Intra-abdominal botryomycosis in a dog

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- ➤ Botryomycosis (bacterial pseudogranuloma) is a rarely reported and poorly understood disease found in many species.
- ➤ Most cases of botryomycosis in dogs involve the skin or muscles.
- ➤ Diagnosis of botryomycosis requires histologic examination and bacteriologic culture of the lesion.
- ➤ Treatment of this condition requires surgical intervention and administration of antimicrobials.

A 6-year-old 41.8-kg (92-lb) sexually intact male German Shepherd Dog used as an attack and pursuit dog by the local police department was examined because of general malaise and sudden onset of ataxia. Vaccinations were current, monthly heartworm preventative was being given, and the dog's history was unremarkable except for a transient episode of vomiting and diarrhea approximately 5 months previously that had responded to treatment.

The primary handler was out of town, but the substitute handler informed us that the dog had appeared normal earlier in the day but was now stumbling and having difficulty walking. On physical examination, the dog was alert and bared its teeth when approached. The dog was markedly ataxic and stumbled every few steps. During a limited neurologic examination, proprioception and deep pain were normal in all 4 limbs, no signs of pain were elicited on manipulation of the limbs, back, or neck, and otitis was not evident. Differential diagnoses at this time included otitis interna, intervertebral disk disease, meningitis, stroke, tick paralysis, trauma, and toxicosis. Rectal temperature was 40.2 C (104.4 F). Blood was drawn and sent to a commercial laboratory for CBC and biochemical analysis; a urine sample was not obtainable. Because of the dog's unpredictable temperament, he was treated with enrofloxacin (2.5 mg/kg [1.1 mg/lb], IM) and dexamethasone (1 mg/kg [0.45 mg/lb], IV) and discharged on enrofloxacin (2.5 mg/kg, PO, q 12 h). Results of the biochemical analysis were within reference range limits, but the WBC count was extremely high, with a total WBC count of 54,700 cells/ml (reference range, 5,900 to 16,900 cells/ml) and segmented neutrophil count of 74% (reference range, 51 to 67%). Red blood cell parameters were within reference range limits. Hematologic results were discussed with the substitute handler, and agreement was made to withhold further diagnostics to see how the dog responded to treatment with antibiotics pending return of the handler.

At the time of reexamination 3 days later, ataxia was still present but was not as pronounced as before. The dog now weighed 39.5 kg (86.9 lb) and had a rectal temperature of 38.5 C (101.3 F). While catheterizing the dog to obtain a urine sample, a mass was palpated in the central portion of the abdomen. Results of urinalysis were unremarkable. The handler agreed to return the following day for further diagnostics to better define the mass.

The following day the dog was sedated with butorphanol (0.16 mg/kg [0.07 mg/lb], SC), atropine (0.022 mg/kg [0.01 mg/lb], SC), and acepromazine (0.022 mg/kg, SC). Abdominal radiography revealed a large mass in the mid-dorsal portion of the abdomen displacing the abdominal viscera and a slight reduction in detail on the lateral radiographic view, suggestive of peritoneal effusion, was noticed. Thoracic radiography revealed no abnormalities. Ultrasonographic examination of the abdomen revealed a mass with walls of varying thickness and mixed echogenicity and approximately 8 cm × 10 cm (3.2 in × 4 in) in size. In real time, a fluid movement could be detected in several areas of the mass, and a small amount of free fluid in the abdomen was evident. The mass appeared not to be attached to any of the viscera in the central portion of the abdomen. Because the exact nature of the mass was unknown, the dog was given a guarded prognosis. Fine-needle ultrasound-guided biopsy and exploratory surgery were discussed as options, and the handler opted for exploratory laparotomy and possible surgical excision of the mass.

A catheter was placed, and lactated Ringer's solution (20 ml/kg/h [9 ml/lb/h], IV) was administered before and during surgery. Anesthesia was induced with sodium pentothal to effect and maintained with isoflurane. The dog was prepared for a laparotomy via a ventral midline approach. Immediately after the abdominal cavity was entered, a small amount of purulent serosanguinous fluid seeped out of the abdomen. The mass was isolated and gently dissected from the necrotic omentum that enveloped it and was exteriorized in 1 piece. The abdominal cavity was explored for further pathologic changes, but none were found other than signs of peritonitis. The abdomen was flushed with lactated Ringer's solution containing cefazolin; 3 Penrose drains were placed, and the abdomen was closed in a routine manner. The mass weighed 2.2 kg (4.5 lb), had a fibrous outer core, and was filled with pockets containing purulent fluid. Sections of the mass and its fluid were submitted for histopathologic evaluation and bacteriologic culture and susceptibility testing.

The day after surgery the dog was still slightly ataxic but was anxious to eat and drink and had a rectal temperature of 38 C (100.4 F). The abdomen was flushed with lactated Ringer's solution containing cefazolin via the Penrose drains, and cefazolin (33 mg/kg [15 mg/lb], IV, q 8 h) was administered. Treatment

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with cefazolin and abdominal flushes was continued for 3 days after surgery, then the Penrose drains were removed, and the dog was discharged on cephalexin (10 mg/kg [4.5 mg/lb], PO, q 8 h). Ten days after surgery, the dog was returned for staple removal and weighed 41 kg (90.2 lb). The ataxia appeared to have completely resolved, and the owner reported that the dog was returning to normal activity. Cephalexin was continued at the same dosage for 1 additional week. The handler failed to return the dog for a repeat examination after the course of antibiotics was completed.

