WHAT HAS THE AORTA BEEN TRYING TO TELL US ABOUT SYSTEMIC HYPERTENSION?

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Abstract:

Systemic hypertension (SH) is an insidious cause of target organ damage in our canine and feline patients. Human and veterinary literature provides clues about aortic remodeling in dogs and cats with SH. Aortic undulation and aortic knob formation will be discussed as a sentinel sign of SH in dogs and cats. Alterations in the size and shape of the three aortic cusps provide a reliable indicator of SH. Hopefully, recognizing aortic remodeling on thoracic radiographs and echocardiographic studies will provide earlier recognition, monitoring, and treatment for SH.

Keywords: Aortic remodeling, systematic hypertension, thoracic radiographs, echocardiogram, dog, cat

In canine patients, systemic hypertension can be seen in patients with diabetes mellitus, hyperadrenocorticism, acute or chronic renal disease, adrenal tumors, and associated with some medications (Palladia®, Proin®). SH in feline patients is typically found secondary to renal disease and hyperthyroidism. Idiopathic causes may account for up to 20% of cases in cats.

Systemic hypertension is currently classified as mild (150-159 mm Hg), moderate (160-179 mm Hg), or severe (greater than 180 mm Hg). Target organ damage (TOD), outlined by the 2018 ACVIM SH consensus statement, has listed the cardiovascular changes as concentric left ventricular hypertrophy, left-sided heart failure, and rarely aortic aneurysm. A minimal risk of target organ damage is thought to occur in animals with blood pressure under 150 mmHg.

Since the 1960s, uncoiling and dilatation of the thoracic aorta have been recognized on lateral radiographic studies in people with systemic hypertension. A bulge of the aortic arch/proximal descending aorta seen on the posteroanterior radiograph in people is referred to as the aortic knob. The aortic knob is considered target organ damage in people with SH. Echocardiographic aortic root dilatation and asymmetric dilation of the sinuses at the level of the aortic valves have been more recently identified as target organ damage in people with SH. Abdominal ultrasound and advanced imaging (computed tomography or magnetic resonance imaging) of the abdomen have been used to identify dilatation/aneurysm formation of the aorta secondary to systemic hypertension in people.

In canine patients, lateral thoracic radiographs showed variable aortic undulation and disproportionate enlargement of a portion of the aorta between the ascending and proximal descending aorta compared to the descending aorta cranial to the diaphragm. In dogs with SH, a ratio of the thoracic cavity caudal to the 3rd rib to aortic knob width from the ventrodorsal view was developed to account for the variability in the dog's sizes. The median ratio of the thoracic cavity to aortic knob was 3.4 in dogs with SH vs 4.1 in dogs with normal blood pressure. The asymmetric size of the three aortic cusps (>1.0 mm) strongly indicates SH in dogs. The ratio of the caudal abdominal aorta to the caudal vena cava is ~ 1:1 in unsedated canine patients with blood pressure in the normal range. The abdominal aorta may be increased in size relative to the

caudal vena cava in unsedated dogs with SH. The caudal vena cava can be decreased artifactually due to external pressure when scanning and volume depletion.

There is an overlap of radiographic and echocardiographic findings in cats with cardiomyopathy, thyrotoxic cardiomyopathy, and systemic hypertension, all having varying degrees of left ventricular hypertrophy. Identifying unique radiographic and echocardiographic parameters is needed to differentiate these diseases. Alterations in the size and shape of the aorta in cats with SH have been recognized in various case reports of echocardiographic studies and anecdotally noted as undulant or prominent aortic arches on thoracic radiographs. In contrast, a paper from 1993 deemed aortic undulation as an aging change in older cats without documenting normal blood pressure.

Echocardiograms were performed in all cats to rule out underlying cardiac diseases. Cats were assigned to the group with normal blood pressure if systolic blood pressure was less than 150 mmHg and to the SH group if it was over 150 mmHg. Seventy-six cats were included in each group for evaluation of the echocardiographic data. No differences were noted in the thickness of the interventricular septum or left ventricular free wall between the groups. The aortic diameter was significantly different between the groups. The average differences between the size of the three aortic cusps, in cats with normal blood pressure ranged from 0.15-0.25 mm. The aortic cusp size was considered altered when the differences in cusp size varied between ≥.5-3.5 mm. The aortic cusps were abnormal in size in 62/76 SH cats, 12/62 with ≥0.50 mm, and 50/62 at ≥1.0 mm differences.

Radiographic studies of the same cats were available for evaluation by two blinded reviewers for 46 cats with normal blood pressure and 49 cats with SH. The vertebral heart score was measured from the lateral projection and showed no difference between the groups. The

maximum distance of the aortic arch/proximal descending aorta to the spine and trachea on the ventrodorsal/dorsoventral projection showed significant differences between the groups. The best cut-off for detection of SH when measuring the aorta to the spine was 0.77 cm, a sensitivity of 84% and a specificity of 44%. The aorta to the trachea (when the margins were visualized) had the best cut-off of 1.12 cm with a sensitivity of 81% and specificity of 69%.

Variable left ventricular hypertrophy and occasional increased aortic diameter have been reported in cats with SH. No significant echocardiographic differences in left ventricular thickness were noted between the cats with normal blood pressure and cats with SH in this study. Left ventricular hypertrophy occurs as a late remodeling change in people with SH. Waiting for echocardiographic evidence of left ventricular hypertrophy has limited clinical application for preventing progressive TOD. Aortic root dilatation, including asymmetric aortic sinuses, is recognized as TOD due to SH in human medicine. Aortic dilatation precedes aortic aneurysm formation in people. In veterinary medicine, only aortic aneurysm has been included as TOD and likely underestimates the cardiovascular risk to our veterinary patients once aortic dilatation begins.

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