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Signalment and Presentation

- “Copper”
- 9 year old intact male Golden Retriever
- Presented on 8/3/12 to UTCVM with acute hind limb paresis
Diagnostics

- MRI: Linear hyperintensity from L3-L6

- CT: Mild spinal cord compression at the thoracolumbar junction

- Cerebrospinal fluid tap: Marked numbers of neutrophils and macrophages
Clinical Outcome

• Minimal improvement over 2.5 months of hospitalization and various treatments
  ▫ Euthanasia elected

• Submitted for necropsy
Gross Findings

- Marked bilateral hind limb muscle atrophy
- Marked bilateral adrenocortical atrophy
- Degenerative joint disease
- Tricuspid and mitral valve endocardiosis
Gross Findings

• Marked dorsoventral flattening of the spinal cord at T11-12

• Bulging disks at T11-12 and T13-L1
  ▫ Minimally compressive
  ▫ Clinically insignificant
Morphologic Diagnosis

- Fibrocartilaginous emboli with myelomalacia and Wallerian degeneration

- Multiple segments affected
  - T10-13 and L3-6
Fibrocartilaginous Embolism (FCE)

- Emboli are histochemically identical to the nucleus pulposus
- Infarction and necrosis of affected spinal cord segments
  - Peracute paresis
Signalment

- Large and giant breed dogs most commonly affected
  - Miniature schnauzers predisposed
  - No sex predilection
- Median age affected 5-6 years old
- Peracute, nonprogressive, nonpainful paresis
  - Frequently asymmetric
- L4-S3 most commonly affected
Pathophysiology Hypotheses

1. Direct penetration of spinal cord or vertebral vessels by the nucleus pulposus
   • Precipitated by increased intrathoracic and intraabdominal pressures by trauma, exercise, etc.

2. Chronic neovascularization of a degenerate intervertebral disk
   • Abrupt increase of intervertebral disk pressure higher than arterial blood pressure results in embolism of fibrocartilage
Pathophysiology Hypotheses

3. Patent embryonic remnant vessels in the nucleus pulposus

4. Herniation of the nucleus pulposus into the vertebral bone marrow
   • Enters basivertebral vein and internal vertebral venous plexus

• Remains controversial
Thank You!

- Dr. Linden Craig
- Resident mates
- UTCVM Histology Lab