



Research Article

Further Insights into the Clinical Aspects of *Angiostrongylus vasorum* Natural Infection in Symptomatic and Asymptomatic Dogs

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Abstract

Canine angiostrongylosis is a cardiopulmonary disease emerging in Europe, which can be fatal if left untreated. An early diagnosis and appropriate treatment is auspicious, not only in symptomatic dogs because it may lead to a complete clinical resolution, but also in asymptomatic dogs to reduce the risk of parasite importation in new areas suitable for parasite establishment. The aim of the present work is to investigate the different clinical and paraclinical findings in both symptomatic and asymptomatic dogs naturally infected by *Angiostrongylus vasorum*. Twelve dogs were included in the study. Diagnosis was made by means of larval morphological identification on faecal samples. Pathological paraclinical findings were registered both in symptomatic and asymptomatic dogs. In particular, the increase in β -globulin fraction at serum protein electrophoresis and radiographic alterations were reported as useful findings to increase the suspicion of infection in asymptomatic dogs.

Keywords

Angiostrongylosis; Asymptomatic infection; Dogs; Clinical pictures; Paraclinical findings

Introduction

Canine angiostrongylosis is an emerging disease whose expansion is probably due to climatic factors, to the presence of foxes in urban areas that have facilitated the spread of the parasite, or more simply, to more accurate diagnostic methods [1]. In Italy, data on the prevalence of canine angiostrongylosis are scant [2], however, recent cases suggest a spread of the infection [3-6].

Despite the parasite *Angiostrongylus vasorum*, defined as the "great imitator" [7], is responsible for very different clinical pictures, it is generally associated with respiratory, neurological and coagulative disorders [1]. The severity of symptoms can greatly vary, ranging from severe to asymptomatic forms [7]. The identification of asymptomatic dogs is an important task to reduce the risk of parasite

importation in new areas suitable for parasite establishment [8].

Laboratory [9,10], radiographic [11,12], echocardiographic [13-15], and thoracic computer tomographic [16,17] abnormalities are not-specific. The gold standard to achieve the diagnosis still remains the first-stage larvae (L_1) detection, using the Baermann technique in faecal samples, preferably collected on three consecutive days. Recently, both serological and PCR tests have been developed, but are either not commercially available or not yet fully validated [18]. As treatment milbemycin oxime and a combination of moxidectin with imidacloprid in a spot-on formulation (Im/Mox) are licensed, while fenbendazole, which is widely used in naturally infected dogs and appears to be effective, and is unlicensed [19].

It is known that the infections with *A. vasorum* are of great importance in veterinary medicine (potentially fatal if left untreated), thus any additional information that may help to increase the suspect of infection to reach a tempestive diagnosis may be of value. On this view, the aim of the present work is to investigate the clinical and paraclinical changes of twelve naturally *A. vasorum* infected symptomatic and asymptomatic dogs. Furthermore, the reverse to normal of clinical and paraclinical findings after therapy are reported, when available.

Materials and Methods

Dogs were referred to the Clinical Unit of the Veterinary Faculty of Bari (Southern Italy). Diagnosis was reached by means of *A. vasorum* L_1 detection in faecal samples on direct smears, or using Baermann test; in one dog, larvae were also found in pleural effusion (Case 1). Identification was possible, following the morphological and morphometric parameters [20]. Asymptomatic dogs were revealed because Baermann test was introduced as routine test during clinical practice. Dogs were also tested for *Dirofilaria immitis* infection, resulting negative.

A blood sample was collected for haematology, blood coagulation profile, biochemical analysis, and serum protein electrophoresis in all animals. Thoracic radiographs and echocardiography were performed.

Specific therapy was administered choosing fenbendazole (Panacur, Intervet, Animal Health), and/or Im/Mox (Advocate spot-on, Bayer), according to the severity of symptoms. Fenbendazole was used in monotherapy, as previously reported (50 mg/kg, PO daily) [21], in case of severe clinical symptoms (i.e. dyspnea), while Im/Mox was used in monotherapy, with three applications at 15 days interval in asymptomatic/paucisymptomatic (i.e. sporadic coughing) dogs, or in combination with fenbendazole at 25 mg/kg, PO, once a day. No other drugs were administered.

