Bacterial septic arthritis in 19 dogs

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Objective To provide information on the clinical features, diagnosis and treatment of bacterial septic arthritis in dogs.

Design A retrospective study examining case records of all dogs diagnosed with bacterial septic arthritis at Murdoch University Veterinary Hospital between 1988 and 1997.

Results Nineteen dogs were diagnosed with bacterial septic arthritis, which most commonly occurred after surgery involving the stifle joint. Haematogenous infection occurred in only five dogs. Diagnosis was based on clinical signs, joint fluid analysis, radiography, microbiology and/or response to treatment. Chronic lameness was the most common problem at presentation. Analysis of joint fluid invariably revealed large number of nucleated cells, which consisted primarily of neutrophils. In all but one case the neutrophils were nondegenerate. Culture of joint fluid was frequently successful. *Staphylococcus* spp were the most common bacteria isolated. Treatment involved antimicrobial drugs only in five dogs. Other dogs received antimicrobial drugs in combination with surgical procedures such as joint lavage and removal of nonabsorbable suture material (eight), arthrodesis (two) or amputation (one). Two dogs were euthanased. Most dogs responded well to treatment and were free of signs of septic arthritis at follow-up.

Conclusion Bacterial septic arthritis may often be mild and manifest as chronic lameness. Analysis of joint fluid will detect an inflammatory arthropathy but the presence of toxic neutrophils should not be relied on as an indicator of sepsis. Culture of infected joint fluid is likely to be successful if antimicrobials are not given prior to collection and if the sample is inoculated into enrichment broth. Treatment should involve antimicrobial drugs, open-joint lavage and removal of joint prostheses if the infection is associated with previous surgery.

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CCL	Cranial cruciate ligament
MUVH	Murdoch University Veterinary Hospital

Bacterial septic arthritis is an inflammatory arthropathy that may result from haematogenous spread, an accidental or surgical wound or, rarely, by extension from surrounding tissues.[1] Unlike in cattle, horses and other farm animals, joint infection in dogs is usually monoarticular and is typically associated with acute onset of lameness.[1,2] However, joint infection may manifest as chronic lameness, which may be misdiagnosed as osteoarthritis if arthrocentesis is not performed.[2] Early recognition of joint infection is important to limit the damage to articular cartilage caused by proteolytic enzymes released from damaged synoviocytes and the formation of pannus which invades through the articular cartilage into subchondral bone.[3]

Analysis of joint fluid from septic joints reveals markedly increased counts of nucleated cells, usually in excess of 50 x 10^9/L, consisting primarily of neutrophils. In chronic cases the increase in cell numbers may be less dramatic.[3,4] A diagnosis of septic arthritis is confirmed by demonstration of bacteria in the joint fluid or capsule by Gram stain and/or culture.[3,5] Culture of joint fluid is not always successful in septic arthritis,[2,6] but incubating joint fluid in enrichment broth increases success.[7] Culture of capsule is a more sensitive method of detecting bacteria but more invasive and expensive than arthrocentesis.[2,8]

Treatment of septic arthritis involves an extended course of antimicrobial drugs based on sensitivity testing.[3,5] In addition, sepsis associated with a penetrating wound, whether surgical or accidental, should be treated by joint lavage using 0.9% saline. This may be accomplished by arthrotomy and open lavage or by insertion of ingress/egress drains.[2,5] The former is more likely to result in complete debridement and removal of purulent material and avoids difficulties associated with managing drains in small animals.[4]

This study examines the case records of 19 dogs that were diagnosed with septic arthritis at MUVH between 1988 and 1997. It provides information on clinical features, diagnosis and treatment and highlights some difficulties associated with differentiating septic arthritis from other arthropathies.

Materials and methods

Case records of all dogs that had been computer coded with a diagnosis of septic arthritis between 1988 and 1997 at MUVH were examined. The records of all 612 animals that had undergone arthrotomy during this same period were also examined. Diagnostic evaluation for each case included one or more of the following: physical examination, palpation of the affected joint, rectal temperature measurement, blood cell counts and serum biochemical tests, arthrocentesis and joint fluid analysis, radiography, arthrotomy and joint capsule biopsy. Bacterial culture of joint fluid, joint capsule or any prosthesis that may have been used at a previous surgery was performed as applicable.

