Aldosterone synthase variant predisposes HF patients to AF

29 July 2008

MedWire News: A polymorphism in the promoter region of the gene encoding aldosterone synthase is associated with the presence of atrial fibrillation (AF) in patients with heart failure (HF), research suggests.

The importance of the renin-angiotensin-aldosterone system in vascular health is well established where it appears to play a role in the pathogenesis of both HF and AF.

"The final step in the aldosterone synthetic pathway is through an enzymatic reaction catalyzed by aldosterone synthase," say the researchers.

The promoter of aldosterone synthase gene (*CYP11B2*) contains a polymorphism that reportedly influences aldosterone synthase activity. The CC genotype of this polymorphism (T344C), which is associated with increased aldosterone levels, has been linked to poor prognosis in HF patients.

Basil Lewis (Lady Davis Carmel and Lin Medical Centers, Haifa, Israel) and team identified this genotype in 55 of 196 patients with symptomatic HF of at least 3 months duration.

They found that 45% of patients with the CC genotype had AF, compared with just 27% of patients with other genotypes ($p=0.01$).

The CC genotype remained associated with AF after accounting for confounders including age, gender, HF severity, and echocardiographic parameters. It increased the likelihood of carriers having AF 2.35 fold ($p=0.03$).

"Beta-blocker therapy, with known renin-angiotensin system antagonistic characteristics, has been suggested to decrease AF prevalence in patients with systolic HF," the researchers comment in the *American Journal of Cardiology.*

"More specific therapy with direct aldosterone antagonists may offer stronger anti-remodeling properties," they speculate. "This concept, especially in the *CYP11B2* CC genotype subpopulation, and may potentially decrease AF prevalence in these patients."

Am J Cardiol 2008; **102**: 326-329