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Implantation of cardioverter defibrillator in post-myocardial infarction patients: when and how?

We read with great interest the recently published guidelines in the European Heart Journal ‘for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death’. In chapter XIV, page 2115, it is recommended (class I) that an implantable cardioverter defibrillator (ICD) should be implanted at least 40 days post myocardial infarction (MI) in patients with left ventricular dysfunction with an ejection fraction (EF) of 30–40% and NYHA functional class II or III. We presume that this recommendation is based on the results of MADIT II trial.

MADIT II trial clearly demonstrated that the Kaplan–Meier estimates of survival in the two groups began to diverge at approximately 9 months and continued their separate paths thereafter favouring the group who received an ICD. In our opinion, there is no obvious or reasonable explanation for this delay. If we wanted to follow strictly the outcome of MADIT II, we would recommend an ICD implantation not earlier than 9 months post MI. There is strong evidence to support this hypothesis although its mechanism is not entirely clear. Furthermore, according to the results of MADIT I trial, clear benefit from ICD implantation was documented in post MI patients with NYHA functional class I, II, or III, a left ventricular EF <0.35, documented episodes of asymptomatic non-suppressible ventricular tachycardia (VT) and inducible non-suppressible VT on electrophysiologic study. Therefore, based on the results of these two large-scale trials, it seems reasonable to treat post MI patients according to MADIT I data during the first 9 months, and according to MADIT II data thereafter. This strategy may reduce the number of unnecessary ICD implantations.

In view of the recent developments in the treatment of acute MI, it is more than evident that ischaemic cardiomyopathy is an evolving area. Does this mean that new studies are required to resolve this issue?

References


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Role of delayed enhancement MRI in patients with acute coronary syndrome and unobstructed coronary arteries

We read with interest the paper by Assomull et al. on the use of magnetic resonance
imaging (MRI) in patients with acute chest pain, raised cardiac troponin, and angiographically normal coronary arteries. A diagnosis of myocarditis and cardiomyopathy was made in 50 and 3.4% of cases, respectively. Somewhat unexpectedly, only 11.6% of the population showed myocardial infarction, although it is conceivable that, among patients with a normal MRI scan, myocardial ischaemia might have caused a small increase in circulating troponin.

The authors, however, do not provide any detail on the underlying cause of myocardial damage in patients with myocardial infarction and in those without delayed enhancement at MRI. Was any additional test performed to identify non-obstructive coronary atherosclerosis, vasospasm, or microcirculatory dysfunction? Because therapeutic measures vary between these conditions, their discrimination is extremely important. Indeed, the authors should have qualified more conclusively the definition of 'unobstructed coronary artery'. Did the coronary arteries appear homogeneously smooth at angiography, or rather, did they present an irregular contour, pointing to potentially unstable atherosclerotic plaques?

Pathophysiology cannot be inferred, though, from coronary 'lumenography' alone, and additional indicators must be sought with other imaging modalities. Combined positron emission tomography/computed tomography, for example, could become the gold standard for this analysis, though its drawbacks due to cost, radiation exposure, and lack of general availability. MRI, on the other hand, is not limiting. However, in patients with ischaemic heart disease and normal coronary arteries, delayed enhancement MRI alone provides insufficient information on the pathogenesis of myocardial damage and, by extension, is hardly able to guide therapy. In conclusion, myocardial ischaemia needs to be qualified in its underlying cause.

In the present study, although it is conceivable that, among patients with a normal MRI scan, myocardial ischaemia might have caused a small increase in circulating troponin.

We thank Coceani and L'Abbate for their comments on our paper. We concur that 'pathophysiology cannot be inferred from coronary lumenography alone', and this was one of the key motivations in conducting the present study.

Our aim was to examine typical patients who present with chest pain, troponin elevation, and unobstructed coronaries. Their clinical evaluation was that routinely performed by their attending cardiologists, thus retaining a 'real-life' practical format to the study. In 65% of cases, CMR was found to yield an underlying aetiology to account for the troponin elevation. In the remaining 35%, no obvious explanation was identified although, within the resolution of the scan, a number of clinical possibilities could be excluded or deemed of relatively low clinical significance.

No patients during the study underwent additional tests to look for vasospasm or microcirculatory dysfunction. This reflects the normal practice of those referring patients for CMR scans. In addition, the incremental yield of performing such tests is contentious. Both spasm and microcirculatory dysfunction may be seen in the absence of troponin elevation and there is variable reproducibility. Many patients may have a dual pathology and indeed myocarditis may be a mechanism for spasm: establishing a causal relationship is difficult.

The assessment of non-obstructive coronary atherosclerosis was based on expert comments by experienced operators. All patients had completely normal coronary arteriograms with smooth luminal margins, without irregularity or stenosis. Although we concur with Coceani and L'Abbate that further evaluation for unstable atherosclerotic plaques would have been ideal and this is acknowledged as a potential limitation, we point out that during follow-up, at a median of 398 days, no new or alternative diagnosis became apparent in any of the patient cohort. It would be difficult to counter therefore, as suggested by Coceani and L'Abbate, that MRI is 'hardly able to guide therapy'. Notwithstanding, we agree that further evaluation to examine pathophysiology would be merited in patients with a non-diagnostic CMR scan who were persistently symptomatic and in those presenting with recurrent episodes of troponin elevation—a cohort not represented in our study. Potentially, MR perfusion could be helpful here also but further work is required.

References


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