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Introduction to Orthopaedics Mini Series

Session One: Lameness Assessment and Non-Surgical Orthopaedics

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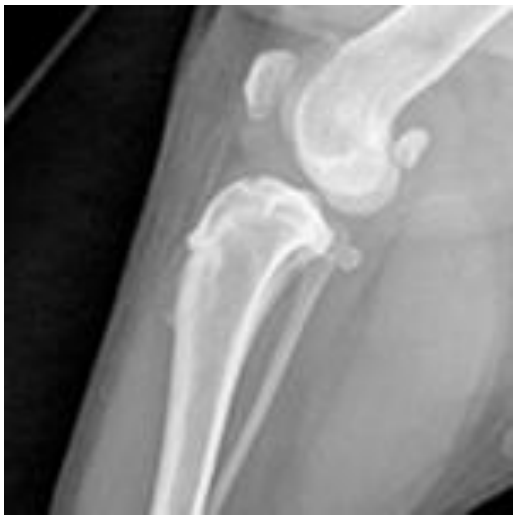

Orthopaedic Examination

History

Any orthopaedic examination must begin with adequate history taking and a full general examination. A systematic approach is essential so multiple problems are not missed. This ensures that the animal's general health is ascertained before focusing on the orthopaedic problem.

- Breed, age, gender
- Occurrence of trauma
- Owner identification of limb(s) involved
- Chronological progression
- Efficacy of treatments
- Variability – weather, exercise, after rest
- Other features- fever, inappetance, lethargy, weight loss

Any deviation from the 'normal' is important to note at this stage. For example an old dog sustaining a fracture with minimal trauma minimal trauma should set alarm bells ringing for a pathological fracture or an old osteoarthritic dog in severe pain should set alarm bells ringing for a tumour as chronic osteoarthritis is unlikely to cause severe pain.

Patient Observation

The next stage of the examination is observation of the patient noting the following (this can be done before the general examination).

- Weight status
- Body Conformation
- Decreased weight bearing
- Trembling
- Swellings
- Muscle atrophy
- Digit and joint alignment

Gait Observation

Gait observation is next and should be before the limbs are examined. A chronic low-grade lameness will often disappear in the consulting room. The animal should be observed at walk and trot. A covert lameness can be unmasked with exercise, tight circles and stair climbing. Cats' gaits can be challenging to observe and it is often best to encourage them to run to their basket, jump up and down to assess their movement.

Lameness typically presents with many different gait alterations

- Head bobbing (Thoracic limb lameness)
    - Head elevates as the painful leg strikes the ground
- Hip/pelvic elevation/drop (Pelvic limb lameness)
    - Increased hip/pelvis elevation/drop compared to non painful limb
- Shortened stride
- Limb circumduction
- Abnormal sounds - Clicks, snaps

A bilateral lameness can be challenging to spot for the inexperienced eye. Weight can be shifted away from the painful limbs to the thoracic or pelvic limbs, the animal may just look stiff, may bunny hop or even hand stand.

Neurological disease must be assessed for now and ataxia and paresis can be mistaken for lameness and vice versa. Stumbling, falling over, dragging of toe nails, crisscrossing of legs and hypermetria are all signs of a neurological component to the gait abnormality. However be aware that the animal could have neurological disease and orthopaedic disease concurrently.

Patient Palpation

The animal should be palpated whilst standing and in recumbency. For standing palpation the animal should be stood as symmetrical as possible and you should palpate the contralateral limb at the same time noting

- Asymmetry- swelling, heat, malalignment, crepitus
- Muscle atrophy
- Bilateral conditions (experience or radiography to distinguish)

It is essential to examine as many normal dogs and cats as possible so subtle changes can be picked up.

A basic neurological examination can be completed at this stage to help differentiate neurological disease. Conscious proprioceptive positioning, i.e. paw position or knuckling response and hopping reaction are two simple quick tests to assess for subtle neurological deficits.

Recumbent Examination

Ideally the recumbent examination should be done in the lateral recumbency. Many tests can be done in the standing animal but it is easier to restrain and manipulate them when they are lying down. Always do the normal side first.

