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Do Statins Decrease the Arrhythmia Burden in Patients with Paroxysmal Atrial Fibrillation?

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Hypothesis

Atrial Fibrillation could be secondary to inflammation of atrial tissue. Since statins have known anti-inflammatory properties, can they affect prevent episodes of arrhythmias in patients with known paroxysmal atrial fibrillation?

Methods & Materials

This was a prospective randomized trial of 106 patients who had an atrial based pacemaker therapy initiated for significant bradycardia. Fifty-two patients (23 males, 70 +/- 13 years old) were randomized to the statin group (atorvastatin 20 mg/d) and 54 (25 males, 72 +/- 13 years old) to the non-statin group. The effect of atorvastatin on time to the first attack of AF or AHE (> or =180 per minute and > or =1 or 10 minutes), which was accurately detected by pacemaker interrogation at 1,3,6 and 12 months of follow-up.

Results

Event-free survivals from AHE > or =1 minute were not significantly different between the statin and non-statin groups (55% vs 60%, p = ns). AHE ≥ 10 minutes were significantly lower in the statin group (n= 3 (5.8%)) compared to the non-statin group (n=10 (19.2%)) after 1 year of follow-up (p = 0.041). Mean monthly AHEs lasting > 10 minutes was significantly lower in the statin group compared to the control group (0.06 vs 0.25, p=0.028). The mean left atrial volume of the statin group was significantly lower than that of the nonstatin group at the end of follow-up (39.7 +/- 1.7 vs 43.7 +/- 1.9 cc, p < .0001).

Conclusions

Atorvastatin is effective in preventing significant AF (> or =10 minutes) and left atrial enlargement in patients with bradyarrhythmias and implantation of a pacemaker.

Commentary

This prospective study by Tsai CT et al is a good effort studying the effects statins on the arrhythmia burden in patients with atrial fibrillation. Statins have been shown the promise of decreasing AF in previous observational studies. If atrial

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high rate episodes (AHE) can be equated to true paroxysms of AF, the data from the pacemakers can accurately quantify the true arrhythmia burden in patients with AF. The strengths of this study are its prospective randomized control design and accurate quantification of arrhythmia burden. Some of the limitations are the lack of pre study arrhythmia burden assessment and relatively small number of patients. There were no details regarding the type of antiarrhythmic drug therapy prior to the initiation of statins. This is limited to a subset of patients who mostly have tachybrady syndrome and those with bradycardia without known AF. Although, they are an important subgroup of patients with AF, they may not represent all AF patients across the board. It is unclear how statins affect the atrial substrate and arrhythmia burden in those more prolonged persistent forms of the arrhythmia. Additional details on changes in inflammatory markers between the two groups would have been helpful. Despite the low incidence of AHE/AF episodes interestingly resulted in significant reduction of LA volume in patients treated with statins. The results are promising and a large well designed prospective study is required to meaningfully validate the results of this study.