DISEASES OF BONES

OSTEODYSTROPHY

Osteodystrophy is a general term used to describe those diseases of bones in which there is a failure of normal bone development or abnormal metabolism of bone that is already mature. The major clinical manifestations include distortion and enlargement of the bones, susceptibility to fractures, and interference with gait and posture.

ETIOLOGY

The common causes of osteodystrophy in farm animals include the following.

1- Nutritional Causes

Calcium, Phosphorus, and Vitamin D Absolute deficiencies or imbalances in calcium–phosphorus ratios in diets cause the following conditions:

1• Rickets in young animals (e.g., growing lambs fed a diet rich in wheat bran)

2• Absolute deficiencies of calcium in beef calves on intensive rations with inadequate supplementation

3• Osteomalacia in adult ruminants Osteodystrophia fibrosa in the horse occurs most commonly in animals receiving a diet low in calcium and high in phosphorus. Osteodystrophia fibrosa in pigs occurs as a sequela to rickets and osteomalacia, which may occur together in young, rapidly growing pigs that are placed on rations deficient in calcium, phosphorus, and vitamin D following weaning. Copper Deficiency

4• Osteoporosis in lambs

5• Epiphysitis in young cattle Other Nutritional Causes

6• Inadequate dietary protein and general undernutrition of cattle and sheep can result in severe osteoporosis and a great increase in ease of fracture.

7• Chronic parasitism can lead to osteodystrophy in young, rapidly growing ruminants.

8• Hypovitaminosis A and hypervitaminosis A can cause osteodystrophic changes in cattle and pigs.

9• Prolonged feeding of a diet high in calcium to bulls (such as high-quality alfalfa) can cause nutritionalhypercalcitoninism, replacement of trabecular bone in the vertebrae and long bones with compact bone, and neoplasms of the ultimobranchial gland.

10• Multiple vitamin and mineral deficiencies are recorded as causing osteodystrophy in cattle. The mineral demands of lactation in cattle can result in a decrease in bone mineral content during lactation with a subsequent increase during the dry period.

2- Chemical Agents

1• Chronic lead poisoning is reputed to cause osteoporosis in lambs and foals.

2• Chronic fluorine poisoning causes the characteristic lesions of osteofluorosis, including osteoporosis and exostoses.

3• Grazing the poisonous plants Setaria sphaceleta, Cenchrus ciliaris, and Panicum maximum var. trichoglume causes osteodystrophia in horses.

4• Enzootic calcinosis of muscles and other tissues is caused by the ingestion of Solanum malacoxylon, Solanum torvum, Trisetum flavescens (yellow oatgrass), and Cestrum diurnum, which exert a vitamin D–like activity.

5• Bowie or bentleg, a disease caused by poisoning with Trachymene glaucifolia, is characterized by extreme outward bowing of the bones of the front limbs.

3- Inherited and Congenital Causes

There are many inherited and congenital defects of bones of newborn farm animals, which are described and discussed later in this chapter. In summary, these include:

1• Achondroplasia and chondrodystrophy in dwarf calves and some cases of prolonged gestation

2• Osteogenesis imperfecta in lambs and Charolais cattle. There is marked bone fragility and characteristic changes on radiologic examination.

3• Osteopetrosis in Hereford and Angus calves

4• Congenital chondrodystrophy of unknown origin (“acorn” calves)

5• Inherited exostoses in horses; inherited thick legs and inherited rickets of pigs, which are well-established entities Angular deformities of joints of long bones as a result of asymmetric growth-plate activity are common in foals and are commonly repaired surgically. The distal radius and distal metacarpus are most often affected, the distal tibia and metatarsal less commonly. Physiologically immature foals subjected to exercise may develop compression-type fractures of the central or third tarsal bones. Some of these foals are born prematurely or are from a twin pregnancy. Retained cartilage in the distal radial physis of foals 3 to 70 days of age presents without apparent clinical signs. Physitis is dysplasia of the growth plate,

characterized by an irregular border betweenthe cartilage and the metaphyseal zone of ossification, an increase in the lateromedial diameter of the physis, and distoproximally oriented fissures at the medial aspect of the metaphysis, which originate at the physis. In some cases, these may result in bilateral tibial metaphyseal stress fractures in foals. Abnormal modeling of trabecular bone has been recognized in prenatal and neonatal calves. Abnormalities included growth retardation lines and lattices, focal retention of primary spongiosa, and the persistence of secondary spongiosa. Intrauterine infection with viruses such as bovine virus diarrhea (BVD) may be a causative factor.

