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SEALPOX IN CAPTIVE GREY SEALS (*HALICHOERUS GRYPUS*) AND THEIR HANDLERS

Brad D. Hicks[1] and Graham A. J. Worthy[2]

ABSTRACT: Histopathologic, ultrastructural, and negative-staining studies indicated that nodular lesions on the flippers, head, and necks of recently weaned, captive grey seals (*Halichoerus grypus*) were similar to sealpox lesions reported from several other species of seals. Virions associated with the nodules were characteristic of the parapoxvirus subgroup of pox viruses. Two of the three persons handling the seals developed nodular lesions similar to "milker's nodules," the characteristic lesion in persons infected with parapoxvirus. The clinical course of the parapoxvirus infection in both the grey seals and their handlers is described. It was concluded that although sealpox is transmissible to man, the mild clinical manifestations place it in the nuisance category of zoonotic diseases.

INTRODUCTION

Sealpox infections have been described in harbor seals (*Phoca vitulina*) (Wilson et al., 1972b), South American fur seals (*Otaria byronia*) (Wilson and Poglayen-Neuwall, 1971) and northern fur seals (*Callorhinus ursinus*) (Hadlow et al., 1980). Diagnosis is based on the characteristic gross and histopathologic appearance of the lesions (Okada and Fujimoto, 1984) and on finding typical virions in negatively stained preparations in the electron microscope (Wilson and Sweeney, 1970). The gross and histopathologic appearance of the lesions and the size and shape of the virions seen in the sealpox lesions are similar to those seen in sheep with Orf (contagious pustular dermatitis), in calves with bovine papular stomatitis (BPS) and in cattle with pseudocowpox (Cheville, 1975). These diseases, classified in the Parapoxvirus subgroup, have been reported to be transmissible to humans (Lane et al., 1981). This report describes an outbreak of sealpox in recently weaned captive grey seals (*Halichoerus grypus*) and documents two cases of human infection resulting in lesions similar to milker's nodules in persons working with the infected seals.

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MATERIALS AND METHODS

Eleven recently weaned grey seal pups were collected from Amet Island, Nova Scotia on 2 February 1983 and transported to holding facilities at the University of Guelph, Guelph, Ontario, where they were to be used in a study of the energetics of the post-weaning fast. Upon arrival in Guelph on 4 February 1983, the seals were placed in a 500 m^2 outdoor enclosure for 10 days. They were then divided into three groups. On 14 February, two groups, group A consisting of four seals and group B consisting of three seals, were placed in separate cement tanks (10 m × 10 m × 1.5 m) containing 2% salt water. The third group (C), consisting of four seals, remained in the outdoor enclosure for an additional 10 wk.

Skin samples from the seals were obtained by surgical biopsy. The freshly excised tissue was divided in half. One piece was placed in buffered 10% formalin, the other was placed in a petri dish containing 2% glutaraldehyde (cacodylate buffered, pH 7.2) and cut into about 1 mm cubes. Tissue samples were processed for light and electron microscopic examination, as previously described (Geraci et al., 1979).

Negatively-stained preparations of lesions from two seals and both seal handlers were examined also. The negatively stained samples were prepared by scraping the lesions with a fresh sterile scalpel blade and suspending the scrapings in 0.1 ml of sterile distilled water. A small droplet of this suspension was then placed on a Formvar coated grid and allowed to dry at room temperature. The grids were then neg-

FIGURE 1. The palmar surface of a young grey seal's flipper covered with large single and coalescing pox nodules.

atively stained with 2% phosphotungstic acid at pH 6.5 and viewed in an Hitachi HS-9 electron microscope.

RESULTS

On 11 February, while the seals were still in the outdoor enclosure, three 1 cm diameter raised lesions were noticed on the right flipper of seal #7 from group A. These lesions increased slightly in diameter over the next week and by 2 March, similar nodular lesions were present on the lower jaw and both forelimbs (Fig. 1). On 7 March, two 1 cm diameter lesions were noticed on the right flipper of another group A seal, #10, and within 1 wk six more lesions appeared on this seal. At this time several raised 1 cm diameter nodules developed on the right hind flipper, the tail and around the identification tag on the left flipper of the remaining seal in this group.