According to owner availability, clinical and paraclinical changes were monitored till normalization of clinical-pathological alteration, and/or till the end of treatment.

The animals were being kept under their usual housing conditions; they were being handled and sampled with the owners' consent, and with the approval of the Ethical Committee of the Faculty of Veterinary Medicine of the University of Bari.

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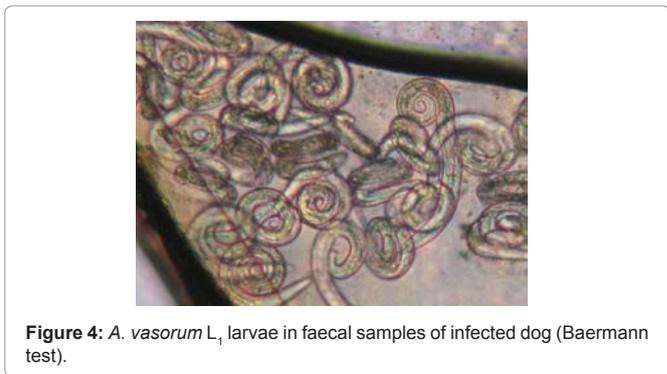
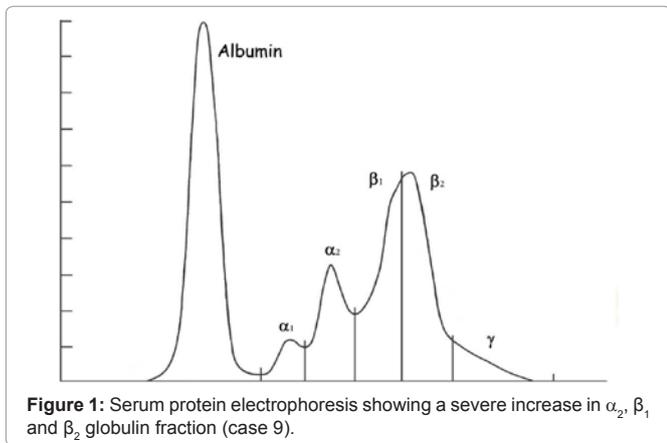
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Results

Dogs described in this study were aged between nine months and 12 years (Table 1). All dogs were from central-southern Italy and never moved from. Presenting complaint was heterogeneous (Table 1), and five cases were presented for routine control visit and one case for eye examination, to investigate a long lasting reduction of virus. Thoracic auscultation was normal in seven cases, while in the other animals, muffling of heart sounds (2/12), rales (3/12), wheezing (1/12), and loud bronchovesicular sounds (2/12) were detected (Table 1).

The complete blood count showed anemia (5/12), leukocytosis (4/5), eosinophilia (6/12), and basophilia (3/12) (Table 2). Serum protein electrophoresis showed a variable increase in β -globulin fraction (Figure 1) in all animals, except for two dogs (Table 2). The biochemical examination showed non-specific and non-uniform alterations, but in four cases, an increase in phospho-creatine kinase (CK) was found. Primary hemostasis disorders, characterized by thrombocytopenia, were detected in three cases, associated or not to alterations of clotting profile that were registered in 6 cases.

Thoracic radiographs showed pathological findings in both symptomatic and asymptomatic dogs, except for one asymptomatic dog (Table 1). In particular, in four asymptomatic dogs, thick radiodense circles or S shaped radiodensities were observed on radiographs (Figures 2 and 3). Among symptomatic dogs, a variable



pulmonary pattern was registered (Table 1). Enlargement of the right heart was observed in two cases, associated to dilatation of pulmonary artery trunks in one of them. The echocardiographic examination was normal in all dogs, except for 2 cases 3, where signs of mild and severe pulmonary hypertension were registered, respectively.

