CHRONIC PULMONARY LESIONS IN THE EXPERIMENTAL INFESTATION WITH ASCARIS SUUM IN DOMESTIC PIG

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Summary

The aim of the study was to investigate the histopathologic lesions caused by the migration of Ascaris suum larvae through lung. Fragments of the lung were collected from a number of 10 swine (four months old), and histologically processed. Animals in this study were divided into 2 groups of 5 individuals: a group artificially infested with 1000 eggs of Ascaris suum, and another control group. Lesions present in the lungs collected from the artificially infested group varied in intensity from one area to another following the larval migration. Lungs from the control group were apparently normal. Lungs presented lesions in the bronchioli and lung interstitium. Bronchiolar epithelium showed zonal necrosis, with cellular debris in the lumen of bronchioli, whose intensity differed from one area to another of the lung parenchyma. Chronic inflammation represented by lympho-eosinophilic infiltrate (consecutive to active migration of Ascaris spp. larvae through the lung parenchyma) evolved into a chronic fibrous peri-brochiolitis that led to a local bronchiolar fibrostenosis. The chronic morphological changes lead to the transformation of the bronchiolus in a rigid tube that no longer has the ability to adapt its lumen diameter and has an altered aspect. Lesions were chronic and had an irreversible nature.

Key words: Ascaris suum, swine, pulmonary lesions.

Introduction

Although the modern growing systems have reduced the impact of many parasitic diseases, the Ascaris suum infestation continues to be a problem for animal breeders. It is hard to find a farm without infestation and in some regions the prevalence of infestations has increased (Roepstorff and Nansen, 1994). In the United States of America epidemiologic studies have shown a prevalence of 60% of the infestation with Ascaris suum in the market swines.

Another important matter is that of the zoonotic aspect of this parasitosis. Anderson (1995) and Maruyama et al. (1996) have presented new evidence regarding the infestation of humans with Ascaris suum. Public health problem resides in the longevity of the infestation eggs and in the usage of contaminated pig feces as field fertilizer mostly in US and Europe.

Ascaris suum is a nematode located in the domestic and wild pig intestine, accidentally in sheep (Cozma, 2000) as well as in other species (experimental ascariasis in calves - Mccraw, 1970). The adult form lives in the intestine of the pig, and the female lays eggs that are eliminated with feces. Although undeveloped eggs are eliminated in the environment, if in favorable conditions, they become able to infest, containing the stage 2 larva inside. Once in the body of the host, under the action of digestive juices, eggs release larvae that will undergo a migration, arriving in the gut as adult parasites.

Complexity of pathogenic phenomena induced by Ascaris suum in host correlates with the evolutionary phases: larvae, migrating and adult. Ascariasis, induced by larvae is characterized by
mechanical intervention, toxic-allergic and inoculation effect.

The mechanical action is usually minor and transient and is perceived as micro-haemorrhages through the parenchyma of organs, especially the liver and lung. Somatic migration in the encephalon, muscles is perceived as clinical manifestations characteristic to the injured organ. Larval toxins have a necrotic effect, translated in intra-lobular hepatic necrosis (Şuteu and Cozma, 2004).

Larval migration to the lungs produces hemorrhagic lesions and intense eosinophilic infiltration around alveoli, through which they migrate toward the upper bronchial tree. Re-infestations will produce extensive bleeding, pulmonary edema and emphysema.

Eosinophilic granuloma formation, especially in the lung, is consequent to the allergic action. The consecutive eosinopenia probably causes excitation of hematopoietic organs, the peripheral leukocytosis and eosinophilia being the response of the next phase. Sometimes allergic skin and/or nervous manifestations occur (Şuteu and Cozma, 2004).

Pathological diagnosis of pig ascariasis is based on the detection of characteristic lesions in the liver with diagnostic value (Straw, 2006) and those from different tissues caused by infestation with Ascaris suum. In terms of histopathology, ascariasis is characterized by lesions in the organs (especially lung and liver) caused by larval parasite migration in the presence of a massive eosinophilic infiltration.

Adult parasites cause inflammation of the duodenum and jejunum, intestinal obstruction, volvulus, chronic cirogene hepatitis, so called milk spots, pancreatitis, jaundice tissue, obstruction of common bile duct and liver degeneration (Baba, 1996).

Inoculation action is demonstrated by numerous experimental studies and epidemiological facts confirming the role of porting and inoculation of larvae with pathogens. Lesions caused by larval development may exacerbate the evolution of enzootic pneumonia and potency latent infection with swine influenza viruses. Other larval migration associated infections are possible: Salmonella, with amoebas in the liver, the fungus etc. (Vasiu, 1998; Perianu, 2003).

