
Department of Veterinary Pathology, Bombay Veterinary College, Maharashtra Animal & Fishery Sciences University, Parel, Mumbai-12.

Introduction
Spirocerca lupi is a parasitic disease caused due to nematode *Spirocerca lupi* and is worldwide in occurrence. It is endemic in some warm climates. Domestic dogs and other wild carnivores (fox, wolf, coyote and jackal) are most commonly affected, although natural infections have also been reported in domestic and wild felids, man, goats, horses and donkeys. Target organs are the oesophagus and aorta, resulting in gastrointestinal, respiratory and circulatory signs (Oryan et al., 2008). The major epidemiologic factors of the disease include canine population density and environmental conditions (e.g., soil type and pH, temperature, rainfall, solar radiation) that sustain the source of the intermediate hosts (dung beetle) and transport hosts (reptiles, birds, rodents) as stated by Chhabra et al., (1973).

Carnivores are infected by ingestion of beetles or a variety of paratenic hosts including numerous amphibians, reptiles, lizards, domestic and wild birds and small mammals such as hedgehogs, mice and rabbits (Soulsby, 1986). In the definitive host, the larvae usually follow a specific migratory route, reaches to the stomach of the dog and then penetrate the gastric mucosa of the host and begin a predictable migratory route. They migrate along arteries, follow their life cycle in the thoracic aorta and about three months post-infection, the larvae leave the aorta and migrate to the caudal esophagus where they provoke the development of granulomas as they mature to adults over the next three months (Soulsby, 1986 and Urquhart et al., 1996). Here they form granulomatous tumor-like growth in which they reach maturity and reproduce eggs. The eggs are passed via a small opening (or openings) into the esophagus and their cycle repeats itself (Evans, 1983 and Lobetti, 2000).

The clinical manifestations due to spirocercosis vary greatly, depending on the stage of the disease, aberrant migrations and possible complications. Clinical signs, in endemic areas include vomition or regurgitation, dysphagia, dyspnoea, pyrexia and anorexia (Bailey, 1972; Jubb et al., 1993 and Lobetti, 2000). Definitive diagnosis is made by detection of typical embryonated eggs in fecal smears, and/or finding the parasite in the granulomatous lesions (Fox et al., 1988). This paper reports the case of oesophageal granuloma caused by *Spirocerca lupi* in a stray dog.

Materials and methods
A One year old female non-descript stray dog was presented for postmortem examination to the Department. The dog showed restlessness before death. Detailed necropsy was conducted and gross lesions were recorded. The affected tissue sample were collected in 10% formalin, processed and embedded in paraffin blocks. Section of 5 µm were taken on slides and stained with haematoxylin and eosin (Culling, 1963). Intestinal contents and worms from the lesions were collected for parasitological examination.

Result and Discussion
Clinical signs observed before death were restlessness and anorexia. On necropsy, external examination of dog revealed pale mucous membranes, ticks on the body and dehydrated carcass. Grossly, Aorta showed aneurysm and cut section of the aorta revealed red brown colored round worms (Fig. 1). Moreover, oesophageal mucosa showed nodules of about 3x4 cm and cut section of the nodule showed round worms (Fig. 2 and 3). The length of worms varied from 1-4 cm in both, aorta and esophagus. There were approximately 6-12 numbers of worms in aorta and esophageal nodule. The present findings are in accordance with the observation of various researchers (Fox et al., 1988; Dvir et al., 2001; Moulton, 2002) in which they reported that the most common pathological...
lesions of spirocercosis includes aortic scars and aneurysms, and esophageal nodular granulomas.

Moreover, Bailey, (1972) and Chandrasekharan et al. (1958) were of the opinion that there are generally three to six worms containing nodules were seen in the submucosa of the wall of the oesophagus, a few centimetres cranial to the diaphragm. However, the number of worms present in a nodule varies from a few to 30, but there are typically between three to six (Chandrasekharan et al. (1958).

It is suggested that the pathologic lesions may result from normal or aberrant migration of parasite. In the normal migration route along the gastro-aortic arterial system, the lesions include rupture of the aorta resulting haemothorax and sudden death, or rupture of other major blood vessels, that cause haematomas (Riberio et al., 1994; Dvir et al., 2001;). Migration through the aortic wall to the oesophagus may result in mediastinitis, pneumomediastinum, pleuritis or pyothorax with clinical sign of dyspnoea (Stephens et al., 1983; Hamir, 1984; Dvir et al., 2001;). However, we did not observe these lesions in the present study.

Stomach showed hemorrhages on the mucosa and there were streaks of hemorrhages noticed on the intestinal mucosa. Liver was enlarged, congested and cut section showed blood tinged exudates. Kidneys were puffy and capsule peeled off with difficulty. Cortical surface showed white focal areas. Other visceral organs did not show significant changes. These lesions observed in the present investigation are non specific and could be due to secondary complication.

Histopathological examination of the nodule from oesophagus revealed a typical granulomatous inflammation surrounding the

Fig. 1: Aorta showing aneurysm  Fig.2: Oesophagus showing nodules along with worms  Fig.3: Esophageal nodules containing worms  Fig.5: Thickening of the wall of aorta and aneurysm
worm (Fig. 4). Aorta showed aneurysm and thickening of the wall (Fig. 5). Kidneys showed large focal lymphocytic aggregations along with diffuse tubular degeneration of epithelial cells, diffuse hypercellularity of the glomeruli, multifocal interstitial edema and proteinaceous casts in glomeruli. Liver showed diffuse cloudy swelling of the hepatocytes and infiltration of the mono nuclear cells.

Necrosis, haemorrhages and exudation followed by fibrosis, scar formation and sometimes mineralization and aneurysm formation in the aortic and other blood vessels are other usual histopathological findings in canine spirocercosis (Jubb et al., 1993). This may be the reason for aortic aneurysm in this case.

Fig. 4: Granuloma formation around the worm (oesophagus)

Metaplastic ossification of the aorta, to the extent of bone marrow formation, has been reported (Kumar et al., 1981). This is not in agreement with the present study. Aortic lesions are the most common lesion associated with spirocercosis and are considered pathognomonic (Bailey, 1972; Ramachandran et al., 1984).

Summary

Spirocercosis is a serious condition in endemic areas. Clinical signs usually develop late in the disease except in those cases presenting with peracute aortic rupture. Even though there is very good drugs are available for spirocercosis treatment, still there is high prevalence of spirocercosis among stray dogs. This communication is an attempt to record the common gross and histopathological findings of spirocercosis in stray dogs.

References


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