Arthrocentesis was performed aseptically and joint fluid placed into plain or EDTA-coated tubes. Total and differential nucleated cell counts were performed on all joint fluid samples that were not clotted. The proportion of neutrophils and whether they were degenerate were also recorded. Gram stains were performed on all samples. Culture was performed by direct inoculation onto blood agar plates (Oxoid

trypticase soy agar) before and after inoculation into enrichment broth (Oxoid signal blood culture medium). Joint capsule samples were crushed and mixed with enrichment broth as well as being inoculated onto blood agar plates. Samples were cultured aerobically and anaerobically in all cases.

All dogs were treated using antimicrobial drugs with or without ancillary therapy such as arthrotomy, joint lavage and removal of prostheses where appropriate. Follow-up for outcome of treatment was either by revisit examination or telephone consultation with the owner.

Results

History and patient details

Nineteen dogs (10 male and 9 female) were diagnosed with septic arthritis. Eleven different breeds were represented and ages were from 6 months to 8 years (median 5 years). Fourteen cases were associated with one or more previous surgical procedures involving the stifle, 11 of which were extracapsular CCL stabilisation procedures. Suture material used to stabilise the joint was prolene in one, braided polyester tape in one, teflon-coated braided polyester suture in two, nylon leader line in four and not recorded in three animals. Eight postoperative stifle infections occurred following surgery at MUVH. The overall prevalence of joint sepsis following stifle arthrotomy at MUVH was 2.5% (8/320). Five cases which developed signs of heat, pain and swelling in the immediate period after stifle arthrotomy at MUVH were discarded from the study because joint fluid analysis was not performed. These cases all responded to antimicrobial therapy. Of all MUVH arthrotomies performed the only joints affected with postoperative sepsis were stifles.

Of the 14 dogs with postoperative infections, 3 developed signs of infection within 7 to 14 days of surgery at MUVH. The 11 others had a gradual onset of lameness or, alternatively, did not progress well postoperatively for 6 weeks to 16 months before diagnosis was made. Six had undergone arthrotomy at other clinics and five at MUVH. These cases were classified as chronic or low-grade septic arthritis similar to the category described by Bennett and Taylor.[2]

Haematogenous joint infection was diagnosed in five dogs. Two had been chronically lame and previously treated for radiographically confirmed osteoarthritis, whereas three were presented as acutely lame. The femoral head had been excised from one of these 18 months earlier but the dog had been sound until presentation for the current problem. This case was considered a haematogenous infection because of the delay between surgery and onset of signs and because *Escherichia coli* was cultured from the blood.

Physical examination

Rectal temperature was recorded in 15 dogs and was increased (> 39.2°C) in 12. The affected joint was painful and swollen in 19 dogs and recorded as being palpably hot in 11 cases. The owners reported lethargy and depression in 10 of 19 cases. A discharging sinus was present on the lateral aspect of the stifle in only two dogs. Both had undergone extracapsular CCL repair using teflon-coated braided polyester suture (Table 1). The 20 affected joints comprised 15 stifles, 2 hips, 2 hocks and 1 elbow. On one occasion the stifle and hock were both involved as a complication of a tibial fracture repair. In this case the pin had penetrated both joints and infection had spread from osteomyelitis at the fracture site.

Arthrocentesis

Arthrocentesis was attempted in 17 cases. On four occasions little or no joint fluid was collected. One of these was a stifle that on exploratory arthrotomy was found to have severe destruction of articular cartilage and little joint fluid, one case was a hock and the other two cases were hips with severe acetabular osteophyte formation that may have increased the difficulty of entering the joint space. Total nucleated cell counts performed on nine samples averaged 100 x 10^9/L with a range of 15.2 to 152 x 10^9/L (normal less than 5.0 x 10^9 / L). Differential cell counts were performed on 12 samples. Neutrophils made up 90% or more of the cells in 10 samples with a range of 77 to 95%. Degenerate (toxic) neutrophils were detected in only one sample. Joint fluid was collected during necropsy in one case and was only cultured.

