Australian Registry of Wildlife Health
Taronga Conservation Society Australia
PO BOX 20 Mosman NSW 2088
02-9978-4749
www.arwh.org

PATHOLOGY REPORT
Australian Registry of Wildlife Health

Taronga Zoo

Status: Final
Report Date: 23/01/2020

Submitter

WIRES - Northern Rivers
P.O.BOX 1356
LISMORE
2480

Business Phone:
Mobile Phone:
Fax:
Email: wiresnr@wiresnr.org

Submission Details

Submitter's Ref:
Date Submitted: 12-Dec-2019 00:00:00
Date Received: 12-Dec-2019 00:00:00
Lab. Case/Spec ID:
Previous Lab. ID:
Specimen ID: TARZ-13389.1

Animal Detail

Animal ID:
ARKS No:
Rehab ID:
Animal Name:
Species: Macropus rufogriseus banksianus
Common Name: Red-necked Wallaby
Sex Class: Female
Age Class: Pouched Young
Enclosure No:

Epidemiology

Number Dead: 1
Number At Risk:
Number Sick:
Number Submitted:
Date Died: 12-Dec-2019 00:00:00
Death Circumstance: Found Dead

Parameter measurements were not included in report.

SPECIMEN HISTORY

Animal in care at Rock Valley near Lismore, NSW since 23/08/19 when recovered from a dead hit by car dam near Casino. The joey died suddenly overnight 12/12/19 and was examined post mortem by Dr Phil Kemsley, LLS. Tissues were sent to the Registry for investigation.

"This one died suddenly after some initial symptoms of the illness (hypersensitivity, panicking) and had had no prior treatments, so would be ideal for testing. We are attempting to get the body in to someone locally to do a necropsy." Renata Phelps

GROSS PATHOLOGY

No notes supplied

HISTOPATHOLOGY

Tissues are generally well preserved.

Kidney (slide 1A): Frequent tubules are dilated and tortuous, and lined by attenuated or necrotic epithelial cells with luminal clusters of karyorrhectic debris and sloughed epithelial cells, small amounts of proteinaceous material and occasional fragments of basophilic mineral. Scattered, tubules are lined by plump, karyomegalic epithelial cells with slight cytoplasmic basophilia and marginated chromatin (regeneration). The basement membrane of occasional tubules is expanded by deeply basophilic mineral material and scattered tubules contain luminal aggregates of indistinct amphophilic crystalline material, rarely accompanied by multinucleate giant cells.

Liver (slide 1A): Increased numbers of neutrophils percolate through hepatic sinusoids.

Spleen (slide 1A): Small numbers of lymphocytes multifocally cluster around blood vessels in the adjacent mesentery, accompanied by scattered neutrophils. The red pulp is diffusely contracted,

Pancreas (slide 1A): Small numbers of lymphocytes and plasma cells multifocally cluster around blood vessels scattered throughout the parenchyma.

Lymph node; NOS (slide 1B): Lymphoid follicles are mildly expanded with distinct germinal centres cuffed by a darker mantle cell zone.

Lung (slide 1B): Airways are variably occluded by intense infiltrates of degenerate neutrophils, accompanied by fibrin and florid colonies small cocci. Small numbers of neutrophils occasionally cluster within the pulmonary interstitium, which is diffusely congested. The tunica adventitia of pulmonary arteries is mildly loosened by clear space (oedema) and alveolar spaces multifocally contain extravasated erythrocytes.

Brain including cerebral cortex, hippocampus, internal capsule, cerebellum and brainstem (slides 1D-F): Glial cells multifocally cluster around neuronal cell bodies within the superficial lamina (satellitosis).

No significant lesions are present in the following tissues: Colon (slide 1B), skeletal muscle (slide 1C), heart (slide 1C), stomach (slide 1D)

DIAGNOSIS

Kidney: Severe, acute tubular degeneration and necrosis with tubular proteinosis and mineralisation
Lung: Moderate, acute, diffuse, fibrinosuppurative bronchopneumonia
Liver: Peripheral neutrophilia

COMMENTS

Histologic changes are most significant within sections of kidney, which are characterised by severe, acute tubular degeneration and necrosis with protein loss and tubular mineralisaton. Evidence of tubular regeneration is consistent with a subacute time course (injury occurring within days of death). Acute tubular injury is an aetiologically non-specific change suggestive of either an ischaemic (eg. poor renal perfusion, severe anemia, shock) or toxic insult. It is possible these changes resulted in acute uremic encephalopathy; however, whether these changes account for the reported behavioural changes is uncertain.

Sections of lung are characterised by an acute inflammatory infiltrate centred on airways and accompanied by pulmonary oedema and mild haemorrhage. Bronchioles are variably occluded by fibrin and degenerate neutrophils intermingling with florid bacterial colonies; however, the majority of the pulmonary parenchyma is spared. It is possible bacterial infection resulted in toxemia and sudden death; however, the extent of the renal change suggests this was the more clinically relevant change and proximate cause of death.

Given the severity and extent of the acute tubular injury, exposure to an environmental toxin (eg. plant, drug) warrants consideration. Testing of any frozen tissues for possible infectious agents is also indicated, given concern regarding an emerging syndrome.

Pathologist: Karrie Rose
Date Finalised: 21-Jan-2020 00:00:00
Copy To: renata.phelps7@gmail.com; phil.kemsley@lls.nsw.gov.au; tgrillo@wildlifehealthaustralia.com.au; ofir.schwarzmann@dpi.nsw.gov.au; claire.harrison@dpi.nsw.gov.au