4- **Physical and Environmental Causes**

Moderate osteodystrophy and arthropathy may occur in rapidly growing pigs and cattle raised indoors and fed diets that contain adequate amounts of calcium, phosphorus, and vitamin D. Those animals raised on slatted floors or concrete floors are most commonly affected, and it is thought that traumatic injury of the epiphyses and condyles of long bones may be predisposing factors in osteochondrosis and arthrosis in the pig (leg weakness) and epiphysitis in cattle. Experimentally raising young calves on metal slatted floors may result in more severe and more numerous lesions of the epiphysis than occurs in calves raised on clay floors. Total confinement rearing of lambs can result in the development of epiphysiolysis and limb deformities. However, the importance of weight-bearing injury as a cause of osteodystrophy in farm animals is still uncertain. In most reports of such osteodystrophy, all other known causes have not been eliminated. Chronic osteodystrophy and arthropathy have been associated with undesirable conformation in the horse. Vertebral exostoses are not uncommon in old bulls and usually affect the thoracic vertebrae (T2 and T12) and the lumbar vertebrae (L2 to L3), which are subjected to increased pressure during the bending of the vertebral columns while

copulating. The exostoses occur mainly on the ventral aspects of the vertebrae, fusing them to cause immobility of the region. Fracture of the ossification may occur, resulting in partial displacement of the vertebral column and spinal cord compression. The disease is commonly referred to as spondylitis or vertebral osteochondrosis and also occurs less commonly in adult cows and in pigs. It is suggested that the annulus fibrosus degenerates and that the resulting malfunctioning of the disk allows excessive mobility of the vertebral bodies, resulting in stimulation of new bone formation. A similar lesion occurs commonly in horses and may affect performance, particularly in hurdle races and cross-country events. The initial lesion may be a degeneration of the intervertebral disk. Some types of growth-plate defects occur in young, rapidly growing foals, andthese are considered to be traumatic in origin. Failure of chondrogenesis of the growth plate may be the result of crush injuries in heavy, rapidly growing foals with interruption of the vascular supply to the germinal cells of the growth plate. Asymmetric pressures as a result of abnormal muscle pull or joint laxity may slow growth on the affected side and result in limb angulation. Femoral fractures occur in newborn calves during the process of assisted traction during birth. Laboratory compression of isolated femurs from calves revealed that the fracture configurations and locations are similar to those found in clinical cases associated with forced extraction. The breaking strength of all femurs fell within the magnitude of forces calculated to be created when mechanical devices are used to assist delivery during dystocia. It is suggested that the wedging of the femur in the maternal pelvis and resulting compression during forced extraction accounts for the occurrence of supracondylar fractures of the femur of calves delivered in anterior presentation using mechanical devices in a manner commonly used by veterinarians and farmers. Tumors Osteosarcomas are highly malignant tumors of skeletoblastic mesenchyme in which the tumor cells produce osteoid or bone. Osteosarcomas are the most common type of primary bone tumor in animals

such as dogs and cats but are rare in horses and cattle. Most tumors of bone in large animals occur in the skull. A periosteal sarcoma on the scapula has been recorded in the horse and an osteosarcoma of the mandible in a cow.

PATHOGENESIS

Osteodystrophy is a general term used to describe those diseases of bones in which there is a failure of normal bone development or abnormal metabolism of bone that is already mature. There are some species differences in the osteodystrophies that occur with dietary deficiencies of calcium, phosphorus, and vitamin D. Rickets and osteomalacia have a similar pathogenesis, with the end result being decreased or defective bone mineralization. In broad terms, rickets is the failure of endochondral ossification in growing bone, whereas osteomalacia is disrupted remodeling in mature bone. Rickets and osteomalacia occur primarily in ruminants fed a deficient diet, osteodystrophia fibrosa occurs in horses, and all three may occur in pigs.

