Osteoarthritis

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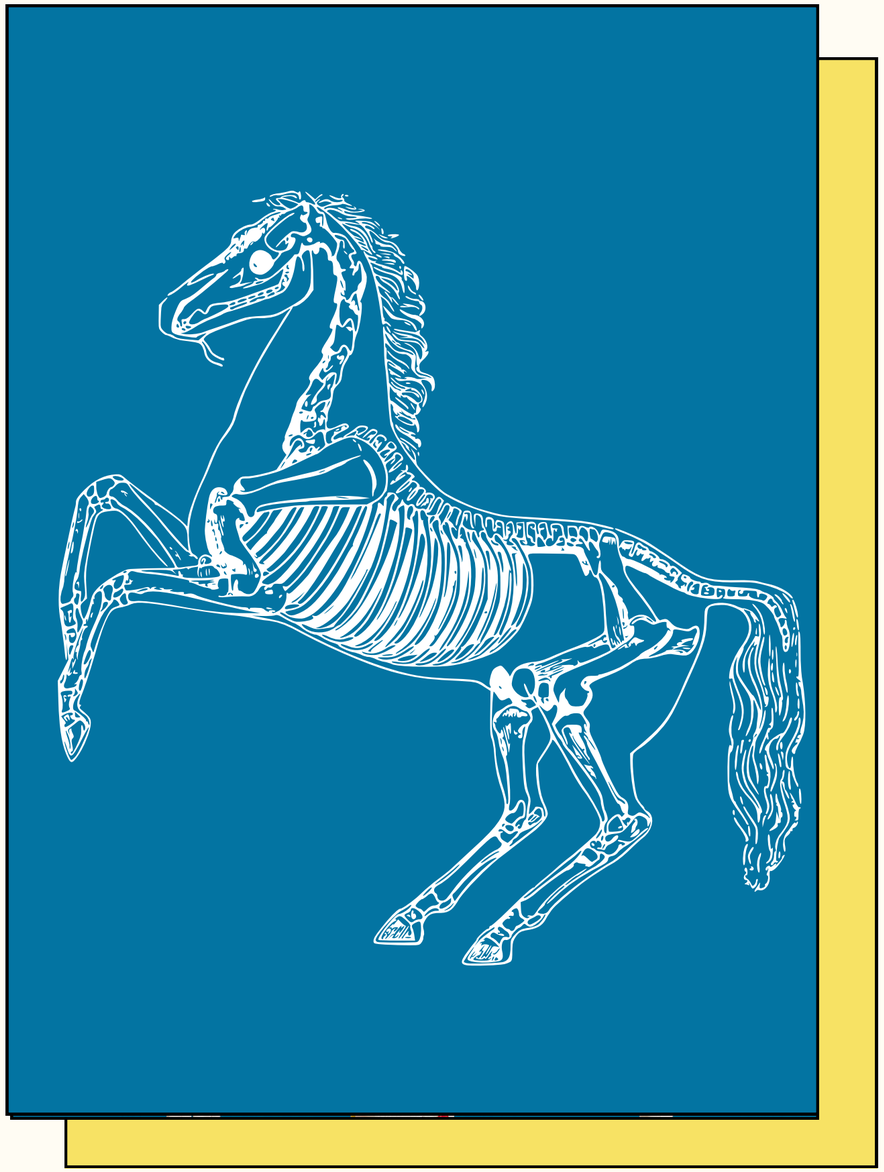
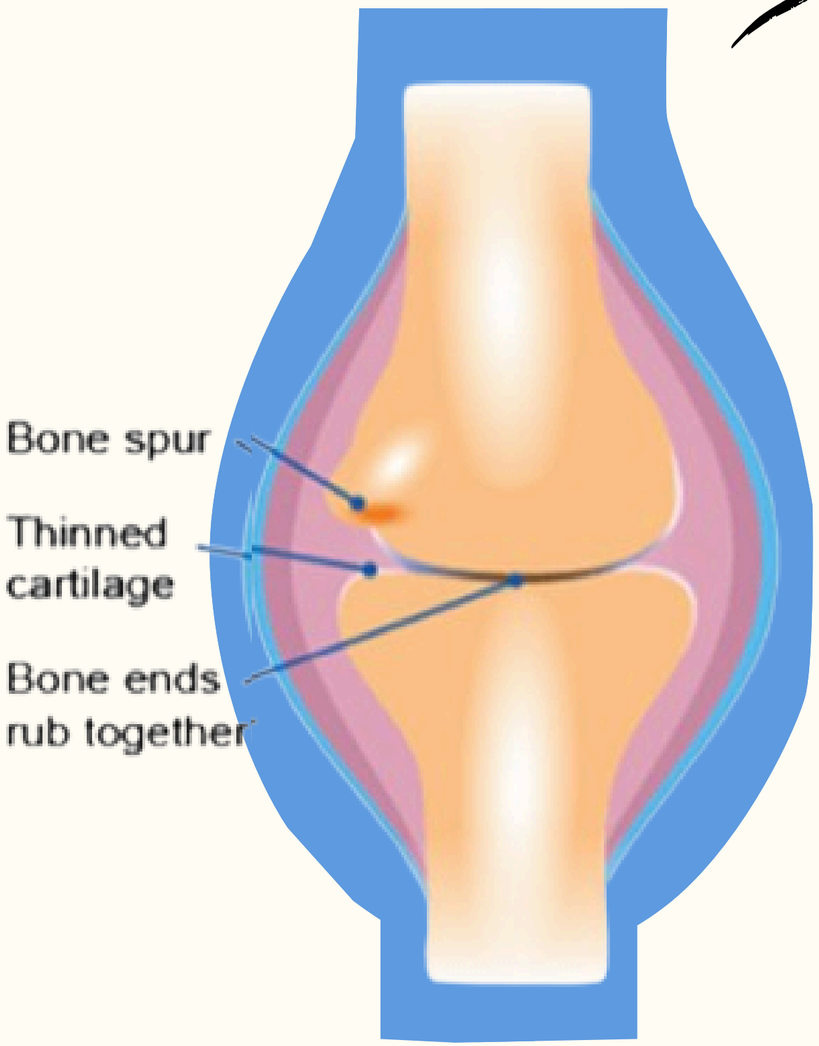




