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Aelurostrongylus abstrusus induced pneumonia in cats: pathological and epidemiological findings of 38 cases (1987-1996)

Pneumonia induzida por Aelurostrongylus abstrusus em gatos: achados patológicos e epidemiológicos de 38 casos (1987-1996)

Selwyn Arlington Headley

Abstract

A retrospective study was designed to obtain epidemiological information and describe the main histological alterations of Aelurostrongylus abstrusus induced pneumonia in cats submitted for routine necropsy during 1987-1996 at the Federal University of Santa Maria (UFSM), Santa Maria, Brazil. Prevalence varied from 5.9 to 25%, sexual predominance was not observed, infestation was more predominant in younger than in older cats, and only domestic short hair cats were diagnosed positive for A. abstrusus. Granulomatous pneumonia and hyperplasia and hypertrophy of the smooth muscles of pulmonary arteries and parenchyma were the predominant histological lesions observed, and occurred more frequently in cats that were less than 4 years old. The pathogenesis of the histological alterations is also described.

Key words: Cats, Aelurostrongylus abstrusus, epidemiology, histopathology

Resumo

Um estudo retrospectivo foi realizado para obter-se dados epidemiológicos e descrever as principais alterações histológicas na pneumonia induzida por Aelurostrongylus abstrusus em gatos submetidos para necropsia de rotina durante 1987-1996 na Universidade Federal de Santa Maria (UFSM), Santa Maria, Brasil. A prevalência variou entre 5,9 a 25%, predominância sexual não foi observada, a infestação foi predominantemente em gatos jovens em relação aos velhos e somente gatos sem raça definida foram diagnosticados como positivos para a infestação por A. abstrusus. A pneumonia granulomatosa e a hiperplasia e hipertrofia da musculatura lisa das artérias pulmonares e no parenquima pulmonar foram as alterações histológicas predominantes e ocorreram mais frequentemente em gatos com menos de quatro anos de idade. A patogenia das alterações histológicas também é discutida.

Palavras-chave: Gatos, Aelurostrongylus abstrusus, epidemiologia, histopatologia

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Introduction

The lungworm, *Aelurostrongylus abstrusus*, produces severe subclinical granulomatous pneumonia in cats (HAMILTON, 1963; DUBEY; BEVERLEY; CRANE, 1968; DUNGWORTH, 1993), which is frequently confused clinically with other pulmonary infestations (IDIART et al., 1986; HAWKINGS, 1995). Clinical signs, when present, associated with this disease include severe coughing, sneezing, ocular-nasal discharge, and progressive weight loss (HAMILTON, 1963; HAWKINGS, 1995; BOWMAN, 1999); however, most cases are asymptomatic (IDIART et al., 1986; CASTELLANO; VENTURINI, 1993; HAWKINGS, 1995). Severe infestations in which there is cough, dyspnea, and polypnea may terminate fatally (BOWMAN, 1999).

Gross lesions are characterized by yellow, firm, slightly salient, multifocal nodules (1-10 mm in diameter) distributed throughout the pulmonary parenchyma; the sectioned pulmonary surface normally demonstrates variable amounts of creamy exudate (DUBEY; BEVERLEY; CRANE, 1968; DUNGWORTH, 1993). Microscopically, collections of eggs and larvae packed within the alveolar space with a mixed influx of macrophages, eosinophils, and giant cells are observed (DUNGWORTH, 1993). Hypertrophy and hyperplasia of the smooth muscle of the walls of the bronchus, bronchioles, arteries, and alveolar ducts are progressive lesions (DUNGWORTH, 1993), and considered as manifestations of chronic infestation by *A. abstrusus* (HAMILTON, 1963; DUBEY; BEVERLEY; CRANE, 1968).

Infestation by the lungworm *A. abstrusus* in cats is relatively common with worldwide distribution (DUBEY; BEVERLEY; CRANE, 1968). Cases have been described in countries such as Scotland (HAMILTON, 1963), England (DUBEY; BEVERLEY; CRANE, 1968), Australia (COMAN; JONES; DRIESEN, 1981), the USA (LOSONSKY; THRALL; PRESTWOOD, 1983), Turkey (TÜZER et al., 2002), and Argentina (SCHIAFFI et al., 1995). However, there are not many detailed descriptions of the feline lungworm in Brazil. A review of published cases of feline lungworm pneumonia in Brazil revealed a recent description of the occurrence of this parasite in Uberlândia (MUNDIM et al., 2004), a short report of epidemiological findings in Santa Maria (HEADLEY; CONRADO, 1997), and reports of coprological findings in Rio de Janeiro (LANGENEGGER; LANZIERI, 1963), and São Paulo (CAMPEDELLI, 1972; FENERICH; SANTOS; RIBEIRO, 1975).

