Fatal red-bellied black snake envenomation: clinical and pathological findings

Dr Matthew Wun[1], Dr Mirrim Kelly-Bosma[2], Dr Rachel Allavena[2], Dr Andrew Padula[3,4], Dr Ellie Leister[5]

[1]Veterinary Specialist Services, Underwood, Australia, [2]School of Veterinary Science, The University of Queensland, Gatton, Australia, [3]Australian Venom Research Unit, Department of Pharmacology & Therapeutics, University of Melbourne, Melbourne, Australia, [4]Padula Serums Pty Ltd, Bairnsdale, Australia, [5]Pet Intensive Care Unit, Underwood, Australia

Most cases of red-bellied black snake (RBBS) envenomation in dogs respond favourably to treatment comprising of tiger-brown snake antivenom (TBAV), intravenous fluid therapy (IVFT), analgesia and, if indicated, mechanical ventilation and/or blood transfusion.[1]

This report describes the clinical and pathological findings in a dog fatally envenomed by a RBBS, despite receiving intensive veterinary care. An eight-year old Staffordshire Bull Terrier dog presented for a 1-day history of vomiting, diarrhoea, haematochezia and weakness. Further investigation revealed evidence of rhabdomyolysis, haemolytic, regenerative anaemia with spherocytosis, azotaemia, bilirubinaemia, pigmenturia, oliguria and a mild coagulopathy. TBAV, analgesia, IVFT, a mannitol infusion and multiple transfusions of packed red blood cells were administered. A gradual improvement in clinical status was achieved, however, 64 hours post-presentation pigmenturia worsened and hypoxaemia, dyspnoea and anuria developed. Serum biochemistry at this time showed worsening azotaemia, and cardiorespiratory arrest occurred shortly afterwards. Post-mortem examination and histopathology revealed diffuse jaundice, bicavitary effusion, renal tubular necrosis and haemosiderosis, hepatic submassive necrosis with cholestasis, adrenal cortical necrosis, pulmonary oedema and skeletal muscle injury. Pre- and post-TBAV serum and urine RBBS venom antigen concentrations were determined, confirming the diagnosis of RBBS envenomation.

This is the second report of acute kidney injury secondary to RBBS envenomation in the dog despite appropriate treatment,[2] and the first with evidence of hepatic and adrenal cortical necrosis. Delayed presentation post-envenomation may be a contributing factor to the poor outcome in this case, and repeated TBAV administration and/or early institution of renal replacement therapy may be indicated in future cases.

References

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The Australian and New Zealand College of Veterinary Scientists

Building 3, Garden City Office Park
2404 Logan Road
EIGHT MILE PLAINS QLD 4113

Telephone: (07) 3423 2016
Email: marketing@anzcvs.org.au
Web: anzcvs.org.au

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