Most of the manipulations described do not cause pain in the normal animal however if no pain response is noted this does not mean it is normal. Any manipulation that causes pain should be carefully and gently repeated to reduce risk of misinterpretation. It is also essential to immobilise the surrounding tissues and joints as much as possible so that the manipulation you are carrying out truly tests that area and not the adjacent areas (this can be difficult). You should palpate all bones and joints starting with the digits and working proximally. Assess for pain, instability, crepitus and altered ROM

Paw, carpus, hock

- Pads and interdigital webbing examined
- Maximally flex and extend digits, carpus and tarsus
- Varus and valgus stress applied to carpus and hock
- Achilles tendon palpated during flexion and extension
- Palpate tendon up to musculotendinous junction

Elbow

- Huge ROM in normal elbow (approx 130°)
- Maximally flex and extend
- Rotational stability
    - Apply internal and external rotation to elbow
    - In full extension the anconeus is the primary stabiliser of pronation.
    - The lateral collateral ligament is the primary stabiliser of supination
    - In 90° flexion the medial collateral ligaments are responsible for rotational stability

Shoulder

- Flex and extend shoulder
- Grasp antebrachium and stabilise cranial shoulder with other hand. Ensures manipulate shoulder and not just move the scapula
- Biceps brachii tendon palpated proximal medial humerus as limb pulled caudally along body wall
- Shoulder abduction angles
    - Needs sedation at least
    - Compare to contralateral limb
- Don't forget axillary palpation

Stifle

- Flex, extend

Patellar luxation

- Some animals have normal mediolateral movement of patella within trochlea
- Subluxation where patella rides onto trochlear ridge
    - May cause lameness
- Luxation out of trochlea is abnormal
    - Medial or lateral
    - Need to differentiate between luxation out of trochlea and reducing a permanently luxated patella

Medial patellar luxation

- May be permanently luxated medially- assessed by palpation
- To luxate a patella medially
    - Stifle is extended, pes internally rotated, digital pressure applied to patella in a medial direction
    - May luxate with just internal rotation

Lateral patellar luxation

- May be permanently luxated laterally- assessed by palpation
- To luxate a patella laterally
    - Flex stifle slightly, pes externally rotated, pressure applied in a lateral direction

Always assess for cruciate ligament instability with patella reduced

Cruciate ligament instability

- Testing can be painful
- May require sedation
- Drawer movement is craniocaudal sliding of the tibia in relation to the femur
- Normally no drawer movement
    - Puppy laxity, drawer movement but definite end point to test
- Diminished or no drawer movement does not rule out cruciate ligament disease
- Diminished with chronicity, animal tenseness, partial tears, meniscal injury

- Indirect Drawer movement, Tibial Compression Test
    - Compresses femur and tibia together
    - Cranial cruciate ligament incompetence tibia slides forward
    - To perform the stifle is slightly flexed, one hand flexes and extends the hock. The index finger of other hand lies cranial to femur, patellar ligament and tibial tuberosity. It detects tuberosity sliding cranially
    - Repeated several times

- Direct Drawer test (Cranial and caudal)
    - Index finger of one hand on patella with thumb on lateral fabella. Index finger of opposite hand on cranial aspect of tibial tuberosity with thumb on fibular head
    - Place fingers as close to bone as possible not on soft tissues
    - Keep wrists straight and femur held stable
    - Tibia is pushed forwards and then backwards
    - Do not rotate the tibia
    - Perform in extension and flexion
    - Repeat several times but quickly

Interpretation of direct drawer test can be difficult

- Normal ligaments
    - Sudden thud at end of cranial or caudal movement
    - Definite end point
- Diseased or ruptured ligaments
    - End point is soft
    - No sudden stoppage
- Mistakes of drawer testing
    - Wrists not straight so inadequate force applied
    - Fingertips alone so proper force not applied
    - Finger medial/laterally placed skin moves and misinterpreted as drawer movement
    - Performed slowly so detection of small 1 to 2mm of motion is impossible
    - Rotation of tibia misinterpreted as drawer motion

