
We thank Drs Chhabra and Spodick for their interest in our publication.1 Indeed they raise interesting questions for which we provide some answers in the following paragraphs.

We do think there may be important differences in the mechanism of post ablation pericarditis and of idiopathic pericarditis and thereby for recurrences also. For example, recurrences are highly common in idiopathic pericarditis (10%–30%),2 but occurred in only 1 of 85 patients (1%) in the present study.

Two additional diagnostic criteria for pericarditis are mentioned by Drs Chhabra and Spodick, namely, pericardial effusion on echocardiography and pericardial rub. We think these criteria are not as valuable in this context as there is always some amount of fluid on echo after epicardial ventricular tachycardia ablation, and a pigtail catheter is left in situ in the pericardial space. In addition, much care was taken to distinguish between pain at the postoperative site (entry point of the pigtail catheter) and pericarditic pain. As we do not puncture the pleura, there is no reason for truly pleuritic pain.

Although we recognize the value of controlled trials, there were few patients with new-onset atrial fibrillation, and we thus opted to be careful with our conclusions. We fully agree that the presence of underlying structural heart disease may increase the likelihood of postprocedural atrial fibrillation. Of the patients with new-onset atrial fibrillation, 2 had no structural heart disease, whereas 1 had dilated cardiomyopathy and 4 had scars of unknown origin. For interest, there were 2 patients with new-onset atrial fibrillation in the no steroid group, 2 in the systemic steroid group, and 3 in the epicardial steroid group. Unfortunately, further answers can only be speculative as the numbers are too small for statistically meaningful comparison and for correction for underlying heart disease. Furthermore, the limited number of subjects precludes any subgroup analyses in patients with and without beta-blockers and with and without antiarrhythmic drugs.

The higher incidence of NSAIb use in patients who received intra-pericardial steroids may be because of increased awareness of pericarditic chest pain after epicardial mapping in more recent patients. However, NSAIDS were generally avoided in patients with a poor ejection fraction and with renal failure, which may have contributed to the distribution of NSAIb use among the treatment groups. Indeed, this is a limitation related to the observational nature of our study.

Colchicine may indeed be a promising therapeutic option in patients undergoing epicardial mapping.3,4 Whether a single intra-pericardial injection of triamcinolone attenuates the effect of colchicine treatment, as has been observed in patients who had received prolonged treatment with systemic steroids,5 remains uncertain. However, the single use of triamcinolone without any associated gastrointestinal side effects renders it an appealing therapeutic option.

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