Aquatic Mammals 1987, **13.3**, 105–108

Cutaneous mycobacteriosis in a Southern sealion

J. C. M. Lewis*

International Zoo Veterinary Group, Al Ain Zoo and Aquarium, P. O. Box 1204, Al Ain, Abu Dhabi, United Arab Emirates

Summary

A clinical case is described suggesting that cutaneous lesions in pinnipeds may result from mycobacterial infection.

Mycobacterium fortuitum was isolated from biopsied skin nodules that arose in a juvenile Southern sealion, and from sediment of centrifuged pool water. The histopathology of lesions is described, and possible lines of treatment discussed.

Introduction

Captive pinnipeds are susceptible to a variety of skin problems: these include traumatic wounds, bacterial folliculitis and pustular dermatitis, pediculosis, demodicosis, seal pox and perhaps less commonly mycotic infections. A clinical case is described which suggests that cutaneous mycobacteriosis could be added to the list.

Case report

Two Southern sealions (*Otaria flavescens*) are maintained at Al Ain Zoo and Aquarium in the United Arab Emirates. The exhibit consists of a 160 000 litre, kidney shaped pool measuring approximately 19 metres (length) by three metres (width), by 2.2 metres (depth) plus an adjacent land area of 70 square metres. The water is mechanically filtered at 30 cubic metres per hour continuously and completely changed twice weekly. It is not salinated. Only one male and one female are kept at any one time. Diet consists mainly of sardines, with mackerel when available, plus salt and fisheater multivitamin tablets (Mazuri) daily.

In October 1985 a juvenile female Southern sealion, weighing approximately 50–60 kg was brought to replace an adult female that had died 12 months previously from unknown causes. On arrival it was in apparently good health and remained so until May 1986 when multiple raised nodules, $\frac{1}{2}$–1 cm in diameter, appeared in the skin over the back and flanks with a single lesion on the head (Fig. 1). Neither overlying hair loss nor ulceration were seen at this stage. No systemic illness was noted, appetite and weight remaining normal. It was caught by net and two lesions removed by excision biopsy under local anaesthesia. The skin was prepared with chlorhexidine ('Hibitane', ICI) and 70% alcohol, followed by local infiltration with xylocaine. 125 mg nortestosterone proprionate ('Anabolin-forte', Farvet Labs) were given intramuscularly and vitamin supplements increased to four Mazuri Fisheater tablets daily.

A direct smear of biopsy material was negative for acid fast bacilli (AFB), but Mycobacterium fortuitum was isolated from homogenised biopsy tissue on both acid egg and pyruvic acid egg media at 37°C within seven days. Slower growth was noted at 26°C. Mycobacterium fortuitum was also grown on these media from sediment obtained by centrifuging six litres of pool water. Cultures were maintained at 37°C on both media by serial seeding and species identification was carried out by the PHLS Regional Centre for Tuberculosis, Dulwich (Collins *et al*, 1985). No other significant bacterial or fungal pathogens were isolated. Biopsy material for histopathology was fixed in 10% buffered formal saline and 4 μ sections prepared from paraffin wax blocks and stained with haematoxylin and eosin. Histologically, the epidermis displayed parakeratosis with marked acanthosis, and incontinence of pigment in the germinative layer. A mixed inflammatory cell infiltrate was seen throughout upper layers of the dermis (Fig. 2), with patchy infiltration of the lower layers concentrated mainly in perivascular areas and apocrine sweat glands. Focal accumulations of acute inflammatory cells were present in both dermis and epidermis, in some cases constituting microabscesses. Fibrosis was not a feature. Further sections stained by Periodic Acid Schiff, Grocott-Gomori silver and giesma techniques failed to reveal either parasitic or dermatomycotic infections. Acid fast bacilli were not identified in Ziehl-Nielson preparations.

Oxytetracycline was given at 1 gram b.i.d. per os for two weeks followed by a combination of rifampicin (600 mg, u.i.d.), trimethoprim/sulphadiazine (240 mg/1200 mg, u.i.d.) and oxytetracycline (1 gm

*Present Address: Department of Veterinary Science, Zoological Society of London, Regent's Park, London NW1 4RY.