All dogs were treated; fenbendazole was administered in monotherapy in five cases; the Im/Mox was administered in monotherapy in five cases, and in combination with fenbendazole (25 mg/kg, PO, once a day for 21 days), in other two cases (Table 1).

Clinical pictures significantly improved in all symptomatic dogs, after 1-2 weeks of treatment (Table 1). Fecal monitoring was not available in 2 animals; all the other dogs reached negative results between 3 and 8 weeks after treatment, except for two dogs remaining positives. The radiographic monitoring showed an improvement in quite all dogs, while the reverse to normal of radiographic abnormalities, when reached, was registered at different times (Table 1). The radiodensities of asymptomatic dogs were no longer visible after two months of treatment in two cases. In cases 3 and 6, radiographic controls were available only at the end of treatment, and in other two cases (case 4 and 5), radiographic controls were totally unavailable. The laboratory monitoring after treatment was incomplete and fragmentary, available in only 6 out of 12 dogs, and the reverse to normal was variable (Table 2).

Discussion

In this study, different clinical presentations were associated with the presence of *A. vasorum* in dogs, ranging from asymptomatic

Table 1: Case load of *A. vasorum* naturally infected dogs enrolled in the study.

Case	1	2	3	4	5	6	7	8	9	10	11	12
Signaling	Male Castrated 1 year Mixed breed 26 Kg	Female 1 year Épagneul breton 18 Kg	Female sterilized 3 years Mixed breed 14 Kg	Male 1 year Mixed breed 12 Kg	Female 2 years Mixed breed 22 Kg	Male 3 years Spinone 36 Kg	Female sterilized 4 years Mixed breed 31 Kg	Female sterilized 12 years Mixed breed 26 Kg	Male 9 months Mixed breed 15 Kg	Female 6.5 years Labrador 29Kg	Male 4 years Mixed breed 31 Kg	Male 2 years German Shepherd 32 Kg
History and clinical complaint	Cough; fever; depression; and inappetence of 1 month duration + Respiratory crisis in the last 2 days	Exercise intolerance and cough of 2 months duration	Cough for 5 months + Progressive abdomen distention from 2 months	Capricious appetite and cough from 2-3 months + Haemoptysis	Severe cough from 2 months	Gradual reduction of visus + sporadic cough	Routine visit	Routine visit	Epistaxis; hemoptysis cough and depression of 2 months duration + inappetence and unilateral hyphema	Routine visit	Routine visit	Routine visit
Clinical findings	Dyspnea + fever (hemotorax)	Expiratory dyspnea	Abdominal effusion	Fever + polypnea + hemothoma after blood collection	Cough induced after stimulation	Cough induced after stimulation	Normal	Normal	Unilateral hyphema + Pale mucous membranes	Normal	Normal	Normal
Thoracic auscultation	Muffling of the heart sounds + loud bronchovesicular sounds	Rales and wheezing	Few rales	Muffling of the heart sounds in the left cardiac area	Normal	Rales on stimulation of cough + loud bronchovesicular	Normal	Normal	Normal	Normal	Normal	Normal
Thoracic radiography	Moderate bilateral pleural effusion + Diffuse interstitial pulmonary pattern + Alveolar pattern in the cranial left lobe	Diffuse bronchial-interstitial pulmonary pattern + Dilatation of the right heart	Diffuse interstitial-bronchial pulmonary pattern + pulmonary artery trunks + Dilatation of the right heart	Alveolar pattern in the diaphragmatic left lobe	Diffuse interstitial pulmonary pattern	Diffuse interstitial pulmonary pattern	Circular areas of radiopacity	Circular areas of radiopacity	Diffuse interstitial-bronchial pulmonary pattern	Circular areas of radiopacity	Circular areas of radiopacity	Normal
Diagnosis	L1 in the pleural effusion and in faeces	L1 with Baermann test	L1 on direct fecal smear	L1 on direct fecal smear	L1 on direct fecal smear	L1 with Baermann test	L1 with Baermann test	L1 with Baermann test	L1 on direct fecal smear	L1 on direct fecal smear	L1 with Baermann test	L1 with Baermann test
Therapy	Fenbendazole 25 mg/kg; PO twice a day for 21 days	Fenbendazole 25 mg/kg; PO twice a day for 21 days	Fenbendazole 25 mg/kg; PO twice a day for 21 days	Fenbendazole 25mg/kg; PO; once a day for 21 days + Im/Mox every 15 days for 3 times	Fenbendazole 25mg/kg; PO once a day for 21 days + Im/Mox every 15 days for 3 times	Fenbendazole 25 mg/kg; PO twice a day for 21 days	Im/Mox every 15 days for 3 times	Im/Mox every 15 days for 3 times	Fenbendazole 25 mg/kg; PO; twice a day for 21 days	Im/Mox every 15 days for 3 times	Im/Mox every 15 days for 3 times	Im/Mox every 15 days for 3 times
Clinical recovery	After 1 week	After 1 week	After 2 weeks-	After 1 week	/	After 2 weeks-	/	Occasional cough	1 week; (ocular problems solved in 2 months)	/	/	/
Remission of radiography signs	N.K.	5 months	N.K.	N.K.	N.K.	N.K.	N.K.	3 months	N.K.	2 months	2 months	/
Radiography follow up	Two weeks: persistence of interstitial pulmonary pattern	1 month 2 months 5 months	Three weeks: persistence of a interstitial pulmonary pattern	N.A.	N.A.	1 month: persistence of diffuse interstitial pulmonary pattern	2 months	2 months 3 months 6 months	4 months: persistence of mild interstitial-bronchial pulmonary pattern	2 months	2 months	/