Rickets

Rickets is a disease of young, rapidly growing animals in which there is a failure of provisional calcification of the osteoid plus a failure of mineralization of the cartilaginous matrix of developing bone. There is also failure of degeneration of growing cartilage and formation of osteoid on persistentcartilage, with irregularity of osteochondral junctions and overgrowth of fibrous tissue in the osteochondral zone. Rickets is most commonly caused in ruminants by a deficiency of vitamin D or phosphorus. Genetic causes of rickets exist, one of which is a simple autosomal-recessive inheritance in Corriedale sheep in New Zealand. Failure of provisional calcification of cartilage results in an increased depth and width of the epiphyseal plates, particularly of the long bones (**humerus, radius, ulna and tibia)** and the costal cartilages of the ribs. The uncalcified, and therefore soft, tissues of the

metaphyses and epiphyses become distorted under the pressure of weight-bearing, which also causes medial or lateral deviation of the shafts of long bones. There is a decreased rate of longitudinal growth of long bones and enlargement of the ends of long bones as a result of the effects of weight, causing flaring of the diaphysis adjacent to the epiphyseal plate. Within the thickened and widened epiphyseal plate, there may be hemorrhages and minute fractures of adjacent trabecular bone of the metaphysis, and in chronic cases the hemorrhagic zone may be largely replaced by fibrous tissue. These changes can be seen radiographically as "epiphysitis" and clinically as enlargements of the ends of long bones and costochondral junctions of the ribs. These changes at the epiphyses may result in separation of the epiphysis, which commonly affects the femoral head. The articular cartilages may remain normal, or there may be subarticular collapse resulting in grooving and folding of the articular cartilage and ultimately degenerative arthropathy and osteochondrosis. Eruption of the teeth in rickets is irregular, and dental attrition is rapid. Growth of the mandibles is retarded and is combined with abnormal dentition. There may be marked malocclusion of the teeth.

Osteomalacia

Osteomalacia is a softening of mature bone as a result of extensive resorption of mineral deposits in bone and failure of mineralization of newly formed matrix. There is no enlargement of the ends of long bones or distortions of long bones, but spontaneous fractures of any bone subjected to weightbearing are common.

Osteodystrophia Fibrosa

Osteodystrophia fibrosa may be superimposed on rickets or osteomalacia and occurs in secondary hyperparathyroidism. Diets low in calcium or that contain a relative excess of phosphorus cause secondary hyperparathyroidism. There is extensive resorption of bone and replacement by connective tissue. The disease is

best known in the horse and results in swelling of the mandibles, maxillae, and frontal bones (the "bighead" syndrome). Spontaneous fracture of long bones and ribs occurs commonly.

Radiographically there is extreme porosity of the entire skeleton. Osteoporosis Osteoporosis is defined as a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility and increased susceptibility to fractures.5 In osteoporosis, the bone becomes porous, light, and fragile, and it fractures easily. Osteoporosis is uncommon in farm animals and is usually associated with general undernutrition and intestinal parasitism rather than specifically a deficiency of calcium, phosphorus, or vitamin D. Copper deficiency in lambs may result in osteoporosis as a result of impaired osteoblastic activity. Chronic lead poisoning in lambs also results in osteoporosis as a result of deficient production of osteoid. In a series of 19 lactating or recently weaned sows with a history of lameness, weakness, or paralysis, 10 had osteoporosis and pathologic fractures, and six had lumbar vertebral osteomyelitis. Bone ash, specific gravity of bone, and the ratio of cortical to total bone were significantly reduced in sows with osteoporosis and pathologic fractures. Ovariectomized sheep that are fed an acidogenic (calcium-wasting) diet and administered corticosteroids develop osteoporosis, which is being used as a model to study the disease in humans.

Osteodystrophy of Chronic Fluorosis

Osteodystrophy of chronic fluorosis is characterized by the development of exostoses on the shafts of long bones as a result of periosteal hyperostosis. The articular surfaces remain essentially normal, but there is severe lameness because of the involvement of the periosteum and encroachment of the osteophytes on the tendons and ligaments.

Congenital Defects of Bone

Congenital defects of bone include complete (achondroplasia) and partial (chondrodystrophy) failure of normal development of cartilage. Growth of the cartilage is restricted and disorganized, and mineralization is reduced. The affected bones fail to grow, leading to gross deformity, particularly of the bones of the head.

