SPIROCERCA LUPI INDUCED AORTIC ANEURYSM AND RUPTURE IN TWO GERMAN SHEPHERD DOGS

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SUMMARY

Cases of sudden deaths attributed to *Spirocerca lupi* induced aortic aneurysm and rupture in two German shepherd dogs are described and discussed. The dogs, one aged 12 and the other 2 years, were suspected to have died of either poisoning or snake bites. However, autopsy revealed copious hemothorax in both cases together with perforated aneurysms in the aortic walls at points about 15cm from the aortic arches. The perforation sites were about 5-10mm in diameter. *Spirocerca lupi* worms were seen in the aortic wall at the affected site as well as in the oesophageal lumen. In addition, numerous small pyogranulomas were seen in the oesophageal wall, more of them in the aged bitch than in the juvenile one. Histopathologically, lesions in the aortic wall were severe destruction and disruption of tissue elements in all layers of the aortic wall, coupled with hemorrhage, a heavy neutrophil infiltration, hyaline degeneration of the tunicae media and adventitia. Surprisingly eosinophils were only sparsely present. This report serves to provide a useful adjunct to differential diagnosis of sudden deaths in dogs.

INTRODUCTION

Spirocercosis is a common, well recognized disease in dogs in which it is variously characterized by formation of nodular lesions of pyogranulomas in distal oesophagus (Ndiritu and al-Sadi, 1976, Khan & Ajmal, 1977) aneurysms that may lead to fatal ruptures of the aorta (Ivoghli, 1977-Hamir, 1984) as well as perforation of the oesophagus which has been seen to result in pyothorax (Hamir, 1986). The histological features of the nodular lesions have characteristics ranging from pyogranulomatous lesions to frank fibrosarcomas (Gupta & Singh, 1975; Ndiritu & al-Saidi 1976; Singh & Tewari, 1976). The manifestation and severity of the presenting clinical signs seem to depend on the kind of lesions involved. They range from dysphagia, megaesophagus, vomiting following meals and sudden death on event of an aortic rupture (Chhabra, 1973, Hamir, 1986, Wandera, 1976; King, 1990). In addition reports exist on other rather rare manifestations like paraplegia which occur when the parasite migrate into the spinal cord (Smith and Knottenbelt, 1989). This report contributes further to the existing knowledge on the pathological features a rapidly fatal form of the disease when its development involves an aneurysm and rupture of the aorta.

MATERIALS AND METHODS

Carcasses of two German Shepherd bitches one aged 12 and the other 2 years were submitted to the department for postmortem examination less than 12 hours after they had died suddenly. The dogs were described to have been in very good health before the nightfall only to be found dead in the morning. Their body conditions were very good. Either poisoning or a snake bites had been suspected to be the possible causes of their deaths. The bitch was autopsied systematically and each one organ system was carefully examined after the other and characteristic lesions especially in the thoracic cavity, the oesophagus and aorta were carefully photographed onto a Svema FN 64 black and white film. Appropriate tissue samples from lesions in the aorta and oesophagus were preserved in 10% neutral buffered formalin. The formalin fixed samples were later retrieved and processed into paraffin sections by standard procedures as outlined by Bancroft and Stevens (1990).The paraffin sections were stained with haematoxylin and eosin (H & E), mounted in DPX® and examined under an Olympus BH2 PM-10AD photomicrographic system.

RESULTS

Post mortem examination revealed copious hemothorax in form of abundant dark red clotted and non clotted blood. The clotted blood was estimated to be a mass of about two kilogrammes which completely obscured the view of the organs in the left half of the thoracic cavity. After removing the mass of the clotted blood and placing it beside the carcase the volume of non clotted blood estimated at about two litres of volume was exposed.

