CASE NUMBER: 03F-96-3-51
DATE: Dec 19, 2003

HISTOPATHOLOGY:
Slide 21, 3-51-1:
1). Kidney: Nephritis, interstitial, severe, diffuse, subacute, fibrinohemorrhagic and lymphohistiocytic, with dispersed melanin granules and scattered intra and extracellular bacilli
2). Spleen, capsule: Peritonitis/serositis, marked, focally extensive, acute, with florid intralesional bacilli
3). Spleen, ellipsoids: Lysis, moderate, multifocal, acute with intralesional coccobacilli
4). Heart, endocardium: Embolism, septic, moderate, multifocal, random, acute
5). Liver, hepatocytes: Degeneration and necrosis, moderate, multifocal, acute

Slide 22, 3-51-2:
There are no significant lesions within the heart, kidney, peripheral vasculature, spleen, liver or peripheral nerves.

Slide 23, 3-51-3:
There are no significant lesions within the kidney, heart, spleen, liver, peripheral nerves or peripheral vasculature.

Slide 24, 3-51-4:
1). Heart, epicardium: Epicarditis, mild, multifocal, granulomatous, chronic

There are no significant lesions within the posterior kidney, liver, peripheral nerves, spleen and peripheral vasculature.

Slide 25, 3-51-5:
There are no significant lesions within the kidney, heart, spleen, liver, peripheral nerves or peripheral vasculature.

Slide 26, 3-51-6:
There are no significant lesions within the heart, posterior kidney, spleen, liver, peripheral nerves and peripheral vasculature.

Slide 27, 3-51-7:
1). Liver, sinusoids: Disseminated intravascular coagulation, moderate, multifocal, acute with hepatocellular degeneration and necrosis

There are no significant lesions within the heart, anterior kidney, spleen, peripheral nerves and peripheral vasculature.

COMMENTS:
In slide 1, the multisystemic inflammatory and necrotising process is profound and likely would have contributed significantly to antemortem morbidity. Based on the relatively

paucity of observed bacteria within the renal interstitium relative to the heart and peritoneum, the possibility of an underlying viral or other disease process with secondary bacterial involvement cannot be entirely discounted. Although only 1 of 7 sections exhibited this process, follow up routine bacterial and viral culture, possibly coupled with polymerase chain reaction for select viral pathogens (such as IHNV and VHSV) may be considered. Based on the lack of attendant infiltrate associated with the septic emboli in the ventricular myocardium, furunculosis (due to *Aeromonas salmonicida*) is a prime consideration. Although there are no discernible pathogens in slide 7, the sinusoidal accumulation of fibrin is consistent with an acute fulminating inflammatory process. This change may reflect the incipient stages of the more fulminating process in slide 1.

FINAL REPORT