

CASE NUMBER: 03F-63-A3.3-47

DATE: Aug 16, 2003

**MORPHOLOGIC DIAGNOSES:**

Slide 42, A3.3-47-1:

- 1). Heart: Myocarditis, mild to moderate, multifocal, random, necrotising, acute with intralesional bacilli
- 2). Kidney: Nephritis, interstitial, moderate, multifocal, necrotising, with intralesional bacilli
- 3). Spleen: Splenitis, mild, multifocal, embolic, acute, necrotising, with intralesional bacilli
- 4). Liver: Hemorrhage, mild to moderate, multifocal, random, acute

There are no overt lesions within the peripheral nerves.

Slide 43, A3.3-47-2:

- 1). Vasculature, liver: Embolism, septic, mild, multifocal, random, acute
- 2). Kidney: Nephritis, interstitial, moderate, multifocal, necrotising, with intralesional bacilli

There are no significant lesions in the spleen, heart or peripheral nerves.

Slide 44, A3.3-47-3:

There are no significant lesions in the kidney, spleen, heart, liver, peripheral vasculature, or peripheral nerves.

Slide 45, A3.3-47-4:

- 1). Heart, endocardium: Hypertrophy, mild to moderate, multifocal

There are no significant lesions in the kidney, spleen, liver, peripheral vasculature, or peripheral nerves.

Slide 46, A3.3-47-5:

- 1). Liver: Lobular collapse, moderate, multifocal, with apoptosis, compensatory hepatocytes hypertrophy, karyomegally, ceroidosis and biliary ductular hyperplasia

There are no significant lesions in the kidney, spleen, heart, peripheral vasculature, or peripheral nerves.

Slide 47, A3.3-47-6:

- 1). Kidney: Nephritis, interstitial, moderate, multifocal, necrotising, with intralesional bacilli

There are no significant lesions in the liver, corpuscle of Stannius, spleen, heart, peripheral vasculature, or peripheral nerves.

#### COMMENTS:

In 3 of 6 sections the multisystemic bacteremia is consistent with furunculosis (*Aeromonas salmonicida*) which would have been sufficiently severe to result in increased morbidity and mortality. The reactive endocardia noted in slide 45 likely represents a sequela to antigenemia. The hepatopathy in slide 46 is provocative and would have contributed significantly to antemortem impaired liver function; the process is consistent with toxic (possible algal) exposure. Follow up clinical evaluation of the stock and additional histopathology to assess the overall prevalence of this condition may be considered. In addition, tissue analysis for Microcystin toxins may be undertaken.

\*FINAL REPORT\*