Septic polyarthritis caused by Erysipelothrix rhusiopathiae in a dog

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Summary
A 14-month-old, male German Shepherd dog was admitted with a six-week history of lameness and swelling of the right hindlimb. Clinical examination revealed polyarthritis, fever, petechiae and ecchymoses of the abdominal skin and prepuce. The haematology and blood chemistry were indicative of sepsis. Mediolateral radiographic views of both of the stifles joints revealed signs of bilateral articular capsule swelling. The radiographic, bacteriological and necropsy findings confirmed a diagnosis of septic polyarthritis due to infection with Erysipelothrix rhusiopathiae.

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Introduction
Erysipelothrix rhusiopathiae (E. rhusiopathiae) is a nonsporulating, Gram-positive, rodshaped bacterium which was identified more than 100 years ago as the aetiologic agent of swine erysipelas (1). E. rhusiopathiae are widely disseminated in nature, and have been isolated from soil and water, as well as from humans, fish, dolphins, turkeys and various other animal species. These bacteria are facultative pathogens for a broad range of species with the domestic swine as one of the major reservoir hosts (2). In swine, infections with E. rhusiopathiae can cause sepsis, generalised skin infection, arthritis, and endocarditis. In dogs, E. rhusiopathiae is known to cause sepsis and endocarditis (3, 4). Although overt clinical disease associated with this bacterium in dogs is rare, one study found an Erysipelothrix-antibody prevalence of five percent in a population of stray dogs in Belgium (5). In humans, the mortality rate of endocarditis due to E. rhusiopathiae infection is surprisingly high (38%) and has been attributed to the common use of vancomycin. Erysipelothrix spp. are inherently resistant to vancomycin; the treatment of choice is penicillin (2).

Natural infections with E. rhusiopathiae have rarely been reported to cause arthritis in dogs (6). Nevertheless, the morphological and clinical features of the arthritis in these cases were similar to human chronic relapsing, erosive synovitis. Based on these findings, several infection models of Erysipelothrix arthritis in dogs have been developed (7–9). Young animals seem to be predisposed to the development of bacterial arthritis because of the unique anatomy of growing joints (8). In the joints of young animals, the cartilage is well perfused, facilitating bacterial seeding. The synovial membrane is close to the avascular articular cartilage, which acts as a barrier to leukocyte migration. The first stage in the establishment of experimental E. rhusiopathiae chronic arthritis is characterised by bacterial growth and spreading within the joint as well as multiplication in the cartilage layer and the entheses. The second stage is characterised by intense inflammation, acute destruction of the cartilage matrix and subsequent elimination of the bacteria. In this stage of diffuse dystrophy, bacteria are mostly eliminated but can still be found in the periarticular connective tissues and musculature. The chronic stage is characterised by lymphoplasmatic synovitis and subchondral pannus tissue. The interior of the joint capsule is usually free of detectable bacterial antigen (8). In another study using nine Beagles, experimental E. rhusiopathiae infection induced polyarthritis of the stifle, hip and elbow joints, but not the digital joints (9). The stifle joint was most often affected, and the infection caused swelling and hindlimb lameness. All dogs developed fever and later discospondylitis. Examination of the synovial fluid of the affected joints revealed an increased cell count. None of the dogs developed endocarditis.

Here we present a rare case of septic polyarthritis caused by E. rhusiopathiae in a dog with a fatal outcome.

Case report
A 14-month-old, 31 kg male German Shepherd dog was presented to the Small Animal Clinic of the Freie Universität Berlin with a six-week history of lameness and swelling of the right hindlimb. No recent trauma or other diseases had been noted by the owner. Initial treatment with antibiotics and carprofene led to improvement for one week. Nevertheless, one week after initiation of treatment, the severity of lameness increased and the dog developed scrotal oedema.
As these signs progressively worsened, physical examination revealed normal body temperature and bilateral hindlimb lameness, as well as petechiae and ecchymoses of the abdominal skin and prepuce. A comprehensive orthopaedic examination revealed swelling of the right stifle joint that was associated with signs of pain on manipulation. A complete blood count found a non-regenerative anaemia, leukocytosis (26,690/μl; reference range 5,600–14,000/μl) with left shift, and severe thrombocytopenia (42,000/μl; reference range 165,000–400,000/μl). The coagulation profile was normal. Serum chemistry revealed a mild hyperbilirubinaemia (6.5 μmol/l, reference range ≤5.1 μmol/l) and urinanalysis showed a mild haematuria, a mild bilirubinuria and bacteriuria on microscopic examination. However, a urine sample was found to be negative for bacterial growth and positive for the inhibitory test.

Medio-lateral radiographic examination of both of the stifle joints revealed signs of bilateral articular capsule swelling (Fig. 1), indicative of synovitis and arthritis. Radiographs of the abdomen and the thorax did not reveal any abnormalities.

Amoxycillin with clavulanic acidb (12.5 mg/kg IV BID), marbofloxacin c (2mg/kg/day IV), metamizold (20mg/kg IV TID), and a continuous rate infusion of crystalloid fluids e (2ml/kg) were administered.

