### **Equine Laminitis**

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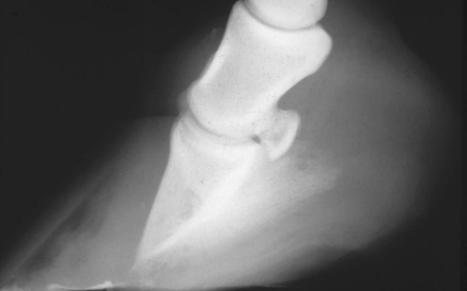
### **Definitions**

- Inflammation of the laminae
  - Numerous inciting causes
  - Laminitis vs. "founder"
- Separation of the dermal and epidermal junction
  - Attachment of coffin bone to hoof wall
- Displacement of the coffin bone
  - Rotation
  - Sinking
  - Combination of the two



# Coffin Bone Displacement



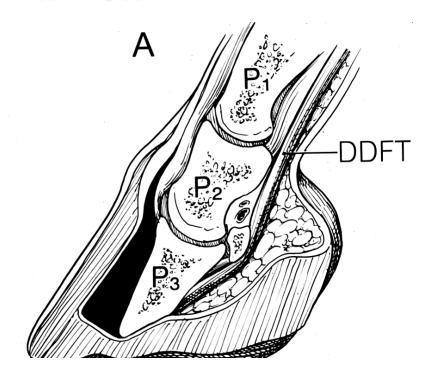






### **Clinical Forms**

- Gastrointestinal/toxic
  - Grain overload, grass founder
  - Metritis, retained placenta, colitis, colic
- Musculoskeletal/mechanical
- Metabolic/local
  - Cushing's disease
  - Metabolic syndrome
  - Corticosteroids
- Idiopathic





### **Predisposing Factors**

- Systemic illness (toxemia)
- Excessive carbohydrate/grass
- Metabolic/weight problems
  - Increased glucose/insulin resistance
- Older horses and mares
  - Non-Thoroughbred breeds
- Unilateral lameness
- Long toe/low heel
  - Hard surfaces



### **Stages of Laminitis**

- Developmental before clinical signs
- Acute
  - Clinical signs only
  - No movement of coffin bone on radiographs
- Chronic
  - Movement of coffin bone within hoof
- Subacute?
  - In between the acute and chronic stages



### Clinical Signs

- Lameness especially when turned
  - Worse on hard surfaces
  - Shifting weight between limbs
- Increased digital pulses
- Heat over dorsal hoof wall
- Both front feet commonly affected
  - All four feet (very sick horses)
  - Both rear feet (draft horses?)
  - Single foot (excessive weight-bearing)



### Laminitis







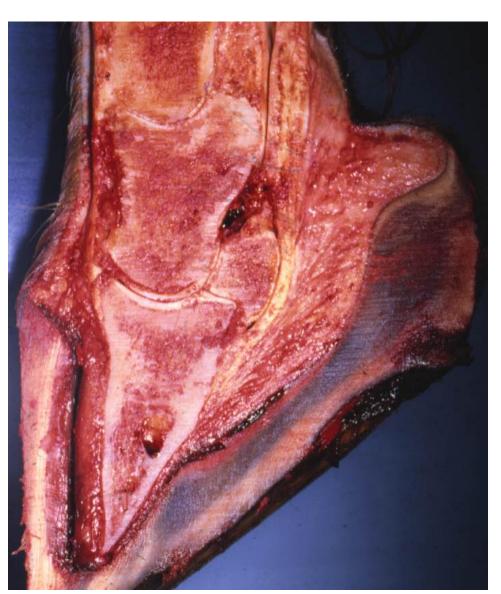


#### **Disease Process**

- Variable developmental period
  - Grain overload 24-72 hours
- Much happening in foot before laminar damage and clinical signs
- Minimal control over initial laminar damage
- Many horses with mild acute/subacute laminitis may not be recognized



### **Gross Pathology**



- 1. Edema compartment syndrome
- 2. Hemorrhage
- 3. Laminar separation
- 4. Structural damage



# **Physiologic Alterations**

- Hypertension
- Sympathetic stimulation/tachycardia
- Systemic/local coagulopathy
- Metabolic alterations





### **Causes of Laminitis?**

- Several potential theories
  - Vascular theory
  - Toxic/enzymatic theory
  - Traumatic/mechanical theory
  - Glucose/insulin resistance theory
- Common end-result in the foot?
  - Inflammation
  - Breakdown of basement membrane of laminae
  - Separation of sensitive and insensitive laminae



### **Vascular Theory**

- Too little blood to foot
- Laminar ischemia
- Reflex hyperemia compartment syndrome/reperfusion
- Secondary inflammation
- Structural failure of laminae