CLINICAL FINDINGS

In general terms there is weakening of the bones as a result of defective mineralization and osteoporosis, which results in the bending of bones, which probably causes pain and shifting lameness—one of the earliest clinical signs of acquired osteodystrophy. The normal weight and tension stresses cause distortion of the normal axial relationships of the bones, which results in the bowing of long bones. The distortions occur most commonly in young, growing animals. The distal ends of the long bones are commonly enlarged at the level of the epiphyseal plate, and circumscribed swellings of the soft tissue around the epiphyses may be prominent and painful on palpation. The effects of osteodystrophy on appetite and body weight will depend on the severity of the lesions and their distribution. In the early stages of rickets in calves and pigs the appetite and growth rate may not be grossly affected until the disease is advanced and causes considerable pain. Persistent recumbency as a result of pain will indirectly affect feed intake unless animals are hand-fed. Spontaneous fractures occur commonly and usually in mature animals. Common sites for fractures include the long bones of the limbs, pelvic girdle, femoral head, vertebrae, ribs, and transverse processes of the vertebrae. Ordinary hand pressure or moderate restraint of animals with osteomalacia and osteodystrophia fibrosa is often sufficient to cause a fracture. The rib cage tends to

become flattened, and in the late stages affected animals have a slab-sided appearance of the thorax and abdomen. Separations of tendons from their bony insertions also occur more frequently and cause severe lameness. The osteoporotic state of the bone makes such separations easy. Any muscle group may be affected, but in young cattle in feedlots, separations of the gastrocnemius are the most common. Thickening of the bones may be detectable clinically if the deposition of osteoid or fibrous tissue is excessive or if exostoses develop, as in fluorosis. Compression of the spinal cord or spinal nerves may lead to paresthesia, paresis, or paralysis, which may be localized in distribution. Details of the clinical findings in the osteodystrophies caused by nutritional deficiencies are provided later in this chapter. Calcinosis of cattle is characterized clinically by chronic wasting; lameness; ectopic calcifications of the cardiovascular system, lungs, and kidneys; ulceration of joint cartilage; and extensive calcification of bones.

DIAGNOSIS

The laboratory analyses that are indicated include the following:

• Serum calcium and phosphorus concentration

• Serum alkaline phosphatase activity

• Feed analysis for calcium, phosphorus, vitamin D, and other minerals when indicated (such as copper, molybdenum, and fluorine)

• Bone ash chemical analysis

• Histopathology of bone biopsy

• Radiographic examination of the skeleton

• Single-photon absorptiometry, a safe and noninvasive method for the measurement of bone mineral content, is now available. Radiographic examination of the affected bones and comparative radiographs of normal bones are indicated when osteodystrophy is suspected. Radiographic examination of slab sections of bone is a sensitive method for detecting abnormalities of trabecular bone in aborted and young calves. Serum calcium and phosphorus concentrations in nutritional osteodystrophies may remain within the normal range for long periods, and not until the lesions are well advanced will abnormal levels be found. Several successive samplings may be necessary to identify an abnormal trend. Serum alkaline phosphatase activity may be increased in the presence of increased bone resorption, but this is not a reliable indicator of osteodystrophy. Increased serum levels of alkaline phosphatase may originate from osseous tissues, intestine, or the liver, but osseous tissue appears to be the major source of activity. Nutritional history and feed analysis results will often provide the best circumstantial evidence of osteodystrophy. In vitamin D–dependent rickets, serum 25(OH) D3 concentrations will be decreased. In phosphorus-dependent rickets, serum 25(OH)D3 will be normal or increased with normal to decreased parathyroid (PTH) concentrations. Urine calcium-to-phosphorus ratios below 0.05 suggest a calcium or vitamin D deficiency, whereas ratios above 1 reflect phosphorus deficiency. The definitive diagnosis is best made by a combination of chemical analysis of bone, histopathologic examination of bone, and radiography. The details for each of the common osteodystrophies are discussed under the appropriate headings.

