Case Report

Tuberculosis of the parotid salivary gland in a kudu *Tragelaphus strepsiceros*

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Tuberculosis is a bacterial disease which has a worldwide distribution and affects most animal and bird species. In the eastern Cape this disease has been known to affect the kudu since the turn of the century. Pre-existing lesions on the ears become infected with mycobacteria which causes swelling of the parotid gland and lymph nodes draining the area. In this article a case of tuberculosis is described affecting the parotid salivary gland and surrounding pre-auricular lymph nodes. The importance of tracing these lesions is stressed in order to prevent infection of people consuming the meat.

Key words: tuberculosis, kudu, *Tragelaphus strepsiceros*, parotid salivary gland.

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Introduction

Tuberculosis remains one of the most widespread fatal bacterial diseases in man and animal, despite intensive research to combat the disease and to treat victims. Although susceptibility to tuberculosis differs between species, the disease occurs in all domestic animals and birds, as well as in most wild animals that are in contact with tuberculous livestock or man. Especially in cattle, pigs and domestic fowl, the disease is of great concern. It is caused by acid-fast bacilli of the genus *Mycobacterium*. The three most important species of mycobacteria that cause tuberculosis are *M. tuberculosis* which is maintained in man, *M. bovis* maintained in most animals and *M. avium* maintained mainly by domestic fowl. The organisms differ from each other with regard to culture characteristics, antigenic composition and pathogenicity in different species (Jones & Hunt 1983). Tuberculosis is transmitted in different ways by infected animals, viz. air or saliva, faeces, urine, vaginal secretions, milk and draining fistulas of primary lesions on the skin (Boever 1986).

Kudu *Tragelaphus strepsiceros* contracted tuberculosis from infected cattle and consequently spread the disease over large areas. Specific habits of the kudu and the way it reacts to the infection contributed to the spread of the disease. High humidity, limited water holes (mud holes) and the continuous release of infected material from draining lesions of infected kudu on the vegetation adjacent to game paths and water holes promoted the transmission of tuberculosis to game animals. The dense, low vegetation in large parts of the eastern Cape influenced the spreading of the disease by forcing animals to make use of the same routes to move through the contaminated vegetation and by providing a suitable environment for the causative organisms.

Paine & Martinaglia (1928), described tuberculosis in the kudu for the first time (Thorburn & Thomas 1940). Robinson (1944) and Himes *et al.* (1976) made further contributions. Paine & Martinaglia (1928) and Thorburn & Thomas (1940) determined that *M. bovis* was the causative agent in the kudu. In most reported cases of tuberculosis in the kudu, uni-

Fig. 1. Tuberculous granulomas (black arrows) with cystic degeneration (white arrows) in the parotid salivary gland measuring 9x8x6 cm.

lateral or bilateral swelling of the parotid areas was the first visible lesion. The lesions gradually increase in size, become softer and then drain a thick, creamy exudate onto the covering skin under the ear for a long period. The infection eventually spreads to the cervical, retropharyngeal and other distant lymph nodes with consequent disseminated infection. It is noteworthy that in most cases the disease has little influence on the general health state of the kudu and infected animals cannot be easily identified in a herd

