the 1st cervical vertebra. Histopathologically, a neutrophilic infiltration was encountered in the bone. Aerobic culture of swabs collected from the abscesses yielded *R. equi* in pure culture that was identified on biochemical tests. *R. equi* has become important as the cause of an opportunistic infection in people suffering from HIV.

Key words: caprine infection, purulent vertebral osteomyelitis, *Rhodococcus equi.*

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INTRODUCTION

Rhodococcus equi infections in goats have been reported from Australia[1], America[2] and India[1]. In this study, *R. equi* was isolated from an inflamed vertebra in a Boer goat in South Africa.

CASE HISTORY

A 1-year-old female Boer goat from the Molecular Biology section of the Onderstepoort Veterinary Institute (OVI) was brought to the *post mortem* room for euthanasia. It was weak and recumbent. The goat had been 1 of a group of 38 goats in a heartwater experiment. They had been at Onderstepoort for 3 months. Other than deworming and vaccination against *Pasteurella* and bluetongue that all the animals had received, this goat does not appear to have received any other treatment. It had not shown any abnormalities throughout the 3-month period that the group was at Onderstepoort, and the problem with the neck was noticed just 1 day before euthanasia was requested. The goat stopped eating and it was assumed that it had hurt its neck. On physical examination, it was noticed that the slightest pressure on the 1st cervical vertebra, the atlas, was extremely painful for the goat. It vigorously resisted handling in that area by moving its head and neck, and bleating loudly.

MATERIALS AND METHODS

After euthanasia with intravenous barbiturates, the atlas was examined more closely. Incisions were made with a bone saw into the atlas[9], revealing a purulent inflammatory reaction within the bone. This consisted of a few, large (more than 1 cm across) areas containing yellowish, creamy pus. No other lesions were encountered. The pus was aseptically collected on swabs for bacterial isolation and identification. Samples of the vertebra, surrounding muscles and the section of the spinal cord within the vertebra were fixed in 10 % buffered formalin. After decalcification and trimming, histological sections were made and examined microscopically.

Swabs were processed for routine isolation using blood tryptose agar (BTA) with MacConkey agar to detect any Gram-negative contaminants that may be present. Blood tryptose agar plates were incubated in a CO_2 incubator at 37 °C for 24 to 72 hours. The isolate was subjected to standard biochemical tests, including catalase, oxidase and indole. A pure colony of the isolate was inoculated on biochemical substrates and incubated at 37 °C for 24 to 48 hours. The substrates used were Hugh and Leifson's oxidative/fermentative medium (OF), deoxyribonuclease agar (DNA), phenolphthalein diphosphate medium (PDP), nutrient gelatin stab medium, egg yolk agar, glucose, aesculin, nitrate, urea, casein, maltose and sucrose.

RESULTS

Histopathology

Resorption of the mineralised component of bone was a major feature in these lesions. There were widespread areas with only fibrous connective tissue where resorption of the osteoid had taken place. Within such fibrous connective tissue, there was an intense inflammatory reaction characterised by a marked infiltration of inflammatory cells, particularly neutrophils. Many multinucleated osteoclasts could be seen in resorption lacunae (also known as Howship's lacunae) aggressively corroding the osteoid. (see Figs 1, 2). There were no lesions in the spinal cord, not even in the segment of spinal cord passing through the affected vertebra.

Bacteriology

Macroscopic examination of BTA plates after 24 hours revealed the presence of a pure culture of small, smooth, shiny and non-haemolytic colonies. The colonies became larger, mucoid and salmon-pink after 72 hours. Microscopic examination of Gram-stained smears from the 24 hour colonies showed pleomorphic, Gram-positive coccobacilli with cocci predominating.

Negative reactions were observed on the following substrates: aesculin, deoxyribonuclease agar (DNA), casein, gelatine, egg yolk agar (lecithinase and lipase activities negative), no gas formation, Hugh and Leifson's O/F basal medium, carbohydrates that include glucose, maltose and sucrose. The isolate was oxidase negative. The positive reactions observed were catalase, splitting of urea, nitrate reduction and presence of phosphatase.

Based on its macroscopic characteristics, microscopic characteristics and reactions on biochemical substrates, the isolate was identified as *Rhodococcus equi* [5,7].

DISCUSSION

Vertebral osteitis occurs in domestic animals, usually involving 1 or 2 vertebrae. It is caused by a variety of microorganisms. It often takes the form of vertebral abscesses or granulomatous lesions. In the horse, *Rhodococcus equi* has been reported as a cause of vertebral osteomyelitis or vertebral osteomyelitis[3].