N.K.: Not Known; N.A.: Not Available

Table 2: Abnormal laboratory findings registered at diagnosis and in follow up post treatment in the 12 dogs included in the study.

		Complete blood count	Biochemical	Globulin fraction increased	Clotting profile
CASE 1	T0	↓RBC - ↓Hgb - ↓Hct - ↑WBC ↑NEU - EOS - ↑BAS - ↓PLT	↑Pt - ↑AST - ↑CK - ↑LDH - ↑Amylase - ↑Globulins - ↓A/G	↑α ₂ - ↑β ₂	↑PT - ↓AT - ↑FDPs - ↓Fibrinogen
	T28	↑EOS	Not evaluated	Not evaluated	Not evaluated
CASE 2	T0	↑RBC - ↑Hgb - ↑Hct - ↑WBC - ↑EOS - ↑LYM	↑AST - ↑CK - ↓Glu	↑β ₁ - ↑β ₂	Not evaluated
	-	Not evaluated			
CASE 3	T0	↓Hgb - ↓Hct ↑WBC - ↑NEU	↑GGT - ↑Urea - ↑Chlorides	↑α ₂ - ↑β ₂	Not evaluated
	T21	↑WBC - ↑NEU - ↑EOS	↑Urea	↑β ₂	Not evaluated
CASE 4	T0	↑EOS - ↓PLT	↑Pt - ↓A/G - ↑Prot C	↑β ₁ +β ₂	↓AT - ↑FDPs - ↑D-D ↑Fibrinogen
	-	Not evaluated			
CASE 5	T0	↓RBC - ↓Hct - ↑EOS - ↑BAS	↓ALT	↑β ₁	Normal
	-	Not evaluated			
CASE 6	T0	↓Hct ↑BAS	↑Pt - ↑Glob - ↓A/G - ↑Urea - ↑Crea - ↑LDH - ↑Amylase	↑β ₁	Not evaluated
	-	Not evaluated			
CASE 7	T0	↑EOS	Normal	↑β ₁ +β ₂	↓aPTT - ↓D-D
	T53	↑EOS - ↓PLT	Normal	↑β ₁	Not evaluated
CASE 8	T0	↓WBC ↓NEU	↑Mg ↑Crea	↑α ₂ - ↑β ₁ +β ₂	↓AT - ↑Fibrinogen
	T48	↓WBC	↑ALP - ↑LDH - ↑Chlorides	↑α ₂	Not evaluated
	T90	normal	Not evaluated	Not evaluated	Not evaluated
CASE 9	T0	↓RBC - ↓Hgb - ↓Hct - ↓PLT	↑Pt - ↓A/G - ↑CK - ↑P	↑α ₂ - ↑β ₁ - ↑β ₂	↑aPTT - ↑PT - ↓AT - ↑FDPs - ↑D-D - ↓Fibrinogen
	T35	Normal	↑ALP	↑α ₂ - ↑β ₂	Normal
CASE 10	T0	Normal	Normal	Normal	Not evaluated
	T45	Normal	Not evaluated	Normal	Not evaluated
	T152	Normal	↑ALP	↑α ₁ - ↑α ₂	↓D-D
CASE 11	T0	Normal	Normal	↑γ	Normal
	T45	Normal	Not evaluated	↑γ	Not evaluated
CASE 12	T0	↑WBC - ↑NEU - ↑EOS	↑CK	↑β ₁ +β ₂	↓FDPs - D-D
	-	Not evaluated			

