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Avascular Necrosis of Femoral Head in Dogs

Karishma K.
BVSc&AH graduate student
College of Veterinary and Animal Sciences, Mannuthy, Thrissur, Kerala, India
Kerala Veterinary and Animal Sciences University
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Introduction

Avascular necrosis (AVN) of the femoral head, also known as Legg-Calvé-Perthes disease (LCPD), is a debilitating orthopedic condition primarily affecting young, small to toy breed dogs. This disease is characterised by the death of bone tissue in the femoral head due to an interruption of its blood supply. The lack of blood flow leads to the collapse of the femoral head and subsequent degenerative changes in the coxofemoral joint, resulting in pain and lameness.

The Anatomy of a Compromised Joint

The femoral head is the spherical, proximal end of the femur (thigh bone) that articulates with the acetabulum, the socket of the pelvis, to form the ball-and-socket coxofemoral joint. This joint is crucial for hip movement and weight-bearing.

The femoral head's primary blood supply is delicate and comes from the medial circumflex femoral artery, which sends branches along the femoral neck. This region is particularly susceptible to poor circulation because it's served by end arteries, meaning there are few collateral vessels to provide backup blood flow if the main artery is compromised. This poor collateral circulation makes the femoral head highly vulnerable to ischemic events, which can lead to avascular necrosis.

In dogs with LCPD, the microscopic architecture of the bone begins to break down. Necrosis of the trabecular bone (the spongy, inner part of the bone) occurs in the subchondral area (the bone directly beneath the articular cartilage). This leads to a weakening of the bone structure. The articular cartilage, which provides a smooth, low-friction surface for joint movement, can also thicken. Over time, the structural integrity of the femoral head is lost, leading to fragmentation and collapse.

Etiology and Predisposition

The precise cause of AVN is often debated and can be multifactorial. LCPD in small breeds is believed to have a **hereditary component**, with an autosomal recessive inheritance pattern. This genetic predisposition, combined with a unique anatomical vascular fragility, sets the stage for the disease to develop before the **femoral growth plate** fully closes.

While LCPD is typically a developmental disease, avascular necrosis in larger breeds or older dogs may be a secondary condition resulting from:

- **Trauma:** Proximal femoral fractures, especially those involving the femoral neck, and hip dislocations can sever the critical blood supply.
- **Iatrogenic Injury:** Damage to the medial circumflex femoral artery during surgical procedures can also cause necrosis.

- **Infections:** Although rare, certain bacteria like *Brucella*, *Staphylococcus* and *E. coli* can trigger inflammatory responses or abscess formation that compress and damage blood vessels.
- **Hormonal Imbalances:** Long-term or high-dose **glucocorticoid** use can inhibit new blood vessel formation (**angiogenesis**), leading to vascular damage.
- **Metabolic Disorders:** Conditions like hyperlipidemia or abnormalities in bone marrow stem cells may contribute to the disease's development.

Clinical signs

The animal shows lameness which is progressive and can be subtle, intermittent or non weight-bearing. Pain will be exhibited on extension and abduction of hip joint. In chronic cases muscle atrophy on the affected side can be observed. The range of movements in the hip joint will be reduced. The affected limb may appear shorter than the other in some cases. Crepitation can be felt upon the hip joint on palpation. Sometimes both limbs may be affected but signs will be shown only on one limb. As the disease advances it can even lead to degenerative joint disease.

Grading of avascular necrosis of femoral head in dogs

Grade 1: Acetabulum and contour of the femoral head and of the femoral neck are apparently normal, and there is a wide joint space, decreased density of thefemoral head and of the femoral neck.

Grade 2: There is femoral head flattening and the presence of multiple density, the acetabular rim may contain a small spur.

Grade 3: There is irregularity in the articular surface of the femoral head and the presence of multiple low-density foci, the spur is more prominent in the acetabular rim.

Grade 4: There is loss of the normal shape of the femoral head, increased prevalence of areas with decreased density.

Grade 5: There is fragmentation of the femoral head with discontinuity of the articular surface, and the acetabular changes are more pronounced than those observed in Grade 4.

Treatment

Femoral head ostectomy (FHO) is a surgical intervention designed to alleviate pain and restore mobility in a compromised hip joint. The procedure involves surgically excising the femoral head and femoral neck. A healthy hip joint is a ball-and-socket joint, with the femoral head fitting snugly into the pelvic acetabulum. By removing the damaged femoral head, the FHO procedure effectively eliminates painful bone-on-bone contact. Initially, the surrounding leg muscles hold the femur in place. As the animal recovers, fibrous scar tissue develops between the femur and the acetabulum, creating a cushioned connection known as a "false joint" or pseudoarthrosis. This allows the patient to regain a functional, pain-free range of motion.

Conclusion

In conclusion, avascular necrosis of the femoral head, particularly as it manifests as Legg-Calvé-Perthes disease, is a significant orthopedic challenge in small-breed dogs. The unique anatomy of the coxofemoral joint and its delicate blood supply make it highly susceptible to this condition. Early recognition of clinical signs like progressive lameness and hip pain is crucial for a timely diagnosis. While the pathology involves the intricate breakdown of bone and cartilage, the prognosis is excellent with prompt and appropriate surgical intervention. A femoral head ostectomy (FHO) effectively removes the source of pain, allowing for the formation of a functional false joint and a return to a good quality of life for the affected animal. This highlights the importance of understanding both the underlying etiology and

the effective treatment options available to veterinary professionals in managing this debilitating disease.

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