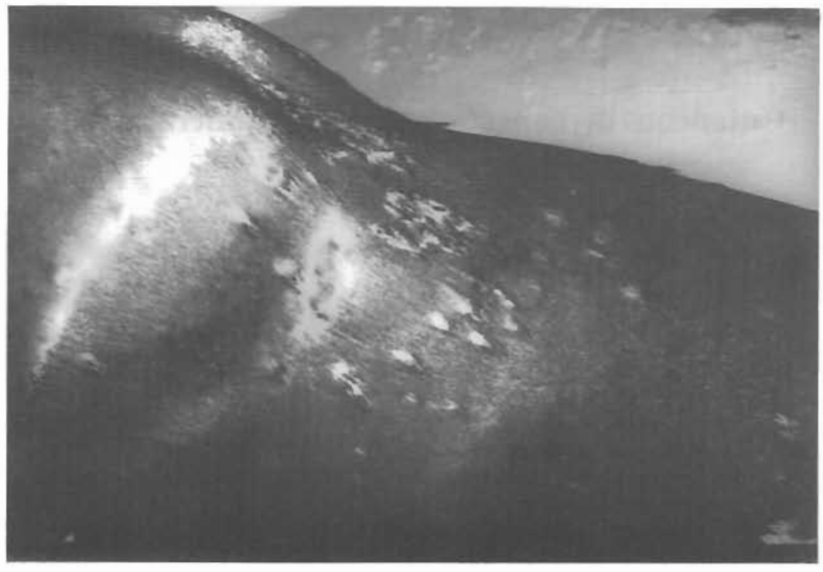

Figure 1. Gross appearance of early skin lesions.

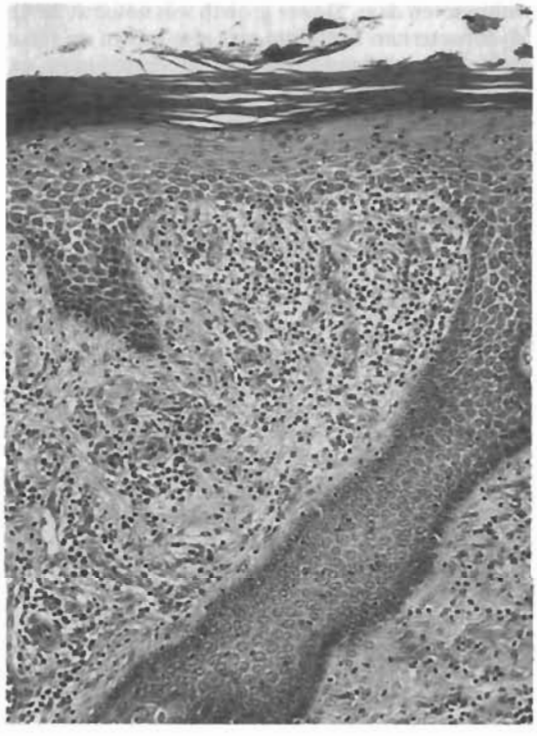

Figure 2. Dermal infiltrate of early skin lesion.

b.i.d.) per os, for a further four weeks. The lesions showed considerable regression over the first ten days of triple drug therapy, but recurred over the next three weeks. During this period lesions gradually increased in size, number and severity, with a wider distribution over the head, neck flanks and flippers. Ulceration and hair loss was noted over larger nodules. Hyporexia and weight loss became apparent and triple drug therapy was stopped after thirty days. A wider variety of fish was offered and Img ethylestrenol given daily by mouth. Lesions resolved fully over the following month and the animal regained its lost weight and appetite. Steroid therapy has been continued to date (January 1987) and the animal's general health and skin condition remain satisfactory.

Discussion

So called typical mycobacteria—M. tuberculosis, M. bovis, M. leprae—are obligate animal pathogens and do not multiply outside their hosts. Thus tuberculosis and leprosy are directly transmitted between animals. In contrast, the large number of 'atypical' mycobacteria species are free living soil and water saprocytes and only occasionally opportunistic pathogens. However, many are capable of causing skin lesions— typically granulomas or pyogranulomas— in a wide variety of mammals, reptiles, amphibians and fish under appropriate conditions. Species involved include M. fortuitum, M. chelonei, M. marinum, M. xenopi and several others (for further

details refer to Fowler, 1986; Jubb *et al*, 1985; Klös & Lange, 1982 or other standard veterinary textbooks). Mycobacterial skin disease has been reported in marine mammals previously; for example a cutaneous infection with M. chelonei was described in a Natterer manatee (Boever *et al*, 1976). Multiple pyogranulomatous lesions were seen in the skin, although cause of death was attributable to pulmonary lesions. Unidentified mycobacteria were also seen in nodular skin lesions in association with Pseudomonas aeruginosa in an Atlantic bottle-nosed dolphin which died of bronchopneumonia (Diamond *et al*, 1979).

In the case described above a diagnosis of cutaneous mycobacteriosis is offered based on three pieces of evidence. Firstly, M. fortuitum was cultured from biopsied material in the absence of other bacterial or fungal pathogens. That AFB were not seen in Ziehl-Nielsen stained tissue sections is of little significance as they are often very difficult to demonstrate in cutaneous mycobacterial lesions after paraffin processing (Arai *et al*, 1984; Jubb *et al*, 1985). Secondly, M. fortuitum was demonstrated in the pool water, thereby establishing environmental contamination. Thirdly, the histopathology described is consistent with a subacute bacterial infection. Although many cutaneous mycobacterial infections are characterised by granuloma formation, this is not invariably so in the early stages (Philpott *et al*, 1963; Lever & Schaumburg-Lever, 1983). Unfortunately, lack of suitable handling facilities precluded later biopsies which may have demonstrated further histopathological changes in older or aged lesions. Whilst clinically, a list of differential diagnoses must include sealpox, San Miguel virus lesions, mycotic infections, neoplasia, ectoparasitism, foreign body reactions and even leishmaniasis, these are easily discounted in the case described by the histological and microbiological investigations.