RBC: Red Blood Cells; Hgb: Hemoglobin; Hct: Hematocrit; WBC: White Blood Cells; NEU: Neutrophils; EOS: Eosinophils; BAS: Basophils; LYM: Lymphocytes; PLT: Platelets; Pt: Total Protein; AST: Aspartate Aminotransferase; CK: Creatine Kinase; LDH: Lactate Dehydrogenase; A/G: Albumin Globulin ratio; Glu: Glucose; GGT: Gamma-Glutamyl Transpeptidase; Prot C: Protein C; ALT: Alanine Aminotransferase; Crea: Creatinine; Mg: Magnesium; ALP: Alkaline Phosphatase; PT: Prothrombin Time; =: Antithrombin III; aPTT: activated Partial Thromboplastin Time; FDPs: Fibrin Degradation Products; D-D: D-Dimer

to severe forms. Baermann test has been used to reach diagnosis because it is a simple, widely available, economic and specific test. The occasional detection of *L*₁ (Figure 4) in the feces of five dogs referred for a routine control visit, suggests that faecal samples can reveal asymptomatic subjects [7]. The identification of those subjects that could act as diffuser of infection is an important task to break the cycle of the parasite, by using successful therapy. From data of this study, pathological paraclinical findings have been registered both in symptomatic and asymptomatic dogs. The repetition of the same pathological changes supports their association with the infection.

In particular, the increase in β-globulin fraction at serum protein electrophoresis and the serpiginous/circular areas of radiopacity on thoracic radiographs were documented as useful findings, that may help to reveal asymptomatic infections. It is quite common during clinical practice to perform routine laboratory tests on healthy dogs, during annual control visit or before small surgery (hysterectomy, castration, dental cure etc). Moreover, in some countries, electrophoresis is included in the routine laboratory tests because of the endemicity for canine vector borne diseases (i.e. Southern Italy). Thus, it assumes a great importance to have information on

that changes that may raise the suspicion of *A. vasorum* infection in asymptomatic dogs.

Angiostrongylosis is characterized by a highly variable clinical picture, and the absence of characteristic findings on clinical examination. In this study, the hemothorax (Case 1), the hemoptysis associated to the formation of a large jugular hematoma after blood collection (Case 4), and the unilateral hyphema may be caused by the primary and secondary hemostasis alterations [22-24], and it has been suggested that the presence of haemostasis alterations is associated to poor prognosis [25]. Causes of the coagulopathy associated with angiostrongylosis are not fully known, but chronic Disseminated Intravascular Coagulation (DIC), as well as an immune-mediated thrombocytopenia has been proposed as possible mechanisms [26]. It is important to note that angiostrongylosis should be included in the differential diagnosis, when alterations of haemostasis are registered at the clinical examination. The abdominal effusion of case 3 is the consequence of the severe pulmonary hypertension, probably caused by the presence of adult worms responsible for thrombosis of the pulmonary vessels [13].

