DIAGNOSIS AND TREATMENT OF LEGG CALVÉ PERTHES DISEASE

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Harry Scott, Philip Witte describe the symptoms of this hip condition in small breed dogs, its progression and therapeutic choices, including surgery.

LEGG Calvé Perthes disease (LCP) is a relatively common cause of hindlimb lameness in small breed dogs nearing skeletal maturity. Affected breeds include toy breeds and terriers. The disease is bilateral in up to 16 per cent of cases (Demko and McLaughlin, 2005).

Synonyms for this disease include “avascular necrosis of the femoral head” and “aseptic necrosis of the femoral head”, “Perthes disease” and “Legg Perthes” disease, “osteocho-n-dritis”, “coxae juvenilis”, “coxa plana” and “idiopathic osteosis”. The terms “avascular/aseptic necrosis of the femoral head” are also used to describe a process of femoral head necro-sis in dogs following traumatic fractures of the femoral neck.

Aetiology and disease progression

In 1910 Arthur Legg, working in the US, Jacques Calvé (in France) and Georg Perthes (in Germany) independently described atrophy in the hip of skeletally immature children, resulting in characteristic deformation of the proximal femur. The underlying mechanisms they suggested for the signs they observed included traumatic disruption of the blood supply to the femoral capital epiphysis (Legg), rickets (Calvé) and infectious osteomyelitis (Perthes).

The aetiology of the condition in humans and dogs is now thought to be associated with disruption...
of the blood supply to the femoral capital epiphysis, as surmised by Legg. The blood supply to the epiphysis and metaphysis are wholly independent in the immature animal (Figure 1). After closure of the femoral capital physis, metaphyseal vessels cross the physis and anastomose with the epiphyseal vessels. Before physeal closure, the epiphyseal blood supply is almost exclusively dependent on vessels in the synovial membrane, with a minor and variable contribution from the arteries in the round (teres) ligament.

In-vitro experiments have shown that a relatively mild increase in intra-articular pressure results in occlusion of the blood supply via the joint capsule (Bassett et al, 1969). It is assumed by some authors that an elevation in intra-articular pressure in the juvenile dog, in the absence of a significant alternative vascular supply, initiates the process of avascular necrosis of the femoral head.

Whether LCP relates to fluctuating intra-articular pressure or not, the disease process results in necrosis of the femoral capital epiphysis. Trabecular necrosis results in resorption and space formation.

The overlying articular cartilage, deriving its nutrition and oxygen requirements from the synovial fluid, remains unaffected and, initially retains its shape as the subchondral bone is lost, resulting in clefts or cavities deep to the cartilage. The remaining necrotic trabeculae are unable to support the articular surface under the normal forces transmitted through the joint and, eventually, the cartilage collapses.

In the long term, once the physis closes, metaphyseal blood vessels may restore the blood supply, allowing the bone to heal, albeit in an abnormal shape. Osteoarthritis will inevitably progress with accompanying hip pain and lameness.

**Diagnosis**

Diagnosis is by history, physical examination and radiography.

LCP should be considered for all young (six to 15-month-old) toy and terrier breed dogs presenting with hindlimb lameness. On the basis of hindlimb lameness alone, LCP must be differentiated from traumatic femoral capital physeal fractures, small epiphyseal fractures (including avulsion fractures of the round ligament), femoral neck fractures, hip luxation and patellar luxation (which causes hindlimb lameness in dogs with a similar signalment). Hip dysplasia is rare in small breed dogs.

A history of a traumatic event increases the suspicion for a fracture or luxation, although trauma can exacerbate mild, unnoticed lameness, resulting in presentation to the veterinary surgeon, and chronic conditions should not be completely excluded on the basis of an apparently acute onset.

Physical examination will typically reveal marked muscle atrophy on the affected hindlimb,
particularly evident in the gluteal and hamstring muscle groups. Patellar luxation should be excluded by manual attempts to luxate the patella medially and laterally – but bear in mind that patellar luxation may be an incidental finding. Resentment on manipulation of the hip is often severe, particularly in extension.

Radiography reveals an irregular widened joint space, a widened femoral neck and a patchy femoral head with areas of increased and decreased opacity (Figure 2). The radiographic appearance of irregular patchy radiodensity throughout the femoral head in young small breed dogs is pathognomonic for LCP, and relates to the regions of dense new trabeculae and cavities.

More chronic cases may show periarticular new bone formation consistent with osteoarthritis. Occasionally, in subtle cases (Figure 3), computed tomography may be required to confirm the presence of clefts in the subchondral bone (Figure 4).

Some attempts have previously been made in the literature to categorise the changes apparent radiographically and to relate these to the duration of the condition (Lee, 1970). However, in the authors’ experience, the condition progresses at different rates and to different extents from one individual to the next, and attempts to differentiate between the severity of femoral head radiolucency, femoral neck widening, increased coxofemoral joint space and deformity of the femoral head are largely irrelevant in terms of management.

**Treatment**

Options for treatment of LCP include conservative management and surgical interventions. The surgical alternatives are essentially salvage procedures of the hip joint – femoral head and neck ostectomy and total hip replacement – although innominate osteotomy has been described in humans and is (infrequently) mentioned in the older canine literature.