# **Toxic/Enzymatic Theory**

- Excessive blood to foot
- "Trigger factors" within the blood (from gut or uterus) initiate damage
- Several enzymes/mediators damage basement membrane
- Secondary inflammation
- Structural failure of laminae



### **Mechanical Theory**

- Different from "classic" GI form
- Traumatic tearing of laminae from excessive weight bearing?
- Pain contributes to increased cortisol
  - Increased insulin?
  - Vasospasm of digital vessels?
- Secondary inflammation
- Structural failure of laminae



### Glucose/IR Theory

- Hyperglycemia/insulin resistance
  - Impaired blood flow
  - Direct effect of increased insulin
- Impaired glucose uptake
  - Laminar cells have high glucose requirement
- Secondary inflammation
- Structural failure of laminae



### **Diagnosis**

#### Clinical signs

- Increased digital pulses
- Heat over dorsal hoof wall
- Variable lameness but often severe
- Heel-toe landing/hoof distortion in chronic cases
- Pain over toe with hoof testers

#### Local anesthesia

Improve with palmar digital, basi-sesamoid or abaxial blocks

#### Radiography

Rotation, sinking or both



### **Chronic Laminitis**



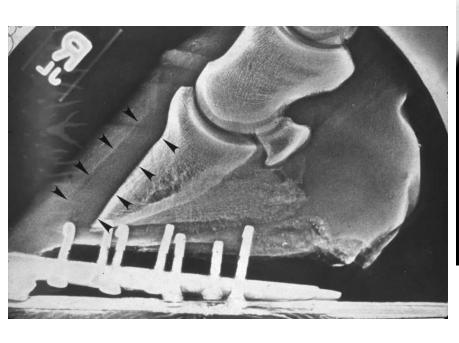


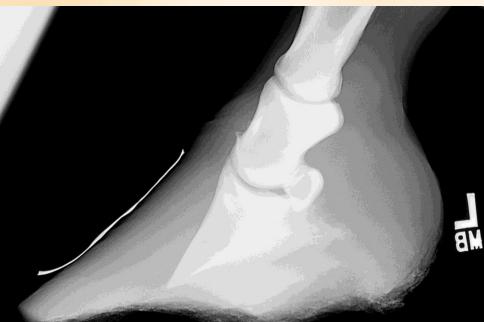


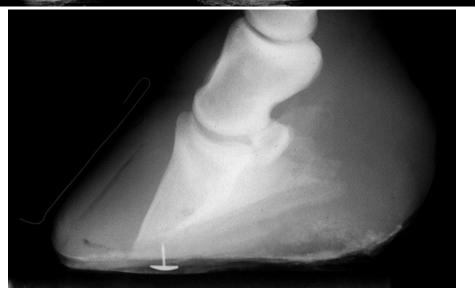




### Radiology - "Rotation"



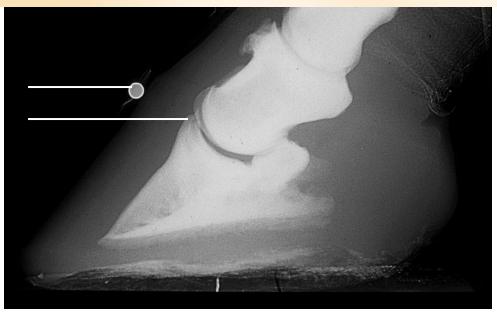


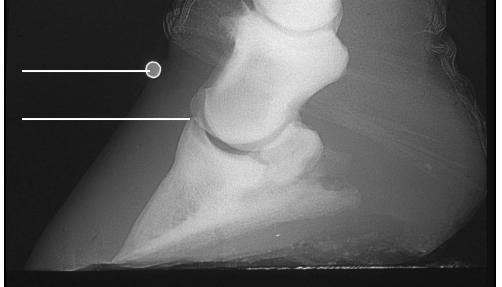




## Radiology - "Sinking"



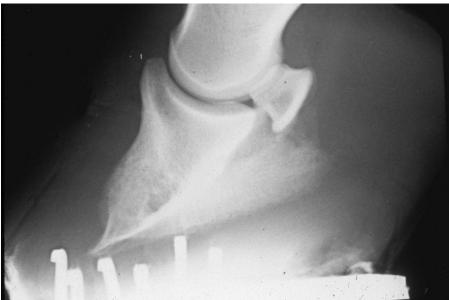


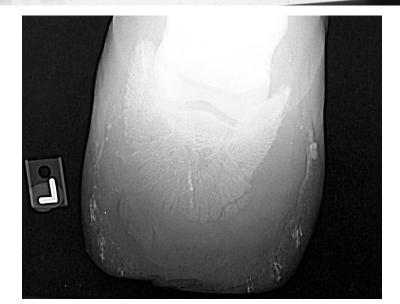




# Radiology - chronic

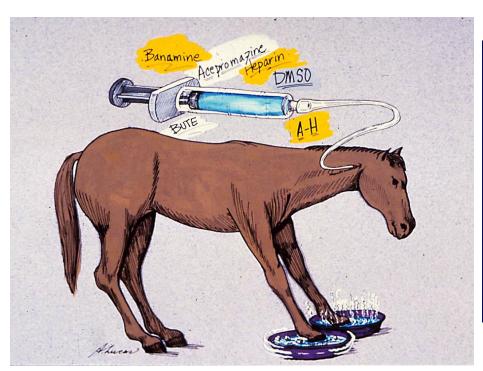


