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Introduction to Orthopaedics Mini Series

Session Three: Introduction to Joint Surgery - Getting Comfortable with Common Joint Diseases

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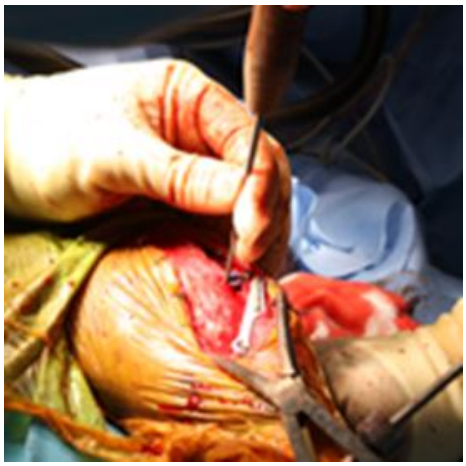

Session 3: Introduction to Joint Surgery – Getting Comfortable with Common Joint Diseases

Joints are designed to afford the greatest stability to the body during weight bearing and motion. A full range of motion that is painless is required for normal function. However any interruption of normal joint mechanics leads to painful osteoarthritis which results in physical incapacity, a reduced quality of life and cost to the owner.

There are different types of joints- synarthroses (fibrous joints) with little motion, amphiarthroses (cartilaginous joints) with limited motion and diarthroses (synovial joints) with the greatest motion. Most joint diseases involve diarthrodial joints.

As with all diseases there are many different underlying causes of joint pathology- degenerative, traumatic, congenital/developmental, infectious, immune mediated, neoplastic, metabolic, anomalous etc...

Osteochondrosis

Osteochondrosis is a developmental condition of the growth cartilages in skeletally immature animals. It is a disorder of Endochondral Ossification, which briefly consists of chondrocyte proliferation, matrix mineralisation, chondrocyte death, vascularization, and ossification. It may be a focal or multifocal condition.
Endochondral ossification takes place at the cartilage of the epiphysis at the end of bones (which account for 20-25% of bone length) and the physis or growth plates (which account for 75-80% of bone length). It has been argued that osteochondrosis occurs at both the epiphysis and physis but other argue the term should apply only for disorders at articular surface.

Osteochondrosis of physis (Proposed/Presumptive)

- Caudal glenoid OCD
- Ununited caudal glenoid
- Fragmented coronoid process
- Ununited anconeal process
- Retained endochondral cores of distal ulna
- Asynchronous growth of radius and ulna
- Slipped capital femoral epiphysis
- Many more

Osteochondrosis of articular cartilage

- Shoulder
    - Humeral head
- Elbow
    - Medial aspect of humeral condyle
- Stifle
    - Lateral or medial femoral condyle
- Hock
    - Medial or lateral trochlear ridge of talus

Pathogenesis

The pathogenesis of osteochondrosis is not clear and there are many proposed theories, which can be split into generalised disease theories or focal disease theories

- Generalised disease

  The first theory suggested that dyschondroplasia, an abnormality of chondrocyte development and maturation, prevented matrix mineralisation and ossification. The osteochondrosis lesions developed secondary to biomechanical stresses. However there is no reported evidence supporting this.

  The second theory suggests that relative osteopaenia of subchondral bone secondary to overnutrition and rapid growth results in the bone being unable to provide mechanical support. Chondrocyte metabolism is consequently disrupted leading to osteochondrosis.

Both of the general theories do not address the fact of site-specific lesions.

- Focal disease

  It has been hypothesised that vascular trauma leads to necrosis of the subchondral bone which can then affect the overlying cartilage. This has been shown to occur in humans however there is no histological evidence in animals.

  The most compelling theory and the one that is most accepted is that there is focal necrosis of the epiphyseal cartilage vascular canals which leads to cartilage ischaemia and necrosis and these areas are susceptible to microtrauma. This may result in a cartilaginous infarct, which prevents endochondral ossification. The resultant thickened cartilage is less resistant to mechanical stress and is metabolically deprived and so degenerates. There are a few possible sequelae, the necrotic focus is organized and heals through granulation tissue which is finally converted to bone by intramembranous ossification, the weakened cartilage may deform leading to altered joint contour and congruency or fissures, clefts or cracks appear. The flap may be dissecated off subchondral bone- Osteochondrosis/Osteochondritis Dissecans lesion. Some may heal or the flap may detach and the defect heals with fibrocartilage.

