

Rosuvastatin slows the development of diastolic dysfunction in calcific aortic stenosis

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Authors

Luis M Moura, Sandra F Ramos, Steen D Kristensen, Fausto J Pinto, Isabel M Barros, Francisco Rocha-Gonçalves

Abstract

Background and aim of the study

The study aims were to test the effect of rosuvastatin on the progression of left ventricular (LV) diastolic function in patients with aortic stenosis (AS), and to evaluate the use of natriuretic-peptide (BNP) as a marker of diastolic dysfunction in this condition.

Methods

Sixty-one hypercholesterolemic, consecutive new referrals with moderate AS were administered rosuvastatin (Crestor™) 20 mg/day for 18 months, while a further 60 subjects with normal cholesterol levels remained untreated. The LV diastolic function was determined using conventional Doppler echocardiography, tissue Doppler imaging (TDI); BNP plasma levels were monitored when subjects entered the study and then assessed prospectively at six-month intervals until the study end.

Results

After an 18-month (mean 73 ± 24 weeks) period of treatment with rosuvastatin (Tx group), patients showed a significantly better diastolic function than untreated subjects (uTx group), as indicated by an isovolumic relaxation time (IVRT) (Tx 102.0 ± 42.8 versus 97.2 ± 19.1 ; $p < 0.001$; uTx 99.7 ± 21.7 versus 95.2 ± 21.8 ms; $p = 0.032$), E/A ratio (Tx 1.0 ± 0.6 versus 0.9 ± 0.3 , $p = 0.52$; uTx 1.2 ± 0.40 versus 0.9 ± 0.30 versus, $p = 0.006$), and E/E' ratio (Tx 11.4 ± 1.5 versus 11.4 ± 1.8 , $p = 0.19$; uTx 15.4 ± 1.2 versus 12.3 ± 1.5 , $p < 0.001$). Similarly, at study end, plasma levels of BNP were significantly lower in the Tx group than in the uTx group [median (1st-3rd quartiles): 37.0 pg/ml (20.1-65.2 pg/ml) versus 57.1 pg/ml (46.9-98.2 pg/ml); $p = 0.017$].

Conclusion

The results of this prospective follow up study of asymptomatic patients showed that rosuvastatin treatment delays the progression of diastolic dysfunction in moderate AS when assessed using hemodynamic echocardiographic parameters or by the release of plasma physiological markers. Hence, the benefits of statin treatment in AS, which are known to affect the valve endothelium, also extend to changes affecting myocardial function itself. © Copyright by ICR Publishers 2012.