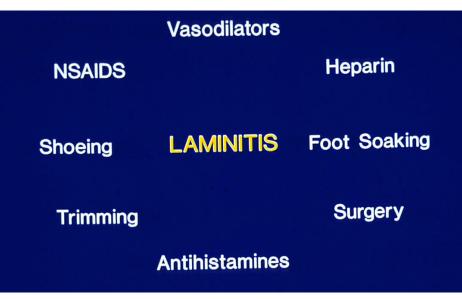






#### **Treatment/Prevention**





Developmental – to prevent disease progression Acute – to minimize severity of laminar damage Chronic – to limit further movement of coffin bone



#### **Treatment**

- Remove or treat inciting cause
  - Minimize toxemia or "trigger factors"
  - Mineral oil, flunixin meglumine, DMSO
- Cushing's pergolide
- Metabolic syndrome/IR
  - Decrease glycemic index (CHOs)
  - Pergolide and thyroxine?
- Mechanical
  - Sling/ improve weight bearing on other limb
  - Support of contralateral foot

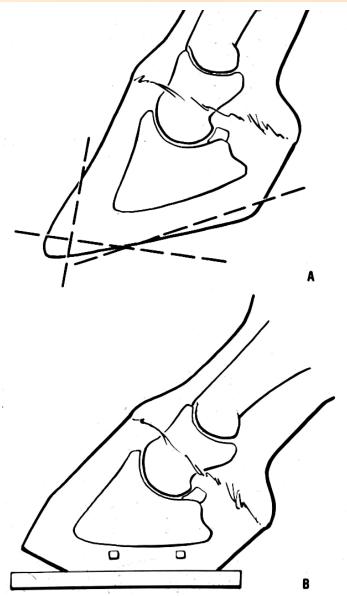


#### **Treatment**

- Dependent on stage of disease
  - Cryotherapy (ice) in acute stage?
  - Vasodilatory drugs?
  - Anti-inflammatory drugs
  - Pain control (decrease sympathetic response)
- Minimize toe length (remove shoe)
  - Bevel dorsal hoof wall of toe
- Increase weight bearing surface area
  - Styrofoam, sand, frog pads, soft putty
  - Corrective shoeing



## **Initial Foot Support**









### **Corrective Shoeing**











# **Foot Preparation**

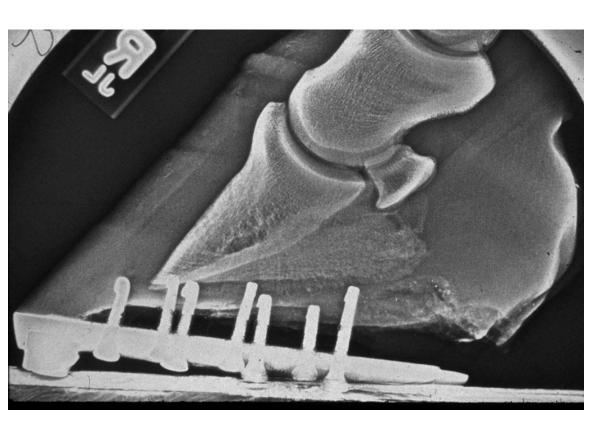






### 6-year old App. Stallion

- Unilateral right forelimb laminitis
- No known predisposing cause (toe grabs and long toe?)



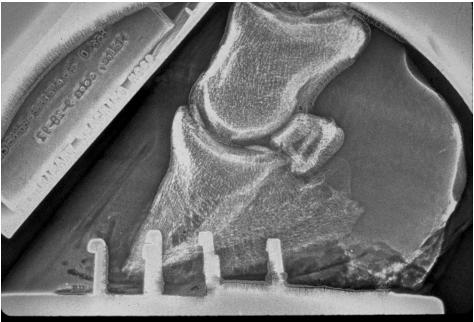
Removed shoe, shortened toe, and applied a lily pad for frog support



### Follow-up Radiographs

10 days 4 weeks





Progressive rotation of coffin bone
Abscess developed at toe region
Performed unilateral deep digital flexor tenotomy
Applied extended heel shoe with a treatment plate



### Thank you from CSU-VTH