Pox-type lesions were first noticed in group B on 7 March. At this time, each seal had two small lesions on their right foreflippers. Within 10 days the remaining seal in this group had several small nodules on the dorsal and ventral surfaces of both front and hind flippers.

None of the group C seals that remained in the outdoor enclosure for the entire 10 wk study period developed pox lesions. On 15 April, all seals were killed.

On 4 February of the following year, 1984, an additional 14 grey seals collected from St. Georges Bay, Nova Scotia were brought to Guelph and held in the same facilities as the seals in 1983. All seals were held in the dry outdoor enclosure for 14 days. The seals were then divided into three groups. Two groups, each consisting of four seals, were placed in the saltwater-filled cement tanks and the third group of six seals remained in the outdoor enclosure. Six of the eight seals placed in tanks developed typical pox lesions 17 to 20 days after transfer to these tanks. As in 1983, none of the seals held in the dry enclosure developed pox lesions.

The appearance and distribution of lesions were similar in all seals. The nodules were present on the head, neck, and ventral surface of the flippers. The lesions which began as small raised nodules 0.5 to 1 cm in diameter increased in size to about 1.5 cm in diameter, and the surface became eroded over a period of 2 to 3 wk. By the fourth week, the lesions began to recede. However, all seals were euthanized 55 days after capture, before recovery was completed.

The microscopic finding in biopsy specimens from two seals and necropsy specimens from another seal were similar. Hyperkeratosis and parakeratosis were prominent features of the stratum corneum. Numerous bacterial colonies often were seen along the skin surface and

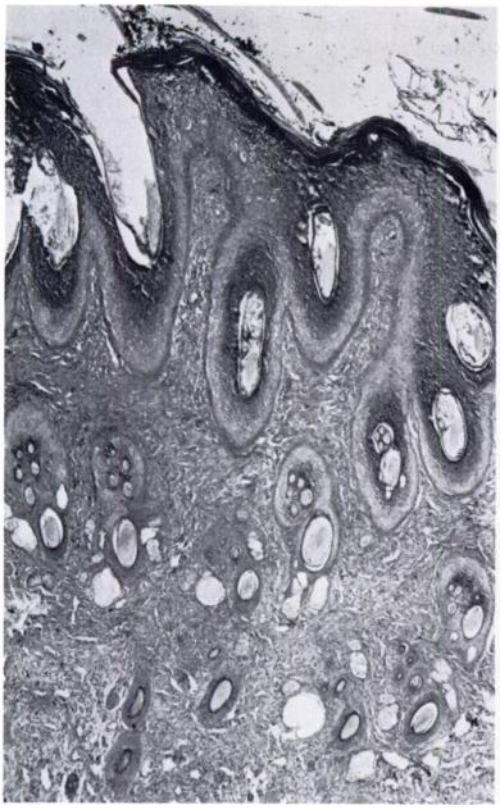

FIGURE 2. Low power photomicrograph illustrating surface debris hyperkeratosis and perakeratosis of the stratum corneum, vacuolization of the stratum spinosum and marked hypercellularity and fibroplasia within the dermis of a grey seal. ×80. H&E.

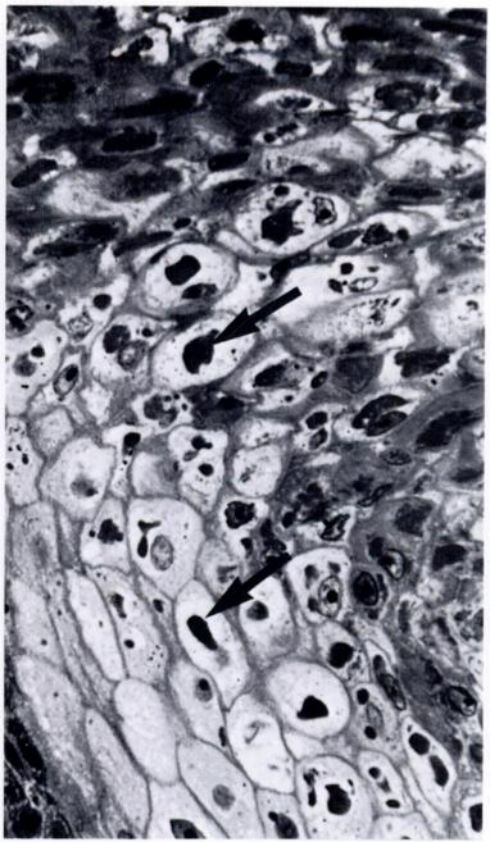

FIGURE 3. Large, dense, irregularly-shaped eosinophilic intracytoplasmic inclusion bodies (arrows) within degenerating keratinocytes of the stratum spinosum of a pox nodule from grey seal #7. ×380. H&E.

