Reply to the Letter to the Editor

Reply to Paraskevas

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Keywords: Aortic stenosis; Statins; Aortic valve replacement

In reaction to 'Prescribing statins in aortic stenosis: Little to lose, much to gain' [1], we would like to provide the readership of this journal with some additional information concerning statin use – or underuse – in our study population.

In our observational prospective cohort study, we described treatment strategies in symptomatic patients with severe aortic stenosis in the Rotterdam area, the Netherlands [2]. In the patients observed, the so-called medical or conservative treatment was mostly aimed at relief of symptoms by diuretics, treatment of atrial fibrillation and systemic or pulmonary hypertension. To prevent endocarditis, all patients were treated with prophylactic antibacterial treatment before starting non-sterile surgical procedures.

Approximately only half of the patients received lipid-lowering drugs: 54% of the 76 patients in the aortic valve replacement (AVR) group and 47% of the 101 patients in the 'medical/conservative' group.

Although we only documented drug prescriptions and have not studied why certain patients received statins (and why many did not), we doubt statin usage was aimed at slowing the progression of aortic stenosis. It is more likely statins were prescribed for (cardio-)vascular co-morbidity or dyslipidaemia.

Statins may interfere with the progression of aortic stenosis, but to what degree and until which disease stage remains uncertain and has yet to be established in larger prospective series. Dr Paraskevas refers to a cohort study in which statins slowed the haemodynamic progression in patients with asymptomatic moderate-to-severe aortic stenosis [1], others reported no clear effect on the progression of moderate-to-severe aortic stenosis in a recent randomised trial [3]. Further, the SEAS trial he refers to, concerned patients with asymptomatic mild-to-moderate aortic stenosis [1,4]. In his comprehensive review, Dr Paraskevas concludes statins improve cardiovascular outcomes in surgical patients (either coronary artery bypass graft (CABG) or patients who need valve replacement or other thoracic surgery) [5]. However, it remains to be seen whether statin therapy is useful in the cohort we studied: the symptomatic patient with severe aortic stenosis in whom the decision to operate or not is yet to be made. Although interesting, this will be difficult to study because co-morbidities, age, the advanced stage of the valve stenosis and treatment selection probably play a major role in clinical outcome.

On the other hand, of course, one could also argue there is not much to lose. Since we have no hard data of our own to either support or reject this statement, we leave this subject open for debate. Nevertheless, we are grateful for Dr Paraskevas’ enthusiastic comments and discussion [1].

References


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Letter to the Editor

Independent predictors of postoperative atrial fibrillation

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An interesting study performed by Choi et al. [1] concluded that a low left ventricular ejection fraction (LVEF), highest C-reactive protein (CRP) before the onset of atrial fibrillation (AF), vasopressor therapy and packed red blood cell (pRBC) transfusion remained as independent predictors of postoperative AF. There has been no definitive cause for postoperative AF identified. Large shifts in fluids, pericarditis and changes in autonomic nervous system have been implicated. Inflammation triggers and maintains AF. Studies have demonstrated an association between AF and CRP concentration [2]. CRP concentration is elevated in patients with left atrial dysfunction without AF, and atrial dysfunction itself can trigger AF after surgery [3]. Prophylactic usage of beta blockers, amiodarone, corticosteroids and statins has reduced the incidence of postoperative AF [4,5]. These medications should be considered in postoperative patients with low LVEF, increased CRP concentration and those receiving vasopressor therapy and increased pRBC transfusion. Authors can use this study or conduct larger trials to identify risk calculator for postoperative AF.
Reply to Letter to the Editor

Reply to Ramaraj

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Keywords: High sensitive C-reactive protein; Postoperative atrial fibrillation; Transfusion

We thank Dr Ramaraj for his comments [1] and interest in our article. We agree with his opinion that prophylactic strategies should be considered in postoperative patients with independent predictors of postoperative atrial fibrillation (AF), which were low left ventricular ejection fraction, increased high sensitive C-reactive protein, vasopressor therapy and packed red blood cell transfusion, according to our study [2]. As Ramaraj points out, evidence in the current literature strongly supports the efficacy of various prophylactic strategies on reducing the relative risk of AF following cardiac surgery (by 20–50%), which include β-blockers, amiodarone, sotalol and atrial pacing [3,4]. However, regardless of the efficacy in AF prevention, controversies exist regarding the subsequent improvement of patients’ outcome and reduced resource utilisation. As mentioned in our article, an inherent pro-arrhythmic property of these various agents has resulted in bradyarrhythmias, hypotension and QT prolongation, probably nullifying the beneficial effects of AF prevention on patients’ outcome. Therefore, identifying patients at an increased risk of developing postoperative AF could lead to more targeted preventive interventions, thereby reducing anti-arrhythmic-related side effects and possibly improving patients’ outcome. Based on our results, further studies addressing the efficacy of perioperative strategies aimed at attenuating inflammation or minimising risk of transfusion on reducing the risk of AF development as well as improving patients’ outcome would add value to the literature.

References


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