Diagnosis

With osteochondrosis of the articular surface, clinical signs are often seen when an OCD flap is formed. Lameness is seen in young dogs with joint effusion and joint pain. It is often a bilateral condition and there may be concurrent orthopaedic disease so care is required. The diagnosis is made by plain radiography, where disruption of the subchondral bone is seen by flattening or concavity of the bone contour or mineralised flaps can be seen. Contrast arthrograms can demonstrate unmineralised flaps. CT or MRI can be used but are not usually necessary. Arthroscopy is useful to visualise the cartilage but is not often necessary to make the diagnosis.

Treatment

Conservative treatment can be attempted early in the disease with exercise and NSAIDs. Short periods of rest may improve clinical signs but it won't improve the disease state. Exercise may actually detach or destroy the flap, allowing the defect to heal. In animals with advanced osteoarthritis the results of flap removal is equivocal so these animals should be treated as for osteoarthritis.

Surgical treatment is aimed is aimed at flap excision or retrieval which can be achieved either via arthrotomy or arthroscopy with no evidence one is better than the other. The diseased cartilage and subchondral bone is also removed removed by curettage. The joint is then

extensively lavaged to remove the debris and inflammatory mediators. Flap removal is extremely successful in the shoulder but less so in other joints.

Restorative treatments have been described with osteochondral transplants (OATS system), synthetic implants or autologous chondrocyte implantation. OATS system is designed to replace the defect with donor osteochondral plugs and therefore restore the joint contour with hyaline cartilage. However donor site morbidity is an issue and the cartilage transplants are taken from non-weight bearing areas of joints (normally the knee) so the quality of the hyaline cartilage has been questioned. There is no evidence this is better than traditional surgery. The synthetic implants (carbon, metal, plastic, ceramic etc.) or autologous chondrocyte implantation have not been well evaluated in dogs.

Prevention of osteochondrosis is difficult as it has an uncertain aetiology. There is a genetic contribution so breeding from affected individuals should be advised against. The role of diet with controlled energy intake and relatively low Ca has not been established but pups should not be allowed to become overweight. Exercise was proposed as a risk factor but restriction of exercise is unlikely to prevent osteochondrosis.

Elbow Dysplasia

Elbow dysplasia is a syndrome, which has been defined by the International Elbow Working Group to consist of any or a combination of Fragmented Coronoid Process (Medial coronoid disease), Osteochondrosis or OCD, Ununited Anconeal Process or Joint Incongruency.

Medial compartment disease

Medial compartment disease consists of medial coronoid disease, humeral cartilage disease, either osteochondrosis or kissing lesions and joint incongruency. They are commonly combined and initially affect only the medial compartment hence the term medial compartment disease. Medial coronoid disease is the preferred terminology instead of fragmented medial coronoid as not all affected coronoids fragment. Kissing lesions are the lesions on the humerus as a result of eburnation of cartilage as the humerus rubs on the exposed subchondral bone of the medial coronoid.

Medial compartment disease affects immature large to giant breed dogs typically from 6-18 months old with males being almost twice as likely to be affected as females. It is a multifactorial disease with complex genetics and environmental influences. Medial coronoid disease was originally thought of as an osteochondrosis lesions but it is now thought of as overloading with fatigue microdamage of subchondral bone. Joint incongruence either with an abnormal trochlear notch of the ulna (humeroulnar incongruence) or uneven alignment of the radius and ulna articular surfaces (radioulnar incongruence, humeroulnar conflict), leads to abnormal load distribution across the joint surfaces and pathology of the weight bearing areas.

Diagnosis

Physical examination may reveal an altered stance with the elbow slightly adducted and the antebrachium rotated laterally. There can be varying degrees of lameness with bilateral lameness being easy to miss. Also young dogs often stay very active increasing the chance of a lameness being missed. There can be joint pain with deep palpation of the medial compartment or on full flexion with supination but the absence of pain does not rule it out. The joint can be effusive or thickened and there may be muscle atrophy. In chronic cases there can be a decreased ROM and crepitus.

