Cardiac manifestations of borreliosis in a dog: a case report

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ABSTRACT: In Europe Lyme disease is caused by the spirochete *Borrelia burgdorferi* sensu lato. It presents with a variety of clinical manifestations including heart disease and problems of the nervous system, skin and joints. Lyme carditis occurs in 4–10% of infected humans and is characterized by a plethora of cardiovascular syndromes such as arrhythmia, myocarditis, and pericarditis among others. This article is a case report of a serologically positive (*B. burgdorferi* s.l.) dog, in which the antibody dynamics correlated with clinical, electrocardiographic, and echocardiographic findings of heart disease resembling human Lyme-induced dilated cardiomyopathy (DCM).

Keywords: borreliosis; atrial fibrillation; serology; cardiomegaly; dogs

List of abbreviations

AF = atrial fibrillation; **ALP** = alkaline phosphatise; **ANA test** = antinuclear antibody test; **AV** = atrioventricular; **BID** = twice daily; **CBC** = complete blood count; **CDCD-VFU** = Clinic of Dog and Cat Diseases, University of Veterinary and Pharmaceutical Sciences, Brno; **DCM** = dilated cardiomyopathy; **DNA** = deoxyribonucleic acid; **ECG** = electrocardiography; **IgG** = Immunoglobulin G; **IgM** = Immunoglobulin M; **PCR** = polymerase chain reaction; **SF** = shortening fraction; **SID** = once daily; **TBE** = tick-borne encephalitis; **TID** = three times daily; **VHS** = vertebral scale size

Borreliosis is a zoonotic, tick-borne, bacterial disease of domestic animals and humans caused by a Gram-negative spirochete (*Borrelia burgdorferi* sensu lato), which includes at least three pathogenic genospecies: *B. burgdorferi* sensu stricto, *B. garinii* and *B. afzelii* (Londono et al., 2005). In the Czech Republic the main genospecies that have been detected are *B. garinii* and *B. afzelii* (Kybicova et al., 2009). The clinical form of borreliosis occurs in human beings and domestic animals, especially in dogs, horses and cattle (Burgess et al., 1987; Cohen et al., 1992; Skotarczak et al., 2005). Clinical signs depend on the individual host response and vary widely; however, they are generally rare (Levy and Magnarelli, 1992). Numerous clinical syndromes have been reported including limb and joint disease, neurological, cardiac, ophthalmic and renal abnormalities (Deibener et al., 2001; Skotarczak et al., 2005; Greene and Straubinger, 2006). The involvement of the heart was first described in dogs by Levy and Durray (1988) in North America; their diagnosis was based on clinical, serological and pathological findings. Interestingly, human and primate *B. burgdorferi* infections have been associated with cardiomyopathies in Europe, but not in the United States (Cadavid et al., 2004; Bartunek et al., 2007). This case report describes a borrelia serologically-positive Boxer dog, exhibiting heart disease that improved following antibiotic treatment and cardiac support. To the authors' knowledge, borrelia-induced cardiac manifestations in dogs have not been described in Europe.

Case description

A five-year-old male Boxer dog, weighing 36 kg was presented to the Clinic of Dog and Cat Diseases at the University of Veterinary and Pharmaceutical Sciences (CDCD-VFU), Brno, Czech Republic with a three-day history of lethargy and generalized pain. The dog was at that time on a regime of vaccinations and deworming. The patient had never received a vaccination against borreliosis, and had not travelled outside the country. The initial clinical evaluation revealed only a generalized hyperesthesia (Day 1). Blood samples for complete blood count (CBC) and for establishing the biochemical profile were taken. Serology for borreliosis (Table 1) was also performed and the results sent by request to his private veterinarian. CBC revealed mild normocytic normochromic anaemia (hematocrit: 34%, haemoglobin: 9.9 g/dl, erythrocytes: 4.4 × 10^6/µl; reference range: hematocrit: 37–55%, haemoglobin: 12–18 g/dl, erythrocytes: 5.5–8.5 × 10^6/µl) with anisocytosis and hypochromasia. On Day 36, the patient's private veterinarian treated the dog with carprofen following an episode of lethargy, lameness and swelling of the carpal joint of the right forelimb. According to the owner, the patient's condition seemed to improve within the next few days. On Day 61 the patient had episodes of diarrhoea, anorexia, weakness, fainting, tachycardia and hypothermia, and was treated for an assumed immune-mediated disorder with prednisone, vitamin K_1, and doxycycline (doses unknown). On Day 63, the dog was examined at the CDCD-VFU because of worsening health status. During the clinical examination, signs of circulatory collapse, pale mucous membranes, slow capillary refill time, tachycardia (220 beats per minute), irregular heart rhythm, pulse deficits and hypothermia (37.1 °C) were observed. Tachypnoea (68 breaths per minute), decreased heart sound intensity, intermittent muffled bronchovesicular murmurs and painful abdominal palpation were also present. CBC results showed normocytic normochromic anaemia (hematocrit: 33%, haemoglobin: 10.8 g/dl, erythrocytes: 4.47 × 10^6/µl). The biochemical profile was normal. Serology for borreliosis was also carried out (Table 1). Thoracic radiographs demonstrated mild pleural effusion, cardiomegaly (VHS-vertebral scale size: 12) and a mild bronchial-to-interstitial lung pattern. Electrocardiography (ECG) showed atrial fibrillation (AF) and prolonged QRS complexes (Figure 1). Hospitalization was recommended but the owners declined. The dog was then treated with co-amoxicillin (25 mg/kg SC BID), enrofloxacin (5 mg/kg SC BID), furosemide (6 mg/kg *i.v.* once, followed by 2 mg/kg TID *p.o.*) and digoxin (0.25 mg/m^2 BID *p.o.*).

