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Short communication

PARALYSIS OF A DROMEDARY CAMEL CAUSED BY *Hyalomma dromedarii* INFESTATION

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ABSTRACT

A heavy tick infestation is reported, causing paralysis in an old female dromedary camel with all stages of *Hyalomma dromedarii*. The diagnosis relied solely on clinical signs due to a lack of specific methods for identifying tick neurotoxin(s). No gross or microscopic lesions were observed except for mild to marked demyelination of the spinal cord white matter.

Key words: *Hyalomma dromedarii*, hind leg paralysis

Tick paralysis is an acute, progressive, symmetrical, ascending motor paralysis caused by salivary toxins from specific tick species. A wide range of mammals, birds, reptiles, and even humans can be affected. In humans, tick paralysis is primarily caused by ticks of the genus Ixodes, *Dermacentor*, and *Amblyomma,* and has been reported in Australia, South Africa, North America, and Europe (Merck, 2016). Young domestic animals heavily infested with ticks often experience paralysis (Radostits *et al,* 2007).

It is important to distinguish tick paralysis or toxicity from tick-borne diseases or tick-borne fever like those caused by Rickettsiae, transmitted through tick bites. Wernery (2022) recently reviewed rickettsial infections in dromedaries, highlighting limited knowledge on Rickettsiosis in camels with reported infections but no confirmed disease. Even less is known about tick paralysis in camels.

We report here a possible case of tick paralysis in an adult female dromedary camel caused by *Hy. dromedarii*.

Materials and Methods

A female adult dromedary camel weighing approximately 300 kg was found in a sitting position at a small camel farm in the Dubai Emirate. The animal was unable to stand despite attempts to lift it with a crane. A neurological examination revealed a complete loss of deep sensation in the hind legs. Due to ethical concerns regarding the animal's suffering and limited chances of recovery, a decision was made to euthanase the camel. The euthanase protocol followed established guidelines. Following euthanasia, the camel was transported to the Central Veterinary Research Laboratory (CVRL) in Dubai for further investigation.

Results

Necropsy findings

Examination of the camel post-mortem revealed multiple *Hy. dromedarii* ticks attached throughout the body, including larvae, nymphs, and engorged females (Fig 1). The urinary bladder was distended with urine, and the rectum contained a large amount of faecal material. No other significant macroscopic lesions were observed in internal organs, including the brain and spinal cord.

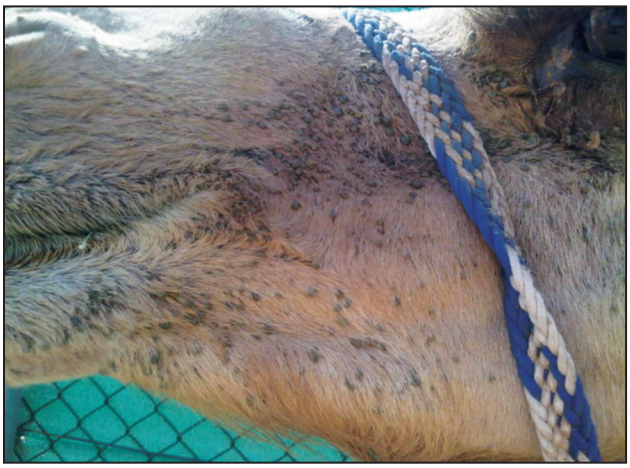

Fig 1. Female dromedary camel head infested with *Hyalomma dromedarii*.

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Blood and histopathology

Blood parameters from blood obtained before euthanasia, were within normal ranges except for an elevated creatine kinase (CK) level.

Blood trace elements of selenium and zinc did not show any abnormality, while the serum copper level was found to be 10.83 µmol/l (ref.-value between 9.00-14.00 µmol/l, Wernery *et al,* 2009). The copper level of the liver was below the reference value (18.00 – 140.00 ppm per weight per gram) with 13.40 ppm wet weight copper per gram liver.

Histological examination revealed no abnormalities in the brain but showed mild to moderate demyelination in the spinal cord white matter.

Discussion

We report a possible case of tick paralysis in an adult female dromedary camel caused by *Hy. dromedarii* ticks.The animal exhibited a rapid progression of hind leg paralysis, culminating in complete recumbency within 48 hours of onset. The attempt to lift the animal up by crane did not succeed, necessitating the decision to euthanase the camel for animal welfare reasons. The severe, progressive nature of the paralysis, coupled with the presence of numerous *Hy. dromedarii* ticks in various life stages on the camel's body points to a possible tick paralysis. Specific diagnostic tools for identifying the salivary neurotoxin responsible for tick paralysis are currently not available. Therefore, epidemiological and clinical context is needed to support the theory. As haematology and biochemistry values were largely within the normal range, except for the elevated CK. The increased CK can have various causes like muscle damage from trauma, hypoxia, thrombosis, recumbency, vitamin E and selenium deficiency, inflammatory myositis and neoplasia. The toxins may also have an effect on the CK levels in the blood. Mineral values like calcium, sodium, magnesium and potassium were in the reference value range. Blood trace elements of selenium, zinc and copper did not show any abnormality. However, the copper levels in the liver were below the reference value (18.00 – 140.00 ppm wet weight per gram) with 13.40 ppm wet weight copper per gram liver. This decreased copper value might have contributed to the severe paralysis. Camels have shown to exhibit a higher tolerance for mineral deficiencies compared to other ruminants (Bengoumi *et al,* 2002).

Previous reports have documented cases of tick paralysis in camels. Manefield and Tinson (1996) described mild paresis in Australian camel calves infested with *Ixodes holocyclus* ticks. Barre and Uilenberg (2010), suspected *Hy. dromedarii* in paralysis cases in North-East Africa. Additionally, Musa and Osman (1990) reported an outbreak in Southern Darfur, Sudan involving 251 camels with clinical signs of incoordination, unsteady gait, paralysis and recumbancy attributed to infestation with *Hy. dromedarii* and *Rhipicephalus* ticks.

Even though our investigation did not yield new insights into the gross and histopathological changes associated with tick paralysis in camels, there is a potential for underdiagnosis of this condition, warranting further research in this area. Notably, the successful utilisation of tick antitoxin serum (TAS) and antibiotic treatment in canine tick paralysis cases (Atwell and Campbell, 2001) suggested promising possibilities for future exploration in camels.

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