Dipyridamole echocardiography test in patients with normal or near normal coronary arteries

We read with great interest the paper by Sicari et al.1 dealing with the prognostic value of dipyridamole echocardiography test in patients with chest pain syndrome and angiographically normal or near normal coronary arteries and the accompanying editorial by Marwick.2 This study, based in a large cohort with long-term follow-up, shows that dipyridamole echocardiography can identify a subgroup with a less benign prognosis.

Intravenous aminophylline is a potent coronary vasodilator that has been extensively investigated over the past several years in the non-invasive assessment of patients with suspected coronary artery disease. On the other hand, the ability of dipyridamole to provoke coronary artery spasm in patients with variant angina and angiographically normal coronary arteries is less well known.

Although the mechanisms of dipyridamole-induced coronary artery spasm remain unclear, this 'false-positive' test response in patients with normal coronary arteries can be induced by dipyridamole3,4 or by aminophylline.5,6 Aminophylline is a dipyridamole antidote, routinely infused at the end of the test, which produces an abrupt withdrawal of coronary artery vasodilatation which may trigger coronary artery spasm in patients with variant angina.

Fujita et al.7 found a sensitivity of 69% (11 of 16) for dipyridamole echocardiography in patients with coronary artery spasm and normal or nearly normal coronary arteries. Sicari et al.1 and Marwick2 do not mention the ability of dipyridamole echocardiography to provoke coronary artery spasm in patients with normal or near normal coronary arteries and variant angina. However, it is conceivable that the prognostic value of this test could be due, in part, to the detection of patients with spontaneous coronary artery spasm.

References


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Manuel Martı́nez-Sellés
Department of Cardiology
Hospital Universitario Gregorio Marañón
Dr Esquerdo, 46
28007 Madrid
Spain
Tel/Fax: +34 915868276
E-mail address: mmselles@secardiologia.es

Tomás Datino
Department of Cardiology
Hospital Universitario Gregorio Marañón
Dr Esquerdo, 46
28007 Madrid
Spain

Ana Pello
Department of Cardiology
Hospital Universitario Gregorio Marañón
Dr Esquerdo, 46
28007 Madrid
Spain

Dipyridamole echocardiography test in patients with normal or near normal coronary arteries: reply

We thank Dr Martı́nez-Sellés for the interest in our work and on the very appropriate comment focusing on this important issue, i.e. the potential correlation of coronary vasospasm in determining true ischaemia and wall motion abnormalities in patient with angiographically non-significant coronary artery disease. It is well known for 20 years that aminophylline administration may trigger coronary vasospasm in about one-third of patients with variant angina.3 The patient population under investigation pooled together subjects with different clinical conditions and heterogeneous angiographic patterns. Patients with variant angina were also enrolled in the study (the vasodilating stress test is used in this set of patients to exclude significant forms of coronary artery disease) but none of the patients experienced coronary vasospasm after aminophylline infusion. Moreover, none of the patients in our study with a positive test experienced an ST-segment elevation during dipyridamole infusion (prior to aminophylline infusion), indicating the presence of coronary vasospasm. However, as Martı́nez-Sellés correctly points out, there have been anecdotal reports of coronary vasospasm—documented by simultaneous electrocardiographic monitoring—occurring at the end of dipyridamole testing in patients subsequently shown to have variant angina. Nonetheless, the occurrence of this event is very uncommon and it is unlikely to have occurred in the patient population under investigation.

In our opinion, the results of the study by Fujita et al.,2 describing reversible perfusion abnormalities during dipyridamole stress in patients with variant angina and normal or near-normal coronary arteries are not consistent with vasospasm occurrence. In fact, 14 out of 16 patients had history of hypertension (n = 5), and/or diabetes mellitus (n = 2), and/or hypercholesterolaemia (n = 9). All these factors are well-established causes of altered microcirculation as evidenced by perfusion scintigraphy and/or coronary flow reserve studies.1,4 In the same patient population, authors could not demonstrate a significant agreement between the presence of a perfusion defect and the angiographic site of coronary vasospasm, indicating that the altered coronary flow reserve could be attributed to other causes. In their conclusions, the authors state that perfusion defects are due to a concomitant microvascular dysfunction in patients with variant angina. In this particular case, we can evoke the ‘alternate’ or ‘inverse’ ischaemic cascade5 which refers to a sequence of clinical events during which the occurrence of wall motion abnormalities usually cannot be proved in spite of frequent occurrence of chest pain and ST-segment depression, although in a subset of patients, a reduction in coronary flow reserve and/or a metabolic evidence of inducible ischaemia has been described.

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