Atrial Volume, Ventricular Shape Predict Mortality

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TORONTO — Increased left-atrial volume and abnormal left-ventricular geometry were each independent predictors of death among elderly patients with preserved systolic heart function in a study

with more than 11,000 subjects.

The findings suggest a potential role for left-atrial volume index and assessment of left-ventricular geometry in evaluating elderly patients, Dr. Dharmendrakumar A.



Patel said at the 14th World Congress on Heart Disease. Both parameters are measured by echocardiography.

A high left-atrial volume index may be an indicator of diastolic dysfunction, said Dr. Patel, a researcher at the Ochsner Clinic in New Orleans. But as of today, no interventions have proved to reduce left-atrial volume and thereby improve prognosis.

His study used echo results from 11,039 patients aged older than 70 years (average age 78 years) who were referred for an

echocardiographic examination at the Ochsner Clinic during 2004-2006. All of the patients had a left-ventricular ejection fraction of at least 50%, and their average ejection fraction was about 60%. None of the patients had severe valve disease.

During an average follow-up of 1.6 years, 1,531 of the patients (14%) died.

Analysis of mortality by left-atrial vol-

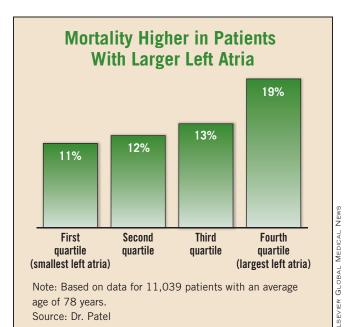
Left-atrial volume index and abnormal left-ventricular geometry contributed to mortality.

DR. PATEL

ume index showed that the patients in the quartile with the largest left atria had a 19% mortality rate, significantly higher than the 11% death rate among the patients in the quartile with the smallest left

atria. (See box.) The average left-atrial volume index was 32.5 mL/m² among the patients who survived during follow-up, and 35.7 mL/m² among the patients who died, a statistically significant difference, Dr. Patel said at the congress, sponsored by the International Academy of Cardiology.

Patients with abnormal left-ventricular geometry also had worse survival, compared with those with normal geometry. The mortality rate during follow-up was 12% among those with normal left-ven-



tricular geometry at baseline (about 50% of all people in the study), compared with 19% mortality among the 5% of patients with concentric, left-ventricular hypertrophy at baseline, the geometry that carried the highest mortality risk. Patients with concentric remodeling and those with eccentric hypertrophy also had significantly increased death rates, about 15%-16%, during follow-up.

A multivariate analysis showed that left-atrial volume index and abnormal left-ventricular geometry were significant, independent factors that contributed to mortality in these patients. Other significant mortality determinants were age, gender, body mass index, and left-ventricular ejection fraction.

An additional analysis further documented the additive risk posed by the two echo parameters that Dr. Patel studied. Among the quartile of patients with the highest left-atrial volume index,

those who also had a left ventricle with a concentric, hypertrophic shape had a strikingly high, 50% mortality rate during follow-up, he reported.

A limitation of this study was that it included only people who had been referred for an echocardiography examination. Also, Dr. Patel did not have information on the causes of death nor on the prevalence of comorbidities.

Tissue Doppler Imaging Identifies Heart Failure Risk

CHICAGO — Tissue Doppler imaging of the heart may be a way to safely and noninvasively screen asymptomatic people who are at risk of dying from heart failure, according to results from an initial study with 1,100 people.

In this pilot study, people in the lowest tertile of left ventricular motion had a 2.6-fold increased risk of dying over the next 5 years, compared with patients with the highest level of ventricular wall motion, in an analysis that controlled for age and gender, Dr. Rasmus Mogelvang said at the annual meeting of the American College of Cardiology.

Although the finding suggested that tissue Doppler imaging (TDI) may be effective for the early detection of heart failure and an increased risk of death due to heart failure, the data collected so far are preliminary and did not allow Dr. Mogelvang and his associates to calculate a threshold value for increased risk.