Microbiology

Gram stains were performed on 13 joint fluid samples. Gram-positive cocci were detected in six and a combination of Gram-positive rods and cocci and Gram-negative rods in one. The most commonly cultured bacterium was *Staphylococcus intermedius.* Other bacteria isolated included *S aureus,* beta-haemolytic *Streptococcus* and *Pseudomonas aeruginosa* (Table 2). Joint fluid alone was submitted for culture from 12 dogs (including two samples that had insufficient volume to allow cytological examination) and bacteria were cultured from all samples. In addition, joint fluid and joint capsule submitted from four dogs resulted in positive culture from the capsule only in one, from the fluid only in another and negative cultures from both in the other two dogs. In two cases of sepsis following CCL surgery, only the prosthesis was submitted for culture and bacteria were cultured in each case. Sepsis of the joint rather than just suture prosthesis contamination was confirmed in these cases by radiographic and surgical findings of subchondral bone loss, articular erosion and pannus formation. *E coli* was successfully cultured from the blood of one dog where arthro-

Table 1. Summary of clinical findings in 19 dogs with septic arthritis

Clinical findings	Number of cases
Lameness	19
Chronic	13
Acute onset	6
Painful, swollen joint	19
Previous surgery	15
CCL stabilisation	11
Pyrexia (> 39.2°C)	12[a]
Lethargy, depression	10
Discharging sinus	2

[a]Rectal temperature recorded in 15 dogs only.

Table 2. Bacteria isolated from septic arthritis in 19 dogs.

Bacteria	Number of joints
S intermedius	6
S aureus	2
P aeruginosa	2
Streptococcus spp	2
Mixed	3
E coli	1

E coli was isolated from blood culture; all others from joint fluid, joint capsule and/or suture.

centesis had been unsuccessful. All Gram-positive species cultured (13 cases) were sensitive to amoxycillin-clavulanate, and all except one, which had been treated with cephalexin previously, were sensitive to cephalexin.

Radiography

Radiographs were taken of 16 of the 20 affected joints. Soft-tissue swelling associated with periarticular thickening and joint effusion were commonly seen as was periarticular growth of new bone. Patchy lysis of subchondral bone was seen in four cases. A radiographic diagnosis of septic arthritis was made in only three cases. In these, the septic process continued for several months and there was gross destruction of subchondral bone and severe periarticular formation of new bone.

Treatment

Haematogenous infection – The five dogs in this category were treated with antimicrobial drugs only for between 10 days and 8 weeks. The condition of all dogs had improved at time of follow-up (8 weeks to 6 years). The owners of two dogs reported the dogs had not been lame since the end of treatment 4 and 6 years previously. The three others were examined 2, 10 and 12 months after treatment and had mild lameness attributable to osteoarthritis that had been diagnosed previously in the affected joint.

Postoperative joint infection – Of the 14 dogs that developed postoperative septic arthritis, the diagnosis was confirmed at necropsy without treatment in one, and another was lost to follow-up before commencement of treatment (Table 3). Amputation was performed on one dog that had osteomyelitis of the tibia and septic arthritis of the stifle and hock. Successful arthrodesis was performed on two others. Another case was treated with cephalexin for 4 weeks but 2 weeks later was diagnosed with a ruptured CCL. At this time Gram-positive cocci were seen in joint fluid. The lameness did not resolve despite a further 6 weeks of cephalexin and extra-articular CCL repair. The dog was euthanased because of persistent lameness and multiple joint abnormalities associated with osteochondrosis. Four other low-grade cases were treated initially with antimicrobial drugs alone. These failed to improve and were subsequently treated more aggressively.

Table 3. Treatment of 14 cases of postoperative infection involving the stifle joint.

Treatment	Number of cases
Antimicrobial drugs and joint lavage	8
Arthrodesis	2
Amputation	1
Euthanasia, antimicrobial drugs failed	1
Euthanasia without treatment	1
Lost to follow-up	1

In six dogs treatment included suture removal, open-joint lavage with 0.9% saline for 5 to 7 days and delayed primary closure. Two others were similarly treated by suture removal and intra-operative joint lavage with 0.9% saline but primary closure was performed. The condition of all eight dogs was improved at time of follow-up (1 to 36 months). Two had no discernible lameness, one was lame after heavy exercise only, four had a mild lameness responsive to nonsteroidal anti-inflammatory drugs and one was chronically lame due to grade four patellar luxation.