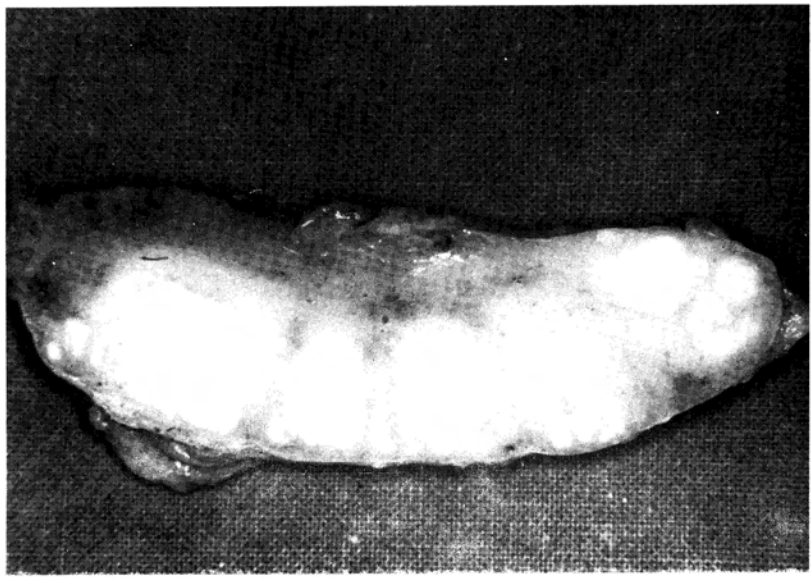

Fig. 2. A surrouding lymph node measuring 7x2x2 cm, with multiple caseating granulomas.

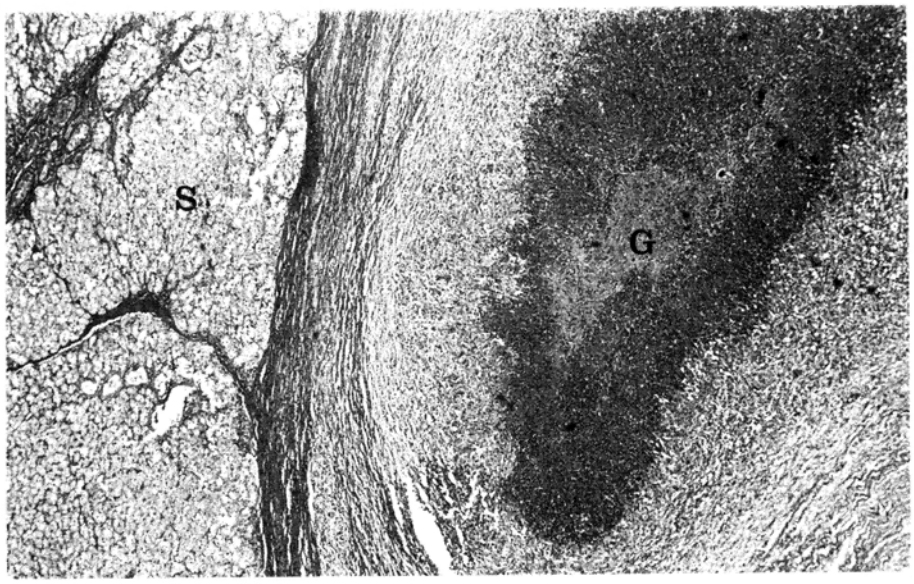

Fig. 3. Tuberculous granulomas (G) in the parotid salivary gland (S). Note the area of central caseating necrosis (arrow). (Haematoxylin and eosin x 30).

(Thorburn & Thomas 1940). The fact that the primary lesion occurs in the parotid area, which is situated below the ear is explained by the above authors who state that the primary inoculation of organisms takes place through injury to the skin of the exceptionally large ears by means of thorny branches, ticks or horn-flies (*Lyperosia* sp.). The animals scratch the irritating ear lesions with the hoof of the hind limb and in doing so transmit soil contaminated with mycobacteria onto the lesions. Tuberculous lesions eventually develop

