NECROTISING SYNDROMES IN POSSUMS

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Introduction

Common brushtail (BTP) and ringtail possums (RTP) can suffer a range of severe necrotising lesions. A common and perplexing one is 'necrotic paw' syndrome where RTP and very occasionally BTP suffer necrosis and amputation of extremities including the tail tip and one to four paws, sometimes all at the same stage of deterioration or at very different stages ranging from normal tissue to complete necrosis and amputation. Early lesions show severe inflammation with swelling, oozing and ulceration possibly with a moist crust. Advanced lesions include gangrenous necrosis and exposed bones. Sometimes amputation stumps are closed with scar tissue and furred skin. Other RTP present with widespread alopecia, crusting and scarring or ulcers near their paws. Pouch and back riding young are unaffected.



Figure 1. Necrosis of a forepaw in a RTP

Aetiological agents for these common conditions are unknown. Microbiological and histological investigations have revealed non-specific inflammation and necrosis and a range of likely secondary bacterial invaders. The affected tissue may be sharply demarcated from normal epidermis and subcutaneous tissues.



Figure 2. BTP with severe facial necrosis affecting both eyes

BTP rarely present with paw necrosis but dermatitis and focal ulcerations are common and can be severe and even fatal affecting much of the skin and subcutaneous tissues of the face, including one or both eyes.

The asynchrony of affected paws, the varying stages of disease, the lack of spread to offspring and non-specific findings from histopathological and microbiological investigations have perplexed wildlife veterinarians and carers for decades. Here we describe these syndromes and discuss a chance

finding on a chronically debilitated adult ringtail possum that may lead to the identification of potential primary agents for some of these distressing diseases.

Discussion

An adult female RTP with generalised alopecia and mildly crusting dermatitis that had not responded to intensive care for over 6 months was euthanised. Fortuitously she was brought within her artificial drey over which strange insects were noticed crawling. Crushing one released bloody fluid so the remaining three were preserved in alcohol. Images of these insects were identified by entomologists at the Australian Museum and the UNSW as Cleradini insects. Five *Clerada* (Heteroptera: Lygaeidae) species have been recorded in Australia almost exclusively from the nests of mammals and are repeatedly recorded to take vertebrate blood. *C. apicicornis* is incriminated in transmitting *Trypanosoma cruzi* (Chagas' disease) to people in South America.



Figure 4. Non-engorged Cleradini insect

Figure 3. Engorged Cleradini insects

Finding the insects with a RTP with generalised non-responsive dermatitis led to speculation about their association with "necrotic paw syndrome" either directly through hypersensitivity reactions and secondary bacterial infections or mediated through transmission of other agents through the insects' feeding habits of. A haematophagous agent may explain the irregular distributions and temporal stages seen with many of the necrotic lesions.

Heavy growths, predominantly of a weakly haemolytic *Staphylococcus* sp (probably *S. aureus*) were cultured from several swabs taken from beneath the crusts of a BTP with severe facial necrosis. Resident skin bacteria may contribute to the superficial necrosis secondarily following damage and inflammation by the Cleradini insect or this insect may be a vector for necrotising bacteria.

While examining a RTP drey for live Cleradini insects to test their pathogenicity on rats, 7 adult leeches were also found in the moist base of the deep drey. Presumably they feed on any possum sleeping in the drey and may contribute to the development of necrotising lesions and perhaps other superficial lesions in possums. The presence of both Cleradini insects and leeches in the nest indicates a potential synergy between these parasites in causing disease may have to be tested as well as their individual disease potentials. Pathogenic bacteria have been isolated from medicinal leeches (e.g., *Aeromonas veronii*). Could leeches lead to the severe ulcerations seen in BTP?

Lesions with similar early presentations of necrotic paw syndrome in RTP have been reported in dogs and severe indolent necrotic lesions are reported in people. Various aetiological agents have been hypothesised for these, including white-tipped spiders, but these have not been confirmed and remain hypothetical and controversial. Cleradini insects and leeches are non-specific vertebrate blood suckers. Could they be involved in the transmission of agents of disease responsible for necrotising lesions in humans and other animals?

Research into the pathogenic potential of Cleradini insects and leeches to cause severe necrotic lesions is soon to be underway using a rat model.

DR DEREK SPIELMAN BVSc I began my professional involvement in treating Australian wildlife as the Veterinary Intern at Taronga Zoo in 1986. I finished an MVSc and BSc while at the zoo, majoring in zoology and genetics for the latter. I progressed to veterinary officer then chief veterinarian with overall responsibility managing the zoo's Veterinary Quarantine Centre and the NRMA Wildlife Clinic before leaving to manage the NRAC Vertebrate Fauna Audit and report of north-eastern NSW for the NSW NPWS. I then undertook a PhD in conservation genetics under Profs Dick Frankham and Dave Briscoe at Macquarie University. In 1997 I accepted a position with the Parks and Wildlife Commission of the Northern Territory as the Senior Curator/Veterinarian at the Territory Wildlife Park where I progressed to Senior Veterinarian and Park Manager. I left the NT to take up a position as Chief Veterinarian for the Ocean Park Corporation in Hong Kong. After 3 years I returned to Australia and private veterinary practice also commencing as the Consultant Veterinarian for the Wildlife Assistance and Information Foundation (WAIF). I accepted a position as Lecturer in Veterinary Pathology at the University of Sydney where I have worked since 2006