The source of bleeding was identified in perforations of about 5-10mm in diameter in the walls of the aorta at points located about 15cm from the aortic arches (Fig.2). The perforation site was surrounded by a wider zone of aneurysm and hemorrhage in the subserosal tissues (Fig.3). The intimal excavation into the fistula was about one centimetre in diameter. The cut surface at the perforation revealed an ovoid aneurysm of dimensions of 3 x 2 centimetres. The intimal surface of the aorta was speckled with lesions of hyperaemia and hemorrhage. Lesions in the thoracic oesophagus consisted of adult worms about 10 of them in the oesophageal lumen (Fig.4) and a chain of 5 intramural granulomatous nodules of sizes ranging between one and three centimetres and spaced one to three centimetres apart in the distal 15cm of the thoracic oesophagus between the aortic arch and the diaphragm (Fig.4). Histologically the aortic lesions were characterized by severe destruction and disruption of the tissue elements in all layers of the aorta. Acute lesions were characterized by a heavy neutrophil infiltration, fibrin exudate, hemorrhage and hyaline degeneration of the tissues in tunica media and tunica adventitia. Other inflammatory cells in the exudate included lymphocytes aud plasma cells. Eosinophils were only sparsely present. In older lesions, there was fibroplasia, granulation tissue formation and fibrosis.

Oesophageal lesions consisted of granulomatous tissue formation around the worms and in some areas several small individual granulomas appeared to

have fused into a larger conglomerate. In between the individual granulomas was a heavy infiltration of neutrophils interspersed between the proliferating fibroblasts and necrotic cell debris heavily laden with bacterial colonies. Other inflammatory cells included many plasma cells and a few lymphocytes. Surprisingly eosinophils were only sparsely present.

DISCUSSION

The gross and microscopic lesions described in connection with the sudden death of the dog highlighted in this report are closely compatible with those described by a number of other workers (Ivoghli, 1977; Hamir, 1984) on the fatality of *Spirocerca lupi* induced aortic aneurysms and rupture. It is surprising however, that in the acute lesions described in here eosinophils were not as a prominent feature as has been described in a report by Ivoghli(1977).

The relative uniformity in the age of lesions in the aorta and oesophagus serve to indicate that the worms involved were all in the same wave of migration. Whereas the presence of so many free worms lying close together in the lumen of the oesophagus without being washed down with ingesta indicates that they were of recent arrival in the oesophagus. This observation tempts the proposition that the rapid, unimpeded mass migration of the worms across the aorta are responsible for the perforation and tearing of the aorta. The absence of chronic atheromatous like lesions in the aorta argues in favour of perforation and tearing of the aorta. It is further tempting to suggest that the paucity of eosinophils in the inflammatory exudate indicates lack of formidable defense against the parasitic migration and thus allowing for a rapid unimpeded mass migration resulting in an extensive tissue disruption.

This report serves to illustrate the need to consider *Spirocerca lupi* induced aortic rupture in events of sudden deaths in healthy dogs. The rapidity with which the deaths occur does not allow for treatment measures thus prevention through regular screening and deworming of dogs would be the most feasible approach towards minimizing such deaths.

It has been reported that fatal aortic lesions in spirocercosis are a rare event. However, our observations show that out of a total of 19 cases followed up at this University so far, as much as five (26.4%) of them featured fatal aortic aneurysms and rupture. Four others (21.1%) featured fibrosarcomatous lesions, whereas the rest ten (52.5%) were characterized by ordinary pyogranulomatous lesions beset in the oesophageal walls.

The specific circumstances leading to variations in types of lesions produced by *Spirocerca lupi* in dogs remain unknown (Ndiritu & Al-Sadi, 1976; Chhabra, 1973). It would however, appear that German Shepherds are the ones most predisposed to the fatal aortic aneurysms and perforations as seen in reports by Ivoghli (1977) and Hamir (1984). Factors responsible for the apparently high susceptibility of German Shepherds to fatal aortic aneurysms and rupture remain unknown.

