

Canine Polyarthritis  
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## **Introduction**

Canine polyarthritis (PA) is an inflammatory disease that affects multiple joints. This is distinguished from polyarthropathies, which are any disease that affects multiple joints (not necessarily inflammatory). Canine polyarthritis can be further divided into two categories: those that are caused by infectious processes and those that are caused by non-infectious processes. The non-infectious categories can be further divided into erosive and non-erosive processes. The vast majority of non-infectious cases of canine polyarthritis fall under the category of immune-mediated disease. This subcategory has been recently divided in four different types. Type I immune-mediated polyarthritis (IMPA) is the most common of the types and can not be associated with any underlying process. Type II IMPA is associated with chronic infectious diseases, type III IMPA is associated with underlying gastrointestinal disease, and type IV IMPA is associated with underlying neoplastic disease. Canine PA can affect dogs of any age depending on the underlying cause. Many forms of PA show a predilection for dogs of a younger age. There is been no sex predilection demonstrated in the literature.

## **Clinical Signs**

The typical clinical signs can include reluctance to walk (80%), spontaneous vocalization (33%), exercise intolerance (20%), stiffness (100%), lameness, inability to stand, weight loss, and anorexia. Typical physical exam finding can include pyrexia, warm, swollen, and painful joints, lymphadenopathy, depression, lameness, and joint instability with subluxation or luxation.

## **Differential Diagnoses**

### **Infectious**

Many infectious agents can cause canine PA and include bacterial, protozoal, spirochetes, and fungal organisms. There are a couple of reasons that joints can become a destination for infectious diseases. The synovial lining of most joints has a very high blood flow and therefore any hematogenous microorganisms can end up coasting through. In addition to high blood flow, the synovium is a very important area of filtration and phagocytosis of blood born pathogens. These two characteristics make joints a very common destination for blood borne pathogens. Most bacteria can infect joints, but some of the more common species include *Streptococcus*, *Staphylococcus*, coliforms, *Corynebacteria*, *Erysipelas*, and *Pasturella*. Bacterial infections must find a way into the joint and often result from

penetrating wounds, extension from bone or soft tissue infections, or from hematogenous spread. The primary source of hematogenous bacteria can be lung, gastrointestinal and urinary tract, umbilicus, or endocardium.

The largest collection of infectious causes of canine PA are the tick-borne diseases. Lyme disease, Rickettsia, and Ehrlichia are the most common. These are diseases that often present with PA but can also be associated with lymphadenopathy and fever. Fungal infection is not very common and would likely occur from extension of bony infections. Possibilities include *Blastomyces*, *Histoplasma*, *Cryptococcus*, and *Coccidioides*. Protozoal infection with *Leishmania* has also been reported to cause canine PA.

### Non-infectious

The vast majority of cases of canine PA are considered immune-mediated. The pathophysiology of immune-mediated PA is complex and incompletely understood. Immune complex formation either occurs in the joints themselves or they are formed distantly and then deposited in the joints. Immune complex deposition leads to joint tissue destruction and influx of inflammatory cells. Neutrophils release lysosomal enzymes that lead to further tissue destruction. In certain types of IMPA (rheumatoid arthritis), the production of altered self-antigens can lead to loss of tolerance to self and the production of autoantibodies and antinuclear antibodies.

IMPA can be divided into those disease processes that cause erosion of the joint and those that do not. Most cases of IMPA are of the non-erosive variety. Recent literature has divided non-erosive IMPA into four different categories. (See above). IMPA is now recognized as the most common cause of fever of unknown origin seen at referral hospitals. Most cases (50%) of IMPA are of the idiopathic variety. Type II, III, and IV are related to disease processes elsewhere. It is thought that these distant disease processes are responsible for the production of immune complexes. These complexes can become lodged in the joints and serve as the catalyst for PA.

The most common form of erosive IMPA is rheumatoid arthritis. This is usually a disease of younger, small breed dogs. These animals generally present with variable degrees of fever and malaise and most of the time have a bilaterally symmetrical swelling of the joints. The carpus and the tarsus are the joints most commonly affected. The disease can be cyclical in nature and eventually leads to erosion followed by complete destruction of the affected joints. The exact etiology remains unknown.

Another recognized form of erosive IMPA is known as polyarthritis of greyhounds. This is a disease that looks very similar to idiopathic non-erosive PA. This is a disease of greyhounds that is not as destructive as Rheumatoid arthritis and tends to affect the proximal interphalangeal joints, carpus, tarsus, elbow and stifle.

Systemic lupus erythematosus (SLE) is another cause of IMPA. PA is the most common manifestation of SLE but it can also affect the skin, central nervous system, and the kidneys.

Young beagles, boxers, pointers and rottweilers can get a form of polyarthritis/ meningitis that is manifest as cyclical bouts of fever, depression, distal joint and neck pain. This tends to be self-limiting after several cycles.

Drug reactions can also lead to PA. These are often seen in conjunction with skin rashes, fever, and lymphadenopathy. It is important to recognize the recent addition of drugs with onset of clinical signs. Any medication can cause this but the sulfa drugs tend to be the most common.

In additions, there are also many breed-associated cases of PA. These have been reported in Akitas, Weimaranars, Boxers, SharPeis, and spaniels.

### **Diagnostic work-up**

When working up a case of PA, the diagnostics that are chosen will likely be dictated by signalment, history, physical exam findings, etc.

### **Minimum Data Base**

The first thing that should be considered is a minimum data base (CBC, chemistry, urinalysis). Things to look for include leukocytosis, a left shift, urinary tract infections, or any other changes that could be linked to systemic diseases.

