Sudden Death in a Rottweiler Puppy with Myocardial Yersiniosis

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KEY WORDS
Myocarditis, Yersinia enterocolitica, dog.

SUMMARY
A previously healthy 4-months-old male Rottweiler puppy collapsed and died shortly after exercise. Post mortem examination revealed a large area of transmural chronic active myocarditis in the lateral wall of the right ventricle. Yersinia enterocolitica was isolated from this unusual cardiac lesion. The puppy also had microscopic portal hepatitis. It is speculated that these lesions resulted from random localisation from a clinically silent yersinial bacteraemia.

INTRODUCTION
Myocarditis in animals is most commonly a sequel to haematogenous infection of the heart, and a variety of specific causes (viruses, bacteria, protozoa) are recognised. In dogs, myocarditis associated with parvovirus infection was common at one time. In contrast, bacterial myocarditis of dogs is not commonly recognised, and would not have a high index of suspicion in dogs with signs of heart failure.

This case report is of a young dog that died suddenly and unexpectedly with active myocarditis from which Yersinia enterocolitica was isolated.

CASE REPORT
A 4-months-old male Rottweiler puppy, with no previous clinical history, had been seen by a veterinary surgeon only when examined prior to routine vaccination one month earlier. At that time he was judged to be healthy, and had no adverse reaction after vaccination against canine distemper, hepatitis, parvovirus and leptospirosis. One month later this puppy was fed his usual evening meal and was taken out for exercise. On returning home the dog suddenly appeared distressed, collapsed and died within a few minutes.

Post mortem examination carried out 19 hours after death revealed a normally grown puppy (body weight 14.75kg) with a gross lesion confined to the heart. The heart was of normal size (136g), with unremarkable atria and left ventricle, valves and great vessels. However, in the upper part of the lateral wall of the right ventricle there was a large protruding discoidal epicardial area (65mm diameter) with a rough erythematous surface (Fig. 1). Cut surfaces of the right ventricle revealed an area of firm, pale, transmural tissue (2cm diameter) that elevated the overlying smooth ventricular endocardium (Fig. 2). Cut surfaces of the transmural mass were even, firm and pale. Measurements of cardiac parameters merely confirmed the increased right ventricular weight, but provided no morphometric evidence of hypertrophy, nor of dilatation. There was no evidence of congestive cardiac failure and no other gross abnormalities were found.

Histological examination of the right ventricle revealed transmural infiltration by neutrophilic leukocytes, macrophages, lymphocytes, plasma cells and activated fibroblasts (Figs. 3 and 4). There was extensive necrosis of inner myocardial fibres. There were no histological lesions in the other parts of the heart sampled (atria, interventricular septum, left ventricular free wall). The only other histological abnormality was in the liver in which there was multifocal portal and capsular accumulation of mixed inflammatory cells, similar to those in the myocardium. The histological diagnoses are chronic active myocarditis and portal hepatitis.

The epicardial surface of the right ventricular lesion was seared and incised with a sterile scalpel. The underlying deep myocardial lesion was swabbed, plated on blood agar and put into brain
heart infusion broth as an enrichment. Bacterial growth on the agar the next day consisted of colonies of non-haemolytic, oxidase-negative, Gram-negative rods. The organisms were identified, using a commercial biochemical kit (Apo 20E), as *Yersinia enterocolitica*. The culture identity was confirmed at a specialist laboratory by reference to current standard taxonomic criteria and further identified as serotype 09, biotype 3.

**DISCUSSION**

This puppy had neither premonitory clinical signs, nor post mortem evidence of congestive heart failure (eg oedema, ascites, vascular congestion), suggesting that death was associated with cardiac dysrhythmia secondary to a large myocardial lesion. Histological examination revealed chronic active myocarditis, and intralesional *Yersinia enterocolitica* was cultured.

No other bacteriological examination was carried out so the origin and route of infection of the heart remain speculative. *Yersinia enterocolitica* is known to be part of the intestinal flora of asymptomatic dogs and has been associated with chronic enteritis in this species. In experimental infection of beagles, *Yersinia enterocolitica* can be shed in faeces for up to three weeks without producing any clinical or haematological abnormalities. Haematogenous dissemination of this organism occurs occasionally in man, and can localise in the heart and liver. It is a matter for speculation that, in the case reported here, there was a silent bowel infection with *Yersinia enterocolitica* followed by secondary bacteraemic localisation in the heart (and possibly in liver) causing fatal myocarditis and cardiac dysrhythmia. An underlying cause of the putative bacteraemia was not apparent from post mortem examination. In man, most cases of yersinial enterocolitis are mild and self-limiting, but infection can be life-threatening, especially in neonates or in immunocompromised patients. The observations in this unusual case are consistent with a standard veterinary text indicating that *Yersinia enterocolitica* exists as a car-
rier state in many species and can cause sporadic enteritis or generalised infections. This report may also add to the suspicion of breed-related immunodeficiencies in the Rottweiler, as suggested by its unusual susceptibility to parvovirus infection and by a case report of persistent neutropenia.

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REFERENCES