Streptococcus phocae infections associated with starvation in Cape fur seals

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ABSTRACT

Mortalities and abortions associated with starvation occurred at Cape Cross, Namibia, in Cape fur seals (*Arctocephalus pusillus pusillus*). Affected seals showed lethargy and emaciation, and the most common pathological signs were those of a respiratory infection, both in adults and offspring. *Streptococcus phocae* was isolated from adult seals, a cub and aborted foetuses.

Key words: infection, seal, *Streptococcus phocae.*

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INTRODUCTION

Streptococcal infections are common in animals both as primary and secondary causes of disease. Infections due to *Streptococcus* species have been described in seals[1–3,10,11]. These include septicaemia[1,6,10], navel ill[1], bronchopneumonia[1–3], oral ulcer[3] and pyoderma[11]. During 1988 an epizootic outbreak of seal (phocine) distemper, characterised by respiratory signs, abortions and deaths, occurred in the seas surrounding northwestern Europe[6,7]. Phocine distemper is caused by a morbillivirus related to canine distemper virus[7]. *Streptococcus phocae* was often found as a secondary invader in these cases[6,7]. The most common pathological signs attributable to *S.phocae* were those of respiratory tract infection.

Seals belong to the order Pinnipedia, the members of which all are aquatic mammals, divided into 3 families: Phocidae, the true seals; Otariidae, the fur seals and sea lions, and Odobenidae, the walrus[5]. With the exception of some members of the Phocidae, all are marine. Only 1 species, the Cape fur seal (*Arctocephalus pusillus pusillus*), is endemic to southern Africa.

Infection with *Streptococcus phocae* has been described in harbour seals (*Phoca vitulina*)[7,10,11] and grey seals (*Haliochoerus grypus*)[1,3], both of which belong to the family Phocidae or true seals[9]. The reported cases of *S. phocae* infection were from seals originating in the north Atlantic Ocean, from the coastline of the British Isles[1–3], Scandinavia[6,7], the Netherlands and Germany[8].

Large seal colonies of Cape fur seal occur along the western and southern coasts and associated islands of southern Africa, from southern Angola to Algoa Bay in South Africa. The total population, estimated at 1.1 million in 1984[5], was increasing at a rate of 3.7 % annually. Cape Cross, in northwestern Namibia, is one of at least 24 major breeding localities of these seals[9]. The total seal population at Cape Cross in 1994 was estimated at 80 000 animals.

The adults feed on schools of small fish, foraging to at least 160 km offshore. In Namibia the small bearded goby (*Sufflogobius bibarbatus*) contributes slightly more than 50 % of the diet[5].

Gestation, including delayed implantation, is 12 months, and most seal pups are born in late November and early December[5,9]. The female seal remains with the pup for the 1st week, during which time mating takes place, before returning to the sea to feed[5].

The South Atlantic waters off the coast of Namibia are well-known as very rich fishing grounds. This is due to upwelled water carried northwards by the Benguela system. This normal upwelling of water, which is most intense in summer, brings nutrients from below the euphotic zone, resulting in dense blooms of phytoplankton that form the base of the food chain[4].

This report documents the isolation of *S. phocae* from large-scale seal mortalities in the Cape Cross area.

CASE HISTORY

During August 1994, mortalities and abortions in Cape Fur Seals started occurring at Cape Cross. By October approximately 10 000 seals had died, approximately 20 000 seals were affected, and approximately 15 000 abortions had occurred. There was an associated shortage of fish in the feeding areas of the seals that started in 1994 and lasted for the whole of 1995 (J H M David, Sea Fisheries Institute, pers. comm., 1998).

Seals that were examined during October and November showed weakness, listlessness, emaciation and apathy. All age groups and both sexes were equally affected. Aborted foetuses were also noticed. Many female seals appeared normal before aborting, whereafter they became emaciated and died after about 10–14 days.

Twelve seals, comprising 9 females, 2 foetuses and 1 cub, were necropsied. All were emaciated. The subcutaneous fat layer in adult females was either absent or less than 1 cm thick. A moderate mucopurulent tracheitis and mild multifocal purulent pleuritis were seen in some cases. Pulmonary oedema and areas of atelectasis were present in others. The gastrointestinal tract of 1 female showed gastric ulcers and another one moderate haemorrhagic enteritis. Both round- and tapeworms were present, but they were not specifically identified. A moderate hydropericardium was seen in some females. No specific macroscopic lesions were discernible in the foetuses and cub.