Overview

Osteoarthritis (OA), also known as degenerative joint disease (DJD), is a **chronic, progressive, noninfectious deterioration of articular cartilage.**

It results in changes within the joint including **cartilage loss, subchondral bone sclerosis, osteophyte formation, and synovial membrane inflammation.**

Signalment

Athletic Horses

Repetitive training that overwhelms repair mechanism

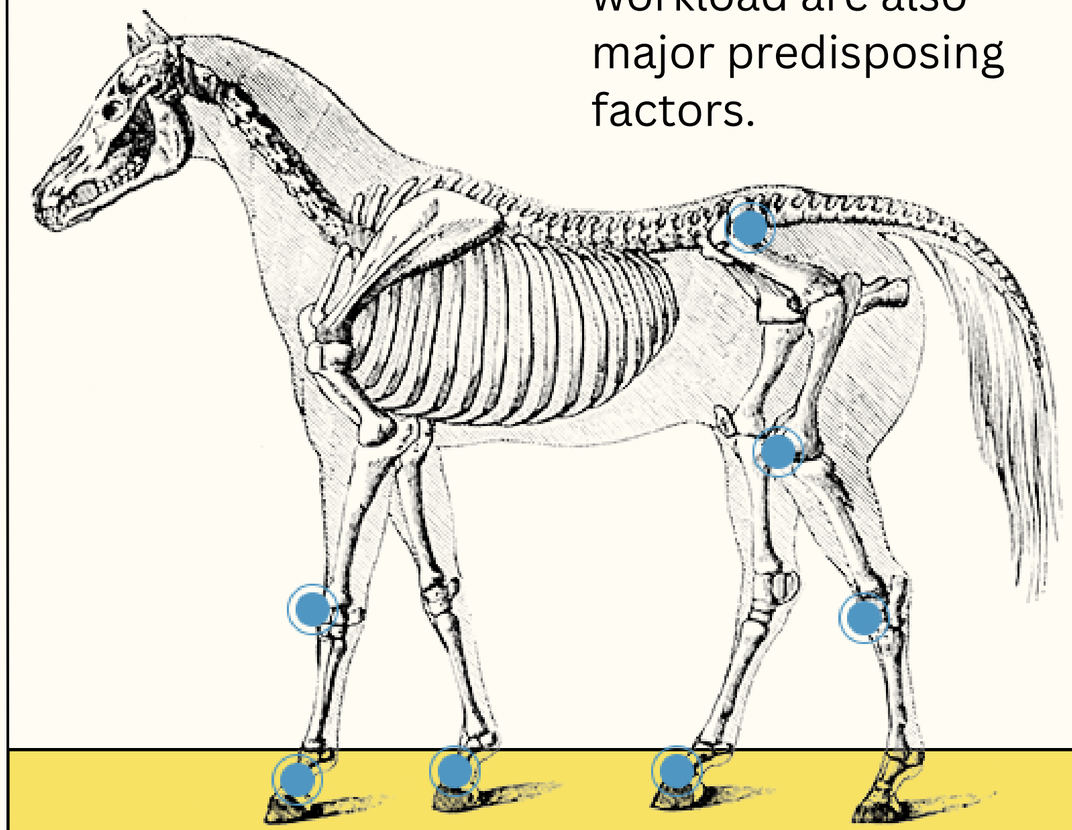
All ages and both sexes

Etiology / Causes of Osteoarthritis

OA commonly develops secondary to **joint instability, trauma, or abnormal biomechanical stress**. These may result from:

- Poor joint conformation or limb deformities
- Repetitive stress or excessive exercise
- Previous injury to ligaments, tendons, or cartilage
- Infectious arthritis or osteochondrosis (in young horses)

Aging and heavy workload are also major predisposing factors.



Pathophysiology

1. Mechanical Overload

- Abnormal repetitive forces (e.g., trauma, racing, jumping) exceed the normal repair capacity of cartilage.
- Results in matrix degradation, chondromalacia, and loss of viscoelastic properties.

2. Synovial Inflammation (Synovitis)

- The inflamed synovium produces inflammatory mediators such as:
- Prostaglandin E_2 (PGE_2)
- Cytokines (IL-1, TNF-α)
- Matrix metalloproteinases (MMPs) and other degradative enzymes
- These promote cartilage breakdown, pain, and joint effusion.

3. Loss of Synovial Fluid Quality

- Inflammation reduces synovial fluid viscosity and hyaluronic acid concentration.
- Leads to poor lubrication and reduced shock absorption.

Pathophysiology

4. Subchondral Bone Changes

- Bone becomes sclerotic and less compliant, transmitting excessive forces to cartilage.
- Remodeling creates subchondral bone sclerosis and periarticular bone proliferation.