NECROPSY FINDINGS

The pathologic findings vary with the cause, and the details are described under each of the osteodystrophies elsewhere in the book. In general terms, the nutritional osteodystrophies are characterized by bone deformities, bones that may be cut easily

with a knife and that bend or break easily with hand pressure, and in prolonged cases the presence of degenerative joint disease. In young growing animals the ends of long bones may be enlarged, and the epiphyses may be prominent and circumscribed by periosteal and fibrous tissue thickening. On longitudinal cut sections the cortices may appear thinner than normal, and the trabecular bone might have been resorbed, leaving an enlarged marrow cavity. The epiphyseal plate may be increased in depth and width and appear grossly irregular, and small fractures involving the epiphyseal plate and adjacent metaphysis may be present. Separation of epiphyses is common, particularly of the femoral head. The calluses of healed fractures of long bones, ribs, vertebrae, and the pelvic girdle are common in pigs with osteodystrophy. On histologic examination there are varying degrees of severity of rickets in young, rapidly growing animals and osteomalacia in adult animals, and osteodystrophia fibrosa is possible in both young and adult animals.

DIFFERENTIAL DIAGNOSIS

In both congenital and acquired osteodystrophy the clinical findings are usually suggestive. There are varying degrees of lameness, stiff gait, long periods of recumbency and failure to perform physical work normally, and progressive loss of body weight in some cases, and there may be obvious contortions of long bones, ribs, head, and vertebral column. The most common cause of osteodystrophy in young, rapidly growing animals is a dietary deficiency or imbalance of calcium, phosphorus, and vitamin D. If the details of the nutritional history are available and if a representative sample of the feed given is analyzed, a clinical diagnosis can be made on the basis of clinical findings, nutritional history, and response to treatment. In some cases, osteodystrophy may be attributable to overfeeding, such as might occur in rapidly growing, large foals. However, often the nutritional history may

indicate that the animals have been receiving adequate quantities of calcium, phosphorus, and vitamin D, which necessitates that other, less common causes of osteodystrophy be considered. Often the first clue is an unfavorable response to treatment with calcium, phosphorus, and vitamin D. Examples include copper deficiency in cattle, leg weakness in swine of uncertain etiology—but perhaps there is weight-bearing trauma and a relative lack of exercise because of confinement—or chemical poisoning such as enzootic calcinosis or fluorosis. These will require laboratory evaluation of serum biochemistry, radiography of affected bones, and pathologic examination. The presence of bony deformities at birth suggests congenital chondrodystrophy, some cases of which appear to be inherited, whereas some are attributable to environmental influences.

TREATMENT

The common nutritional osteodystrophies attributable to a dietary deficiency or imbalance of calcium, phosphorus, and vitamin D will usually respond favorably following the oral administration of a suitable source of calcium and phosphorus combined with parenteral injections of vitamin D. The oral administration of dicalcium phosphate, at the rate of 3 to 4 times the daily requirement, daily for 6 days, followed by a reduction to the daily requirement by the 10th day, combined with one injection of vitamin D at the rate of 10,000 IU/kg BW, is recommended. Affected animals are placed on a diet that contains the required levels and ratios of calcium, phosphorus, and vitamin D. The oral administration of the calcium and phosphorus will result in increased absorption of the minerals, which will restore depleted skeletal reserves. Calcium absorption isincreased in adult animals

following a period of calcium deficiency; young animals with high growth requirements absorb and retain calcium in direct relation to intake. General supportive measures include adequate bedding for animals that are recumbent. The treatment of the osteodystrophies resulting from causes other than calcium and phosphorus deficiencies depends on the cause. Copper deficiency will respond gradually to copper supplementation. There is no specific treatment for the osteodystrophy associated with leg weakness in pigs, and slaughter for salvage is often necessary. Overnutrition in young, rapidly growing foals may require a marked reduction in the total amount of feed made available daily. Oxytetracycline has been used for the treatment of flexural deformities of the distal interphalangeal joints of young foals. It is postulated that oxytetracycline chelates calcium, rendering it unavailable for use for striated muscle contraction. It is considered effective for obtaining a short-term moderate decrease in metacarpophalangeal joint angle in newborn foals. Hemicircumferential periosteal transection and elevation has gained wide acceptance for correction of angular limb deformities in young foals.