Good Drugs for a Bad Heart

With hypertrophic cardiomyopathy (HCM), the most common feline cardiac disease, most cats progress to develop dynamic obstruction of the left ventricular outflow tract (LVOT). Beta blockers, particularly atenolol, are the standard drugs used to decrease LVOT obstruction in cats because of their negative inotropic and chronotropic effects. However, beta-blockers may cause adverse effects on inotropy and lusitropy and little data exist regarding their long-term use. Ivabradine is a new heart rate (HR) lowering drug that selectively lowers the pacemaker current in the sinoatrial node. This study compared the effects of atenolol and ivabradine on LVOT obstruction in cats with preclinical HCM.

Client-owned cats with preclinical HCM and dynamic LVOT obstruction ($n = 28$) received single PO dosages of ivabradine (0.3 mg/kg) or atenolol (2 mg/kg); HR, echocardiographic variables, and systolic blood pressure were evaluated before and 3 hours after administration. Peak velocity in the LVOT was significantly reduced with both drugs compared with baseline values; a greater effect was noted with atenolol versus ivabradine. Although reduction of HR was statistically significant with ivabradine use, the decrease in LVOT obstruction was considered clinically insignificant as echocardiographic indices of systolic function remained largely unchanged; this is in contrast to the clinically significant effects noted with atenolol. In conclusion, ivabradine appears safe to use in cats with preclinical HCM, as it does not worsen obstruction. However, it is inferior to atenolol (the current standard therapy) in its ability to decrease LVOT obstruction.

Commentary

Systolic anterior motion of the mitral valve (SAM) is frequently seen in cats with hypertrophic cardiomyopathy (HCM). When SAM can be reduced, the left ventricular hypertrophy can regress because outflow obstruction has been either reduced or completely eliminated. Beta-blocking agents, specifically atenolol, are used commonly to reduce SAM and are generally thought to be effective at this goal. Long-term follow up, however, in cats with HCM and SAM treated with atenolol have not shown any benefit. Thus, the search for something that makes a difference in survival for cats with HCM continues.

Ivabradine works similarly to atenolol in that it reduces heart rate, but its mechanism of action is such that there are no negative inotropic or lusitropic effects. The theory behind this study was that if SAM can be reduced with ivabradine similar to that seen clinically with atenolol, then it might be worth using ivabradine and avoiding the negative inotropic and lusitropic effects to see if just reducing heart rate has a positive effect on survival in cats with HCM and SAM. This study showed the negative inotropic effect is likely an important part of why atenolol is so effective at reducing the outflow tract obstruction and because ivabradine does not affect inotropic state, it was not as effective. For the practitioner, this means that we should continue to use atenolol for cats with SAM and HCM, and we are still unsure about whether this leads to a reduction in progression or increase in survival.—Amara Estrada, DVM, DACVIM (Cardiology)

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