On Day 64 the patient was hospitalized at the CDCD-VFU because of worsening of the clinical status. According to the owner, the dog recovered from the polypnoea and diarrhoea but the depression and anorexia persisted. The clinical abnormalities observed were lethargy, pale mucous membranes, 8% dehydration, tachycardia, irregular heart rhythm, pulse deficits, ascites, cool limbs and intermittent bilateral hypersensitivity of the facial region. A CBC showed mild normocytic normochromic anaemia (hematocrit: 34%, haemoglobin: 11.6 g/dl, erythrocytes: 4.77 × 10^6/µl) and a slight lymphopenia (0.92 × 10^3/l; reference range 1–4.8 × 10^3/l). The chemical profile showed mild azotaemia (creatinine 261.3 mmol/l and urea 26.4 mmol/l; reference range 30–120 and 2.5–7 mmol/l respectively) and increased activity of alkaline phosphatase (ALP: 2.38 µkat/l; reference range 0.1–1.5 µkat/l). Thoracic radiographs were unchanged. Echocardiography (M-mode) showed a dilated and hypodynamic left ventricle (diameter in systole/diastole: 6.5/5.6 cm. and shortening fraction – SF: 14%) and enlargement of the left atrium (5.4 cm). Investigation of the valves by colour Doppler and pulsed flow Doppler revealed mitral and tricuspidal insufficiency. Abdominal ultrasonography showed free abdominal fluid. Hemocultures, antinuclear antibody test (ANA test), tick-borne encephalitis (TBE) and ana-

Table 1. Results of serological examination for *Borrelia* sp.

Index of positivity	Day 1	Day 63	Day 71	Day 114	Day 122	Day 473
IgM	0.56	2.11*	1.68*	0.78	0.7	0.4
IgG	0.3	4.25*	5.75*	4.11*	3.87*	0.2

Positive: ≥ 1.15; doubtful: 0.85–1.15; negative: ≤ 0.84
*indicates a positive value

plasma serology were negative. Additionally PCR for DNA of *Borrelia* spp. and *Anaplasma phagocytophilum* were negative. During hospitalization the dog received co-amoxicillin (25 mg/kg BID *i.v.*), enrofloxacin (5 mg/kg BID *s.c.*), digoxin (0.25 mg/m^2 BID *p.o.*), furosemide (2 mg/kg BID *i.v.*), enalapril (0.5 mg/kg SID *p.o.*), methylprednisolon (5 mg/kg *i.v.* single dose followed by prednisone 0.5 mg/kg BID *p.o.*) and ranitidine (2 mg/kg BID *p.o.*). Infusion therapy was initiated with saline 0.9% and was continued with Hartmann solution (80 ml/kg/day). The corticosteroid dosage was tapered for four days until the results of borrelial serology were received. During the next five days the dog's clinical condition improved and the owner requested its discharge. Borrelial antibodies were re-checked (Day 71). Therapy continued with doxycycline (10 mg/kg SID for 20 days), digoxin (0.25 mg/m^2 BID *p.o.*), enalapril (0.5 mg/kg SID *p.o.*) and ranitidine (2 mg/kg BID *p.o.* for five days). In the follow-ups done on days 95, 114 and 122 respectively, the patient remained clinically normal; therefore, the heart medication was continued. The anaemia still persisted on Day 95 (hematocrit: 32%, haemoglobin: 10.3 g/dl, erythrocytes: 4.35 × 10^6/µl). On day 114, serology was determined (Table 1) and a subsequent ECG showed conversion to sinus rhythm (Figure 2).