Radiography,CT, Arthroscopy, MRI and ultrasound can all further the diagnosis. Radiographs should include perfect craniocaudal, fully flexed and 90 degrees flexed views but radiographs can appear normal in the early stages. It is also difficult to see the medial coronoid process. Secondary changes such as osteophytes on anconeal process, radial head and medial epicondyle can be seen as the disease progresses. With osteochondrosis a triangular

subchondral defect can be seen on the medial humeral trochlea with sclerosis of the medial condyle on the craniocaudal radiographic view.

CT is required to assess incongruity and allows direct assessment of medial coronoid process but only the bone not the cartilage. However that is usually all that is required to make the diagnosis. Arthroscopy allows direct visualization of the cartilage and allows you to combine diagnosis with treatment.

Treatment

Conservative

- Exercise (important to mitigate weight gain and to improve muscling and proprioception)
- NSAIDs
- Adjunct analgesia
- Physiotherapy
- Nutraceuticals (low quality evidence)

Surgical

- Reconstructive
- Salvage

At present there is very little evidence to show surgical management is better than conservative.

Surgery can be restorative to try and restore joint congruency or loading

- Arthroscopic debridement
- Subtotal coronoidectomy
- Proximal ulnar osteotomy
- Sliding humeral osteotomy
- Bicipital ulnar release procedure (BURP)
- Canine unicompartmental elbow (CUE)

Or salvage

- Arthrodesis
- Total elbow replacement

Ununited anconeal process

Ununited anconeal process typically affects Large to Giant breed dogs but is has been described in small dogs such as French Bulldogs and Dachshunds. It is bilateral in 25-35% of cases with males being almost twice as likely to be affected as females. It typically presents in dogs aged 5-12months old and can be in combination with medial coronoid disease.

In large breed dogs the anconeal process develops as a separate centre(s) of ossification however the pathogenesis of the lack of fusion is unclear. Proposed theories include genetic, osteochondrosis and incongruity (short radius or abnormal trochlear notch.

Diagnosis

The dogs are lame with joint effusion and pain is often elicited on manipulation especially elbow hyperextension. Radiography reveals lucency between the anconeal process and the proximal ulna, which is most obvious on the flexed mediolateral view. However it is important not to diagnose UAP until 22-24 weeks of age. CT and/or arthroscopy are useful to diagnose concurrent lesions esp. medial coronoid disease.

Treatment

Surgery recommended in all cases.

- Fragment excision
- Surgical reattachment
    - Lag screw or K wires
- Proximal ulnar osteotomy (PUO)
    - Alone or with surgical reattachment

The best results seem to come with a combination of surgical reattachment and proximal ulnar osteotomy and the worst with fragment excision. However it is not clear that the process heals with either surgical reattachment or PUO with one study reporting 22% union but another reporting 71% union.

Other Elbow Conditions

Ununited Medial Humeral Epicondyle

Ununited Medial Humeral Epicondyle is longer considered part of Elbow Dysplasia syndrome and it has recently been proposed it should be called Flexor Enthesopathy. It is unclear if it is significant or not as often there are other elbow pathologies present. It is often considered incidental but others have proposed it as a cause of lameness.

Incomplete ossification of humeral condyle (IOHC)

The medial and lateral humeral condyles form separately but ossification is complete at 8-12 weeks. Incomplete ossification of humeral condyle (IOHC) is where there is failure of the condyles to fuse. However it has also been reported as an incomplete fracture. The significance is that IOHC can lead to lateral or medial unicondylar fractures or Y-T bicondylar fractures which can be devastating on the elbow joint. It typically affects Springer and Cocker Spaniels but it is also seen in Labs, GSDs, Pointers, Rotties and others.

The diagnosis is often difficult by radiography due to the superimposition of the ulna however oblique views may help. CT is often required.

Surgery is recommended especially in dogs that are lame. A transcondylar screw is placed but it is unclear how many of the lesions actually heal. The surgery is associated with a high complication rate (59% for lateromedial screw placement) with infection, seroma, screw loosening and screw breakage reported. It would seem that mediolateral screw placement is associated with a lower infection rate.