This article describes the histopathological patterns observed in 38 cases of pneumonia induced by *A. abstrusus* in cats, and presents epidemiological data relative to pathogenesis, breed, age distribution, sexual predominance, and the prevalence of infestation.

Material and Methods

All cases of cats submitted for routine necropsy during January 1987 to December 1996 at the Department of Veterinary Pathology, Federal University of Santa Maria (UFSM), Santa Maria, RS, were reviewed and examined for parasitic forms of *A. abstrusus* (HEADLEY; CONRADO, 1997). The total annual number of cats submitted for routine necropsy was obtained and compared with the number of cats infected by *A. abstrusus* as diagnosed by histopathology. The breeds affected, sexual predominance, age range of cats infected, prevalence of infestation, and the proportion of males to females infected were determined. Three age groups were defined to determine the age distribution of infestation: 1) 0 – 2 years; 2) 2.1 – 4 years; and 3) more than four years of age. The Fischer Test was used to determine a possible sexual predominance between the age groups.

A diagnosis of parasitic feline pneumonia was based on the presence of intralesional parasitic forms of *A. abstrusus*, and marked hypertrophy and hyperplasia of pulmonary smooth muscles.
Aelurostrongylus abstrusus induced pneumonia in cats: pathological and epidemiological... (HAMILTON, 1963; DUBEY; BEVERLEY; CRANE, 1968). The parasitic forms observed in each case were recorded, and gross descriptions of pulmonary lesions were documented.

Histological lesions were re-examined and classified according to the predominant findings observed: 1) severe granulomatous bronchopneumonia, characterized by a mixed influx of alveolar macrophages, eosinophils and giant cells associated with parasitic forms of A. abstrusus; 2) discrete granulomatous bronchopneumonia, where parasitic forms were present but the inflammatory reaction was mild and; 3) adaptive changes, characterized by hyperplasia and hypertrophy of smooth muscle of alveolar septa, bronchioles, bronchus, and the pulmonary blood vessels with and without intralesional parasites.

Results

Pneumonia associated with A. abstrusus was observed in 18.6% (38/204) of the cats submitted for routine necropsy during 1987 to 1996 (Table 1). The annual prevalence of infestation in these cats ranged from 5.9% (1/17 in 1992) to 25% (7/28 and 4/16 in 1988 and 1990, respectively). The seasonal pattern of annual prevalence of A. abstrusus pneumonia observed was characterized by alternate years of peaks and pitfalls occurring from 1987 to 1992; thereafter, a slight increase in prevalence was demonstrated from 1993 to 1996 (Table 1).

Only short hair domestic cats were infected. The age of the cats infested by A. abstrusus varied from 2 months to 10 years. Infestation was predominant in younger (81.4%) than older cats (18.6%). In the evaluated groups, infestation was more frequent in cats that were less than two years old (47.4%; 18/38), followed by those within the 2.1 – 4 year age group (34.2%; 13/38); cats that were older than 4 years represented only 18.4% (7/38) of all cats diagnosed with parasitic pneumonia (Table 2). An apparent sexual predominance was observed, with infestation occurring more in male than female cats, but statistically there were no significant difference (Fischer test; P=0.3285) between sex and the age groups (Table 2).

Table 1. Annual distribution of Aelurostrongylus abstrusus diagnosed by histopathology in cats submitted for necropsy during 1987-1999 at the Department of Veterinary Pathology, UFSM, Santa Maria, Brazil.