Meniscal Injury

- Physical examination does not prove or disprove meniscal injury
- Suspected if hear click, snap clunk or grating when flex, extend or perform drawer tests

Collateral ligament instability

- Invariably associated with cruciate ligament(s) rupture
- Apply valgus and varus stress to stifle and palpate for joint opening abnormally medially or laterally respectively

Hip

- Flex and extend hip, abduct hip.
- Diagnose hip luxation by assessing greater trochanter position relative to ileal wing and tuber ischia
- Hip laxity can be assessed by Ortolani sign
    - Noise or thud as unstable femoral head is replaced into the acetabulum
    - Muscle tension often masks hip laxity
    - Assess again under sedation
    - Hip is subluxated proximally by grasping adducted stifle and pushing proximally while other hand stabilises the pelvis
    - Stifle is then abducted and downward pressure is applied across the greater trochanter. Thud is felt and or heard.

Pelvis

- Gentle manipulation of the ileal wing to assess for sacroiliac instability
- Palpate pelvis for asymmetry, crepitus and pain
    - Rectal palpation may help with ischial and pubic fractures
- Lumbosacral pain
    - Palpation of LS region
    - Rectal palpation
    - Tail lift
    - Lordosis test

Rest of limb palpation

- Long bones and muscles often forgotten about
- All areas of the limb are gently squeezed
    - Bone and muscles
- Do last as pain from neoplasia or panosteitis can be exquisite
- Find muscle planes where can fingers can reach bone
    - Distal and proximal humerus, proximal ulna, distal radius
- Deep palpation of axilla
    - Brachial plexus tumours
    - Severe progressive lameness
    - Mass may be detected or exquisite pain

Further Diagnostic Tools

Radiography, arthrography, synoviocentesis, fluoroscopy, CT, MRI, nuclear imaging, ultrasound, kinetic and kinematic gait analysis, arthroscopy and exploratory surgery can all be utilized to further the diagnosis.

Radiography is the mainstay of further investigations. Properly positioned orthogonal views are essential and sedation or general anaesthesia is required to obtain these. If the view is not near perfect, i.e. rotated, then repeat it. Contrast arthrograms, with positive contrast injected into the joint can be useful especially in the shoulder.

Synoviocentesis is another relatively simple and cheap test to investigate joint disease. This involves the puncture of a joint and aspiration of fluid. This must be performed aseptically with a surgical preparation and wearing sterile gloves. The fluid can be assessed by gross inspection, cytological evaluation (see table below) and bacterial culture. For culture the fluid should be put in blood culture medium to increase the chance of a positive result.

Condition	Total cell count	% of mononuclear cells	% of neutrophils
Normal	< 2 x 10^9/L	94-100	0-6
Osteoarthritis	2-5 x 10^9/L	88-100	0-12
Rheumatoid arthritis	8-38 x 10^9/L	20-80	20-80
Non-erosive IMPA	4-370 x 10^9/L	5-85	15-95
Infective arthritis	40-267 x 10^9/L	1-10	90-100

Miscellaneous Orthopaedic Conditions

Panosteitis

Panosteitis is a self-limiting inflammatory disease of the bone marrow of long bones. It predominantly affects large to giant breed dogs but has been reported in small breeds (Miniature Schnauzer, Scottish Terrier). Typically affects young dogs of between 5 and12 months old but can vary affect dogs from 2mths to 5yrs of age. Males are four times more likely to be affected than females.

It typically causes shifting leg lameness and the lameness can be acute so it can be mistaken for a fracture. The lameness can be mild but it can present as a severe lameness resulting in an inability to walk. There is pain on palpation of the affected bone but care should be taken to avoid compressing muscles and nerves during palpation. Multiple bones can be affected at same time with ulna being the most commonly affected bone followed by the radius, humerus, femur and finally the tibia.