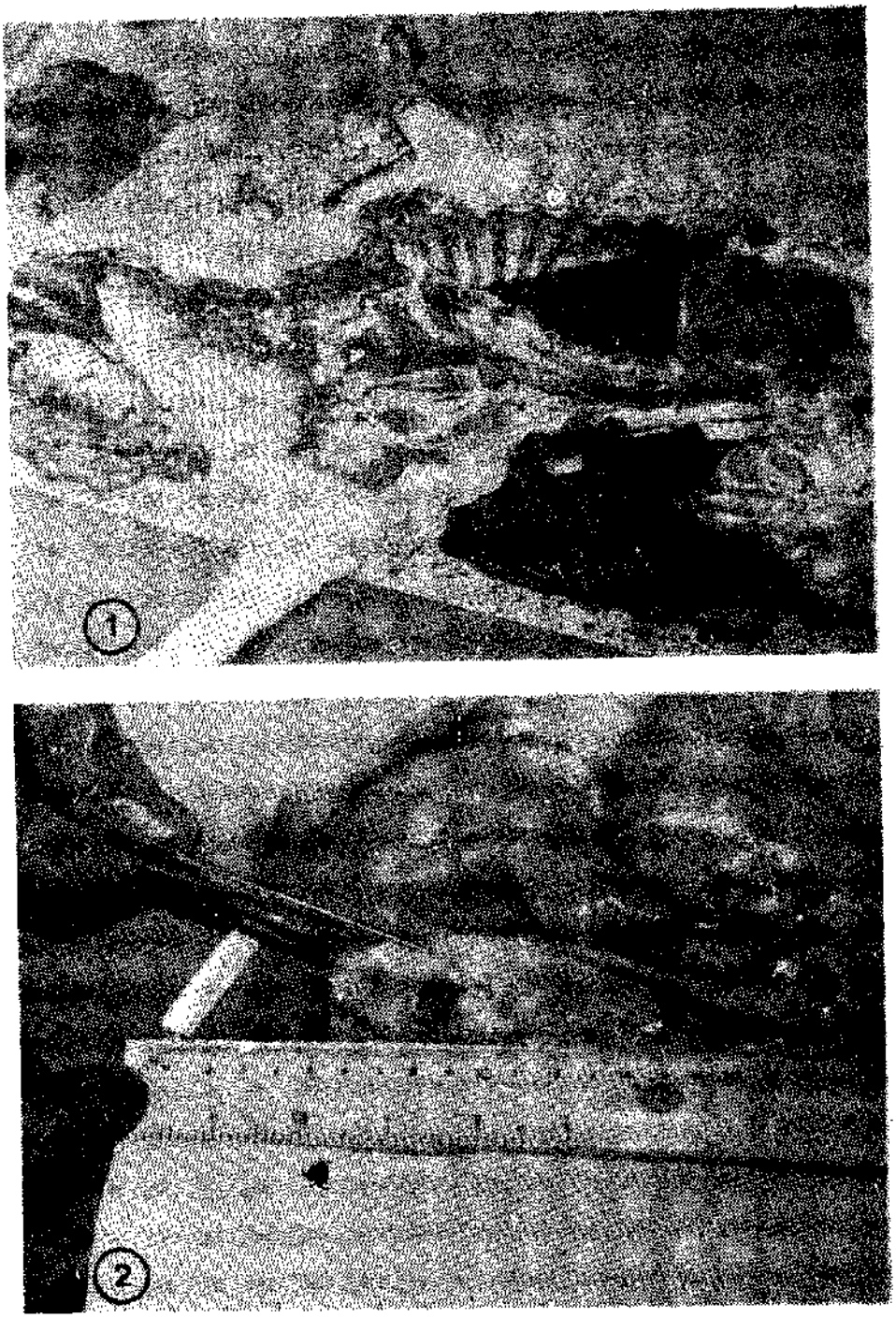

Fig. 1: Hemothorax in a German shepherd carcase due to *Spirocerca lupi* induced rupture of the thoracic aorta. Note the volume of non clotted blood in the thoracic cavity and the mass of blood clots beside the carcase fished out of the chest cavity.

Figure 2: Thoracic aorta with a 10mm wide perforation in its wall at a point about 15cm from the aortic arch.