Abnormalities in complete blood count and serum biochemistry have frequently been described, but are not specific. The most common clinico-pathologic changes are hyperglobulinemia, eosinophilia and anemia [21]. Chapman et al. [21] reports of 70%, with an increase in serum globulin, and this is very similar to the report in this study. Anyway, the report of the β -globulin fraction increase in this study is more specific than previously reported, and it is interesting to note that this pathological finding has been frequently registered also in asymptomatic dogs (3/5). The beta globulins include transferrin, lipoproteins, complement, and immunoglobulins; these are increased with inflammatory, neoplastic, and hepatic disease. In a recent retrospective study on serum protein electrophoresis, the increase of β_2 globulin fraction has been associated with the diagnosis of angiostrongylosis in 3 dogs; an inflammatory/infectious, and/or vascular cause was suggested [27].

Eosinophilia, observed in six cases, should increase the suspicion of the disease, but eosinophils values in the reference range, as observed in the other cases, does not exclude the diagnosis [28]. An increase in CK has been registered in four cases; this finding is reported as an alteration in kinase-myocardial band isoenzyme (CK-MB), possibly related to acute cardiac injury [9]. Thoracic radiographic findings are largely described in the course of angiostrongylosis [11,12,21], with the possibility of abnormalities also in asymptomatic dogs [11]. The particular aspect of the serpiginous/circular areas of radiodensities described in asymptomatic dogs (Figures 2 and 3), could be attributed to fistulas created during larval migration from the pulmonary capillaries into the alveoli, or to bronchiectasis with bronchial walls markedly thickened and misshapen. The latter hypothesis was not supported by the typical aspect of bronchiectasis (i.e., loss of the normal tapering of the bronchial walls) on lateral radiographic view. A not better defined bronchial pattern has been documented in 60% of dogs in natural condition [12], differently in a recent work, no bronchial changes have been documented by using computer tomography under experimental conditions [16], but it is assumed that experimental infection is not identical to natural infection. Furthermore, the association with asymptomatic cases suggests the possibility that these findings represent the earliest

changing on thoracic radiographs before the onset of clinical disease, and the disappearance of these alterations after treatment in two dogs support the correlation with canine angiostrongylosis.

The persistence of some paraclinical alterations after therapy (eosinophilia, and a mild increase in β -fraction apparently persisted in three dogs; reverse to normal of radiographic findings was not available in all dogs), probably depends on the variability of follow up. On the other hand, studies have not definitively shown whether any of the specific used medications completely clear infection [29], thus, the persistence of some alteration is possible.

The detection of L_1 in a dog of 12 years reveals the receptivity to infection also in old dogs, although a greater risk of infection is reported in young subjects, because of their high predation activity that facilitate the ingestion of the intermediate host [21]. In one dog, L_1 were found also in the pleural effusion; it was assumed that L_1 passed from lung tissue to the thoracic cavity, following lung rupture occurred concurrently with acute respiratory distress observed by the owners [3].

A. vasorum infection can be fatal, thus an early diagnosis and appropriate treatment is auspicious, to avoid the onset of potentially lethal lesions. Although none of clinico-pathological findings are specific, a combination of these findings could increase the index of suspicion, also in asymptomatic dogs. In this view, the increase in β -globulin fraction at serum protein electrophoresis, and the circular areas of radiopacity on thoracic radiographs could be useful findings. Given the geographical difference between the previously reported case series from Italy [6], this study also documents the apparent spread of disease in Italy.

Conflict of Interest

None of the authors of this paper has a financial or personal relationship with other people or organizations that could inappropriately influence, or bias the content of the paper.

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