Aortic mineralization in a cat : A case report

Chiaki Morita, MVS[1], Yusuke Yamashita, BVS[2], Takayoshi Miyabayashi, BVS, MS, PhD, Dipl. ACVR[1]
1 : iVEAT Fukuoka Veterinary Specialty Practice, 2 : Aoi Animal Hospital

History

A 9-year-old, neutered male, American shorthair cat had resection of a bladder mass (urothelial carcinoma) at a university hospital and was discharged after three days. The following day, after subcutaneous fluid administration at a primary veterinary clinic, the patient developed dyspnea and diagnosed with pleural effusion, pulmonary edema, and cardiomyopathy. Pleural drainage and cardiac treatment (Pimobendan and ACE inhibitor) were initiated. Seven days later, the patient came to Aoi Animal Hospital for a second opinion.

Clinicopathological findings

The results of complete blood count and blood chemistry tests were within a normal limit except for a high SNAP NT-proBNP value. The blood pressure was a high end of normal (138 mmHg; normal value is <140mmHg).

Radiographic and CT imaging findings

Radiographic findings

On the right lateral view, there was mineralization of vascular wall from the aortic root to the abdominal aorta (Figure 1a). In the ventrodorsal (VD) and dorsoventral views, the left ventricular wall was attached to the thoracic wall, and cardiac enlargement was observed (Fig. 1b). In the VD view, the lengths of the left and right kidneys were 1.7 and 2.5 times of the length of the second lumbar vertebral body, respectively (Fig. 1b).

Computed tomography (CT) imaging findings

Mineralization was seen from the aortic root to the abdominal aorta (Figure 2a). Multi-phased, non-selective, intravenous contrast studies were performed. The interventricular septum and the papillary muscle appeared thickened (Figure 2c).

Discussion

In cats, aortic mineralization on thoracic radiographs was rare.[1-4] Two reports suggested that the causes of aortic mineralization were arteriosclerosis and metastatic calcification due to chronic renal failure.[1,2]

In dogs, aortic mineralization was also rare, and it was considered to have no clinical significance.[4]

In humans, however, the incidence of aortic mineralization on thoracic

radiographs was 1.9% in men and 2.6% in women (the ages of 30 and 89).[5,6] Furthermore, in humans, aortic mineralization was associated with arteriosclerosis, chronic renal failure, aging, and cardiovascular diseases.[6-9]

In humans, arteriosclerosis was the most common cause of aortic mineralization.[10] The risk factors of arteriosclerotic damage include hypertension, dyslipidemia, and diabetes mellitus.[11] In this case, dyslipidemia and diabetes mellitus were negative because of blood test results. In this patient, the blood pressure was within a normal range, but the value was in the high end.[12] Thus, hypertension could not be completely ruled out.

Metastatic calcification due to chronic renal failure was less likely in this patient. Although the patient had the left renal atrophy, serum calcium levels, urea nitrogen, and creatinine were all normal on the blood test. On contrast-enhanced CT imaging, renal excretory function appeared normal.

Aging could cause degenerative changes and dystrophic calcification of aorta. Thus, the aging factor could not be ruled out.

In human reports, aortic calcification was associated with cardiovascular diseases.[6,13] Aortic calcification could cause adverse effects on vascular compliance, vasomotion, and plaque stability.[14] Aortic stiffness including aortic calcification correlated with high left ventricular afterload.[13] In addition, cardiomyopathy could cause turbulence and reduced perfusion of blood in the aorta, which might have resulted in the aortic mineralization in this case.[15,16]

Finally, this patient had urothelial carcinoma of the bladder in the past. The aortic calcification might have occurred due to the presence of carcinoma. In the molecular biological aspects, osteopontin was a multifunctional glycoprotein that was involved in cardiovascular disease, cancer, and biomineralization (the process by which organisms produce minerals).[17] In humans, overexpression of osteopontin promoted carcinogenesis, progression, and metastasis of multiple malignant tumors.[18] Notably, in humans, increased osteopontin expression was observed in urothelial carcinoma of the urinary bladder.[19] We could not examined osteopontin in this patient.

