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Source: Journal of Wildlife Diseases, 19(3) : 289-290

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-19.3.289

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Journal of Wildlife Diseases, 19(3), 1983, pp. 289–290
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Capture Myopathy in a Free-flying Greater Sandhill Crane (*Grus canadensis tabida*) from Wisconsin

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Capture myopathy has been reported frequently in wild mammals (Bartsch et al., 1977, Vet. Pathol. 14: 314–324). There are, however, fewer reports of this disease in wild birds (Young, 1967, Int. Zoo Yearb. 7: 226–227; Bartsch et al., 1977, op. cit.; Henschel and Low, 1978, S. Afr. J. Sci. 74: 305–306; Wobeser, 1981, Diseases of Wild Waterfowl, Plenum Press, New York, 300 pp.). We are reporting a case of skeletal muscle necrosis in a greater sandhill crane found dead 5 days after its capture, radio-tagging, and release. We believe this is the first case of capture myopathy to be reported for this species.

During a disease and parasite survey and migration study of sandhill cranes in 1976 and 1977 in Wisconsin, 130 cranes were captured by rocket-netting. Ten of these cranes were fitted with radio transmitters, nine of which were tracked for a duration of from 48 days to over 16 mo (Melvin, 1978, M.S. Thesis, Univ. of Wisc., Stevens Point, 80 pp.). The subject sandhill crane was the tenth bird fitted with a transmitter; it was an immature female weighing 5.25 kg at capture. Blood samples were collected, tracheal and cloacal swabs were taken, and the bird was fitted with a transmitter and banded with a USFWS leg band before its release. The total time of restraint of this crane was about 1 hr. Telemetry observations indicated that the crane remained within 0.5 km of the banding site for the 4 days following its release, whereas the other cranes similarly captured, radio-tagged, and released, moved much greater distances. On the fifth day, no movement was detected and the carcass was subsequently located. Necropsy was completed at the National Wildlife Health Laboratory the following day. Severe weight loss had occurred; the carcass weighed only 3.8 kg with marked pectoral muscle atrophy and complete absence of subcutaneous fat. Gross lesions included bilateral, pale, streaked areas in the biceps femoris muscle, pale mottled kidneys, and splenic congestion. Routine cultures of the liver, spleen, brain, colon, and cecae showed no pathogenic bacteria to be present. Virological examination of the cloaca and brain were negative when tested for hemagglutinating agents after inoculation into embryonated chicken eggs.

Microscopically, the skeletal muscle had extensive, severe necrosis characterized by swollen myofibers with amorphous, fragmented cytoplasm that was slightly mineralized in some fibers. There was proliferation of nuclei in a few fibers. In some areas all the myofibers in a primary bundle were necrotic, in other areas only a single fiber was affected. There was centrolobular congestion in the kidney, a moderately severe nephrosis characterized by aggregates of eosinophilic debris (urate spherules), and partially mineralized debris in some of the distal convoluted tubules and collecting ducts. There was also a mild granulocytic infiltrate in the interstitium and tubules.

These changes are very similar to Harthoorn's description of the subacute capture myopathy–renal failure syndrome (Wobeser, 1981, op. cit.). In our experience, urate deposits in the lower nephron are often associated with dehydration. Impaired mobility and subsequent dehydration may have been a factor in the renal damage in this crane, although increased uric acid production or myoglobinuria may have occurred as a result of the muscular damage. The final diagnosis based on the above findings was capture-related skeletal muscle necrosis and granulocytic nephritis.

Received for publication 12 January 1983.

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Research was supported by the U.S. Fish and Wildlife Service (contract #14-16-008-2079) and the University of Wisconsin, Stevens Point

The authors thank Ruth Duncan and Douglas Docherty of the National Wildlife Health Laboratory for laboratory assistance.

Journal of Wildlife Diseases, 19(3), 1983, pp. 290–293
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Oxalate Toxicity in a Scaly-tailed Possum, a Patagonian Cavy and a Swamp Wallaby

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In Australia oxalate-containing plants of the families Oxalidaceae and Chenopodiaceae have been shown to be toxic to livestock (Seawright, 1982, Animal Health in Australia, Vol. 2, Chemical and Plant Poisons, Australian Government Printing Service, Canberra, pp. 74–76). Osteodystrophia fibrosa resulting from ingested oxalates interfering with calcium availability has occurred in horses grazing *Panicum maximum* var. *trichoglume* (green panic), *Cenchrus ciliaris* (buffet grass), *Setaria anceps* (setaria), *Brachiaria mutica* (para grass) and *Pennisetum clandestinum* (kikuyu) pastures (Groenendyk and Seawright, 1974, Aust. Vet. J. 50: 131–132; Walthal and McKenzie, 1976, Aust. Vet. J. 52: 11–16). There has been one confirmed report of oxalate nephrosis in cattle grazing *Setaria anceps* (Bua River strain) in Queensland (Seawright et al., 1970, Aust. Vet. J. 46: 293–296). The lesions associated with oxalate toxicity in man, cats and dogs from the ingestion of ethylene glycol (antifreeze) are oxalate nephrosis and renal failure (Kersting and Nielsen, 1965, J. Am. Vet. Med. Assoc. 146: 113–118; Kersting and Nielsen, 1966, Am. J. Vet. Res. 27: 574–582).

Three cases of oxalate toxicity occurred within a 4 mo period at the Zoological Gardens in Perth, Western Australia. The first was in a scaly-tailed possum (*Wyulda squamicaudata*), a cave-dwelling marsupial found in the Kimberley region of Western Australia and rarely exhibited in captivity. The possum was one of a pair presented to the Zoological Gardens in 1977 and placed on exhibition in the nocturnal house. The male died in 1981 of undetermined causes. The female remained the sole occupant of the enclosure until a banded hare-wallaby (*Lagostrophus fasciatus*) was introduced 7 mo prior to the possum's death. The diet of the possum prior to the wallaby's arrival contained no freshly cut grasses. With the addition of the wallaby to the enclosure freshly cut grasses were included in the feed. No signs of ill health were observed in the animals prior to being housed together. The initial sign noted in the possum was lethargy. Twenty-four hr later the keeper noticed an unusual hopping gait and 48 hr later the animal was found recumbent with intermittent episodes of myoclonus. The possum became moribund and died within 90 min. The second oxalate toxicity was in a Patagonian cavy (*Dolichotis patagona*). These cavies are plain-dwellers from Argentina and the Patagonian area of South America and this animal was one of a colony at the zoo. It was found in a comatose state and was destroyed. There had been no history of ill health in this animal. The diet was similar to that provided for the possum including provision of freshly cut green grasses. A third case of oxalate toxicity occurred in a swamp wallaby (*Wallabia bicolor*) which is a forest-dwelling wallaby often found near creeks and swamps in the eastern States of Australia. This wallaby had developed lumpy jaw and was placed in an isolation pen where the jaw lesion was being treated by debridement and injec-

Received for publication 11 October 1982.

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