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of cats Submitted</th>
<th>Infected</th>
<th>Annual Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987</td>
<td>28</td>
<td>3</td>
<td>10.7</td>
</tr>
<tr>
<td>1988</td>
<td>28</td>
<td>7</td>
<td>25.0</td>
</tr>
<tr>
<td>1989</td>
<td>10</td>
<td>1</td>
<td>10.0</td>
</tr>
<tr>
<td>1990</td>
<td>16</td>
<td>4</td>
<td>25.0</td>
</tr>
<tr>
<td>1991</td>
<td>14</td>
<td>3</td>
<td>21.4</td>
</tr>
<tr>
<td>1992</td>
<td>17</td>
<td>1</td>
<td>5.9</td>
</tr>
<tr>
<td>1993</td>
<td>11</td>
<td>2</td>
<td>18.2</td>
</tr>
<tr>
<td>1994</td>
<td>16</td>
<td>3</td>
<td>18.8</td>
</tr>
<tr>
<td>1995</td>
<td>35</td>
<td>7</td>
<td>20.0</td>
</tr>
<tr>
<td>1996</td>
<td>29</td>
<td>7</td>
<td>24.1</td>
</tr>
<tr>
<td>Total</td>
<td>204</td>
<td>38</td>
<td>18.6</td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Age range (y)</th>
<th>Number of cats</th>
<th>Male</th>
<th>Female</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 2.0</td>
<td>18</td>
<td>11</td>
<td>7</td>
<td>47,37</td>
</tr>
<tr>
<td>2.1 - 4.0</td>
<td>13</td>
<td>7</td>
<td>6</td>
<td>34,21</td>
</tr>
<tr>
<td>&gt;4.1</td>
<td>7</td>
<td>6</td>
<td>1</td>
<td>18,42</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
<td>24</td>
<td>14</td>
<td>100,00</td>
</tr>
</tbody>
</table>

Gross pathology reports described the lungs as enlarged and/or consolidated, with small, multifocal to coalescent, or diffused, irregular, creamish-white, slightly raised areas, distributed throughout the pleural surface of the lungs, forming patchy areas of consolidation. The sectioned pulmonary surfaces
were described as containing caseous, firm, solitary or coalescent nodules of various sizes.

Severe granulomatous verminotic bronchopneumonia was the principal histopathological pattern observed (57.9%; 22/38) (Table 3), followed by smooth muscle reactive changes (26.3%; 10/38), and discrete granulomatous verminotic bronchopneumonia (15.8%; 6/38). In most cases intralesional parasites (eggs and/or larvae) were observed by histopathology (Table 3). However, severe reactive alterations (hyperplasia/hypertrophy) were observed in two cases without infectious forms of the parasite.

**Table 3.** Distribution of principal histological patterns and forms of parasite observed in 38 cases of pneumonia induced by *Aelurostrongylus abstrusus*.

<table>
<thead>
<tr>
<th>Principal histological pattern</th>
<th>Cases</th>
<th>Parasitic forms</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Eggs</td>
</tr>
<tr>
<td>Severe granulomatous bronchopneumonia</td>
<td>22</td>
<td>20</td>
</tr>
<tr>
<td>Adaptive changes</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>Discrete granulomatous bronchopneumonia</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
<td>100</td>
</tr>
</tbody>
</table>

The lesions of granulomatous bronchopneumonia were characterized by packed clusters of larvae and eggs of *A. abstrusus* within alveolar spaces. In most cases the interstitial tissues surrounding these parasitic forms were thickened by the proliferation of Type II pneumocytes associated with mononuclear infiltrate, consisting mainly of lymphocytes, macrophages and, giant cells; eosinophils, few neutrophils, and residual debris were also observed (Figure 1); severe bronchial destruction was observed in a few cases (Figure 2). Reactive changes (hyperplasia and hypertrophy) to the smooth muscle were severe and multifocal, and occurred in the intima and media of arteries and arterioles, the smooth muscle of bronchial glands, bronchus, and bronchiolar epithelium, and the walls of alveoli air spaces (Figure 3); in some cases of severe reactive changes, there was occlusion of the vascular lumen and bone metaplasia (Figure 4). Discrete granulomatous pneumonia was similar to the severe type, except that the inflammatory reaction and tissue damage were minimal.

**Figure 1.** Cat, verminotic granulomatous pattern of bronchopneumonia. There are clusters of eggs (arrow head) and larvae (*) of *Aelurostrongylus abstrusus* within the alveolar space associated with an influx of inflammatory cells.

**Figure 2.** Cat, verminotic granulomatous pattern of bronchopneumonia. Observe mixed (neutrophilic and eosinophilic) inflammatory exsudate (e) within the bronchiole, severe destruction of bronchiole epithelium (b), and destruction of the surrounding peri-bronchiole pulmonary parenchyma by eggs (p) and larvae of *Aelurostrongylus abstrusus*. 
Aelurostrongylus abstrusus induced pneumonia in cats: pathological and epidemiological...

