CASE NUMBER: 03F-49-A3.4-23
DATE: Aug 2, 2003

MORPHOLOGIC DIAGNOSES:
Slide 66, A3.4-23-1:
1). Kidney: Nephritis, interstitial, marked, multifocal to coalescing, lymphohistiocytic, subacute to chronic with intrahistiocytic coccobacilli
2). Spleen: Splenitis, moderate, multifocal, random, lymphohistiocytic, subacute
3). Liver: Hepatitis, mild, multifocal, necrogranulomatous
4). Peritoneum: Peritonitis, moderate, focal, granulomatous, with intralesional adjuvant vacuoles consistent with vaccine induced peritonitis

There are no overt lesions within the peripheral nerves or peripheral vasculature.

Slide 67, A3.4-23-2:
There are no apparent lesions within the spleen, liver, heart, kidney, peripheral vasculature or peripheral nerves.

Slide 68, A3.4-23-3:
There are no apparent lesions within the spleen, liver, heart, kidney, peripheral vasculature or peripheral nerves.

Slide 69, A3.4-23-4:
1). Spleen: Congestion and hyperemia, moderate, diffuse, acute

There are no apparent lesions within the liver, heart, kidney, peripheral vasculature or peripheral nerves.

Slide 70, A3.4-23-5:
1). Kidney: Nephritis, interstitial, moderate, multifocal, lymphohistiocytic, subacute to chronic
2). Spleen: Splenitis, moderate, multifocal, lymphohistiocytic, subacute
3). Heart: Myocarditis, mild, multifocal, random, subacute

There are no overt lesions within the liver, peripheral nerves or peripheral vasculature.

Slide 71, A3.4-23-6:
1). Spleen, capsule: Peritonitis, moderate, segmental, fibrinogranulomatous, subacute

There are no apparent lesions within the liver, heart, kidney, peripheral vasculature or peripheral nerves.

Slide 72, A3.4-23-7:
There are no apparent lesions within the liver, heart, kidney, spleen, peripheral vasculature or peripheral nerves.

Slide 73, A3.4-23-8:
1). Heart, epicardium: Epicarditis, moderate, focal, granulomatous

There are no apparent lesions within the liver, spleen, kidney, peripheral vasculature or peripheral nerves.

COMMENTS:
The multisystemic inflammatory infiltrate noted within 2-3 sections is consistent with a fulminating septicemia which would have contributed at least moderately to antemortem homeostatic derangements; based on the lack of discernible pathogens and nature of the inflammatory infiltrate, bacterial kidney disease is a prime consideration. Follow up culture, florescent antibody tests or molecular studies (PCR) for *Renibacterium salmoninarum* may be considered. There were no apparent predisposing or underlying disease processes within the examined tissues.

FINAL REPORT