It is not clear from the case described whether specific antimicrobial treatment for cutaneous mycobacteriosis in pinnipeds is necessary. Whilst the appetite and weight loss observed when lesions were extensive might suggest that such therapy is desirable, the marked response to anabolic steroids implies that improvement in general health status may be more important. This is in keeping with the opportunistic nature of atypical mycobacterial infections. Antibiotic therapy in this case was ultimately unsuccessful. Indeed the initial apparent response to the triple drug therapy may be attributable to the residual effect of parenteral anabolics given at the time of biopsy. Owing to restricted facilities available at Al Ain, *in vitro* sensitivity of the M. fortuitum isolate was not established, hence the use of a broad spectrum triple drug regimen. However, as atypical mycobacteria often demonstrate multiple drug resistance (Kuze *et al*, 1981) *in vitro* sensitivity testing is very desirable if specific antibacterials are to be used. This may only be practical with mycobacteria that grow relatively rapidly *in vitro* such as M. fortuitum and M. chelonei.

In conclusion, it is suggested that M. fortuitum can cause skin disease in pinnipeds kept in poor environmental conditions and fed a restricted diet. The disease is characterised by the acute appearance of multiple cutaneous nodules. Overlying hair loss and epidermal ulceration are not necessarily features, but may occur when the condition is more advanced. Histopathologically, early lesions demonstrate a mixed inflammatory cell infiltrate in the dermis with thickening of the epidermis. Mycobacteria can be cultured from biopsied material. Gradual resolution of skin lesions appears to coincide with improved general health and/or use of anabolic steroids. Systemic signs are seen where lesions have become extensive. Clearly more cases need to be described before firm conclusions should be drawn. However, clinically similar skin lesions have been seen in many pinnipeds (D. C. Taylor-personal communication) although they have not been characterised with respect to histopathology or aetiology. Skin nodules taken from two other Southern sealions maintained at Al Ain Zoo between 1982 and 1986 showed similar histopathological appearances to the case described, although in addition both foreign body and Langhans type giant cells were seen in the dermal infiltrate. Few other details are available, but these may have been more advanced lesions of the same type. It remains to be seen how commonly skin lesions in pinnipeds are caused by M. fortuitum or other atypical mycobacteria, but at least consideration should be given to the possibility of cutaneous mycobacteriosis in cases where skin nodules are a prominent feature.

Acknowledgements

Jeremy Hillsdon-Smith and his staff of the Department of Microbiology, Al Ain Tawam Hospital, for microbiology services and advice.

Maggie Haddon of the Department of Histopathology, Al Ain Tawam Hospital, for her patient technical advice and assistance.

Heinz Elle of Al Ain Zoo for his photography and general help.

Mr Sangam for dietary advice and provisions.

Sheikh Zayed bin Sultan al Nahyan for his continued support of the veterinary department, Al Ain Zoo.

References

Arai, H. *et al* (1984) Mycobacterium marinum infection of the skin in Japan. *J. Dermatol. (Tokyo)*, **11**, 37.

Boever, W. J. *et al* (1976) Mycobacterium chelonei infection in a Natterer manatee. *J.A.V.M.A.* **169**, 927.

Collins, C. H. *et al* (1985) Organisation and practice in tuberculosis bacteriology. p. 79 Butterworths; London.

Diamond, S. S. *et al* (1979) Fatal bronchopneumonia and dermatitis caused by pseudomonas aeruginosa in an Atlantic bottle-nosed dolphin. *J.A.V.M.A.* **175,** 984.

Fowler, M. E. ed. (1986) Zoo and wild animal medicine: 2nd Ed., p. 103, p. 575. W. B. Saunders, London.

Jubb, K. V. F. *et al* (1985) Pathology of Domestic Animals, Vol. 1: pp. 478–480. Academic Press Inc., London.

Klös, H.-G. & Lang, E. M. (1982) Handbook of Zoo Medicine. p. 371; p. 384. Van Nostrand Reinhold Co., London.

Kuze, F. *et al* (1981) *In vitro* and *in vivo* susceptibility of atypical mycobacteria to various drugs. *Rev. Infect. Dis.*, **3,** 885.

Lever, W. F. & Schaumburg-Lever, G. (1983) Histopathology of the skin. 6th ed. Lippincott, Philadelphia.

Philpott, J. A. *et al* (1963) Swimming pool granuloma. *Arch. Dermatol.*, **88,** 158.