Discussion

Septic arthritis can result from penetrating or surgical wounds, by extension from surrounding tissues or via haematogenous spread.[1] The literature is divided as to the most common route. Bennett and Taylor[2] found that most septic arthritides are haematogenously spread whereas others[4,5,9] suggest that most occur secondary to a penetrating wound. In this study, the latter view is strongly supported, as only five dogs had no history of a surgical or other penetrating wound. Three of these cases had severe and long-standing osteoarthritis that is thought to predispose to haematogenous joint infection, perhaps because the increase in synovial vasculature in osteoarthritic joints may favour the penetration of bacteria into the joint.[1]

A male-to-female ratio of 2:1 in canine septic arthritis has been demonstrated previously,[2] though an even distribution was seen in this study. Over half of the present cases developed sepsis after CCL stabilisation procedures. Of these, females outnumbered males by 1.5:1, which is in accordance with the occurrence of CCL rupture in the general population.[10] This may in part explain the relatively more common occurrence of septic arthritis in females in this study.

It has been suggested that synovial fluid of infected joints often contains toxic neutrophils which may help to differentiate sepsis from other inflammatory arthropathies[3] and that large numbers of toxic neutrophils is an indication to culture joint fluid.[11] Toxic neutrophils were only identified in 1 of 12 septic joints (8%), and were thus of minimal diagnostic value in detecting septic arthritis in this series. To the authors' knowledge, no other veterinary reports indicate how often toxic neutrophils are identified in septic joints. Glucose concentrations in synovial fluid, not determined in this series, can be markedly reduced in septic arthritis in humans and animals, but there is substantial overlap between septic and other inflammatory arthropathies.[4,12,13] Given the limitations regarding toxic neutrophils and glucose, it is advisable to culture the joint fluid of any dog with an inflammatory arthropathy involving a single joint.

There is disagreement as to whether culture of joint capsule is more sensitive than joint fluid culture. Bennett and Taylor[2] successfully cultured joint fluid and capsule in 81% and 100% of cases of septic arthritis, respectively, but the joint fluid was inoculated directly onto blood agar plates. In an experimental study where joint fluid and capsule were cultured 24 h after intra-articular inoculation with *S intermedius*, Montgomery and others[7] found culture of joint fluid to be more sensitive than that of capsule when the fluid was inoculated into blood culture medium. In this study joint fluid was successfully cultured in 12 of 15 samples (80%) but in only 1 of 4 joint capsule samples. In two instances, in which neither joint fluid nor capsule were successfully cultured, antibiotics had been given prior to sampling.[14,15]

There is a need to establish the efficacy of culturing joint capsule in comparison to inoculation of joint fluid into blood culture medium in the veterinary clinical setting. Culture of joint capsule is justified when arthrotomy is performed for other reasons such as removal of a joint prosthesis and/or

open-joint lavage. However, biopsy of joint capsule is more difficult to justify in cases of haematogenous joint infections. If culture of joint fluid is negative and there is a strong suspicion of sepsis, the clinician may have to choose between capsule biopsy and empirical therapy using cephalexin or amoxycillin-clavulanate. Given that 70 to 90% (86% in this study) of septic arthritides are caused by Gram-positive bacteria,[2,6] many cases may be successfully treated if the latter course is taken. If Gram-negative bacteria are seen but not cultured then there may be more indication to perform capsule culture as the sensitivities of Gram-negative species such as *P aeruginosa, Klebsiella* spp *and Proteus* spp are less predictable.[16]

In two dogs no bacteria were cultured from joint capsule, fluid or prosthesis which puts the diagnosis of septic arthritis in doubt.[3] However, both dogs received antibiotics prior to collection of the samples. The diagnosis in one case was made on the basis of clinical signs (heat, pain and swelling of the joint 7 days postoperatively, pyrexia), a high polymorphonuclear cell count in the joint fluid and complete response to antimicrobial drugs, open-joint lavage and delayed primary closure. In the other case, diagnosis was based on Gram stain of the joint fluid, which had a mixed population of bacteria. The presence of a mixed bacterial population after collection of the sample at arthrotomy makes it unlikely that the bacteria were contaminants. This case provided dilemmas in other areas. Despite separate courses of amoxycillin-clavulanate, norfloxacin and enrofloxacin, the joint fluid maintained a high polymorphonuclear cell count (> 100 x 10^9/L), although bacteria were not seen in two subsequent joint fluid analyses. A possible explanation for this is the syndrome described in man,[17] dogs,[2] pigs[18] and rabbits[19] where there is persistent joint inflammation in the absence of bacteria. This inflammation is thought to be perpetuated by antigenic bacterial fragments which stimulate the immune response and cause immune complex hypersensitivity.[2] These cases are responsive to corticosteroids. Stifle arthrodesis was ultimately performed in this case and resulted in pain-free use of the limb with no evidence of postoperative osteomyelitis.