"This is a first step toward showing that TDI has promising significance," said Dr. Mogelvang, a cardiologist at Gentofte Hospital in Copenhagen. "Before we can start to use this in daily practice, we need to set cutoff values."

TDI can "clearly measure systolic and diastolic function in the heart. Putting them together [as a single measure of ventricular performance] is novel," commented Dr. Scott D. Solomon, director of noninvasive cardiology at Brigham and Women's Hospital in Boston, and an expert on TDI. "If the results [from Dr.

Mogelvang's study] are validated in another dataset, then TDI could become an important screening test," he said.

Dr. Mogelvang and his associates used TDI data collected on 1,100 apparently healthy people (average age, 60 years) enrolled in the Copenhagen City Heart Study. They all underwent ventricular assessment using both TDI and conventional echocardiography, and were then followed for an average of 5.1 years. During follow-up, 90 people died.

Three TDI measurements were made for each subject: s', which corresponds to left ventricular wall motion at peak systole; e', which is wall motion between systole and diastole; and a', wall speed at end diastole. People with slower ventricular wall motion had worse survival. Older people have reduced wall-motion speed.

In a series of multivariate analyses that adjusted for baseline differences in age and gender, a combined wall-speed assessment that included readings for s', e', and a' was the best correlate of survival, Dr. Mogelvang reported. In absolute terms, 5-year survival was about 96% among people in the tertile with the greatest wall motion, about 92% among those in the middle tertile, and about 85% among those in the tertile with the lowest level of wall motion. The combined TDI value, which integrated s', e', and a', was a powerful predictor of survival even in people who had normal ventricular function based on their conventional echocardiogram, he said.

Atenolol May Equal Carvedilol For Heart Failure Survival

CHICAGO — Atenolol may be as effective as carvedilol for improving survival and reducing hospitalizations in patients with systolic heart failure, based on a retrospective review of more than 1,000 unrandomized patients.

The finding suggests that atenolol should be tested in a prospective, randomized study to definitively test whether it works as well as carvedilol in patients with heart failure, Dr. John R. Kapoor and his associate said in a poster presented at the annual meeting of the American College of Cardiology.

Currently, the only β -blockers approved by the Food and Drug Administration for use in patients with heart failure are carvedilol and metoprolol succinate, an extended-release formulation of metoprolol, said Dr. Kapoor, a cardiologist at Stanford (Calif.) University. Another β -blocker, bisoprolol, has also proved to help patients with heart failure, but in the United States bisoprolol is approved only to treat hypertension. Atenolol, another β -blocker, is not approved by the FDA for treating heart failure but is often prescribed for that purpose, Dr. Kapoor noted.

To estimate atenolol's efficacy in heart failure patients, the Stanford researchers reviewed 1,385 consecutive patients who had their left ventricular ejection fraction measured by echocardiography at the VA Palo Alto Health Care System during 1998 and 2004 and were found to have an ejection fraction of 40% or less. The study then focused on the 1,162 patients from

this group who were treated with either carvedilol, atenolol, or metoprolol tartrate (an immediate-release formulation of metoprolol). The primary outcome of the analysis was death within the following 6 months; secondary end points were heart failure hospitalization, and death plus hospitalization during 6 months of follow-up. The average age of the patients was 68, and virtually all of the patients were men.

The mortality rate was lowest, 1.3%, among the 251 patients (22%) treated with atenolol. Among the 611 patients (53%) treated with carvedilol, 2.5% died; and among the 300 (26%) treated with metoprolol, 6% died.

After adjustment by a propensity score analysis, patients treated with atenolol had a slightly reduced risk of death, compared with patients treated with carvedilol, but the difference between the two drugs was not statistically significant. After propensity score adjustment, patients treated with metoprolol tartrate were about twice as likely to die as were patients treated with atenolol, a difference that just reached statistical significance.

Adjusted analyses were not reported for the secondary end points. Unadjusted findings showed that the patients treated with atenolol consistently fared better than did those treated with either carvedilol or metoprolol tartrate for both heart failure hospitalizations and for hospitalizations plus deaths. Atenolol treatment was linked with superior outcomes at 90 days, 1 year, and 2 years after the start of treatment.