Hip Conditions

Coxofemoral luxation

Hip luxations account for up to 90% of the luxations seen in cats and dogs. They are normally traumatic (road traffic accidents or falls) but occasionally the cause is unknown esp. in dysplastic hips. The luxation is described by the direction of the femoral head and most are craniodorsal.

Diagnosis

Initially there is a non-weight bearing lameness but with chronic luxations the animal will begin to use the limb. The animal may be recumbent if there are bilateral luxations or other injuries. The hip is painful on manipulation often with crepitus. With a craniodorsal luxation the limb is externally rotated and adducted and the greater trochanter is palpably more prominent dorsally. With a ventral luxation the limb is internally rotated and abducted and the limb appears longer.

Radiographs are required to confirm the diagnosis, determine the direction of the luxation and evaluate for other abnormalities (fractures, hip dysplasia etc)

Treatment

Closed or open reduction should be attempted as soon as possible to minimise the damage to the articular cartilage. Closed reduction should be attempted under general anaesthesia but it may be impossible in chronic luxations.

For a craniodorsal luxation an assistant uses a rope in the inguinal region to provide countertraction while the limb is externally rotated and distocaudal traction is applied. The limb is then internally rotated and pressure is applied to the greater trochanter to seat the femoral head into the acetabulum.

For a cranioventral luxation it is easiest to convert the luxation to a craniodorsal luxation then carry on as above. If that cannot be achieved then traction is applied to the limb and then the femoral head is moved laterally then caudally into acetabulum.

For a caudoventral luxation traction is applied to disengage the femoral head from obturator foramen and limb abduction may help with this. The femoral head is then manipulated laterally and cranially into acetabulum. Once the femoral head is reduced in the acetabulum the hip is moved through its ROM to assess stability and radiographs are obtained to confirm reduction. This is followed by either strict rest (50% reluxation rate) or by the placing of an Ehmer sling (reluxation rates of 15-71%). Hobbles can be used for ventral luxations however there is an 80% success rate with closed reduction alone of ventral luxations.

Unstable closed reductions or open reductions should be followed by stabilisation, which can be achieved by either capsulorrhaphy, a prosthetic capsule technique, transarticular pinning or toggle rod stabilisation. There is a progression of osteoarthritis in 55-62% of cases following luxation.

Salvage surgery either femoral head and neck ostectomy or total hip replacement is necessary in failed stabilisation or cases with hip dysplasia, already well established osteoarthritis or unrecontructable femoral head and neck fractures.

Legg-Calve-Perthes Disease

Legg-Calve-Perthes Disease, also known as avascular necrosis of femoral head is due to a non-inflammatory local ischaemia, which leads to necrosis of the trabecular bone and collapse of epiphysis. This heals with new bone but the femoral head and neck are malformed leading to pain and dysfunction.

The cause is unknown but there is a hereditary component. It has been shown to be a simple autosomal recessive trait in Miniature Poodles and Westies. Young dogs between 4 and 11months old are affected and males and females are equally affected. In 12-16.5% cases the condition is bilateral.

Radiography reveals focal bony lysis of the femoral head ('moth eaten' or 'apple coring'), flattening and mottling of the femoral head and collapse and thickening of the femoral neck. Conservative treatment of rest and analgesia only results in the resolution of the lameness in less than 25% of cases. Surgical treatment consists of either femoral head and neck ostectomy or total hip replacement.

Hip Dysplasia

Hip dysplasia is a complex disease which has been defined as a varying degree of hip laxity, permitting subluxation during early life, giving rise to varying degrees of shallow acetabulum and flattening of the femoral head, finally inevitably leading to osteoarthritis.

It has a complex inheritance with multiple genes combined with environmental influences (weight, growth and hormones). At birth all canine hip joints are normal but they develop abnormally based on the genetics of the dog and environmental influences.

Previously it was thought of as a biphasic disease- young dog with hip laxity followed by a quiescent period then an old dog with secondary osteoarthritis. However it is a monophasic disease, which progresses throughout life.

The pain in young dogs is due to joint laxity with stretching or tearing of the joint capsule, ligaments, muscles and microfracture of dorsal acetabular rim. The pain in old dogs is due to osteoarthritis.

