Statin inhibits synthesis of type I collagen in patients with rheumatic heart disease

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Background Rheumatic heart disease (RHD) remains a health burden in developing countries with lack of pharmacological agents to slow this disease process. Increasing of collagen synthesis plays an important role in the progression of fibrosis, thickening and calcification of cardiac valves in patients with RHD. Statins have been known to have anti-fibrotic and anti-inflammatory effect; however their effect on on the synthesis of type I collagen, primary collagen component of human cardiac valves, has not been elucidated.

Purpose To evaluate the effect of simvastatin on the synthesis of type I collagen in patients with RHD

Methods This experimental randomized pretest-posttest control group study was performed in 31 RHD patients confirmed by echocardiographic finding. Patients with age > 75 years old, unstable hemodynamic, previous percutaneous or valvular heart surgery, renal failure, and previous statins treatment were excluded. Treatment and control groups received a standard medical therapy with (16 patients) and without (15 patients) simvastatin 40 mg/day for 4 weeks, respectively. Carboxy-terminal propeptide of type I procollagen (PICP) is used as a marker of type I collagen synthesis. PICP blood serum was taken from peripheral vein blood and measured by ELISA method prior and after 4 weeks treatment.

Results There were no significant differences in clinical and echocardiographic baseline paramaters between treatment and control groups. Most of cardiac valve abnormality was mitral stenosis concomitant with atrial fibrillation. Prior serum level of PICP were similar between treatment and control groups (709±269 versus 671±242 ng/mL, p=0.699). A significantly lower of PICP serum level was found in statin treated group than untreated patients (583±236 versus 879±316 ng/mL, p=0.008). There were minor side effect reported, including mild myalgia and nausea in two (2) patients treated with statin.

Conclusion Simvastatin may reduce synthesis of type I collagen in patients with RHD which could represent potential pharmacological agents to slow the disease process.