Osteoclasts are activated and controlled by factors produced by the osteoblasts. Bacterial metabolites, for example

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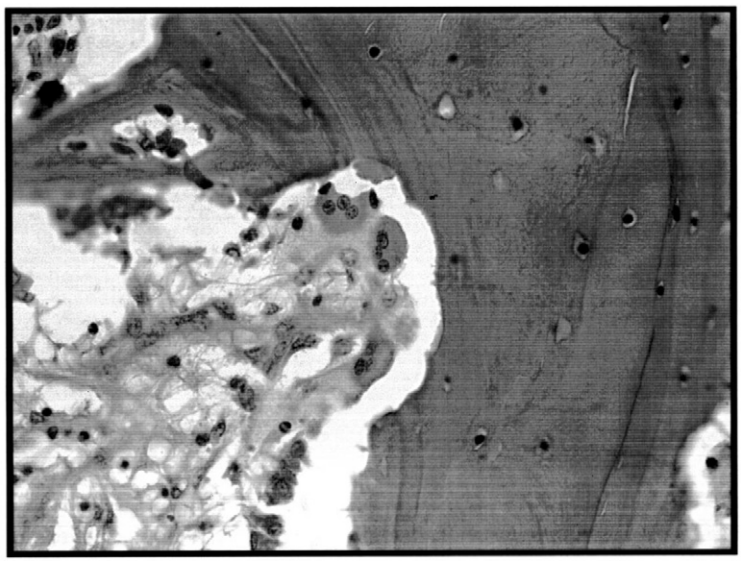

Fig. 1: **Osteoclasts** (multinucleated) **corroding bone, followed in their wake by an acute inflammatory reaction with neutrophils.**

lipopolysaccharide (LPS), are known to influence resorption of bone. They activate osteoblasts to release factors that activate osteoclasts, which are necessary for the resorption of the mineralised component of bone. In this case, there could have been metabolites produced by *R. equi* that may have induced resorption of bone, thereby creating optimum conditions for the bacterium's own spread through the bone[6].

Magnusson was the 1st to describe *R. equi*[1]. It was 1st isolated in Sweden in 1923, where it was associated with purulent pneumonia in foals[4]. The infection was subsequently confirmed in other countries of the world[4]. *Rhodococcus equi* infection is seen in foals from 1–5 months of age, the greatest number of cases occurring between 1 and 3 months of age. The infection usually causes a chronic bronchopneumonia, sometimes with an ulcerative typhlocolitis and mesenteric lymphadenopathy[3].

Rhodococcus equi has been isolated from the cervical lymph nodes of pigs and within hepatic granulomatous lesions in young goats, some of which wasted away and died. Infection in other species is rare, and when it occurs, the animals are often immunosuppressed. Of the 3 goats with a *R. equi* infection that were reported by Carrigan *et al.* in Australia[1], 1 had an osteomyelitis involving the 11th thoracic vertebra. This had led to compression of the spinal cord, which in turn caused posterior paralysis. None of the lesions encountered in that goat were found in any organs or tissues other than bone. The inflammatory reaction in the goat we saw was in the 1st vertebra only, and the section of spinal cord passing through it was unaffected.

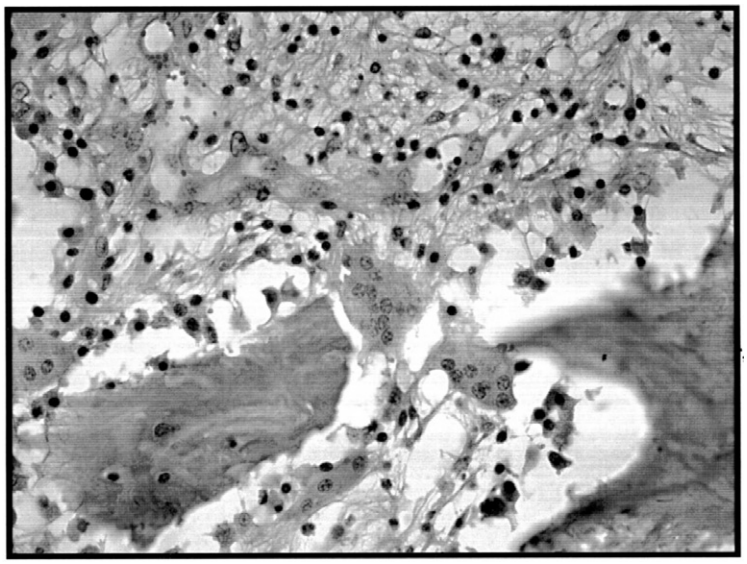

Fig. 2: Bottom half: **two large, multinucleated osteoclasts in the middle, with bone spicules on either side;** top half: **an acute inflammatory reaction with neutrophils. Resorption of bone is facilitating the spread of this infection and inflammation in the bone.**

In humans, its identification is subject to many pitfalls. Incorrect identification as diphteroids or atypical *Mycobacteria* has frequently led physicians to discount stain or culture results as contaminants when *R. equi* was, in fact, the pathogen. For this reason, the spectrum of clinical disease caused by *R. equi* is probably underestimated. It has become an important pathogen of humans infected by the Human Immunodeficiency Virus (HIV), where it causes pulmonary abscesses[2,8]. *Rhodococcus equi* infection in goats has been reported in Australia[1], the United States of America[2] and India[1]. The work reported in this paper shows that *R. equi* infection in goats also occurs in South Africa.

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