Contact with the patient was lost, until Day 461, when the patient returned, presenting a history of lethargy, orthopnea, and anorexia. Clinically the dog had light pink mucous membranes; auscultation revealed a holosystolic murmur (intensity 2/6 with point of maximal intensity at the mitral valve) with pulse deficits and bilateral crackles throughout the lung fields. An ECG showed AF, while the thoracic radiographs revealed mild pleural effusion, generalized cardiomegaly (VHS 12.9) and pulmonary oedema. The patient was treated with oxygen, furosemide 2–4 mg/kg TID and the current standard cardiac medications. The owner declined further hospitalization. On Day 473, the dyspnoea resolved; however, the lethargy and dysrrhythmia persisted. CBC and biochemical examination of the blood revealed mild azotaemia (creatinine 206.3 mmol/l and urea 15.26 mmol/l). Serology for borreliosis was also examined (Table 1). Diltiazem (1 mg/kg BID *p.o.*) was added to the therapy. On Day 484, the clinical status was unchanged. Pimobendan (0.15 mg/kg BID *p.o.*) was then added to the therapy. Despite the fact that the dog required closer monitoring, further contact with the owner was lost.

DISCUSSION AND CONCLUSIONS

The clinical course manifested by the neurological, orthopaedic and cardiovascular findings, in conjunction with serological data is highly suggestive of a borrelial-induced cardiovascular syndrome.

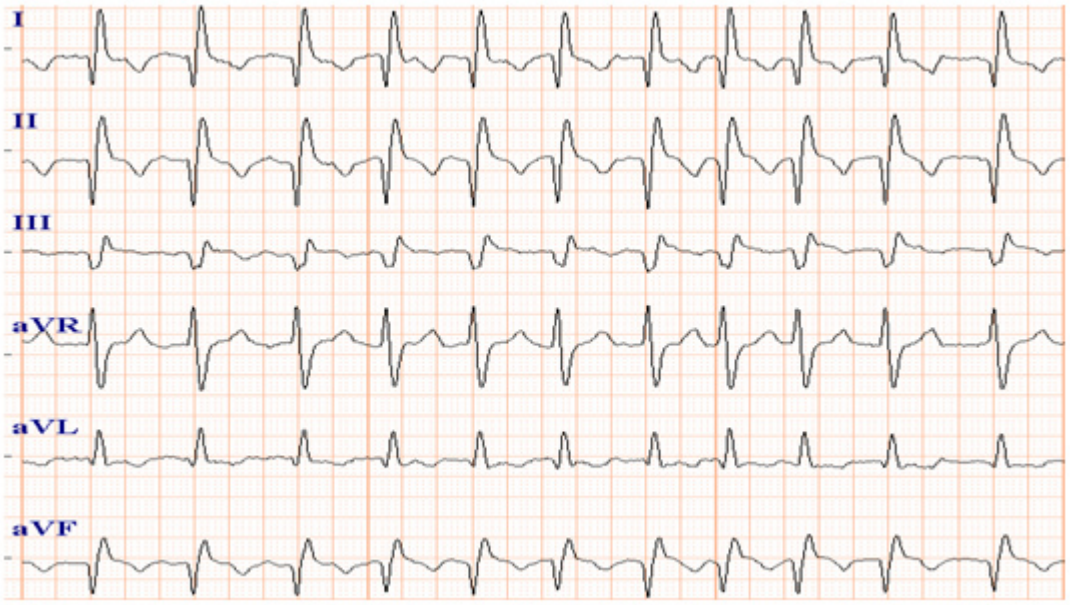

Figure 1. AF with abnormal ventricular activation (partial left bundle branch block-LBBB). Ventricular rate 220 beats/min. Paper speed 50 mm/s

