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An update of lungworm infection in cat with especial reference to *aelurostrongylosis*

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Abstract

Aelurostrongylus abstrusus, the most prevalent species affecting the respiratory system of domestic cats is transmitted through the ingestion of gastropod intermediate or paratenic hosts. The most pathogenic significance is formation of nodules by the masses of eggs and larvae in the alveoli and terminal bronchioles, with fewer adult worms. Clinical signs of lungworm infection range from moderate coughing with slightly increased breathing rates to severe, persistent coughing, labored breathing, and respiratory distress or failure. Coughing and weight loss and, sometimes, severe dyspnoea and death, particularly if there are secondary bacterial infections are the main clinical intervention of chronic respiratory disease. Confirmation of first-stage larvae in the faeces may be the main diagnostic approach. Epidemiological studies may be help in treatment and control of the lungworm infection in cats. Treatment with levamisole, albendazole and fenbendazole has the value in cats.

Keywords: *Aelurostrongylus abstrusus*, alveoli, bronchioles, cats, treatment

Introduction

Aelurostrongylus abstrusus is known as feline lungworm, is a parasite that occurs in cats wherever the necessary slug and snail intermediate hosts are found^[1, 2]. The adults are slender and up to 1 cm long (male 7.5 mm; female 9.86 mm), live in the terminal and respiratory bronchioles^[3]. The male bursa is short and the lobes are not distinct, but all bursal rays can be distinguished of which the dorsal forms two stout branches. The spicules are simple and 0.13 – 0.15 mm long. The eggs form nodular deposits in alveoli, where they embryonate and hatch to release first-stage larvae^[4]. The larvae move to the airways, are coughed up and swallowed, and passed in the faeces. Snails and slugs are intermediate hosts whereas birds, rodents, frogs, and lizards are paratenic (transport) hosts^[3, 5, 6, 8]. The life cycle can be completed if a cat eats either an intermediate host or a paratenic host. Infective larvae migrate to the lungs and reach maturity in about 4-6 weeks after ingestion of the third-stage larvae^[3, 4].

Land snails (*Epiphragmophora* spp., *Helix asperus*, *Helminthoglypta californiensis*, *H. nickliniana*, *H. arrosa*) and slugs (*Agrolimax agrestis*) act as intermediate hosts^[3, 7, 8, 10, 11]. However, because gastropods are not the preferred preys of cats, rodents were suggested to play an important role as paratenic hosts in the biological cycle of *A. abstrusus* and in the epidemiology of aelurostrongylosis^[7].

Other species may also cause the lungworm infection in cats include *Troglostrongylus brevior* reside in the bronchi and bronchioles while *Troglostrongylus subcrenatus* reside in the trachea and bronchi. Moreover, *Eucoleus aerophilus* (synonym *Capillaria aerophila*) adult worms reside in the trachea and bronchi of cat, dog, fox and man^[3]. The pathogenesis, clinical signs, epidemiology, diagnosis, treatment and control of aelurostrongylosis in cats are discussed in the present communication.

Pathogenesis

The characteristic lesions are subpleural nodules which are firm, raised and greyish in colour^[3]. The gross lesions are multifocal, amber, and subpleural granulomatous nodules up to 1 cm in diameter throughout the lungs. In profound infections, which tend to be fatal, there may be creamy yellow areas on the lungs and the pleural cavity may be filled with a thick milky fluid

rich in eggs and larvae [5, 6]. The existence of adult worms, eggs, or larvae in the bronchioles is associated with chronic catarrhal and eosinophilic bronchiolitis [11]. On incision, these nodules may contain viscous milky exudates in acute cases but in the chronic infection calcification is common. Microscopically, the adult parasites, eggs, and coiled larvae are in the bronchioles and alveoli, where they cause catarrhal and eosinophilic bronchiolitis, hyperplasia of submucosal glands, and, later, granulomatous alveolitis, alveolar fibrosis, and fibromuscular hyperplasia [3, 4, 11]. The nodules are formed by masses of eggs and larvae in the alveoli and terminal bronchioles, with fewer adult worms. Eosinophils and neutrophils infiltrate the early lesions, but most cases are dominated by mononuclear cells and giant cells in the alveoli and around degenerating eggs and larvae [11]. Alveoli are dilated, and alveolar septa may be disrupted. Necrosis and mineralization seldom occur. Lymphocytic nodules form around vessels and airways, and there is hypertrophy and hyperplasia of the smooth muscle in the walls of the bronchioles and alveolar ducts [4]. Inflammation may be present within the tracheal mucosa, presumably in response to larvae moving up the trachea to be swallowed [3].