packed into fissures that had developed in the outer surface of the skin. There was a marked mixed inflammatory cell infiltrate in the dermis. Neutrophils were the predominant inflammatory cell in the superficial dermis infiltrating around the remains of hair follicles and associated glands. Deeper in the dermis there was a swirling pattern of activated fibroblasts and mononuclear cells (Fig. 2). Large eosinophilic intracytoplasmic inclusion bodies were present in many of the enlarged degenerating stratum spinosum cells (Fig. 3).

Stratum spinosum cells had ultrastructural alterations characteristic of poxvirus infections. Numerous virions were aggregated within the intracytoplasmic inclusion. The cytoplasm was extensively vacuolated and the tonofilaments were extensively fragmented. Nuclear changes included swelling, chromatolysis, and the intranuclear accumulation of microfilaments.

Two of the three persons caring for the seals in 1983 developed single milker's nodule-like lesions on the fifth finger of the right hand (Fig. 4). One of the seal handlers first noticed a lesion appearing on his finger on 23 February, 19 days after the initial contact with the seals. At this

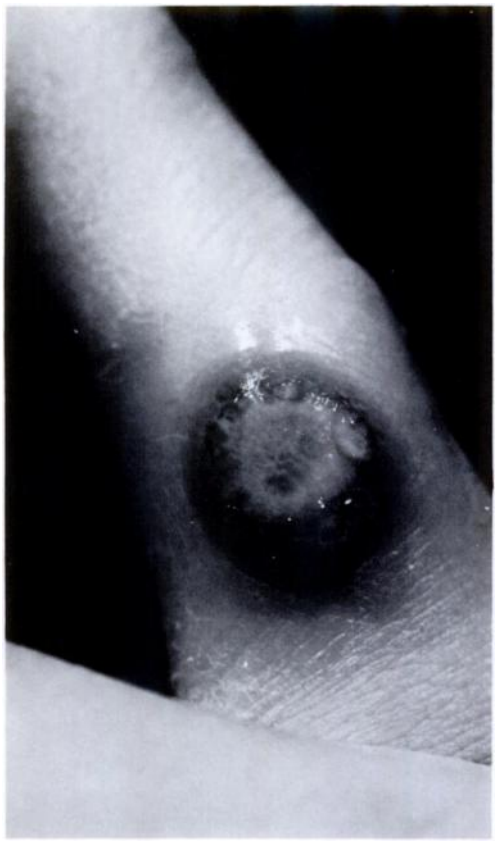

FIGURE 4. Pox nodule, "milker's nodule," on the medial surface of the fifth finger of a seal handler 4 wk after the lesion was first noticed. The nodule had a raised, pale center rimmed by a hyperemic band from which oozed a clear serous fluid.

time the lesion was 5 mm in diameter with a red center and a pale margin. During the next few days, the central region of the lesions became raised (2.5 mm) and slightly wider. Over the course of the next 4 wk the central core became pale while the outer edges became red and edematous. On 24 March (29 days after the lesion was first noticed) the lesion began weeping clear fluid around the periphery and continued to do so for several days. During this time, a scab formed, which fell off 1 wk later (31 March). Beneath the scab there was a dark red area that slowly faded over a period of 3 to 4 mo.

The course of the disease in the second handler was similar to that of the first. In the second case, 35 days elapsed between the time the lesion was first noticed and the scab fell off. This compares well with the 36 days required for the same course of events to take place in the first case. However, unlike the first case where the lesion continued to resolve over the next 3 to 4 mo, the second handler experienced repeated relapses. During the period from 27 April to 2 May, the lesion became active and changed from a flat red blemish to a 3 mm high deep red papule. By 6 May, healing was again proceeding and by 18 May it was again a flat red blemish surrounded by a narrow rim of sloughing epidermis. This person experienced similar less severe relapses during July 1983 and October 1983. The lesion resorbed completely and by January 1984 the skin at the site of the original lesion had a normal appearance.

