Progression of Subaortic Stenosis Detected by Continuous Wave Doppler Echocardiography in a Dog

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Although subaortic stenosis (SAS) in dogs is generally classified as a congenital heart disease,[1,2] previous reports in a colony of Newfoundland dogs[3] suggested that the stenotic lesion is absent at birth and that the obstruction develops and progresses thereafter. However, there are no reports of the clinical progression of SAS in dogs because serial measurements of the pressure gradient (PG) across the obstruction using cardiac catheterization require general anesthesia. This restriction has made it difficult to perform serial cardiac catheterization only for the evaluation of the severity of SAS in dogs. Continuous wave (CW) Doppler echocardiography can be used to measure aortic flow velocity noninvasively in unanesthetized dogs. The accurate maximum instantaneous PG across the stenotic lesion can be calculated from the peak velocity by applying the modified Bernoulli equation.[4,5] These characteristics of CW are advantageous in the serial evaluation of dogs with SAS. We serially measured the maximum instantaneous PG in a dog with SAS using CW over a 24-month period to assess whether there was progression of the severity of the obstruction.

Case Report

An 8-month old female asymptomatic Labrador Retriever with SAS was serially examined without sedation by echocardiography at 8, 12, 18, 24, and 32 months of age by one examiner. CW was performed using a commercially available system (EUB-165; Hitachi, Tokyo, Japan) with a 2.5-MHz transducer. The aortic flow velocity was interrogated from the left apical long-axis view with the dog in left lateral recumbency.

The Doppler beam was aligned to the aortic outflow tract using two-dimensional echocardiographic guidance. The intercept angle between the Doppler beam and the aortic outflow tract was from 10° to 15° on the two-dimensional images. Doppler gain settings were optimized by reducing the gain to the point where background noise disappeared. The peak velocity of aortic flow at each determination was derived by averaging 10 consecutive individual beats whose flow signal was well recorded; the echocardiographic data are summarized in Table 1. The peak velocity of aortic flow was 4.07 m/s at the first examination and progressively increased thereafter to 6.51 m/s at 32 months of age. The maximum instantaneous PG was calculated by applying the modified Bernoulli equation, $\Delta p = 4v^2$, where v is the peak velocity of the aortic flow (m/s); the maximum instantaneous PG increased progressively from 66 to 170 mm Hg during the period (Fig 1).

At 11 months of age, the dog underwent cardiac catheterization under general anesthesia with isoflurane for definitive diagnosis of SAS, and evaluation of its severity. The presence of a stenotic lesion at the left ventricular outflow tract was confirmed by left ventriculography, and a PG of 41 mm Hg was detected just beneath the aortic valve. No cardiac abnormalities other than SAS were detected. Cardiac catheterization was performed again at 32 months of age, along with balloon valvuloplasty; the PG increased to 112 mm Hg under the same condition.

The electrocardiogram remained normal during the study period. The left ventricular internal diameter and wall dimension were measured from the right parasternal short-axis view by M-mode echocardiography, and the values were derived by averaging 5 consecutive beats. The left ventricular end-diastolic posterior wall dimension (LVPWDD) increased from 7 to 14 mm, and the left ventricular internal end-diastolic diameter (LVEDD) decreased from 40 to 37 mm at 8 and 32 months of age, respectively. During the same period, the dog's body weight increased from 14.5 to 22.5 kg due to growth. Thus, to correct for potential differences of the values due to the increase of body size, each cardiac dimension was divided by the body surface area (BSA). The LVEDD/BSA ratio serially decreased, whereas the LVPWDD/BSA ratio increased during the period; therefore, apparent progressive concentric left ventricular hypertrophy occurred (Fig 1). The left ventricular fractional shortening and the ejection fraction calculated by the Teichholz method also increased.

Discussion

The systolic PG is generally an accurate predictor of the severity of the lesion in dogs with SAS.[1,2] Cardiac catheterization used to be performed before the development of Doppler techniques to measure the PG, and is still considered to be a standard method of measurement PG. Because general anesthesia is usually required to perform cardiac catheterization in dogs, it is difficult to justify serial cardiac catheterization to evaluate dogs with heart disease. CW Doppler can measure an instantaneous PG without invasion. It has been reported that the Doppler-derived PG value correlates well with that obtained by cardiac catheterization in both human beings and dogs with SAS.[4,5] Yuill and O'Grady[6] reported that the upper 95% confidence limit of the peak velocity of aortic flow obtained by CW Doppler in 20 normal dogs was 1.46 m/s. In contrast, the peak velocity of aortic flow in the dog in this report was 4.07 m/s at the initial examination, and the instantaneous PG was estimated to be 66 mm Hg, indicating moderate ventricular outflow obstruction. The PG measured by cardiac catheterization at 11 and 32 months of age was 41 and 112 mm Hg, respectively, values about 43% and 66% lower than the values obtained by CW Doppler. Cardiac catheterization on both occasions was performed under general anesthesia, and the anesthetic agent probably depressed cardiac output compared with that in the awake condition, thus decreasing the measured PG.