On the second day after admission to the hospital, the patient developed a moderate fever of 39.7 °C, leucocytosis of 39,730/μl, and thrombocytopenia of 64,000/μl. The stifle joint synovial fluid was sanguineous and opaque with a specific gravity of 1.035, an increased total protein of 4.6 g/dL (reference range less than 2.5 g/dL), and a cell count of 60,000/μl (reference range 1,000–3,000/μl). Cytological analysis revealed abundant neutrophils without any bacteria. The blood culture grew E. rhusiopathiae, whereas the synovial fluid was negative for bacterial growth. The isolated E. rhusiopathiae was sensitive to amoxicillin with clavulanic acid, and marbofloxacin.

On the third day of hospitalisation, the general condition of the dog deteriorated severely despite intensive clinical care. Thoracic radiographs revealed an increased diffuse interstitial and alveolar lung pattern that was interpreted as pulmonary oedema and haemorrhage. Due to the poor prognosis the patient was euthanatized and submitted for necropsy.

Major findings were indicative of septicemia with embolic-purulent inflammation in several organs including lung, brain, kidneys, testicles and spleen as well as a severe diffuse endocarditis of the aortic valves. Furthermore, a severe, diffuse, subacute polyarthritis and synovitis of both stifles and, to a lesser extent, the hock joints were found (►Fig. 2). Post mortem histological examination of the synovial membrane revealed a marked infiltration by numerous neutrophils and lymphocytes that was indicative of subacute to chronic purulent synovitis and arthritis (►Fig. 3).

In summary, based on the radiographic, bacteriological and necropsy findings a diagnosis of septic polyarthritis involving the joints of the hindlimbs was made.

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b Synulox®: Pfizer GmbH, Germany
c Marbocyl®: Vetoquinol GmbH, Germany
d Vetalin®: Intervet, Germany
e Sterofundin®: Braun Melsungen, Germany
Discussion

Septic arthritis is an uncommon disease in dogs. In a previously published retrospective study, most cases of bacterial arthritis involved the stifle and were complications of surgery (10). The second most frequent cause of bacterial arthritis in that study was haematogenous arthritis, most often associated with a predisposing chronic degenerative arthritis (10). Commonly associated bacteria were Staphylococcus spp., Streptococcus spp., Pasteurella spp., Enterobacteriaceae and anaerobes (11–13).

Erysipelothrix spp. are ubiquitous organisms that remain infectious in the soil for months and infection sources are therefore numerous (14–16). In this case, the dog was kept in a rural area without any contact with swine, and the source of infection with E. rhusiopathiae could not be determined. Very few cases of Erysipelothrix spp. infection of dogs have been described in the literature (3,17). The majority of the case reports that have been published have described endocarditis and sepsis, but reports of septic arthritis after natural infection are uncommon (3, 4). Nevertheless, experimental studies have shown that subacute to chronic arthritis can be induced in dogs after intravenous administration of E. rhusiopathiae (9). In swine, chronic arthritis is often a sequela of sepsis (18).

In one case report, a German Short-haired Pointer with E. rhusiopathiae endocarditis had a four-month history of lethargy, intermittent fever, and shifting lameness that had been unresponsive to antibiotic therapy (3). Since the synovial tissue was not examined histologically, it could not be confirmed that the arthritic changes were caused by E. rhusiopathiae (3). Remarkably, the presenting clinical signs in our case included hindlimb lameness, while an apparent systemic infection did not manifest until the final week of the disease. By comparison, dogs that were experimentally infected with E. rhusiopathiae had intermittent fever, lameness (especially of the stifle joint), and changes in the spinal column such as discospondylitis with ventral kyphosis (9). None of these dogs had changes in the heart valves.

For the diagnosis, prognosis and treatment of septic arthritis, collection and analysis of joint fluid provides valuable information including cytology, cell counts, protein content and bacterial culture (19). In the case presented here, the diagnosis of septic arthritis due to E. rhusiopathiae was based on blood culture and histopathology. Bacteria were not detected in synovial fluid. Nevertheless, a review of published experimental studies showed that synovial fluid of infected joints often fails to exhibit bacterial growth, despite the fact that the animals still suffer chronic arthritis (9). In one clinical study, the bacteriological culture of joint fluid of dogs suffering from septic arthritis frequently isolated the causative organism (11). However, in Erysipelothrix spp. associated arthritis, induction and maintenance of the inflammatory lesion in the joint do not rely on intact bacteria. In fact, bacterial wall components alone are potent inducers of arthritis (8). Moreover, in some cases it is not possible to detect bacterial antigen in the joint (9). In the present case, pre-treatment with antibiotic may have hindered the isolation of bacteria from the synovial fluid.

The first important step in treating septic arthritis is the administration of appropriate antibiotics. The next important therapeutic intervention is surgical. Fearnside and Preston recommended arthroscopy, lavage, and drainage of the fibrinopurulent debris; these interventions resulted in early postoperative mobility and minimal morbidity (20).

The literature provides scant prognostic data for E. rhusiopathiae endocarditis in dogs. In one study of 24 dogs with aortic valve endocarditis caused by various organisms, most of the affected dogs died as a result of congestive heart failure, infarction, sepsis, or renal failure (4). Three of the 24 dogs were infected by E. rhusiopathiae, one died two days after admission, another was lost to follow-up and the third one was still alive five months after admission.

In conclusion, septic arthritis caused by natural infection with E. rhusiopathiae in dogs is very rare. In the patient described in this report, we did not identify the source of the infection. Conceivably, the infection might have been eliminated if appropriate antibiotics had been administered earlier in the disease course.

References