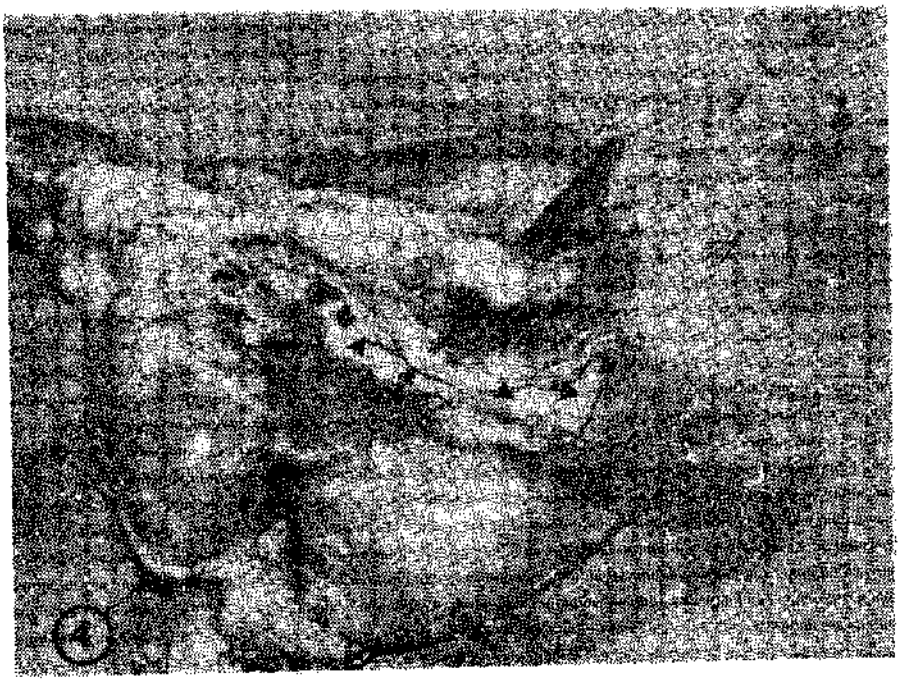

Figure 3: Serosal surface appearance of the perforation site in the aortic wall showing a wide area with subserosal hemorrhage.

Figure 4: Oesophageal lumen showing free lying *Spirocerca lupi* worms. Note also the presence of small granulomatous nodules in the aesophageal wall (arrowheads). The forceps are illustrating the perforation in the aortic wall.

It is however worthwhile to note that aortic perforations appear to be associated with acute lesions only. These observations seem to suggest a greater proneness attributable to a relatively low resistance to infestations with *Spirocerca lupi* in German Shepherds, a phenomenon that has been demonstrated in a number of other dog breeds, cats lambs and rats (Chhabra & Singh, 1972).

REFERENCES

Bancroft, J. D. and Stevens A. (1990). Theory and Practice og Histological Techniques, 3rd edition, Chuchill Livingstone, Edinburgh, 43-80.

Chhabra, R. C. (1973). Symptoms and pathogenesis of spirocercosis in dogs. [*Spirocerca lupi*]. Ind. Vet. J. 50:551-554.

Chhabra, R. C., Singh, K. S. (1972). Development of resistance in Spirocerca infection. [Dogs, cats, lambs, rats]. Indian J. Anim. Sci., 42:355-357.

Gupta, P. P., Singh, B. (1975). A note on fibroma of esophagus associated with *Spirocerca lupi* in a dog. Ind. Vet. J. 52:878-879.

Hamir, A. N. (1984). Perforation of thoracic aorta in a dog associated with *Spirocerca lupi* infection. Aust. Vet. J. 61:64.

Hamir, A. N. (1986). Oesophageal perforation and pyothorax associated with *Spirocerca lupi* infestation in a dog. Vet. Rec. 119:276.

Ivoghli, B. (1977). Fatal aortic aneurysm and rupture caused by *Spirocerca lupi* in a dog. J. Am. Vet. Med. Assoc. 170:834.

Khan, M. Z., Ajmal, M. (1977). Osteogenic sarcoma of the oesophagus in a dog [*Spirocerca lupi* infection]. J.Agric. Res. 15:377-380.

King, J. M. (1990). Esophageal leiomyoma. Vet. Med. 85:120.

Moulton, J. E. (1978). Tumours of Domestic Animals. 2nd. edit. University of California Press. Berkeley. 252-258.

Ndiritu, C. G., Al-Sadi, H. I. (1976). Pathogenesis & lesions of canine spirocercosis [caused by a nematode, *Spirocerca lupi*]. Mod. Vet. Pract. 57:924-934.

Singh, N. P. Tewari, A.N. (1976). Canine spirocercosis, a pathoanatomical and histopathological study [*Spirocerca lupi*]. Indian J. Anim. Sci. 46:211-214.

Smith, D. A. Knottenbelt, D. C. (1989). *Spirocerca lupi* localization in the spinal cord of a dog. Zimbabwe Vet. J. 18:19-22.

Wandera, J. G. (1976). Further observations on canine spirocercosis in Kenya [*Spirocerca lupi*]. Vet. Rec. 99:348-351.