In this dog, the PG measured by both CW Doppler and cardiac catheterization increased during the 2-year period. The development of left ventricular hypertrophy also supported an increase of ejection resistance due to progression of obstruction. These results suggest that the severity of SAS in this dog was progressive, and that it is impossible to

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Accepted July 15, 1995.

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0891-6640/96/1002-0010$3.00/0

diagnose the severity of SAS based on a single measurement of PG, especially in puppies. Pyle et al[3] reported that some puppies in a colony of Newfoundland dogs with SAS and no cardiac murmur at birth developed systolic murmurs, PG, and accompanying lesions of SAS after 8 weeks of age.

In human beings with SAS, PG measured by either cardiac catheterization or CW Doppler also progressively increases.[7,8] It appears that typical SAS may not cause clinically important obstruction in infancy, and may even be absent in early life, and then become evident and progressively more severe. In this dog, progression of the obstruction might not have been recognized without measurements of PG or evaluation of concentric left ventricular hypertrophy, as the dog's general condition did not change throughout the 24-month period. The left ventricular stroke volume might be maintained by increases of the left ventricular fractional shortening and ejection fraction. Dogs with SAS often have few clinical signs of heart disease. However, sudden death may be the first clinical sign of SAS in dogs with severe obstructions.[1,2] Therefore, PG should probably be serially measured using CW Doppler in dogs with SAS.

Table 1. Echocardiographic Data

	Age (mo)				
	8	12	18	24	32
BSA (m^2)	0.60	0.69	0.79	0.79	0.79
Vp (m/s)	4.07	4.87	5.18	5.93	6.51
DPG (mm Hg)	66	95	107	141	170
CPG (mm Hg)	—	41	—	—	112
HR (beats/min)	57	137	60	93	89
LVEDD (mm)	40	34	37	34	37
LVESD (mm)	20	19	17	14	16
LVPWDD (mm)	7	10	12	15	14
LVPWDS (mm)	14	16	21	24	24
IVSD (mm)	11	16	16	17	16
IVSS (mm)	14	17	21	21	21
LVEF (%)	81	75	86	89	87
LVFS (%)	49	43	54	59	56

Abbreviations: BSA, body surface area; Vp, peak velocity of aortic flow; DPG, pressure gradient measured by continuous wave Doppler echocardiography; CPG, pressure gradient measured by cardiac catheterization; HR, heart rate; LVEDD, left ventricular internal end-diastolic diameter; LVESD, left ventricular internal end-systolic diameter; LVPWDD, left ventricular end-diastolic posterior wall dimension; LVPWDS, left ventricular end-systolic posterior wall dimension; IVSD, interventricular end-diastolic septal dimension; IVSS, interventricular end-systolic septal dimension; LVEF, left ventricular ejection fraction (Teichholz method); LVFS, left ventricular fractional shortening.

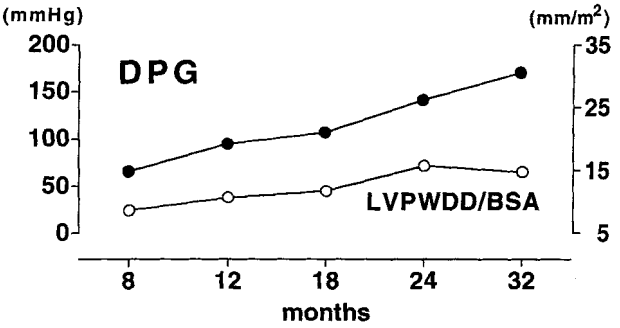

Fig 1. Graphic display of the progression of pressure gradient and left ventricular concentric hypertrophy in a dogs with SAS. DPG, continuous wave Doppler pressure gradient; LVPWD, left ventricular end-diastolic posterior wall dimension; BSA, body surface area.

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