In more general terms, we certainly agree with Dr Martinez-Sellés that coronary vasospasm does exist, and it is a pivotal, often clinically unrecognized, mechanism underlying virtually all forms of ischaemic heart disease, from stable angina to unstable angina to acute myocardial infarction and sudden death. The landmark studies of coronary vasospasm were performed in the Institute of Clinical Physiology by Professor Attilio Maseri in the seventies. After 30 years, we certainly did not forget the Maseri lesson, and we agree with Martinez-Sellés that the single most important factor affecting the frequency with which variant angina is recognized depends on the physician’s awareness of its existence. However, it is also true that it is not enough to be aware of coronary vasospasm to detect it.

The occasional occurrence of coronary vasospasm during dipyridamole infusion and the absence of clear-cut signs of coronary spasm exclude, at least in our patient population, a significant role of coronary vasospasm in the prognostic power of stratification of a positive stress echo with normal coronary arteries.

References


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New evidence of anti-inflammatory interventions in atrial fibrillation

We read with great interest the recent elegant review of Boos et al.,1 which presents an overview of the evidence of inflammation and anti-inflammatory therapies in atrial fibrillation (AF). We agree with the reviewers that some drugs which have anti-inflammatory characteristics, such as ACE-inhibitors, ARBs, statins, steroids, fish oils, and vitamin C, might be useful in the management of AF. In addition, we would like to add some new evidence of anti-inflammatory interventions in AF.

Recently, another two studies evaluated the role of statins in the prevention and treatment of AF. Marin et al.2 studied 234 consecutive patients who underwent coronary artery bypass grafting (CABG) and observed that statin use was significantly associated with a decreased incidence of post-operative AF and increased TIMP1/MMP1 ratio. In a single-blind prospective study, Dernellis et al. 3 randomized 80 patients with paroxysmal AF to atorvastatin or placebo. After 4–6 months of therapy, treatment group exhibited a highly significant reduction in paroxysmal AF and C-reactive protein levels. However, in a recent post hoc analysis of a large randomized clinical trial, MIRACL study,4 intensive statin treatment did not appear to prevent new AF in the 16 weeks following acute coronary syndrome. Therefore, more randomized trials of statins in patients with AF may be warranted.

Another two promising anti-inflammatory therapies have also achieved positive results in the setting of post-operative AF. Merritt et al.5 demonstrated that carvedilol, a new β-blocking agent which has anti-oxidant properties, significantly reduced post-operative AF when compared with metoprolol or atenolol. Furthermore, a randomized open label trial performed by Cheruku et al.6 also showed that non-steroidal anti-inflammatory medications significantly reduced the incidence of AF and shortened the length of hospitalization after CABG. Therefore, inflammation may be a new therapeutic target in the management of AF. More studies of anti-inflammatory interventions are needed to clarify this important issue.

References


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