5. Osteophyte Formation

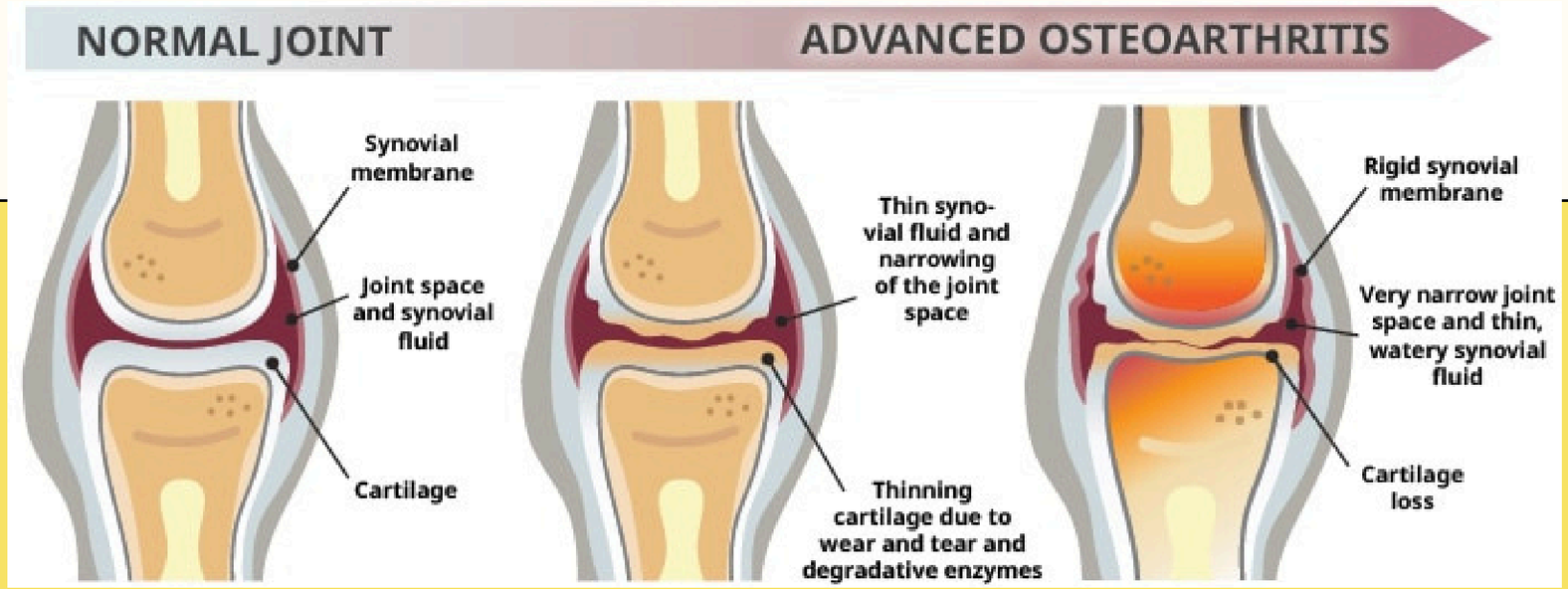
- Bone reacts to instability by forming osteophytes and enthesophytes at joint margins.
- These bony projections can fracture (chip fractures), increasing inflammation.

6. Progression Cycle

- Continuous mechanical stress and inflammation perpetuate the cycle of cartilage degeneration, synovial inflammation, and bone remodeling → chronic OA.

Signs

The hallmark of OA is articular **cartilage degeneration,** occurring in a tissue devoid of innervation. Pain originates from periarticular soft tissues or subchondral bone.



Signs

Historical Findings

- History of intra-articular fracture, osteochondrosis, dislocation, or ligament injury.
- Insidious lameness in low-motion joints (distal intertarsal, tarsometatarsal).
- High-motion joints (fetlock) more likely show acute-onset lameness.
- Lameness may decrease after rest but return with exercise.