In this case, both cardiac disease and urothelial carcinoma might be associated with the aortic mineralization.

Conclusion

When aortic mineralization was observed in cats, chronic renal failure and underlying diseases that triggers arteriosclerosis should be examined. In addition, cardiac diseases should be ruled out for a loss of aortic elasticity and increased

afterload. Molecular biological mechanisms such as osteopontin should be the subject of future research.

Figures and captions

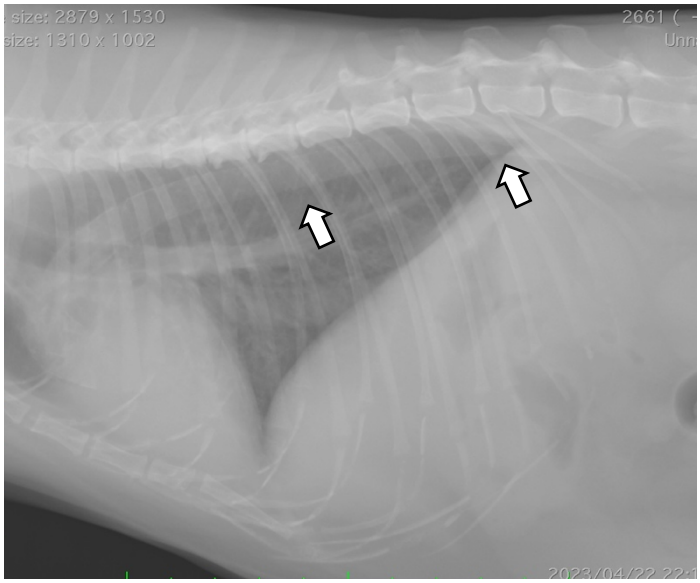

Figure 1 a

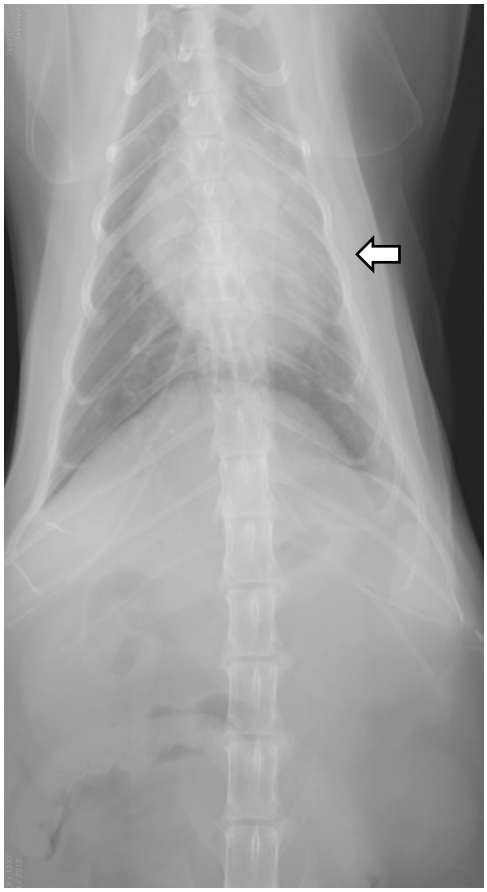

Figure 1 b

1a. Right lateral radiograph of the thorax and cranial abdomen (cropped). Mineralization of the aortic root to abdominal aorta is seen (arrows).
1b. Ventrodorsal radiograph of the thorax and cranial abdomen (cropped). The cardiac shadow is wide and appears enlarged. The left kidney appears smaller than normal (arrow).