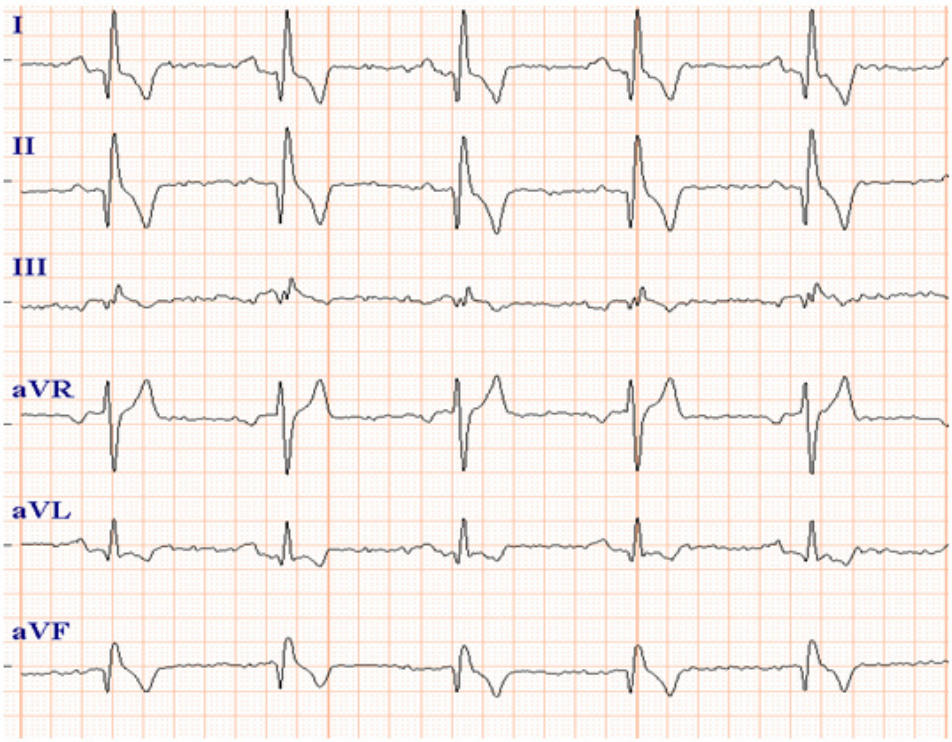

Figure 2. Sinus rhythm. Prolonged P waves and QRS complexes suggesting enlargement of the LA and LV. Split QRS complexes suggest an intraventricular conduction defect (partial LBBB). Paper speed 50 mm/s

Previous studies claim that cardiac manifestations of borreliosis are uncommon in dogs (Nielsen et al., 2002). However, recent information (Schanilec et al., 2010) shows that the incidence of cardiac manifestations could be higher than expected. Studies in experimental animals (mainly rodents) showed that 100% of those infected with *Borrelia* sp. developed myocardial changes. Even though cardiac manifestations have not been described in experimental models of Lyme disease in dogs, it is possible that myocarditis does occur frequently as in other animal models, but without clinical signs (Gasser et al., 1999; Nielsen et al., 2002).

The history of clinical signs and serologic testing are essential for determining whether a current or past infection is responsible for seropositivity. Both in humans and animals the clinical symptoms are divided into three stages (Table 2).

In humans the cardiac involvement in Lyme disease is estimated to be 8–10% in adults (Bateman and Sigal, 2000; Boyle, 2006; Nalmas et al., 2007). The natural incidence of Lyme carditis in veterinary patients has not been established yet. Cardiovascular manifestations usually occur within the first 21 days of exposure to the agent (Lo et al., 2003; Nalmas et al., 2007) and as long as nine months after the onset of disease (Bateman and Sigal, 2000), which is in fitting with our case that had AF, cardiomegaly and decreased SF approximately two months after the onset of neuro-orthopedic signs. The relationship between

Table 2. Relationships between stages of borrelial infection, duration, clinical signs and serological findings

Stage	Clinical signs	Time	Serological positivity	Notes
I	erythema migrans	days/weeks	IgM: 2 weeks after infection	not observed in dogs; high false positive
II	musculoskeletal, cardiovascular, renal and neurological symptoms	months	IgM: up to 2 months IgG: 4–6 weeks to 2 years	observed in dogs
III	recurrent arthritis, chronic heart and neurological disease	years	high IgG	false negative: rare