The microscopic lesions were mild and variable. Two of the adult females had lungworm infections with associated proliferative interstitial pneumonia characterised by mild to moderate lymphocytic, neutrophilic and eosinophilic cell reactions, mainly in the septa. Some pulmonary atelectasis was present in 2 adults. Localised proliferative interstitial pneumonia with multifocal septal lymphocytic proliferation and focal vascular thrombosis were present in another female. This animal also had mild multifocal pyogranulomatous hepatitis in association with a few suspected cocci as well as a hepatic haemorrhage with distinct Gram-positive diplococci. In addi-

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tion, foci of lymphocytic reaction were found in the uterus as well as necrotising bacillary inflammation in the placenta. Hepatic and splenic atrophy, mild mononuclear portal hepatitis, mild centrilobular fatty degeneration and pigmentation of the liver were seen in another female. One seal had deep-seated multifocal, necro-proliferative and eosinophilic ulcerative gastritis. Mild to moderate semi-purulent enteritis with necrotic cryptal debris was present in 2 females, 1 of which had tapeworms. Localised myocardial degeneration, together with a neutrophil reaction, scattered macrophage infiltrates and mild haemorrhage occurred in 1 case. A few small mononuclear foci were found in the renal cortex of 2 animals. Lesions in the brain in 1 adult consisted of mild meningeal haemorrhage, congestion and oedema. One foetus revealed congestion and oedema of the lungs, and the other foetus as well as the cub, had mild multifocal haemorrhages in the cerebellar cortex and meninges. No other lesions were seen in the foetuses and cub.

Samples were cultured for viruses by incubation on monolayers of baby hamster kidney 21, rabbit kidney 13 and foetal cat kidney cells, but no viruses were isolated. Fluorescent antibody staining with canine distemper virus conjugates on various tissues, including lung, urine sediment and buffy coats, were all negative. Culture for *Mycoplasma* was negative. Culture for tuberculosis was negative although an environmental *Mycobacterium* species that could grow only at 27 °C was isolated from 1 animal. The *Mycobacterium* isolate was not further identified.

A β-haemolytic *Streptococcus* was isolated from 5 adult female seals, 1 cub and 3 aborted foetuses. Isolates were made from most of the organs submitted in almost all cases (lung, spleen, liver, kidney, lymph node, uterus, placenta, trachea and conjunctiva), indicating that the seals were suffering from a septicaemic condition. The organs from foetuses were pooled and the same isolate was isolated from each organ pool. The isolate formed white colonies surrounded by large zones of beta haemolysis on bovine blood agar. The isolates grew equally well under aerobic and anaerobic conditions at 37 °C but did not grow at 45 °C. They were all catalase-negative, non-motile Gram-positive cocci that formed chains when grown in liquid medium. The isolates did not produce acid from lactose, mannitol, raffinose, salicin, sorbitol, trehalose, inulin, sucrose, arabinose, galactose, glycerol, dulcitol or adonitol. They produced acid from ribose, maltose, fructose, glycogen and mannose. Acid was weakly produced from xylose after prolonged incubation. Neither arginine, esculin, gelatine nor sodium hippurate were hydrolysed, and only 1 isolate hydrolysed starch weakly. They were all resistant to optochin and sensitive to bacitracin. None of the isolates grew in methylene blue milk, and litmus milk became slightly alkaline. The isolates produced phosphatase. Based on these biochemical characteristics, the isolate was identified as *Streptococcus phocae*[8].

Other bacteria isolated from the seals were *Streptococcus canis* together with *Edwardsiella tarda,* from 1 adult seal, where *S. canis* was isolated from the spleen, liver and kidney and *E. tarda* from the trachea and conjunctiva. *Streptococcus zooepidemicus* was cultured from 2 adult female seals that had septicaemic *S. phocae.* The isolates of *S. zooepidemicus* were made from the conjunctiva and trachea of 1 seal and the conjunctiva of the other. No *Brucella* was isolated, although the culture conditions were conducive for the isolation of *Brucella.*

DISCUSSION

The previously described isolates of *S. phocae* from seals were from cases of phocine distemper virus infection, where secondary infection by bacterial opportunists occurred[8]. In other cases, where the *Streptococcus* isolates were not identified, but were most probably *S. phocae*[2,3,10,11] a secondary role was also attributed to the *Streptococcus.* These were pneumonia[2,3,10] due to a combination of a *Streptococcus* and a heavy nematode infection[2], or an unidentified viral infection[3] or as a sequel to pyogranulomatous laryngitis.

It is unlikely that phocine distemper played a role in the large number of seal deaths occurring off the coast of Namibia, as no virus was cultured, and neither was viral antigen detected using fluorescent antibody techniques. The antigen used in fluorescent tests was prepared from canine distemper virus, but a degree of cross-reaction generally occurs[7]. No other infectious cause of such a large outbreak of mortality and abortion could be established and it is assumed that the primary cause of the deaths and abortions was starvation due to a shortage of fish, and that *S. phocae* was present as a secondary opportunist. The lack of pronounced bacterially-induced pathological lesions supports this. The week that female seals spend ashore after partus as well as the mild worm infection probably exacerbated the weakness due to starvation. Although *S. phocae* was the most common pathogen, the other bacteria may also have played a role. Similar isolates were incriminated in seal deaths in other parts of the world. *Streptococcus canis* and *S. zooepidemicus* have been cultured from cases of pneumonia[2,3] pleuritis[1], conjunctivitis[1] and peritonitis[1] in grey seals and exudative pyodermatitis in northern elephant seals (*Mirounga angustirostris*)[11]. *Edwardsiella tarda* has been isolated from peritonitis in sea lions (*Eumetopias jubatus* and *Zalophus californianus*)[11] and an aborted grey seal foetus[3].

Previous cases of *S. phocae* have all been described in northern hemisphere seals, and not in the Cape fur seal, nor other members of the Otariidae.

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