During routine examination of feline lungs, it is quite common to find fibromuscular hyperplasia in bronchioles and arterioles in otherwise healthy cats [3]. The fibromuscular hyperplasia may be a long-term sequela of subclinical aelurostrongylosis. However, this view has been challenged; thus the pathogenesis and significance of pulmonary fibromuscular hyperplasia in healthy cats remains uncertain. Fibromuscular hyperplasia is grossly visible in the lungs as white subpleural nodules in severe cases [4].

Heavy infections in the prepatent period cause randomly distributed haemorrhages or white foci, which represent an eosinophilic and granulomatous reaction to the migrating larvae [11]. In the patent period, there are 1-10 mm diameter, firm, off-white to pale yellow, slightly raised nodules scattered throughout the lungs. In severe infections, nodules may coalesce to form confluent areas of consolidation. In older lesions from which eggs and larvae have disappeared, alveoli remain epithelialized and septa are persistently thickened by fibrous tissue and smooth muscle [3]. Bronchial glands and smooth muscle in the media of small pulmonary arteries and arterioles may be moderately prominent in cats infected with *Aelurostrongylus*, but these changes are also frequent in clinically healthy cats that have no evidence of parasitism.

Clinical signs

Cats may be subclinically infected and display no clinical signs, but heavy infections cause coughing or increased respiratory rate. However, clinical signs consist of a chronic cough with gradual wasting [3]. It can cause chronic respiratory disease with coughing and weight loss and, sometimes, severe dyspnea, polypnoea with increased lung sound and rales and death, particularly if there are secondary bacterial infections [11]. The clinical effects are slight, and in resting cat are limited to a chronic mild cough; following exercise or handling, there may be coughing and sneezing with slight dyspnea and production of mucoid sputum [4]. In heavy infections young animal may cough dyspnoea, pneumonia, pleural effusion, ascites and suffer from diarrhoea and emaciation, which may be followed by death, or recovery may take place [3, 4, 12]. In very severe infections the simultaneous deposition of a large number of eggs in the lungs may cause sudden death.

In heavy experimental infections the most severe signs have appeared at 6 -12 weeks after infection when egg laying is maximal [4].

Epidemiology

A. abstrusus has been reported from Asia, Australia, Europe, and North and South America [3, 4, 13]. Aelurostrongylosis is widespread partly because it is almost indiscriminate in its ability to develop in slugs and snails, and partly because of its wide range of paratenic hosts. So far all surveys have shown prevalences greater than 5% [4]. Lung worm infection caused by *T. brevior* reside in the bronchi and bronchioles while *T. subcrenatus* distributed in cat of Asia and Europe while caused by *E. aerophilus* in cat, dog, fox and man of Asia, Australia, Europe, and North and South America [3, 13].

Diagnosis

Repeated faecal examination by direct, floatation, or Baermann technique may be necessary to find the characteristic L₁, which bears a subterminal spine on its S-shaped tail [3, 4]. Examination of pharyngeal swabs may be a useful additional procedure. Radiography has revealed the increased vascular and focal parenchymal densities which would be expected from the changes described above [4].

Treatment

1. Levamisole (15 – 100 mg/kg) given on alternate days for five to six treatments suppresses the clinical signs and larvae disappear from the faeces [3].
2. Treatment with albendazole or fenbendazole might be of value.

Control

1. Prophylaxis is in most cases impracticable, since it would imply preventing cats from catching mice, lizards etc.

Conclusion

Impact of *A. abstrusus* is found on the lung tissues and on the health of the cats, despite the presence of only mild haematological abnormalities. Due to the worldwide distribution of feline lung worms, aelurostrongylosis should be considered in the differential diagnosis of lung diseases in spite of the presence of clinical signs and larval excretion.

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