Modified from: Hovius et al. (1999); Deibener et al. (2001); Nielsen et al. (2002); Green and Straubinger (2006)

heart disease and borreliosis, as established in this clinical case is based on serologic tests and the clinical diagnosis (Lamaison, 2007; Lelovas et al., 2008). Generally, a positive Lyme titre and cardiac clinical signs are present concurrently. Simultaneous measurements of IgG and IgM and paired-sample titres are recommended for the detection of borrelial infection (Greene and Straubinger, 2006). The patient's clinical signs of heart failure (lethargy, oedema, etc) and significant radiographic, echocardiographic and ECG changes were highly correlated with the serological data. The serology suggested an initial increase in IgM, which then decreased over time. This could mean that the infection could start in the space of a few days before or after Day 1, though at this time the patient did not produce antibodies yet. The higher IgG in the 2nd and 3rd stages is consistent with the clinical signs, especially the cardiovascular ones, and is highly suggestive of borrelia-induced heart disease. The gradual decrease in the patient's antibodies may be due to a chronic subclinical phase of the disease, or to the normal dynamics of immunological recovery, as reported in human beings and other animals (Ogrinc et al., 2002; Kannian et al., 2007).

Signs of heart failure are considered to be a consequence of borrelial myocarditis (Gasser et al., 1999). In human medicine, it has been shown that up to 25% of dilated cardiomyopathy (DCM) cases may be attributed to Lyme disease (Seslar et al., 2006; Lamaison, 2007). Unfortunately, studies aimed specifically at determining the association between heart disease and borelliosis has not yet been conducted in dogs. The pathogenesis of borrelial carditis is not completely understood (Londono et al., 2005; Raveche et al., 2005, Lamaison, 2007). It is unknown why some patients develop cardiac symptoms as well as cardiac signs of inflammation, while others do not; however, it is believed that susceptibility could be multifactorial. Many cases of borrelial DCM are believed to be caused by infectious myocarditis, direct tissue invasion by the infectious agent, genetic susceptibility, type of borrelial strain, release of cardiotoxic substances by the microorganism (Herxheimer-Jarisch reaction) and activation of autoimmune processes (Gasser et al., 1999; Saba et al., 2001; Lelovas et al., 2008). There is a positive correlation between spirochetal load and loss of cardiomyocytes which suggests that spirochetes or their products are responsible for the loss of cardiomyocytes, through their ability to induce production of cytokines by macrophages and activate endothelial and other host cells (Londono et al., 2005).

Arrhythmia is the most common manifestation of cardiac borreliosis. It includes varying degrees of atrioventricular (AV) block. We have seen a positive dog with a 1st degree AV block and prolonged QT interval that also suffered from neurological signs and had high levels of borrelial antibodies, which suggest that the pathogen was involved in the arrhythmia (Schanilec et al., 2010). In humans, complete AV block has been reported in as much as 80% of cases (Levy and Durray, 1988; Gasser et al., 1999; Bateman and Sigal, 2000; Lo et al., 2003; Nalmas et al., 2007). Interestingly, in North America there is a predominance of various forms of AV blocks in humans, while in the Czech Republic arrhythmias predominate (Bartunek et al., 2007). Other conduction disturbances associated with Lyme disease are rare, but can include sinus arrhythmia, sinus bradycardia, wandering atrial pacemaker, ectopic atrial bradycardia, AF, bundle branch blocks and ventricular ectopy (Appel, 2003; Begon, 2007; Lamaison, 2007; Nalmas et al., 2007; Lelovas et al., 2008).

The reported dog showed acute signs of heart disease including AF, cardiomegaly and decreased SF. AF is the most common arrhythmia in dogs with heart disease. Conversion of AF to sinus rhythm is considered unusual in large breed dogs with advanced heart disease (Saunders et al., 2009). Thus, it is striking that following administration of antibiotics and cardiovascular support the AF converted into sinus rhythm. This can be explained, at least partially, by the fact that the arrhythmia caused by Lyme disease is generally self-limiting, and responds to parenteral antibiotics; therefore, permanent pacing is rarely needed (Saba et al., 2001; Lo et al., 2003; Boyle, 2006; Nalmas et al., 2007).

Other reported ECG findings in Lyme disease include left axis deviation, ST segment and T-wave abnormalities, prolongation of the QT interval and shorter duration of Q and deep S waves (Deibener et al., 2001; Appel, 2003; Seslar et al., 2006; Begon, 2007; Nalmas et al., 2007). Other cardiovascular syndromes caused by borreliosis include myocarditis, DCM (Gasser et al., 1999; Cepelova, 2008; Lelovas et al., 2008), congestive heart failure, pericarditis (Gasser et al., 1999; Deibener et al., 2001), aortic aneurism and coronary artery disease (Gasser et al., 1999; Londono et al., 2005; Lelovas et al., 2008).