Radiography is required to diagnose panosteitis however in the early stages of the disease these could be normal. The typical radiographic sign is an increase in medullary opacity with a granular pattern or with loss of trabecular pattern. Periosteal new bone formation possible. As the lesion progresses the medullary canal becomes more diffuse and homogenous. Finally the densities regress leaving a coarse trabecular pattern.

Treatment consists of rest and analgesia, typically NSAIDs but other analgesics may be required when the pain is most severe.

Hypertrophic Osteodystrophy (HOD)

Hypertrophic Osteodystrophy is a developmental bone disease is rapidly growing dogs, typically large to giant breed dogs. Males are twice as likely as females to be affected. No single unifying cause has been determined. Vitamin C deficiency and over nutrition were proposed initially but these have largely been discounted by more recent studies. Heritability, infection and vaccination (canine distemper virus vaccines) have been investigated as causal factors.

The diagnosis is based on signalment and clinical signs and confirmed by radiography. HOD can affect any bone and the resulting lameness can vary from mild to severe. There is swelling of the metaphyseal region that is warm and can be painful and the condition is often bilateral. Systemic signs are common and include pyrexia, depression, inappetance and diarhhoea.

The pathognomonic radiographic sign is a lucent line parallel to a narrow zone of increased radiodensity immediately adjacent to physis. There may also be periosteal and endosteal proliferation with enlargement of the metaphysis (metaphyseal flare). In severe cases blood culture is indicated.

Most cases are self limiting (days to months) and mild to moderate cases deserve an excellent prognosis however in sever cases death may occur.

Treatment is ensuring the dog is on a balanced diet and analgesia. Severe cases will likely require supportive care and antibiotics are required if a bacteremia is present. In severe cases owners should be warned of angular limb deformities (carpal and tarsal valgus) developing.

Craniomandibular Osteopathy

Most patients are less than 12 months old with puppies under 6 months old being at the highest risk. Typically this affects Cairns, Westies and Scottish Terriers but actually many other breeds can be affected. It is characterized by unilateral or bilateral osseous proliferations of the mandibles and/or tympanic bullae although the temporal bones can also be affected. Male and female dogs are at equal risk for the disease.

The clinical signs vary from minor difficulty in prehending and chewing to a complete inability to open mouth and inability to eat and drink. This can be accompanied by excessive salivation, intermittent pyrexia, depression, pain while eating and weight loss due to inappetance/anorexia. The mandibular enlargement is almost always easy to palpate.
The diagnosis is based on the signalment and clinical findings but is confirmed by radiography and or CT.

The disease may become self-limiting and variable levels of supportive care are required. Soft food, syringe feeding and occasionally feeding tubes are required to ensure the dog receives adequate nutrition. Analgesics, typically NSAIDs are indicated. Unfortunately some dogs are euthanased due to uncontrollable pain, the lesions not resolving enough to allow the dog to eat or if the owners are unable or cannot afford treatment.

Puppy carpal laxity syndrome

Puppy carpal laxity syndrome can present as carpal hyperextension or carpal hypoextension. It affects young growing puppies, typically between 6 and 16 weeks of age and can be uni- or bilateral.

In mild to moderate cases the prognosis is good to excellent. These puppies should be exercised kept on solid floors with good grip and changed to an adult dog food. Splinting has been described but I have never splinted these and never felt the need to.

In severe cases the same management strategies can be adopted and most cases will resolve. Again splinting is described but I do not especially considering these are fast growing pups. If the deformity becomes irreversible in older puppies, then for hypoextension tenotomy of affected tendon can be considered (typically the flexor carpi ulnaris) or for hyperextension pancarpal arthrodesis can be performed as a salvage. The need for surgery is extremely uncommon if these are diagnosed early and appropriate and prompt conservative treatment instigated.

Muscle and tendon disorders

Muscles

Muscle injuries are common and rarely cause a significant problem. Many will heal before a diagnosis is made or despite a diagnosis not being made. A full recovery is expected however exceptions are injuries to athletic dogs (esp. racing greyhounds- gracilis and long head of Triceps) or where muscle contracture develops.

Muscle contracture is extremely challenging to treat. Contracture is abnormal shortening of the muscle tissue rendering it highly resistant to stretching.