Negatively stained scrapings of the lesions from both seal handlers revealed the presence of virus particles similar to virus particles seen from milker's nodules (Friedman-Kien et al., 1973; Dales and Pogo, 1981). These particles were identical also with virus from the seals' pox lesions (Fig. 5).

DISCUSSION

The clinical course, pattern of distribution, gross, histologic, and ultrastructural appearance of the nodular lesions seen in these grey seals were similar to the sealpox lesions reported from other species of seals (Wilson et al., 1969, 1972a, b; Okada and Fujimoto, 1984). The lesions were observed between 9 and 43 days after capture with 75% (9 of 12 affected seals) developing lesions between days 31 and 34 postcapture. This coincides well with the 30 to 35 day period between capture and appearance of lesions described in harbor seals (Wilson et al., 1972b).

None of the 10 seals that remained in the dry enclosure developed pox lesions,

whereas 13 of the 15 seals that were placed in the water-filled cement tanks developed pox lesions. This suggests that the environment in which the seals were housed affected whether or not they developed pox lesions. Since the development of pox lesions is often associated with excoriation of the skin, the rough cement tanks most likely contributed to the development of the lesions in seals kept in them.

The ultrastructural appearance and size of the virions place them in the paravaccinia subgroup of the Poxviruses (Nagington and Horne, 1962). This is the subgroup of poxviruses that infects a wide range of mammalian hosts (Dales and Pogo, 1981) and causes the benign epithelial lesions of milker's nodules in man. We suggest the nodular lesions seen in the seal handlers were caused by the sealpox virus. This suggestion is well supported. The gross appearance and the clinical course of the lesions, as well as the negatively-stained appearance of the virions from the seal handler's lesion, were identical with those found in milker's nodules (Friedman-Kien et al., 1973; Bowman et al., 1981). Since seals with active lesions were handled with unprotected hands and since the seal handlers were not in contact with domestic ruminants, the major source of paravaccinia infections in man (Hanson, 1975), it is highly likely that the source of the parapoxvirus that infected the seal handlers came from the seals.

The lesions on the hands described in this report should not be confused with the serious disease "sealfinger," a disease of unknown cause in which the gross appearance and the clinical course are very different (Beck and Smith, 1976) and the consequences of untreated cases may result in permanent disability. Unlike sealfinger, this parapoxvirus infection had a clinical course similar to that of the milker's nodules in which the uncomplicated localized lesions resolve without treatment. For that reason infection with sealpox can be placed in the nuisance category of zoonotic diseases (Schnurrenburger et al., 1980). The use of rubber gloves when handling clinically affected seals should help prevent infection of seal handlers.

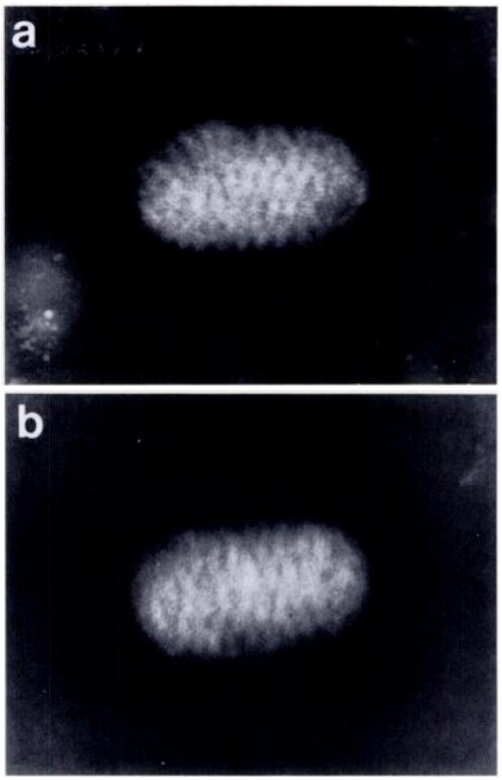

FIGURE 5. Pox virions from (a) grey seal and (b) handler. Both have the oblong shape and the criss-cross pattern characteristic of parapoxvirus. Negative staining with phosphotungstic acid 2%, pH 6.5, ×140,000.

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