Discussion

In this study the prevalence of A. abstrusus during the evaluated period was 18.6%; similar results were described in Uberlândia (MUNDIM et al., 2004). However, more elevated results were described in the outskirts of La Plata and Buenos Aires, 24.3-30% (IDIART et al., 1986), Corrientes, 30% (MARTÍNEZ; CRUZ; LOMBARDERO, 1990), Rio de Janeiro, 35-50%, (LANGENEGGER; LANDZIERI, 1963); while lower infectious rates were reported in London, 3.6%, (DUBEY; BEVERLEY; CRANE, 1968), São Paulo, 8.5% (FENERICH; SANTOS; RIBEIRO, 1975) and Glasgow, 10.6%, (HAMILTON, 1963). These results indicate that this disease is prevalent in Santa Maria, RS, Brazil (Table 1), and probably within other neighbouring cities, since cats submitted to the Veterinary Hospital are not restricted to this city. This prevalence is probably maintained by the regular ingestion of the intermediate and/or the paratenic host by cats in order to complete the biological cycle of the parasite. The difference in results in the various studies could be directly related to the type of investigation realised; however, it was suggested that the real prevalence of A. abstrusus pneumonia is probably more elevated than what has been reported (HAMILTON, 1963; IDIART et al., 1986). In Brazil, this parasite has only been reported in some States (Rio de Janeiro, São Paulo, Minas Gerais, and Rio Grande do Sul); therefore more studies must conducted to determine if the absence of diagnosis of this disease in other States is related to a restricted regional distribution of A. abstrusus and its intermediate and/or paratenic hosts, inadequate parasitological diagnosis, or simply to lack of knowledge of this parasite.

In this study only short hair domestic cats were affected. The predominance of these cats can be related to their free roaming and hunting habits associated with the low socio-economic conditions of the owners, so that these cats would have a greater possibility to ingest the parataenic and/or intermediate hosts. Similar conditions of the occurrence of feline lungworm were described in the outskirts of La Plata and Buenos Aires (IDIART et al., 1986), in Sydney, Australia (BARRS et al., 1999), and in London (SMITH, 1980). The case described in Sydney related a concomitant infestation of salmonellosis indicating that the pulmonary infestation was due to bacteraemia or contamination of migrating larvae of A. abstrusus by Salmonella typhimurium (BARRS et al., 1999). Additionally, A. abstrusus is known to infest principally cats that have a hunting behaviour, being more susceptible to ingest the intermediate (certain
snails and slugs) or paratenic host (rodents, birds, frogs, toads, lizards, and snakes) thereby terminating the biological cycle of the parasite (LAUTENSLAUGTHER, 1976; BOWMAN, 1999).

During this study no significant difference (Fischer test; $P=0.3285$) was observed between sex relative to lungworm infestation. However, the disease was apparently more prevalent in younger cats in comparison with their older counterparts. Why younger than older short hair domestic cats were diagnosed remains unclear. Maybe these animals were already infected by some viral immunodepressive disease that turned these cats more susceptible to a secondary parasitic infestation; or simply the population of cats attended during the period evaluated consisted predominantly of younger cats.

The histological findings described in this study are consistent with infestation by parasitic forms of *A. abstrusus* (DUBEY; BEVERLEY; CRANE, 1968; DUNGWORTH, 1993; HAWKINGS, 1995). Some cases presented severe hypertrophy and hyperplasia of the smooth muscle of arteries and arterioles, hypertrophy of the alveolar septa and bronchial glands without intralesional parasitic forms of *A. abstrusus*. These reactive pulmonary adaptations in cats are considered diagnostic of an infestation by this parasite, since these are the residual effects of an old infestation (DUNGWORTH, 1993), and have been produced experimentally in cats infested by *A. abstrusus* (NAYLOR; HAMILTON; WEATHERLEY, 1984). However, there is a controversy in the relationship between smooth muscle hypertrophy and hyperplasia and infestation by *A. abstrusus*. This finding has been described in all pathological descriptions of this parasite (HAMILTON, 1963; DUBEY; BEVERLEY; CRANE, 1968; IDIART et al., 1986; DUNGWORTH, 1993). Although the pathogenesis of these smooth muscle changes are not entirely elucidated, it has been suggested that the pulmonary arterial lesion, as described in this study, may be related to chronic vasoconstriction which induces an increase in pulmonary pressure, probably mediated by the liberation of histamine during infestation by the parasite (NAYLOR; HAMILTON; WEATHERLEY, 1984). The participation of toxic products, liberated by the parasite that induces irritation to the pulmonary arteries during migration of the parasite, has also been related to the reactive smooth muscle changes observed in this disease (HAMILTON, 1963; DUBEY; BEVERLEY; CRANE, 1968; NAYLOR; HAMILTON; WEATHERLEY, 1984).