### **Radiology**

Radiographs of the affected joints should be obtained. Things that may be identified on films include periarticular osteoporosis, loss of articular cartilage, subchondral bone cysts, subluxations or luxations. Most of the time the only visible changes will include soft tissue swelling and joint effusion.

### **Arthrocentesis**

Joint taps are essential to the diagnosis of PA. Joint taps are very easy to perform and can be done in most patients with simple sedation only. They can be performed quickly and with supplies that are available at most veterinary hospitals. It is recommended that 3-6 joints be tapped to get a sample from multiple joints for comparison. The carpus, tarsus, and stifle are easily accessible and straightforward to tap. Arthrocentesis should be performed using standard sterile technique with adequate hair clipping, sterile prep, gloves, needles and syringes. The tests that can be performed on the fluid collected are dictated by the amount of fluid that can be obtained. Even small amounts can be diagnostic. The minimum analysis should include a fluid analysis for total cell count and a differential. If a very small amount is obtained, direct smears can be performed. A small amount of fluid should be kept for aerobic and anaerobic culture and sensitivity.

Normal synovial fluid should have a cell count of 100-3000 WBC/  $\mu\text{L}$  which translates roughly to about 1-3 WBC/ HPF. Neutrophils should represent less than 10% of the white blood cells present. In most cases of PA, total white blood cell counts are between 4000-400,000 WBC/  $\mu\text{L}$  depending of the initiating cause with the majority of cells being neutrophils. A joint with simple osteoarthritis will have a WBC between 1000 and 100,000 with a majority of mononuclear white blood cells. If enough joint fluid can be collected, other diagnostics to consider include protein quantification and a mucin clot test. These are rarely performed in veterinary medicine due to the low quantity of fluid collected.

### Cultures

As mentioned above, collected synovial fluid should be submitted for aerobic and anaerobic culture and sensitivity. The vast majority of these cultures will wind up being negative. Other sources to consider for culture include blood and urine looking for a hematogenous source of infection (pneumonia, endocarditis) or a hidden urinary tract infection.

### Synovial biopsy

Synovial biopsy can be considered for joints in which the etiology of the PA is yet undetermined. Biopsy of the synovium can help to rule out neoplasia as the cause and direct tissue culture can often lead to a positive culture when a synovial fluid sample has been negative.

### Serology

Serological titers can be performed for the tick-borne diseases (Lyme, Rickettsia, Ehrlichia). These are generally available as a panel and are straightforward to perform. Positive results for Lyme disease only indicate exposure so the results must be interpreted with history, physical exam finding, etc. For Rickettsial disease, we are looking for a 4-fold increase between acute and convalescent titers. Rickettsial titers can take a couple of weeks to increase, therefore a negative result does not rule out disease. Generally any titer to Ehrlichia is considered significant and should be treated for. Anti-nuclear antibody can be performed in cases that are suspected to have SLE. This is sensitive, but not specific for SLE as an ANA can be increase in any systemic inflammatory disease. Rheumatoid factor can also be performed. It is reported as positive in 20-70% of dogs with Rheumatoid arthritis but can also be positive in dogs with systemic inflammatory disease.

### Staging

In any animal with PA, staging for neoplasia should be considered as part of the diagnostics. In addition to the minimum database, chest radiographs and abdominal ultrasound give the best indication of potential neoplastic diseases that could be causing the PA.

## **Treatment**

Treatment for all cases of PA should be aimed at the initiating cause (if possible). In severely affected or debilitated cases, hospitalization with supportive care may be necessary. Antibiotics are chosen empirically (after samples for culture and sensitivity have been collected) or, more ideally, based off of sensitivity of a positive culture. Suspected tick-borne cases should be treated with doxycycline (5-10 mg/kg BID) or tetracycline (22mg/kg TID).

Most cases of PA are going to be considered idiopathic and immune-mediated. The treatment of choice for these cases is immunosuppressive doses of prednisone. Most patients will receive 2-4mg/ kg/day for the first two weeks. This dose is then lowered to 1-2mg/kg/day for the next two weeks. Joint taps are then repeated after one month and further treatment is based on these results. The goal is to taper off of prednisone to the lowest dose possible that controls the clinical signs. For case that are non-responsive to prednisone, treatment with another immunosuppressive such as azathioprine should be considered. Non-steroidal anti-inflammatories can be considered for cases that have their disease in remission but still suffer from the erosive affects of their disease. Additions to the medical treatment can include MSM, glucosamine/ chondroitin, etc. In addition, most patients will benefit from weight reduction and maintenance. In cases with sever erosions and or luxations, surgical arthrodesis of affected joints has been performed successfully.

## **Prognosis**

Prognosis largely depends on the underlying cause and whether it can be treated. Lyme disease tends to have a good prognosis where *Rickettsia* and *Ehrlichia* are a little more variable depending on clinical presentation. Rheumatoid arthritis has a poor prognosis with most cases being presented late in the disease but a reasonable quality of life may be obtainable with surgical arthrodesis. A recent report of 39 cases of idiopathic IMPA showed a good prognosis with reported 56% cure and another 18% managed with continued medications.

## **Vaccinations**

Vaccine administration has been implicated in the pathogenesis of PA but its role remains controversial. There have been multiple cases of IMPA reported following recent vaccination. In addition, distemper virus antigens and antibodies have been isolated from joints affected with PA. However, more recent cases series of IMPA have shown no correlation with timing of vaccine administration and the onset of PA.

## **Conclusion**

Most cases of polyarthritis are of the idiopathic variety and polyarthritis should be considered for any case of fever on unknown origin. Polyarthritis is a complex disease process that is incompletely understood. Treatment should be aimed at the underlying

cause. Most cases of idiopathic IMPA can be cured or made substantially better with treatment. The role of vaccination in PA is still undetermined.

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