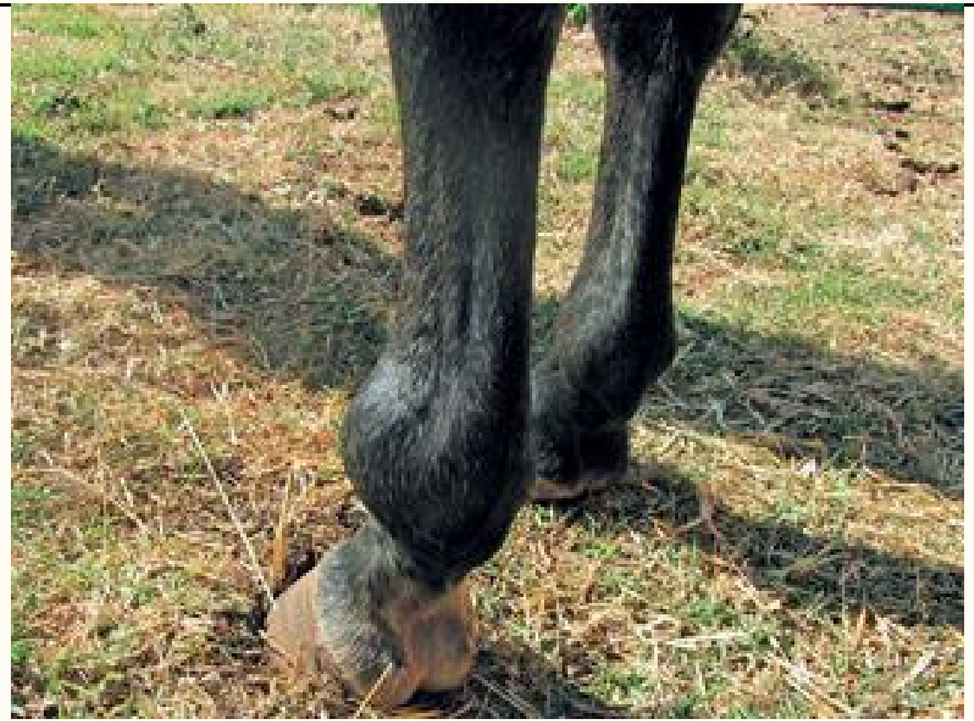
Signs

Physical Examination Findings

- Variable lameness: mild during exercise, severe in advanced stages.
- Pain on flexion of affected joint(s).
- Synovial effusion—especially in high-motion joints.
- Reduced range of motion; stiffness after rest.
- Chronic OA joints: palpable thickening of the joint capsule, hardening of periarticular tissues, crepitus on movement
- Pain and swelling localized to affected joint.

Signs

Physical Examination Findings



OA in the fetlock

Lameness due to OA

Diagnosis

Differential Diagnosis

- Septic arthritis (ruled out via synovial fluid analysis).
- Osteochondrosis or joint effusion (radiography).

Diagnosis

Imaging

- **Radiography:**
  - Narrowing or loss of joint space.
  - Osteophyte formation or fragmentation.
  - Periosteal bone proliferation.
  - Subchondral bone sclerosis.
- **MRI**: best for early cartilage and soft tissue changes.
- **Arthroscopy**: direct visualization of articular cartilage and subchondral bone lesions.