**Conservative management**

During the 1960s a series of papers were published suggesting up to a 25 per cent success rate with conservative management of this condition (Ljunggren, 1966; Ljunggren, 1967; Lee and Fry, 1969). However, objective measurements of long-term function were not made.

Conservative therapy may be attempted in dogs where radiographically apparent changes and lameness are very mild. However, the articular surface of the femoral head will be permanently deformed, resulting in ongoing joint discomfort and the onset and progression of osteoarthritis.

**Femoral head and neck ostectomy**

Lee and Fry (1969) suggested femoral head and neck ostectomy (FHNO) as the optimal treatment
for LCP (Figure 5).

This is a relatively inexpensive procedure with minimal surgical equipment required. A craniolateral approach is made to the hip joint. In small breed dogs most usually affected by LCP, tenotomy or greater trochanteric osteotomy to retract the gluteal muscles are unnecessary, and, indeed contraindicated, and an assistant with a Langenbeck retractor, or the judicious use of Gelpi retractors in the absence of an assistant, can provide sufficient retraction of the gluteal muscles to access and incise the underlying joint capsule.

With the joint exposed via a craniolateral arthrotomy, a small hat spoon or disarticulator is used to cut the round ligament and to elevate the femoral head out of the acetabulum. An assistant is also useful to externally rotate the limb through 90 degrees so the surgeon is presented with the cranial aspect of the proximal femur. A bone spur may be inadvertently made on the caudal aspect of the bone if the ostectomy is made perpendicular to the femoral neck, which may be avoided by making the ostectomy parallel to the base of the femoral head (Figure 6a).

Use of a Gigli wire saw to make the cut is less than satisfactory – potentially traumatising soft tissues around the femoral neck (the surgeon should be cognisant of the location of the sciatic nerve) and often slipping during the process, resulting in a more proximal ostectomy than optimal.

An osteotome or, preferably, oscillating saw allows the surgeon to make an accurate ostectomy in the sagittal plane. The ostectomy should extend from the trochanteric fossa to the level of (and may or may not include) the lesser trochanter (Figure 6).

Inadequate removal of bone can result in continued discomfort (Off and Matis, 2010) presumably as a result of continuing bone to bone contact. The use of muscle flaps interposed between the proximal femur and acetabulum has been described, although is largely discredited.

Management following FHNO is tailored to maximise the flexibility in the developing false joint, or pseudarthrosis. The pseudarthrosis consists of remnants of the joint capsule and surrounding musculature, and is highly fibrous. Encouraging movement as the pseudarthrosis develops will result in greater long-term hip range of motion. Therefore, at the authors’ clinic, a period of three to five days of rest is recommended to allow healing of the skin incision, after which controlled activity is encouraged.

Small breed dogs can cope remarkably well on three legs, and, particularly when moving faster than a slow walk, they will choose to raise a limb following surgery rather than use it. Slow controlled lead walking is recommended to encourage limb placement and frequent physiotherapy should be performed, including a passive range of motion and sit-to-stand exercises and hydrotherapy in an underwater treadmill to encourage hip extension in particular.

Removal of the abnormal proximal femoral tissue significantly improves comfort. However, in one
study, 75 per cent of dogs and cats with FHNO had muscle atrophy suggesting abnormal limb use, 56 per cent had lameness and 32 per cent had signs of pain on manipulation of the hip joint (Off and Matis, 2010). FHNO is acceptable for improving signs of discomfort, but total hip replacement (THR) is the gold standard for restoration of function.

**Total hip replacement**

THR involves replacing the diseased femoral head, femoral neck and acetabulum with cobalt chrome or titanium alloy femoral and acetabular prostheses with an ultra-high molecular weight polyethylene lining in the acetabular component to minimise friction. Cemented and cementless components exist and are routinely used in the UK. For the small breed dogs likely to present with LCP, prostheses are more likely to be cemented (Jankovits et al, 2011). THR has been performed successfully in small breed dogs (Matis and Holz, 2008; Liska, 2010). The veterinary profession has more than 30 years of experience with THR and recorded outcomes are good in small breed dogs.

In the human literature, the expected long-term survival of implants is thought to be around 10 to 15 years, and has been reported up to 20 years (Chougle et al, 2005). Humans bear 50 per cent of their weight on each leg while ambulatory while dogs only bear 20 per cent of their bodyweight on each hindlimb.

It seems reasonable, therefore, to predict similar or better long-term survival of THR in dogs. This is important in cases of LCP, as patients typically present as young dogs.

We are confident performing THR in young dogs, provided that the greater trochanteric physis is radiographically closed, minimising the risk of intraoperative greater trochanteric avulsion. Of the THRs performed at the authors’ clinic during the previous 24 months, 19 per cent have addressed lameness secondary to LCP (Figures 7 and 8). Excellent outcomes have been achieved in more than 90 per cent of cases, with a mean follow-up time of 15 months (range three to 24 months).

**Conclusion**

LCP is an important differential for hindlimb lameness in young small breed dogs. Definitive diagnosis is by radiography. Conservative therapy is unlikely to restore pain-free limb function in most cases. Salvage procedures of the hip include FHNO and THR, the latter of which restores more normal joint function.

**References**

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