SPONDYLOSIS DEFORMANS AS AN UNUSUAL CAUSE OF CRANIAL VENA CAVA SYNDROME IN A FEMALE DOBERMAN PINCHER - A CASE REPORT

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Received for publication July 17, 2007

Abstract

A 4-year-old female crossbreed Doberman pincher was presented to the veterinary clinic with clinical signs of laboured breathing, loss of appetite, and lethargy. Additionally, distension of the veins of the neck (external jugular veins), moderate oedema of the base of the neck and cranial parts of the thorax and mild oedema in forelimbs were found. RTG revealed increased opacity and deformity in soft tissue cranially to the handle of sternum, and massive osteophytes at ventral aspects of vertebral body in thoracic section (Th 3 - Th 13) of the spine, attributable to spondylosis deformans. Autopsy revealed severe deformity of soft tissues beneath the spine (Th1 – Th13) associated with compression of some thoracic structures i.e. heart, blood vessels, and cranial lobes of the lungs. Based on clinical signs and results of thoracic radiography and autopsy, cranial vena cava syndrome secondary to thoracic spinal spondylosis deformans was diagnosed.

Key words: dog, vena cava syndrome, spondylosis deformans.

Cranial vena cava syndrome (CrVCS) is an uncommon, but easily recognised sequel to the obstruction of the cranial vena cava. Clinical signs of CrVCS are characteristic: non-painful, pitting oedema of the head, neck, and forelimb. The syndrome is accompanied by some extra- and intravascular factors interfering with venous blood return through the cranial vena cava. These factors include thrombosis of the vessel along with compression or invasion by pathologic masses (4, 5, 7). In humans, most cases (approximately 95%) described in the literature are caused by mediastinal lymphosarcoma and small-cell bronchogenic carcinoma. Other causes include abscesses, granulomas, mediastinal oedema, haemorrhage, and other thymic lesions. In dogs and cats, there are few cases of CrVCS described in the literature. Thymomas, lymphosarcomas, carcinomas, and aortic body tumours localised in mediastinum are most common etiological factors of the syndrome (5). CrVCS and chylothorax associated with fungal granuloma in male dachshund was described too (4).

Spondylosis deformans (SD) is a degenerative disease of the spine exhibiting the presence of one or more osteophytes, showing different degrees of development at the level of vertebral bodies. Many clinical signs can be related to SD, among them stiffness in the back, lameness, change of gait, and pain (1). The development degree of the lesions is usually age dependent and more severe lesions in older dogs were observed (6). In the present study, a crossbreed Doberman pincher female dog with spondylosis deformans as an unusual cause of cranial vena cava syndrome is described.

Description of the case

A 4-year-old female crossbreed Doberman pincher was presented to the veterinary clinic with clinical signs of laboured breathing, loss of appetite, and lethargy. The owner suggested a sudden onset of clinical signs, but the thorough anamnesis revealed that behaviour alteration and apathy was noted several days ago. Clinical evaluation revealed severe dyspnoea, normal (38.6°C) body temperature, and heart and respiration rates slightly increased. Thoracic auscultation revealed mildly sharpened bronchovesicular sounds, especially over cranial lobes, and additionally dull cardiac sounds were noted. Furthermore, distension of the veins of the neck (external jugular veins), moderate oedemas of the basis of the neck and cranial parts of the thorax and mild in forelimbs were found.

Differential diagnosis suggested vena cava syndrome caused by intrathoracic growth, probably at the heart basis or thymus. To resolve this question thoracic radiography was performed. RTG revealed increased opacity and deformity in soft tissue cranially to the handle of sternum.
Severe osteophytes at ventral aspects of vertebral body in thoracic section (Th.3-Th.13) of the spine, attributable to spondylosis deformans were visible. A pulmonary peribronchial pattern at heart basis and increased opacity in cardiophrenic and cardiosternal angles was also observed. Heart silhouette was normal, but cardiovascular pattern was slightly altered. There were not any pathologic masses at the heart basis, mediastinum, and lungs in thoracic radiography (Fig. 1).