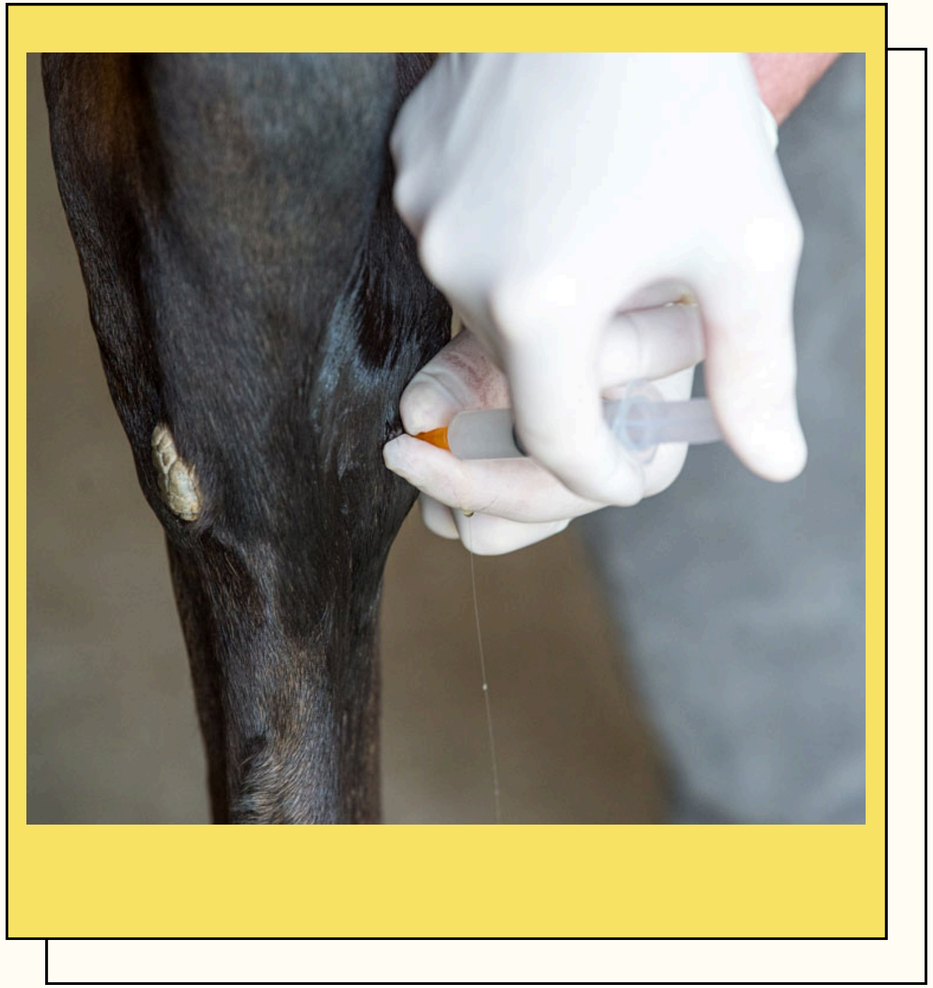
Other Laboratory Findings

Joint fluid viscosity may be reduced.

Treatment

Aims

- Reduce joint inflammation and pain.
- Restore cartilage homeostasis and mobility.
- Delay progression of degenerative changes.



Treatment

Management Strategies

- **Rest and controlled exercise:** Short-term rest during acute inflammation; gradual return to work.
- **Weight management:** Avoid obesity to reduce joint load.
- **Hoof trimming and corrective shoeing:** Balance limb forces and minimize concussion.
- **Cold therapy:** Reduces heat and swelling during acute inflammation.
- **Hydrotherapy or swimming:** Maintains fitness while minimizing joint stress.
- **Physiotherapy and stretching:** Preserve range of motion.
- **Environmental management:** Soft footing in training and turnout areas.

Medications

1. Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

- Control pain and reduce inflammation.
- Phenylbutazone: 2.2 mg/kg PO q12–24h.
- Flunixin meglumine: 0.5 mg/kg PO or IV q12–24h.
- Caution: Avoid prolonged use in dehydrated or ulcer-prone horses.

Medications

2. Intra-Articular Therapy

- Hyaluronic Acid (HA): Restores viscosity, lubricates joint (40 mg IV weekly × 2–3).
- Polysulfated Glycosaminoglycan (PSGAG): Stimulates cartilage repair (500 mg IM q4d × 7 doses).

Corticosteroids:

- Methylprednisolone acetate (20–60 mg/joint) — long-acting.
- Triamcinolone acetate (3–18 mg/joint) — potent anti-inflammatory.