Contracture affects the

- Infraspinatus muscle
  - tendinectomy can result in good to excellent prognosis

- Quadriceps femoris muscle
  - often after femoral fracture in growing animal
  - vigorous physiotherapy required
  - can result in amputation

- Gracilis and/or Semitendinosus
  - conservative management
  - almost universal recurrence after surgery

Tendons

Any tendon can be severed with trauma and tendon injuries are often more debilitating than muscle injuries. Almost all require surgical intervention. The most common tendon injuries are

Severed digital flexor tendons caused by deep lacerations to the palmer or plantar aspect of the paws, these are often missed. All deep wounds in this area require deep surgical exploration. The superficial and deep flexor tendons require separate repair and late presentations are common if the deep flexors were neglected at initial surgery or if the repair breaks down. This results in flattened digits with a sore on metacarpal/tarsal pad.

Injuries to tendon of origin of the biceps brachii. Biceps tendinopathy has been suggested to be most common cause of shoulder lameness. It is thought to be the result of overuse and or a chronic repetitive injury. Diagnosis is made by radiographs, contrast arthrograms and or ultrasound. Medical management is often successful with moderate to full exercise and extended courses of an NSAID. If this fails surgery is indicated with either tenodesis or tenotomy being described.

The biceps brachii tendon can also rupture or be medially displaced.

Injuries to the Common Calcaneal tendon. The common calcaneal tendon (Achilles tendon) compromises three parts- the paired tendons of gastrocnemius, the combined tendon of gracilis, semitendinosus and biceps femoris and the tendon of superficial digital flexor muscle. The tendon can be injured with a complete traumatic rupture or have a chronic Achilles tendinopathy. With Achilles tendinopathy there is an avulsion of the tendon of insertion of gastrocnemius.

This is relatively common in medium to large breed dogs (Typically Dobermans and Labradors)There is typically a history of a chronic lameness without acute trauma. Clinical signs include swelling at the insertion of the tendon at the calcaneus, hyperflexion of the hock and knuckling of the digits ('bird clawed' appearance). Surgical intervention is required when lameness is present and the aim is to temporarily fix the hock in extension with or without surgical re-attachment of the tendon to the calcaneus.

Arthritis

Arthritis is defined as an inflammatory disease process within a synovial joint. It is classically divided into non-inflammatory and inflammatory arthritis.

- Non-inflammatory arthritis
  - Osteoarthritis
  - Traumatic arthritis
- Inflammatory arthritis
  - Immune-mediated
    - Non erosive
    - Erosive
  - Infective

Essentially all types have some degree of inflammation.

Osteoarthritis

Osteoarthritis is most common form of arthritis in dogs and cats. It is estimated that 20% of adult dogs are clinically affected and 60 % of adult cats are reported to have radiographic evidence of osteoarthritis. Osteoarthritis can be idiopathic (primary) or secondary. In reality it is nearly always secondary in dog and in cats often thought of as being secondary but often no initiating cause is identified. Exceptions where it can be primary are osteoarthritis of the small joints in the manus and pes in older dogs and of the elbow joint in older cats. Therefore the diagnosis of osteoarthritis is almost always not sufficient and the underlying joint abnormality should be identified.

Diagnosis

The diagnosis of osteoarthritis and the underlying pathology is based as always on history, clinical signs and diagnostic imaging. The typical history of osteoarthritis is reluctance to exercise, exercise intolerance, inactivity stiffness, lameness, inability to jump and behavioural changes e.g. aggression. In cats these signs can be present but they are often missed by owners so the owner should be questioned on any reduction in activity, jumping, jump height etc. The only signs in some cats are aggression and an unkempt appearance.

The clinical signs are highly variable and one or all of the following are possible- gait alterations, muscle atrophy, joint swelling, periarticular fibrosis, reduced ROM, crepitus and joint pain. Again physical examination may be challenging in cats due to their nature. It is often best to assess the cat's movement by encouraging them to run to their basket, jump up and down from a height etc.