The important microscopic lung lesions revealed by McCraw (1970) in calves infected with Ascaris suum were edema and emphysema in interlobular septa, with a large number of eosinophils in and around the lymphatic vessels, the presence of peri-bronchiolar lymphoid nodules and parasitic granulomas. Many of the microscopic features were consistent with those found in atypical interstitial pneumonia. The changes in alveoli were atelectasis, secretion of plasma proteins, mononuclear cells and eosinophils, and alveolar wall thickening. The lesions found later included fibrosis of the alveolar wall.

Larval migration to the lungs leads to the apparition of different extent bleeding associated with exudate, edema and emphysema with the possibility of occurrence of bacterial pneumonia (Straw et al., 2006).

Material and methods

The study was conducted on a total of 10 pigs (2 groups, n = 5), mixed race of same age (4 months). Group 2 was artificially infested (through nourishment), with 1000 eggs/every individual and group 1 was the control group. Eggs were collected from Ascaris suum female uterus, obtained from slaughtered animals (Eriksen, 1990), and were cultured in plain water at 25°C temperature (after Jungersen, 1998).

At 56 days after infestation, pigs were slaughtered and fragments were collected for conducting investigations on lung histopathology. Pieces were fixed in Stieve mixture for two days, then dehydrated with alcohol, cleared with butyl alcohol (n-butanol) and included in paraffin. 5 µ thick sections were stained with Goldner’s
trichrome method and examined with a Olympus BX41 microscope.

Results and discussion

Lungs harvested from experimentally infected pigs with *Ascaris suum* have shown lesions in the bronchioles and lung interstitium. Bronchiolar epithelium showed zonal necrosis (Fig. 1) with desquamations that can be observed in the lumen of bronchioles as cellular debris (Fig. 2), whose intensity is different from one area to another of the lung parenchyma. A leukocyte infiltrate can be observed in the lung interstitium, with predominantly peri-bronchial arrangement, suggesting that they appeared on the tracts of larval migration. Sometimes bronchioli in the vicinity of leukocyte infiltrate do not present significant changes (Fig. 3), but in many of them the lesions appear in the continuation of leukocyte infiltrate with muscle damage which is missing on large areas, sometimes on over 50% of the circumference (Fig. 4) and with some tendency to collapse. In other situations lesions occur also in the bronchiolar epithelium which suffers metaplasia in some areas (Fig. 5).

In lung interstitium and especially peribronchiolar, the leukocyte infiltration is dominated by lympho histiocytes and eosinophils, which are arranged in large cellular groups that induce compression atrophy of the bronchiolar adjacent structures (Figs. 4 and 5). Furthermore, leukocyte infiltration in the bronchiolar structures has an infiltrative character, invading and tearing the fibrous connective tissue and smooth muscle related to bronchioles. Inflammatory infiltrate also invades the bronchiolar epithelium in some areas (Fig. 5).

Chronic inflammation represented by lympho-eosinophilic infiltrate (consecutive to active migration of *Ascaris spp.* larvae through the lung parenchyma) evolves into a chronic fibrous peri-bronchiolitis that leads to a local bronchiolar fibrostenosis (Figs. 1 and 5) and even pulmonary atelectasis. Also, the papillary hyperplasia of the bronchiolar epithelium occurs, following the chronic and later fibrous lympho-eosinophilic bronchiolitis and peri-bronchiolitis (Fig. 1).

There are also situations when the muscle layer has actually disappeared, only small areas being still obvious (Fig. 6) and epithelial metaplasia is present on large areas.

In this context, bronchiolus does not preserve the characteristic appearance and loses all elasticity, which results in transformation of the bronchiolus in a rigid tube that no longer has the ability to change lumen diameter between physiological limits, as required. Lesions are chronic and have an irreversible nature.

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**Fig. 1.** Bronchiolar fibro-sclerosis with papillary hyperplasia of the bronchiolar epithelium (Goldner’s trichrome stain)

**Fig. 2.** Bronchiolar desquamations with the presence of cellular debris in the bronchiolar lumen (Goldner’s trichrome stain)
Conclusions

Pulmonary lesions observed consequently to the active migration of *Ascaris suum* larvae through the lung parenchyma consist in chronic and later fibrous lympho-eosinophilic bronchiolitis and peri-bronchiolitis, multifocal bronchiolar desquamation of the bronchiolar epithelium, focal necrosis of the bronchiolar muscle and papillary hyperplasia of the bronchiolar epithelium.

References

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