DISEASES OF JOINTS DEGENERATIVE JOINT DISEASE

(OSTEOARTHROPATHY) AND OSTEOCHONDROSIS

There are two common noninfectious conditions of the joint, degenerative joint disease and osteochondrosis. The terms degenerative joint disease and osteoarthropathy are used here to describe noninfectious lesions of the articular surfaces of joints characterized by the following:

• Degeneration and erosion of articular cartilage

• Eburnation of subchondral bones

• Hypertrophy of bone surrounding the articular cartilage, resulting in lipping and spur formation at the joint margins.

In contrast, a separate condition is **osteochondrosis (dyschondroplasia),** which is a degeneration of both the deep layers of the articular cartilage and the epiphyseal plate— **a defect in endochondral ossification**—that occurs most commonly in pigs and horses but also occurs in cattle. Osteochondrosis in horses is one of a number of conditions included in **developmental orthopedic disease**, which is a catch phrase that includes a number of skeletal conditions of the rapidly growing horse.

ETIOLOGY AND EPIDEMIOLOGY

1- Nutritional Causes
2- Toxic Causes
3- Steroid Induced

4- Biomechanical Trauma: Acute traumatic injury, Repeated subacute trauma, Trauma caused by movement,
5- Osteochondrosis, rapped growth in cattle due to decrease in vit A,D or Ca,Ph

PATHOGENESIS

A brief review of the structure and biochemistry of the normal articular joint will serve as background for understanding the pathogenesis of osteoarthropathy. Articular cartilage is a tissue consisting of chondrocytes scattered in **a matrix of collagen fibers** and an **amorphous intercellular substance containing proteoglycans**. Articular cartilage contains no nerves, is avascular, and has a high matrix-to-cell ratio.

The chondrocytes are the only living matter in cartilage, produce the fine strands of collagen, and are engaged in protein and proteoglycan synthesis. The matrix of the cartilage consists of water-soluble proteoglycans interspersed with collagen fibers, which are arranged in parallel rows superficially and crisscross rows closer to the calcified layer.

This enables the cartilage to withstand shearing stresses superficially and compression more deeply.

The proteoglycans are **glycosaminoglycan–protein complexes, bound by a link glycoprotein to a linear hyaluronic acid molecule**. The glycosaminoglycans in articular cartilage are chondroitin 4-sulfate, chondroitin 6-sulfate, and keratan sulfate. About 75% of the proteoglycans exist on aggregates that protect them from degradation, and because of their high water content, they form large polyanionic complexes that have considerable elastic resistance to compression.

Nutrition of the articular cartilage is provided via the synovial fluid and is dependent on the capillary flow to the synovial membrane. Nutrients flow through the synovial fluid and diffuse through the cartilage to the chondrocytes. Proteoglycans are synthesized by the chondrocytes and secreted to the cell exterior.

Proteoglycans are also degraded intracellularly by lysosomes. The normal equilibrium between anabolism and catabolism is maintained by several different low-molecular-weight proteins. When the equilibrium is disturbed and shifts toward catabolism, degeneration occurs.

Primary Osteoarthropathy Primary osteoarthropathy is a result of normal aging processes and ordinary joint usage. The initial lesions occur in the superficial layers of the articular cartilages where, with increasing age, there is loss of the normal resilience of the cartilage, a lowering of the chondroitin sulfate content, and reduction in the permeability of the cartilaginous matrix, which results in progressive degeneration of the articular cartilage.

Secondary Osteoarthropathy Secondary osteoarthropathy appears to be initiated by injuries or congenital conformational defects that create greater shearing stresses on particular points, in contrast to the intermittent compressive stresses typical of ordinary weight-bearing. These irregular stresses result in cartilaginous erosion, increased density of subchondral bone at points of physical stress, and proliferation of bone and cartilage at the articular margins. These following acute trauma

Osteochondrosis Osteochondrosis (dyschondroplasia) is characterized by a focal disturbance of the normal differentiation of the cells in the growing cartilage as a result of failure of the blood supply.

Osteochondrosis should therefore be considered as a disease that occurs multifocally at predilection sites.

CLINICAL FINDINGS

The major clinical characteristic is a chronic lameness that becomes progressively worse over a long period of time and does not usually respond to treatment.

There is usually difficulty in flexing affected joints normally, which results in a stiff and stilted gait.

In dairy cattle, as the lesions become more painful, there is a decline in appetite and milk production, prolonged recumbency, and considerable difficulty in rising from the recumbent state. In the early stages, there may be an apparent remission of the lameness, but relapses are common. The bony prominences of the joint eventually appear more prominent than normal, which is a result of disuse muscle atrophy of the affected limbs.

The joint capsule of palpable joints is usually not painful on palpation.

DIAGNOSIS

1- Joint Fluid:

   a- The changes in the synovial fluid of joints affected with degenerative arthropathy are usually unremarkable and can be readily distinguished from the changes in infectious arthritis.

   b- Total protein concentration and viscosity of synovial fluid of horses can be determined.

   c- Infrared spectroscopy measures the infrared absorption patters of molecules in synovial fluid when exposed to infrared light.

2- Hematology and serum biochemistry should be combined with appropriate hematology and serum biochemistry where indicated, although the results rarely changes.
3- Radiography
4- Arthroscopy

NECROPSY FINDINGS

In degenerative joint disease the joint cartilage is thin or patchily absent, and polished subchondral bone is evident. The articular surfaces are irregular and sometimes folded. Exposed bone may be extensively eroded, and osteophytes (small bony excrescences, appearing like pearls) may be present on the nonarticular parts of the joint on the circumference of the articular cartilage.

DIFFERENTIAL DIAGNOSIS

Osteoarthropathy is characterized clinically by a chronic lameness that becomes progressively worse and usually does not respond to treatment. The gait is stiff, there is disuse muscle atrophy, the bony prominences of the joint are more apparent, but usually there is no marked distension and pain of the joint capsule, as in infectious arthritis. Examination of synovial fluid may aid in differentiation from septic arthritis. Radiographically, there is erosion of articular cartilage, sclerosis of subchondral bone, and periarticular accumulations of osteophytes. In the early stages of the disease in large animals, radiographic changes may not be visible, and repeated examinations may be necessary.

The radiographic changes of osteochondrosis in the shoulder joint of the horse consist of the following:

• Alteration in the contour of the humeral head and glenoid cavity

• Periarticular osteophyte formation

• Sclerosis of the subchondral bone

• Bone cyst formation

TREATMENT

Nonsteroidal Antiinflammatory Agents and Opioids Several nonsteroidal antiinflammatory drugs (NSAIDs), such as phenylbutazone, flunixin meglumine, ketoprofen, naproxen, and carprofen, are available treatment options. Each has associated toxicities.