Radiography is the mainstay of imaging in osteoarthritis. The radiographic signs are

- Osteophytosis
- Enthesophytosis
- Effusion
- Soft tissue swelling
- Subchondral sclerosis
- Intra-articular mineralisation
- Subchondral cyst

Advanced imaging such as CT, MRI or arthroscopy is useful to assess the underlying pathology, as often this is not apparent on radiographs.

Synovial fluid analysis is recommended in the work up of joint disease and should be sampled if there is any doubt as to the underlying disease. In osteoarthritis synovial fluid shows evidence of mild inflammatory change with mild to moderate increases in mononuclear cell numbers.

Management of osteoarthritis

The ideal treatment of osteoarthritis would be to eliminate the underlying cause (perfect reduction of intra-articular fracture, early treatment of hip dysplasia/laxity) however this is very seldom possible. Never the less it is essential that you try to identify the underlying condition and if possible treat it.

With established osteoarthritis treatment of the underlying condition may not be possible and often if it possible it will not alter the progression of the secondary osteoarthritis. However the underlying condition should be identified and treated when possible i.e. cranial cruciate ligament.

Conservative or medical management of osteoarthritis relies on a multimodal approach.

- Weight management
- Exercise
- Physiotherapy/Hydrotherapy
- Medical management
- Nutritional Supplementation
- Complementary Therapies

Obesity is a risk factor for osteoarthritis but weight management is also probably the most important strategy for controlling osteoarthritis. Weight reduction can significantly alleviate the symptoms and animals should be kept at the lean end of acceptable (BCS 4/9).

The effects of exercise on osteoarthritis are largely unexplored. In the short term exercise can worsen lameness in dogs with OA. However the effects of exercise in the medium to long term are unknown. Full exercise mitigates the risk of weight gain, muscle atrophy and joint proprioception loss. A regular exercise programme is essential with rest if allowed at all limited to days when the animal is significantly painful.

Physiotherapy and hydrotherapy can be useful in osteoarthritis however it is essential to understand the difference between physiotherapy and hydrotherapy. Hydrotherapy without a physiotherapist has rather limited benefits restricted to weight management and improving fitness. Physiotherapy has more wide ranging benefits in pain management, improved ROM, improved muscle bulk and improved proprioception.

Medical management can be divided into two categories

- Symptom Modifying
    - Analgesics
- Structure modifying
    - Must demonstrate efficacy in retarding or stopping cartilage erosion
    - Many drugs claim to be structure or disease modifying
    - Many have not met the stringent criteria

Analgesics

NSAIDs are the mainstay of therapy. It is rare that they are not effective and they have minimal adverse effects. There is good evidence for the efficacy of carprophen, meloxicam and firocoxib. These should be given for at least 2 weeks before assessing response. In the long-term use intermittent courses (6-10 weeks) to cover acute flare up episodes.

If after at least two weeks there is minimal improvement consider changing to different NSAID. If NSAIDs fail then start again- confirm the diagnosis, assess its progression, assess compliance, and finally consider surgery.

Alternative analgesics include tramadol, pardale-V, corticosteroids, prednoleucotropin, amantadine and others.

Tramadol is a centrally acting synthetic opioid, which is broken down into at least 30 metabolites. Tramadol, O-desmethyltramadol (ODM) and N,O-didesmethyltramadol (DDM) have pharmacologic effects and multiple receptor systems involved in analgesic effect. Dogs produce small quantities of ODM so have minimal opioid effects whereas cats produce large quantities of ODM so have prominent opioid effects (and therefore adverse effects). There is experimental evidence of its efficacy however there is minimal clinical evidence of efficacy when given orally for chronic pain.

Pardale-V is a combination of paracetamol 400mg and codeine phosphate 9mg. It is licensed in dogs only and designated an NFA-VPS so a vet, pharmacist or SQP can supply this. It is only licensed for 5 days and it is not licensed to be given concurrently with NSAIDs. It has been given with NSAIDs and is s good rescue drug.