Because of worsened clinical state and results of radiographic examination, the animal was euthanised and complete autopsy was performed. The autopsy revealed severe deformity of soft tissues beneath the spine (Th1 – Th13), associated with the compression of some thoracic structures i.e. heart, blood vessels, and cranial lobes of the lungs. This deformity was created by extremely swollen infraspinal muscles and soft tissues. The trachea and oesophagus were dislocated to the right side of the mentioned deformity. Cranial lobes of the lungs were extremely hyperaemic, dark red, and oedematous. There were no macroscopically visible circulatory disturbances within the parenchyma of caudal lobes but their efferent veins were strongly filled with blood. At autopsy, no pathological masses were noted within the mediastinum, lungs, and at the heart basis; and no thrombi were found in thoracic blood vessels. Based on clinical signs, results of thoracic radiography, and autopsy findings, cranial vena cava syndrome secondary to thoracic spinal spondylosis deformans was diagnosed.

Discussion

Cranial vena cava syndrome is seldom described in veterinary literature. However, its surgical treatment in life threat due to the presence of pathological masses within the mediastinum, thymus, or the heart basis, is possible. Even in a case with blood vessels invasion by tumour growth, the prognosis is not always poor (3, 5).

The development of clinical signs in CrVCS is usually slow and the syndrome is characterised by severe oedema of cranial portion of the body. The head, neck, and forelimbs are especially affected. Additionally to oedema formation, jugular venous distension and engorgement of conjunctival and sclerotic vessels are observed. In the case presented herein, the essential problem was dyspnoea, which appeared several days later but at the day of animal presentation the health state of the dog worsened markedly, suggesting sudden onset of disease. Clinical evaluation revealed signs typical for CrVCS, but oedema of the cranial part of the body was only moderate. The neck basis and forelimbs were only slightly affected and the head was unaffected. However, in the literature a case of CrVCS with only mild oedema of the ventral cervical region, lethargy, cough, and reduced exercise tolerance was described (3). In the present case, the most severe manifestation of cranial vena cava blood flow impairment was the distension of the jugular veins. Other sites of oedema formation were soft tissues and muscles beneath thoracic section of the spine, which were affected by vertebral changes. Additionally, autopsy revealed severe passive
hyperaemia of the cranial lobes of the lungs but caudal lobes were macroscopically non-affected. However, efferent veins of the cranial lobes were strongly filled with blood. Oedema of infraspinal muscles and other tissues was probably initiated by the compression due to osteophytes developed at the ventral aspects of thoracic vertebrae in the course of *spondylosis deformans*. On the other hand, oedema formation was strengthened by circulatory disturbances within these structures. Interestingly, in thoracic radiography, the trachea was only slightly displaced downward suggesting that there was no compression of thoracic structures by osteophytes visible in radiograph. However, autopsy revealed that the trachea was displaced laterally to infraspinal deformity.

The differential diagnosis of CrCVS, beside intrathoracic extravascular masses, took into account also thrombosis of the cranial vena cava or any mass, which could obstruct blood flow from *vena* into the right *atrium*, for example atrial neoplasms or adult heartworms *(Dirofilaria immitis)*. CrVCS was detected in 11 out of 17 dogs with cranial vena cava thrombosis, a state, which was caused by different factors including immune-mediated haemolytic anaemia, sepsis, neoplasia, renal disease, and others (7). However, in the presented case, there was no thrombus or other intravascular masses or lesions within the right atrium, which could interfere with venous return through the cranial vena cava. Clinical signs similar to those described in the presented dog are observed with *vena cava* syndrome (CS) associated with heartworm disease (2, 8). The syndrome is a serious complication of chronic heartworm disease where retrograde migration of adult heartworms from pulmonary arteries to the right ventricle, right *atrium*, and *vena cava* causes damage of the tricuspid apparatus (8). This infestation was not described in our geographic region where dogs have no contact with the parasite or do not live in areas of endemic occurrence of *Difilaria* sp.

In author’s knowledge, there are no similar reports in veterinary literature, and CrVCS caused by *spondylosis deformans* is unusual. The vertebral lesions were very distinct, that was surprising because of relatively young age of the affected dog. Clinical signs, which could suggest spinal problems, were absent or masked and vertebral lesions were incidentally discovered during thoracic radiography, which was made rather for the visualisation of thoracic masses. It seems that vertebral osteophytes, though developed markedly, probably could not be the only cause of compression and CrVCS, as mentioned above. In the differential diagnosis of CrCVS, besides different vertebral lesions, *discospondylosis deformans* should be regarded.

References