LCP disease is an osteonecrosis of the femoral head of young, small breed dogs, usually those weighing less than 12 kg. There seems to be no sex predilection in the dog as contrasted to humans, in whom an 80% male incidence of the disease is evident. As in children, however, the condition is usually unilateral, with only about 10% to 15% incidence of bilateral disease. The age of onset varies between 4 months and 12 months, with a peak incidence at about 7 months.

Etiology

The etiology of the condition is unknown. The pathologic features are typical of avascular necrosis of bone. Experimental studies producing avascular necrosis have been performed, but these studies do not duplicate the necrosis that is seen clinically. Legg, in his original thesis, thought that the vascular problem was due to trauma, but Trueta, in describing his "epiphyseal anemia" emphasizes the precarious blood supply of the femoral epiphysis of children and therefore reinforcing of the blood supply as the child grows older. (11) The blood supply to the femoral head of the immature dog seems more complete, as suggested by Basset and co-workers. (1) This multiple origin of the vascular network of the femoral head in the dog allows room for additional hypotheses (Fig. 82-1). Ljunggren (1967) suggested a possible endocrine etiology and showed the osteonecrosis that occurs with a high dosage of steroids (estrogens and/or testosterone). (9) Her hypothesis was predicated on the idea that the morphologic picture of LCP disease in the dog is a manifestation of precocious sexual maturity. Although the experimental evidence in this study is supportive of this idea, reasons are advanced to explain the unilateral nature of the condition or the low incidence in a breed population that is characterized by precocious sexual maturity. The pathology of avascular necrosis followed by revascularization and bony remodeling of the femoral head in the dog certainly suggests a vascular etiology even though the cause of the condition is not completely understood. (4)
Clinical Signs

The animal is usually presented with a limp. Physical examination shows some pain on extension of the hip joint, particularly with internal rotation. The dog will also evidence pain on forced abduction of the hip joint. Advanced signs include muscle contracture and/or apparent shortening of the leg on the affected side associated with collapse of the femoral head.

The first radiographic signs are those of increased joint space. Radiographic signs in the femoral head itself are evident only after vascular resorption of the necrotic bone has begun (Fig. 82-2). It is at this time that danger from collapse of the femoral head begins. If left to follow its normal course, collapse of the femoral head with distortion of the coxofemoral joint will occur, followed by fragmentation of the femoral head (Fig. 82-3). These changes in structure result in significant loss of function with secondary muscle atrophy. (2,5)

Conservative and Surgical Treatment

Conservative therapy has in the past consisted of a general resting of the limb without casts or bandages. Surgical treatment by removal of the femoral head and neck has been advocated, and reported results indicate that surgery is the preferred treatment. (6,8,9)

The only criterion on which to base the choice of treatment modality is the radiographic picture of the affected limb. Clinical signs are considered secondarily.

If the femoral head is round, the joint space parallel, and the femoral head and acetabulum congruent, strict immobilization of the patient in a small space (cage) will usually result in a resolution of the radiographic and clinical problem. During the enforced rest, the animal is allowed out of the cage only to maintain its toilet training. The animal is carried to and from the cage and kept on a leash during evacuation (Fig. 82-4).

Monthly radiographs are taken to follow the progression of the disease. Immobilization of the animal is continued until there is complete resolution of the radiolucent areas. If collapse of the femoral head occurs during the confinement, surgical treatment is performed.

Strict adherence to this form of treatment yields a dog with nearly normal radiographic appearance of its femoral head and completion of pain-free motion and a normal gait. It takes 4 to 6 months before the femoral head heals sufficiently to permit unrestricted weight bearing. Any compromise to complete immobilization will result in the collapse of the femoral head and a poor outcome.

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An animal that is presented with collapse of the femoral head within incongruence of the coxofemoral joint and an uneven joint space with pain is a candidate for femoral head and neck resection (see Fig. 82-3). If left untreated, the animal will undergo extensive muscle atrophy with concomitant disability before there is some partial improvement following revascularization of the collapsed head. This animal will develop osteoarthritis and may become severely crippled.

Following surgery, exercise will help return the animal to reasonable function. Gradual improvement following resection of the femoral head and neck can continue for up to one year. The primary function of the surgery is the relief of pain, but the small size of these patients usually ensures adequate function.

References


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