The use of corticosteroids in osteoarthritis is controversial, and they are generally used intra-articularly, which decreases the likelihood of systemic adverse effects. Rather than being true analgesics they exert a potent but temporary anti-inflammatory effect that will relieve pain. There is some experimental evidence that corticosteroids protect cartilage but also that they suppress cartilage matrix synthesis. If used at all the number of injections should be minimised.

Prednoleucotropin (PLT) is a combination NSAID and corticosteroid and that should set your alarm bells ringing. There is no evidence for its efficacy and anecdotal reports are mixed. There is however evidence of its adverse effects, generally gastroduodenal ulceration but there are the widely known corticosteroid adverse effects and the potential that they are deleterious effect to articular cartilage.

Amantadine was originally a human antiviral drug, which increases CNS dopamine concentrations and is an NMDA antagonist. It does not seem to have any analgesic effect as a sole therapy but it appears to enhance the analgesic effect of NSAIDs and opioids. One veterinary clinical trial showed a subjective effect when used with meloxicam. This study showed that you need to use amantadine for at least 3 weeks before assessing its response. Other drugs including gabapentin and tricyclic antidepressants such as amitriptyline are used to treat neuropathic pain in humans. There are no studies assessing the effect in osteoarthritic pain in cats or dogs.

Structure Modifying drugs

Polysulfated glycosaminoglycans are the structure modifying drugs with the biggest evidence base however it is not licensed in dogs in the UK. Its mechanism of action is unknown. Pentosan polysulfate (Cartrophen) is used extensively in the UK but there have been mixed results in clinical trials. There is only low to moderate evidence for its use and the studies have been poorly designed.

Nutritional Supplementation

Nutraceuticals such as glucosamine sulphate/hydrochloride, chondroitin sulphate and essential fatty acids have become extremely popular in the treatment of osteoarthritis however there is a low level of evidence to support their use.

Other medical therapies

Autologous platelet therapy and adipose derived mesenchymal stem cell therapy have emerged recently as treatment for osteoarthritis. There is some evidence for their effect but more research is required.

Complementary therapies

Laser therapy, shockwave therapy, ultrasonic therapy and acupuncture have all been described as treatment for osteoarthritis but there is limited or no evidence at present for their efficacy.

Surgical management

It is a sad fact that many animals with osteoarthritis may benefit from surgery but yet are denied this for various reasons. More animals will be euthanased for osteoarthritis than have surgery.

As mentioned previously the ideal surgery is aimed at early treatment of the underlying condition however this is often difficult as the diagnosis is made too late in most cases. The cascade of events leading to OA has already begun and the OA progression invariably proceeds regardless of surgical intervention.

In established osteoarthritis salvage surgery is aimed at relieving pain and includes arthrodesis, excision arthroplasty and joint replacement.

Inflammatory arthritis

Immune-mediated arthritis

History and clinical signs

Immune mediated arthritis is typically associated with multiple joint pain or swellings and is usually symmetric but may be asymmetric. Very occasionally it may only affect one joint. The animals often have a shifting lameness and generalised stiffness, with inactivity stiffness typically lasting several minutes in contrast to the inactivity stiffness of OA, which typically only lasts few seconds. The animals often present as pyrexia of unknown origin, in fact immune mediated polyarthritis is the most common diagnosis of PUO.

Diagnostic Tests

Synovial fluid analysis is essential for the diagnosis of immune mediated arthritis. The diagnosis requires evidence of inflammation in several joints and so it is recommended to sample four joints. The larger joints are easier to sample however the carpus and tarsus are often the joints affected. The table earlier in the notes gives the cell count numbers etc. but an over all increased total cell count with an increased percentage of non-degenerate neutrophils. If there is any suspicion of infection then send the synovial fluid for culture.

Radiography has traditionally been recommended in all cases however it may be uninformative early in the disease so is often unnecessary. It may be useful to obtain radiographs as the disease progresses to monitor for erosions, periosteal reactions and deformities.

Other diagnostic tests may be indicated, such as haematology, serum biochemistry, urinalysis, EMG, muscle biopsy and CSF, to probe for an underlying or associated condition. Serology for rheumatoid factor and antinuclear antibody may be useful however RF is not specific for rheumatoid arthritis and ANA is not specific for SLE.