Two cases of haematogenous septic arthritis were manifested as chronic problems. These cases had been treated for osteoarthritis with success for at least 1 year prior to diagnosis. Another case, not included in this study, manifested similarly and had also been treated for osteoarthritis for more than 12 months. This was initially diagnosed as infectious arthritis, despite a negative culture, on the basis of joint fluid analysis, the history of single joint involvement and evidence of severe gingivitis acting as a possible source of bacteraemia. However, the dog failed to respond to amoxycillin-clavulanate and was subsequently placed on immunosuppressive doses of prednisolone, which resolved the lameness and produced a pain-free joint. Whilst analysis of joint fluid was not performed to confirm resolution of the inflammatory arthritis, it is likely that this was an idiopathic inflammatory arthritis associated with a remote infection.[20] These cases highlight the difficulty in differentiating infectious arthritis from other arthropathies. They also demonstrate that a diagnosis of osteoarthritis should be reviewed if standard therapy such as nonsteroidal anti-inflammatory drugs with or without sodium pentosan polysulphate are only partially successful in relieving signs of arthritis.

The only joints affected with postoperative sepsis in this study were stifles. There are a number of possible reasons for this. Stifle arthrotomy involves a large joint incision which is more extensive than the standard arthrotomies of the hip, elbow, hock and shoulder joints.[21] As a result operative times are likely to be longer, increasing the potential for bacterial contamination.[22] Additionally, with extracapsular CCL repair, a nonabsorbable suture is placed in close proximity to the joint and may even be in direct contact with the fabellar extension of the lateral femorotibial joint pouch.[23] This may facilitate entry of bacteria into the joint from the suture material which, like other orthopaedic implants, may be associated with the production of a biofilm or glycocalyx that enhances bacterial adhesion and protects bacteria from phagocytosis.[24] Multifilament suture material is associated with a higher infection rate and formation of discharging sinuses,[25] though in this study there was no obvious association of type of suture material with joint infection.

Four cases of septic arthritis associated with extracapsular CCL repair were unsuccessfully treated with antimicrobial drugs alone emphasising the importance of removing nonabsorbable suture. The antimicrobials may have killed the bacteria within the joint but not those within the suture biofilm. The infection in these cases resolved after more aggressive therapy including removal of nonabsorbable suture and joint lavage was performed.

Overall, 12 cases of joint sepsis that were associated with surgery presented as low-grade infections. Animals are expected to be lame for 4 to 6 weeks following major joint surgery such as CCL stabilisation. It may be difficult to determine whether failure to improve postoperatively is due to mild infection. A recent study found little correlation between infection and the success of CCL repair procedures.[26] Only 2 of 320 CCL repair surgeries performed at MUVH developed low-grade septic arthritis. The remaining cases of low-grade joint sepsis following CCL repair were referred from other practices and the prevalence of postoperative sepsis in these practices is unknown. Despite this low rate, the effects of postoperative joint sepsis are potentially devastating. We therefore believe that joint fluid analysis should be performed in all cases of CCL stabilisation that do not improve, or that deteriorate gradually after a period of postoperative improvement. This would identify those cases that warrant microbiological investigation.

In conclusion, only 19 cases of septic arthritis were diagnosed over a 10 year period at MUVH. The most common cause of septic arthritis in this study was acute and chronic postoperative joint infection involving the stifle joint. Analysis of joint fluid was helpful in establishing the presence of an inflammatory arthropathy, but toxic neutrophils were detected in only 8% of samples and were not considered a reliable indicator of sepsis. Diagnosis was confirmed by culture of blood, joint fluid, joint capsule or periarticular prosthesis. In some cases bacteria were not successfully cultured because antimicrobials had been administered before collec-

tion of samples. In these cases there was sufficient supporting evidence to establish the diagnosis. Haema-togenous infections were successfully treated with antimicrobial drugs alone. Postoperative infections required antimicrobial drugs in combination with removal of nonabsorbable suture material, arthrotomy, joint lavage, arthrodesis or amputation. Overall the response to treatment was good.

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