CASE REPORT: SEVERE PHOTOSENSITIVITY IN A RED STAG FAWN ASSOCIATED WITH A LEPTOSPIRA POMONA INFECTION

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INTRODUCTION:

I would like to present a case of severe photosensitization in a weaner red stag associated with Lepto pomona infection, and draw attention to the similarity of clinical signs to those of facial eczema.

BACKGROUND:

A total of 24 fawns (hinds & stags) were weaned on 15/3/84. Twelve days after weaning, I was presented with one dead (2-3 days) stag fawn and one very sick stag fawn.

CLINICAL EXAMINATION:

Autopsy of dead fawn: All that could be concluded was a possible jaundice and a rubbing around the head.

Sick fawn: Severe photosensitization around the head involving eyelids, ears and lips. Corneal opacity and very yellow mucous membranes. Marked depression. Normal temperature. No haematuria.

Samples collected - Serum, EDTA blood, Urine (from deer shed floor).

DIFFERENTIAL DIAGNOSIS:

Facial eczema was my first choice, as there were several properties in the immediate area with clinical F.E. in sheep and cattle and I had seen one severe case in a yearling hind on another property. As I had once before seen a severe outbreak of Leptospirosis causing jaundice and death in recently weaned fawns this also had to be considered.

TREATMENT:

As Lab results were going to be a day or two away, I decided to cover my options. The patient was treated with streptomycin and supportive multi vitamins. It was locked in shade and provided with hay and water.

The remainder of the mob was vaccinated using 2cc "Leptavoid" and given 2-3cc streptomycin. (I regret that no serum was collected from these). Hind and stag fawns were separated.

Within 48 hours the fawn was much brighter, eating and moving about much better.

Lab Results	Ruakura Normals
PCV 24%	45%
RBC 6.34 x 10^9/litre	8-10
Hb 8.7 g/d l	15-18
WBC 1.3 x 10_{9}/litre	4.5-7.1
Differential 64% Neutrophils	19-57
36% lymphocytes	20-40

Lab Comment - Anaemic.

GGT 170 u/l at 37°
Lab comment - suggestive of bile duct damage
Lepto serology

L. pomona	L. hardjo	L. copenhagani
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Dark ground microscopy - no leptospires seen.
I saw this fawn again two weeks later and euthanasia was carried out because of eye and ear damage.
Serum was collected and submitted for liver enzymes and Lepto CFT.

LAB RESULTS:

GGT 85 u/l at 37°C
LDH 13 u/l at 25°C

L. pomona	L. hardjo	L. copenhagani
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Lab comment - both normal liver function results.

HISTOLOGY:

Liver kuppfer cells contain large amounts of pigment within cytoplasm (probably haemosiderin). Occcasional hepatocyte is necrotic and there is a moderate degree of hepatocellular degenerative change.

Kidney: Most cells lining tubules contain pigment in focal areas. An occasional pigment caste is present within the tubule lumen. In these areas the tubular lumen is often degenerate and attenuated or necrotic, and an occasional affected tubule contains a neutrophil infiltrate. The interstitium is infiltrated by small numbers of mononuclear cells, mainly lymphocytic.

LAB COMMENT:

Facial eczema can be ruled out because large bile ducts are normal.

L. pomona infection appears the most likely cause as there is evidence of intravascular haemolysis and focal interstitial nephritis both typical of L. pomona infection in cattle. There were no further deaths.

COMMENTS AND CONCLUSIONS:

The similarity to F.E. can be appreciated and had I not seen a previous lepto outbreak causing Jaundice and death in recently weaned fawns, I would probably have not treated for lepto on day one - possibly greater losses would have occurred. I presume the source of Lepto was cattle used to cross graze pasture. As cattle are commonly used in Waikato and elsewhere to control excess pasture, it is in my opinion, worth treating and vaccinating cattle before grazing deer pasture. In my opinion, if cattle are being used, deer should be vaccinated annually.

I would like to thank Ruakura Animal Health VIO's for haematological, chemical and histological reports.