Non-erosive immune mediated polyarthritis

Immune mediated polyarthritis (Types I-IV)
Polyarthritis-polymyositis syndrome
Systemic lupus erythematosus and SLE related disorders
Drug-induced immune mediated polyarthritis
Breed associated immune mediated polyarthritis

Immune mediated polyarthritis
Type I – Idiopathic IMPA (by far the most common)
Type II – associated with infection remote from joint
Type III- associated with GI disease
Type IV- associated with neoplasia

Erosive immune mediated polyarthritis

Rheumatoid arthritis

- Rare
- Difficult to diagnose
- Distinguished from other IMPA by erosive changes in joints on radiographs and rheumatoid factor in serum
- Often no radiographically evident changes are visible early on
- List of diagnostic criteria to make working diagnosis

Polyarthritis of greyhounds
Feline chronic progressive polyarthritis

Treatment

The aim of treatment should be to first eliminate the inciting cause however this is often not possible for example in Type IMPA and rheumatoid arthritis. Therefore treatment often requires immunosuppressive drugs. The mainstay of therapy is prednisolone at 1-2mg/kg BID slowly tapering over a number of months dependent on response. Other immunosuppressive drugs may need to be added in refractory cases or in cases with marked corticosteroid side effects. Azathioprine, cyclophosphamide and ciclosporin are the most common additional drugs.

Other treatment modalities such as weight management and physiotherapy can also be used as necessary. With joint deformities salvage surgery such as arthrodesis, excision arthroplasty or joint replacement can be considered.

Infective (Septic) Arthritis

Infective arthritis is an uncommon condition, which is usually bacterial but can be mycoplasmal, protozoal, rickettsial and mycobacterial. Fungal arthritis is seen in the US.

Bacterial septic arthritis

Bacterial septic arthritis can be caused by haematogenous spread, direct penetration (surgical or traumatic) or local spread. Many different bacteria can be isolated but in dogs it is typically Staph. intermedius, Staph. aureus, Strep. spp whilst in the cat it is typically Pasteurella multocida, Bacteroides spp. Risk factors include previous surgery and pre-existing disease e.g. osteoarthritis. The most frequently affected joints are the stifle, elbow and carpus.

History and clinical signs

Septic arthritis is usually a monoarthropathy with moderate to severe lameness. Normally there is joint pain, swelling and heat with local lymphadenopathy. Pyrexia is present in a minority of cases and these dogs can be systemically unwell.

Diagnosis

The diagnosis is based on synoviocentesis. Grossly there is an increased volume of turbid fluid. Cytologically (see previous table) they is an increased cell count with predominately neutrophils that demonstrate degenerative and toxic changes. Synovial fluid should be submitted for bacterial culture (in blood culture medium) before antibiotic therapy is started. A synovial biopsy can be submitted for tissue culture but is unclear if this is any better than synovial fluid at isolating causative bacteria.

The criteria for a diagnosis of definitive bacterial septic arthritis is all three of the following typical history and signs, synovial fluid cytology consistent with bacterial infective arthritis and a positive bacteriologic culture. If the first two are present then a diagnosis of probable bacterial septic arthritis can be made.

Radiography is not required but is helpful at assessing the underlying condition of the joint.

Treatment

There is no clear evidence for what is the best approach. Systemic antibiotics, based on culture and sensitivity, are the standard. Typically you can start with clavulanate-potentiated amoxicillin or cephalosporins. Long courses, 4-6 weeks initially, are required with repeat synoviocentesis towards end. Use antibiotics until the joint fluid is normal or consistent with OA. It is unclear whether joint irrigation is required but this is also normally performed. Needle irrigation is performed in all of my cases. Arthroscopic irrigation can also be performed and foreign material if present e.g. after a penetrating wound, can be removed this way. Open arthrotomy is not recommended unless surgical implants need removal. Infected implants in the surrounding tissue must be removed to resolve the infection.