- Often combined with HA for synergistic effect.
- Antibiotic addition: Amikacin (125 mg) to prevent iatrogenic infection.

Medications

3. Systemic Therapy

- IV or IM HA and PSGAG courses as adjuncts to local therapy.
- Autologous biologics (IRAP, PRP, ACS): Decrease cytokine-mediated cartilage damage.

4. Nutraceuticals and Adjuncts

- Chondroitin sulfate, glucosamine, omega-3 fatty acids—may aid long-term management (evidence variable).
- Topical anti-inflammatories for mild joint pain.

Medications

Contraindications & Precautions

- Avoid intra-articular corticosteroids in horses with laminitis risk.
- Strict aseptic technique during injections.
- Sedate and restrain properly; bandage joint post-injection.
- Repeated corticosteroid use can accelerate cartilage degeneration.

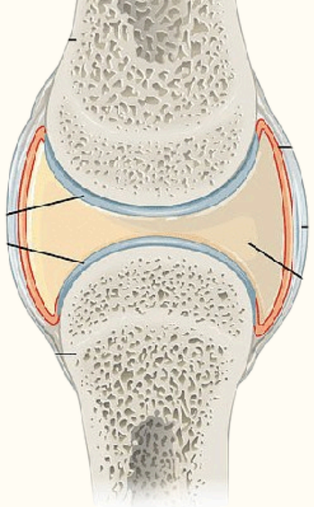
FOLLOW-UP AND PROGNOSIS

Monitoring

- Re-examine after treatment for pain, effusion, and lameness.
- Periodic radiographs to assess progression.
- “Joint flare” (sterile synovitis) after injection.
- Septic arthritis if asepsis compromised.
- Cartilage softening or laminitis with steroid overuse.

Prognosis

- Mild OA in low-motion joints: Good prognosis with early management.
- Severe OA in high-motion joints: Guarded to poor for athletic performance.
- Arthrodesis (joint fusion) may restore comfort for breeding or retirement.



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