Hip dysplasia can affect any breed of dog but is most common in large to giant breeds, German Shepherd Dog, Rottweiler, Saint Bernard and Labrador and Golden Retrievers. There is a very low incidence in sight hounds.

Diagnosis

Many dogs may show no clinical signs or very mild signs depending on the severity of the dysplasia. Lameness is variable and bunny hopping especially in immature dogs is common. There can be difficulty rising or a reluctance to walk, run, jump or use stairs.

Hip manipulation can be painful with crepitus or a reduced ROM especially in older dogs with chronic osteoarthritis. Muscle atrophy is common and there may be a positive ortolani sign. Radiographs are required to define the degree of dysplasia and assess secondary osteoarthritis. The hip extended view is the standard view taken (BVA / Kennel Club, OFA and FCI) however the Penn Hip Program with VD extended, compression and distraction views may better assess joint laxity ion the young patient.

Controlling Hip dysplasia

Selective breeding has tried to reduce the incidence of hip dysplasia. Typically this has been based on phenotype testing (BVA/KC) and has resulted in minimal improvement. Estimated breeding values may improve phenotype testing as these take into account the testing for the dog and all its relatives including progeny. The ideal selective breeding programme would be based on genetic testing but this is a long way off

Treatment

Non-surgical

- Weight control (important from a young age)
- Exercise regimes
  - Rest contra-indicated
  - Regular regime essential
- Physiotherapy
- NSAIDs
- Others
  - Nutraceuticals
  - Regenerative therapies- stem cell therapy

Surgical

- Skeletally immature with laxity
  - Juvenile pubic symphysiodesis
  - Triple pelvic osteotomy
  - Double pelvic osteotomy
- Chronic osteoarthritis
  - Femoral head and neck ostectomy
  - Total hip replacement

There is a subset of young dogs that require salvage surgery and dogs with extreme hip dysplasia can benefit from early total hip replacement.

Cranial Cruciate Ligament Disease

Cranial cruciate ligament disease is the most common cause of pelvic limb lameness in the dog. Traumatic rupture of the CCL as seen in humans is a very rare injury in the dog. In dogs the ligament progressively degenerates but the cause and pathogenesis of this remains elusive. There is loss and metaplasia of the ligamentocytes and failure to maintain collagen fibres, which predisposes the ligament to failure. Multiple factors have been implicated.

- Abnormal conformation and gait
- Increased tibial plateau angle
- Obesity
- Lack of fitness
- Breed variation
- Neutering (more prevalent in neutered animals)
- Sex (females more than males)

Diagnosis

The history of CCL disease is highly variable; the lameness can be severe and non-weight bearing in a complete rupture or be minimal and only noted after strenuous exercise with a stable partial tear. The lameness can be worse after rest or after exercise. There may be stiffness after rest.

Clinical signs

- Stifle pain
    - Flexion/extension
    - Hyperextension with partial tears
- Crepitus
- Quadriceps muscle atrophy
- Periarticular fibrosis
    - Medial buttress
- Loss of patella ligament distinct band
- Sit with affected leg projecting out

CCL disease can be bilateral in up to 40-50% of cases. They don't always present as bilateral cases but it is important to examine both stifles. Mild bilateral lameness and subtle bilateral clinical findings can be misinterpreted as normal.

Severe bilateral cases can present non-ambulatory and be mistaken for a spinal case.

Diagnostic tests

- Palpation of patella ligament
- Pain in hyperextension
- Cranial Drawer Test
- Tibial Compression Test

The cranial drawer and tibial compression test could be normal in conscious painful animal so it is important to repeat under sedation or anaesthesia.

Radiographs are necessary in all cases to assess the osteoarthritis, confirm stifle pathology in challenging cases and to rule out fractures and neoplasia. Secondary radiographic signs are

- Effacement of infrapatellar fat pad
- Caudal distension of synovium
- Osteo and enthesiophyte formation
    - Tibial condyle
    - Femoral trochlear ridge
    - Patella
- Subchondral sclerosis

Stifle arthroscopy, CT arthrograms, MRI or Ultrasound can be used to further document the injuries but are unnecessary in most cases.