Although an echocardiogram is likely to show normal myocardial function in acute Lyme dis-

ease (Bateman and Sigal, 2000; Lo et al., 2003; Manzoor et al., 2008), it may be helpful in chronic Lyme carditis for monitoring the development of cardiomyopathy. However, there is controversy as to whether long-standing DCM is associated with chronic *B. burgdorferi* infection (Manzoor et al., 2008). The echocardiographic changes seen in our patient can be compared with long term borrelial-induced carditis (including left ventricular dysfunction and cardiomegaly); however, it is not possible at this point to differentiate them from idiopathic DCM, to which Boxers have a predisposition (Sisson et al., 1999). We assume that these changes were caused by borrelia since all of them, including the haemodynamics, AF conversion, and the highly acute serological findings, resolved quickly following antibiotic therapy. In our experience, dogs suffering from acute signs of heart failure normally require more time to stabilize, especially when managed as outpatients.

Even though follow-up echocardiograms were not performed, based on the clinical improvement, it is likely that the function of the left ventricular was recovered. Overall, the prognosis of Lyme carditis is very good, although recovery may be prolonged. To date, only one human patient is reported to have died from a borrelia-induced heart disease (Deibener et al., 2001).

The patient's serum was also evaluated for TBE and anaplasmosis, both of which were negative because several tick-borne diseases can occur concurrently (Klimes et al., 2001; Lelovas et al., 2008; Kybicova et al., 2009). The patient was also negative for ANA antibodies. Systemic lupus erythematosus can result in a plethora of clinical signs, including musculoskeletal, neurological and cardiovascular manifestations (mainly pericarditis and myocarditis) (Brehm et al., 2007) and therefore should routinely be tested for in patients showing systemic or organ inflammatory diseases (Batalla et al., 1999; Brehm et al., 2007).

Anaemia and liver disease are not commonly seen in Lyme borreliosis (Nielsen et al., 2002; Greene and Straubinger, 2006); however, they can result from the suppression of bone marrow, bacteraemia or hepatitis (Greene and Straubinger, 2006). Also, passive congestion due to heart disease and to a lesser degree systemic inflammation and anorexia, could lead to an elevation in serum ALP.

Antimicrobial therapy is the mainstay of treatment for Lyme disease. An improvement in the animal's condition is typically expected within 48 hours after the initiation of treatment (Nielsen et al., 2002). In our case, clinical improvement was evident after one week of treatment. It is believed that stabilization of the heart failure prolonged the recovery. No treatment has been shown to reduce or prevent the development of Lyme carditis; however, mild and severe forms are generally treated with oral and intravenous antibiotics, respectively (Cadavid et al., 2004). Furthermore, recent investigations revealed that antibiotic therapy does not eliminate the organism. This may be useful in explaining why most animals treated for Lyme disease persistently have antibody titres against *B. burgdorferi* (Nielsen et al., 2002) and why the patient after almost one year of clinical stabilization of the heart disease relapsed with similar signs. Also, the duration of heart disease before antibiotic treatment is initiated may play a crucial role in the clinical outcome. It is unlikely that a long standing heart disease can be reversed by antibiotic treatment since there are already significant structural alterations of the myocardium (Gasser et al., 1992). Certain cephalosporins (ceftriaxone and cefotaxime), amoxicillins, doxycycline and azithromycin are among the most effective antibiotics against the spirochete, especially when they are applied at an early stage of infection. Treatment is usually continued for 30 days (Nielsen et al., 2002). In some cases where the antibiotic treatment is ineffective by itself, other medications such as steroids or salicylates have been used in the context of severe AV block though with unproven benefits. It should be mentioned that steroids have shown a detrimental effect by causing the recurrence of neurological and orthopaedic symptoms when withdrawn (Batalla et al., 1999; Straubinger et al., 2000; Saba et al., 2001). In our patient, corticosteroids were used initially to treat a suspected immune-mediated disease, but were gradually discontinued once Borrelia was confirmed. It is possible that their administration contributed to the patient's deteriorating condition from day 60 to 65 and was connected with ther recurrence of clinical signs after 411 days; corticosteroids can increase the deposition of IgG and IgM in the heart by persistent higher spirochetal load and impaired complement activation that is required for efficient spirochetal elimination (Cadavid et al., 2004).

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