The histopathologic report described the tissue sections as composed of dense fibrous connective tissue, granulation tissue, and adipose tissue heavily infiltrated with neutrophils and macrophages in a coalescing pyogranulomatous configuration. Discrete pyogranulomas were interspersed with diffuse inflammatory cells consisting of macrophages, neutrophils, and a few lymphocytes. The centers of some of the pyogranulomas contained round-to-oval bodies of colonies of gram-positive cocci embedded in a homogenous eosinophilic matrix. The histopathologic diagnosis was botryomycosis.[a] Results of bacteriologic culture of fluids and tissue revealed only *Staphylococcus intermedius*, which was susceptible to all antibiotics tested.[b]

In a computerized literature search, no references were found regarding intra-abdominal botryomycosis in dogs. An extensive literature search resulted in only 3 case reports of botryomycosis in cats, 2 of the visceral form and 1 of the integumentary form, and only 1 case report of botryomycosis of the integumentary form in a dog.[1]

Botryomycosis is a poorly understood granulomatous bacterial infection that may manifest in either cutaneous (integumentary) or visceral forms. In humans, cutaneous, pulmonary, and hepatic botryomycosis have been reported.[2-7] Mastitis is the most common clinical manifestation of botryomycosis in cattle,[8] although 1 case[9] of pulmonary botryomycosis has been documented. In swine and horses, botryomycosis most commonly manifests as wound infections and mastitis, although several cases of pulmonary botryomycosis have been reported in horses.[8] Botryomycosis has also been referred to as bacterial pseudomycosis and bacterial mycetoma because it mimics mycotic infections both clinically and histologically.[1,2] The cutaneous form usually involves a chronic nonhealing lesion. Often there is a history of trauma to the area, and sometimes a foreign body is involved.[10] The visceral form is usually a chronic disease that is nonresponsive or poorly responsive to treatment.[1] Depending on the stage at initial evaluation, the lesion may have a purulent exudate with small white granules about the size of a piece of sand. These granules are indistinguishable from those of actinomycosis, nocardiosis, and mycetoma unless special stains are used. Microscopically, the grains appear as coarsely lobulated granules covered with club-like projections. Histologically, the botryomycotic grains are surrounded by a granulomatous infiltrate of histiocytes, plasma cells, lymphocytes, giant cells, and neutrophils. The exact composition of the infiltrate depends on the stage of the infection. The grains may have an eosinophilic staining peripheral border (Splendore-Hoeppli material).[1,2,8] It is from these granules that botryomycosis gets its name.[1,2,8] The bacterium most commonly isolated from lesions is *S aureus*, but multiple species of bacteria have been isolated in some cases of botryomycosis, either singularly or in various combinations.[2,11,12]

The pathogenesis of botryomycosis is unclear, but various theories, mostly concerning abnormal host responses to common bacterial agents, have been postulated.[4,5,13,14] Certain strains of mice are more susceptible to botryomycosis, which suggests a genetic predilection[14,15]; immunodeficiency may play a role in botryomycosis, as is suggested by the disease in humans with HIV infection and chronic granulomatous disease.[3,16]

Definitive diagnosis of botryomycosis requires histologic evaluation and use of special stains.[1] Differential diagnoses should include any disease that can result in a pyogranulomatous lesion, such as actinomycosis, nocardiosis, eumycotic mycetoma, systemic mycoses, chronic bacterial abscesses, and foreign body reactions.[1,17,18] Curative treatment of botryomycosis requires complete surgical excision of the lesion followed by appropriate antibiotic treatment.[13,17,18] It is believed by some authors that botryomycosis is more common than is apparent by documented cases.[1,18] It is likely that botryomycosis is either misdiagnosed or undiagnosed, as it requires extensive laboratory analysis for a definitive diagnosis. Many cases may go undiagnosed because tissue is not submitted for histologic evaluation.[1,18]

This case was a diagnostic challenge because the dog resisted examination, history was obtained through a third party not fully familiar with the patient, and the lesion had several atypical features. The staphylococcal infection was most likely responsible for this dog's clinical signs on initial evaluation but appeared to respond to antibiotic treatment. When the dog was examined because of acute onset of ataxia, differential diagnoses high on the list were trauma, toxin, vestibular disease, and meningitis. Because the dog had been maintained under controlled conditions, toxin exposure was deemed unlikely. On physical examination, no evidence of trauma could be found, including tenderness on palpation of the hip and lumbar area. The dog did not have a history of hip dysplasia that would have suddenly been exacerbated and had been previously examined radiographically for hip conformation. While laboratory results were pending, the dog was treated empirically for both infectious disease and intervertebral disk disease, as these seemed to be the most likely rule-outs. Although considered, a spinal tap could not be performed initially because of the dog's temperament, and subsequent findings did not seem to necessitate the procedure.

The high WBC count put a toxic septicemia high on the list of rule-outs for the cause of the ataxia, but the source of the infection was still unknown. Another diagnostic procedure considered was bacteriologic culture of a blood sample, but because an abdominal mass was palpated during urine collection, this was not performed. If the dog had been easier to handle, this mass

probably would have been found on examination when the dog was initially examined for the ataxia. Five months earlier, when the dog was evaluated because of gastrointestinal problems, the abdomen had been carefully palpated (the dog had allowed for it at that time), and no abnormalities were detected. Although no firm link to this episode was documented, it was suspected that some associated event, possibly an intestinal perforation, may have precipitated the lesion.

[a]Laster CP, Mississippi Board of Animal Health, Veterinary Diagnostic Laboratory, Jackson, Miss.
[b]Antech Diagnostics, Memphis, Tenn.

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