Treatment

- Conservative
- Traditional surgery
    - Replace or mimic CCL
    - Eliminate cranial drawer
- Osteotomy procedures
    - Biomechanical modifications
    - Aim is to dynamically stabilise joint
    - Do not eliminate cranial drawer

Conservative treatment consists of a controlled exercise regime with rest being contra-indicated. The degeneration of the ligament continues regardless of exercise. Exercise improves muscle bulk and proprioception. Periarticular fibrosis 'stabilises' the joint in the long term. NSAIDS should be used for extended periods to allow the dog to exercise and physiotherapy is very beneficial.

Surgery

Lots exist

Conventional surgery

- Intra-articular "prostheses"
    - Patsaama type techniques
- Over the Top (OTT)
    - Lots of variants
- Extra-capsular "stabilisation"
    - Lots of variants

Osteotomy Procedures

- Cranial Closing Wedge Ostectomy
- Tibial Plateau Levelling Osteotomy
- Tibial Tuberosity Advancement
- Triple Tibial Osteotomy

There is little evidence that one procedure is better than the others at present but a recent study in the USA showed that the majority of vets wanted an osteotomy for their own dog. The expectation is that dogs will do better with surgery than conservative treatment and the results of the osteotomy procedures are better than the conventional techniques.

Meniscal tears are a common concurrent injury with CCL disease (33-77% incidence). There can be post operative tears (latent, missed at first surgery and postliminary, tear after surgery) with an incidence 3 and 17% depending on the CCL surgery. The menisci have a role in the stability of the stifle and in the transfer of load. The medial mensicus is more firmly attached hence why they are injured more frequently with the shear forces created with cranial tibial subluxation following CCL disease.

The diagnosis is made by visualization of the injury. On physical examination there may be pain in flexion or a meniscal click. The presence of a click is a strong indicator of a meniscal

tear however the absence of one is not a strong indicator of a normal meniscus. Ultrasound, MRI or CT arthrograms can be used to diagnose meniscal injury.

Final diagnosis is made at surgery either by arthroscopy or arthrotomy. Arthroscopy is reported to pick up almost double the numbers of tears by arthrotomy. Either way it is important to probe the meniscus for tears.

Surgical debridement either a partial menisectomy or hemimenisectomy is performed but it is important to preserve as much functional meniscus as possible. Meniscal repair or grafting has been reported but needs further evaluation in the dog.

Patellar luxation

Patellar luxation is common and can be medial (typically in smaller dogs)
or lateral (typically in larger dogs). Patellar luxation results from mal-alignment of extensor mechanism, which causes abnormal “tracking” of patella leading to luxation and subsequent erosion of the trochlear ridge, which leads to easier luxation.
Patellar luxation results in variable lameness, secondary osteoarthritis and secondary joint damage notably erosion of the trochlear ridge.

Diagnosis

Clinical signs

- Palpable instability
- Gross alignment
- Crepitus, joint pain, OA
- Exclude/evaluate other abnormalities

Although patellar luxation is a congenital condition previously asymptomatic dogs may present in later life due to a progression of osteoarthritis or concurrent cranial cruciate ligament disease.

Radiography is used to evaluate alignment assessing for femoral and or tibial deformities. CT evaluation can be useful in combined/complex deformities.

The luxation can be graded according to the following scheme.

1 - Manually luxates; returns to normal

2 - Luxates spontaneously during motion: returns to normal with stifle extended

3 - Luxated most of time but can be reduced

4 – Permanent, irreducible luxation

However this classification system does not give useful information in grading the severity of associated clinical signs or prognosis.

Treatment

The first aim of treatment is to restore functional alignment. Adjunct procedures help enhance patellar stability and improve patellar tracking. Dogs that are asymptomatic do not require surgery.

Surgical treatment options

- "Re-alignments"

    Tibial tuberosity transposition
    Distal femoral corrective osteotomies
    Proximal tibial osteotomies

- Adjunct procedures

    Sulcoplasties
    Erosion
    Recession
    "V"; Wedge; others
    Joint imbrication
    Are these effective/useful??

The choice of surgery is dictated by the specifics of each case but the least "aggressive" surgery likely to restore functional alignment should be selected with additional procedures as required to impart patellar stability/tracking.