The histopathological patterns described in this report should not be considered as different lesions induced by *A. abstrusus*. In fact, they are manifestations of the different degrees of feline lungworm infestation during passage within the pulmonary parenchyma. Active infestation by *A. abstrusus* is directly related to granulomatous bronchopneumonia (mild or discrete) with infiltrating eosinophils and other inflammatory cells, while the residual effect of the passage of this parasite is characterized by hyperplasia and hypertrophy of the pulmonary arteries. However, the results from this study suggest that granulomatous *A. abstrusus* pneumonia and the reactive hyperplasic and hypertrophic changes of pulmonary arteries are probably the most frequently occurring lesions associated with the feline lungworm. The eosinophils observed during this study within the granulomatous pneumonia have been related to early infestations by *A. abstrusus* and may disappear with time, while the reactive changes observed in the walls of bronchioles and alveolar septae produced in the early infectious stage are irreversible, and are maintained after the infestation has terminated (LAUTENSLAUGTHER, 1976; DUNGWORTH, 1993).

In another histopathological study (MARTÍNEZ; CRUZ; LOMBARDERO, 1990), the lesions induced by *A. abstrusus* were classified with relation to the structures affected: large bronchia, small bronchia and lobules, alveolar septae, and blood vessels. However, this study has demonstrated that it is very difficult and almost impractical to provide an adequate
Histopathological diagnosis of pulmonary disease associated with *A. abstrusus* infestation based on the classification proposed by these authors. In most cases of parasitic pneumonia observed in this study, neither the parasitic forms observed nor the lesions described were restricted to a particular anatomic region of the lung. Additionally, the pulmonary inflammation induced by *A. abstrusus* is progressive and irreversible (DUNGWORTH, 1993), and affects most parts of the lung at the same time, because the parasitic forms are not restricted to a definite pulmonary region. Nevertheless, the sequential list of anatomic structures described in that study (MARTÍNEZ; CRUZ; LOMBARDERO, 1990), may explain the progressive passage and subsequent destruction of pulmonary structures induced by parasitic forms of *A. abstrusus*. Therefore, classification of pulmonary disease associated with *A. abstrusus* should be related to the principal inflammatory exsudate in association with the type of pneumonia observed.

Infestation by *A. abstrusus* in cats may be subclinical (LAUTENSLAUGTHER, 1976; DUNGWORTH, 1993), or asymptomatic (IDIART et al., 1986; HAWKINGS, 1995), and also self-resolving (DUNGWORTH, 1993). The subclinical manifestation of this disease has been attributed to the fact that gross lesions at necropsy are comparatively more severe than those observed in the live animal, and also a larger amount of parasites is required to manifest this disease clinically, which normally happens 8-13 weeks post-infection (HAMILTON, 1963; LAUTENSLAUGTHER, 1976). Apparently the persistence of pulmonary lesions after shedding of parasites in the faeces is responsible for the reduced number of clinical diagnosis in cats, and represents a challenge for diagnosis principally in cases of chronic respiratory distress (LAUTENSLAUGTHER, 1976). Consequently, this may result in confusion with other respiratory infestations, such as tuberculosis, mycoses, and tumours (HAWKINGS, 1995). Routine coprological techniques are efficient (DUBEY; BEVERLEY; CRANE, 1968; HAWKINGS, 1995; BOWMAN, 1999), and radiographic evaluations are useful (HAWKINGS, 1995), whenever a clinical diagnosis is desired, or when cats are suffering from chronic respiratory distress accompanied by progressive weight loss (HAWKINGS, 1995). Control is based on restricted contact of healthy cats with intermediate and parataenic hosts (HAWKINGS, 1995; BOWMAN, 1999); anti-parasitic therapy using febendazole, ivermectin, or levamisole is considered efficient (LAUTENSLAUGTHER, 1976; HAWKINGS, 1995; BOWMAN, 1999).

**References**