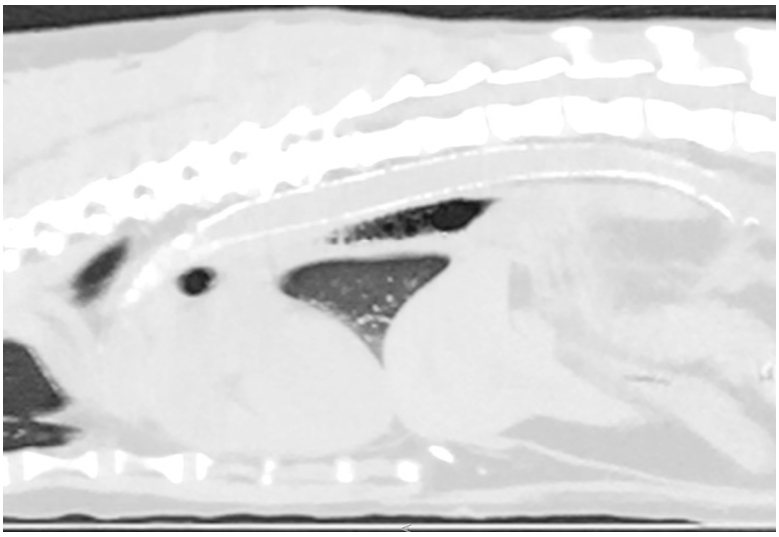

Figure 2a

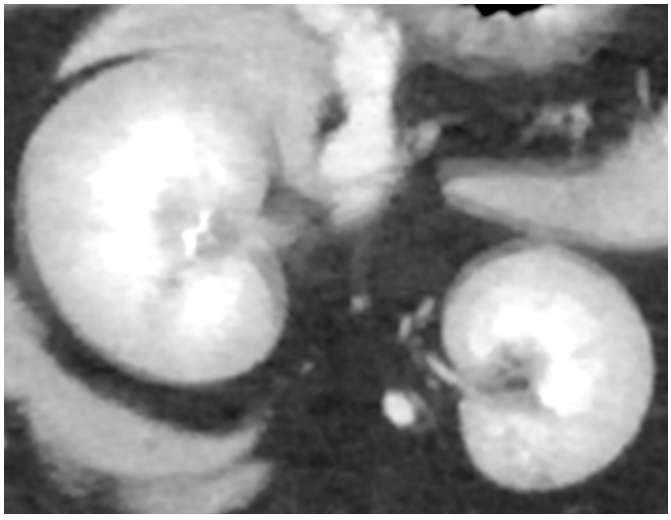

Figure 2 b

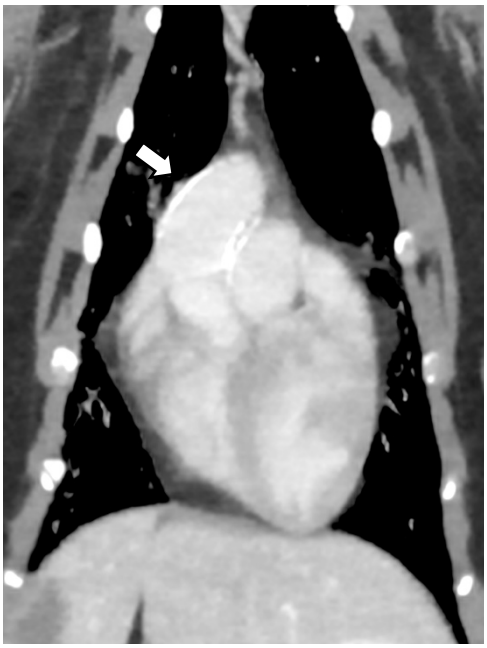

Figure 2 c

2a. Sagittal CT image. Mineralization is seen from the aortic arch to abdominal aorta. 2b. Dorsal CT image of the right and left kidneys in the portal venous phase. The sizes of the left and right kidneys were different, but the renal excretory function appears to be normal. 2c. Dorsal CT image of the thorax in the late arterial phase. Cardiac enlargement and mineralization of the aortic wall (arrow) were noted.

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