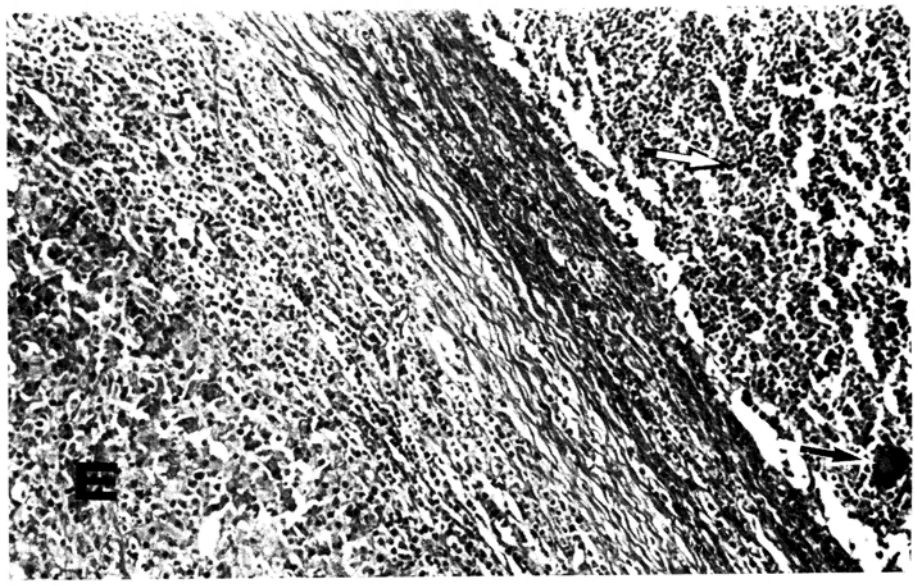

Fig. 4. Multiple epitheloid cells (E) lymphocytes (white arrow) and some Langhans giant cells (black arrow) are visible in the granulomas (Haematoxylin and eosin x 130).

in the regional lymph nodes. This method of infection was experimentally reproduced in cattle (Thorburn & Thomas 1940).

Published reports on cases of tuberculosis in kudu are limited. The purpose of this report is to describe a case of tuberculosis affecting the parotid salivary gland of a kudu in the Addo Elephant National Park.

Materials and methods

During routine culling of kudu in the Addo Elephant National Park, saliva samples of 10 kudu randomly selected were collected for determining amino-acid levels as part of a research project of the Centre for Wildlife Research, University of Pretoria.

The parotid and submandibular salivary glands were dissected after which several mass readings were taken. During the dissection procedures, a swelling, 10 cm in diameter on the left side of a normal adult kudu cow was detected. No conspicuous exudate was visible on the skin but after careful examination a lesion involving the parotid salivary gland was observed, from which a yellow creamy exudate drained after opening. The normal glandular tissue was replaced by granulomatous tissue and nodules of yellow-white caseating necrosis. Extensive cyst formation was evident (Fig. 1). The surrounding preauricular lymph nodes were enlarged and showed nodules of yellow-white caseating necrosis (Fig. 2). No other lesions were found in the head and neck region. The remaining carcass was not examined for tuberculous lesions.

Histopathological examination of haematoxylin and eosin stains of the affected tissues revealed the following lesions:

The acini and stroma of the parotid salivary gland and lymphoid tissue of the lymph nodes were replaced by granulomas in the centre of which areas of caseous necrosis were evident. Multiple epitheloid cells, lymphocytes and a few Langhans gaint cells formed part of the granulomatous reaction (fig. 4). Acid-fast bacilli consistent with that of *Mycobacterium* were identified by means of the Ziehl-Neelsen staining method which is specifically used to identify these organisms. Bacterial isolation was not attempted because field examination of the carcass did not indicate a tuberculous lesion — only histological examination revealed its presence.

Discussion

Our results corroborate previous findings that primary tuberculous lesions occur in the parotid lymph nodes in particularly kudu in the eastern Cape. These lesions could probably originate from direct contamination of pre-existing lesions on the ears which are drained by the parotid lymph nodes. As judged from their body mass, the kudu in this study appeared healthy, confirming that the animals can be asymptomatic carriers of the disease for long periods and spread the organisms over large areas.

The fact that the parotid salivary gland was affected, is unusual. All the surrounding preauricular lymph nodes contained granulomas and the infection could have spread from the lymph nodes to the parotid salivary gland. Alternatively, primary infection of the parotid salivary gland could have taken place via an oral lesion with consequent spread to the surrounding lymph nodes. The yellow creamy exudate in the lesion also corroborate previous finding regarding kudu (Thorburn & Thomas 1940).

Seeing that kudu are often culled or used for biltong, special care should be taken to identify tuberculous lesions and to prevent contaminated carcasses from becoming commercially available.

Acknowledgement

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