also aim to update these guidelines on a regular basis taking into account the results of fully published papers as they become available and also of subsequent guidelines published after our first version such as those by the ACCP [3].

We agree with your first point that aspirin is recommended at a low dose by the ACC and AHA in addition to warfarin, and that the ACCP are more careful with their recommendation. Our recommendation for aspirin in addition to warfarin was based on our literature review of the 11 original trials performed in this area together with consideration of these trials by 12 meta-analyses or other guidelines [4]. We caution that this policy would increase the incidence of bleeding complications but reduce thromboembolic events with a number needed to treat of 19.

Our systematic review of the dosage of aspirin after coronary artery surgery was also summarised and published in the ICVTS prior to our recommendation and we discussed in some detail the difficulties in the literature and also the controversy regarding the dosage of aspirin in these large trials, which are now in some cases almost 20 years old. While some guideline agencies recommend lower doses, many others recommend higher doses. In particular the high quality meta-analysis by Lim et al. [5] published in the British Medical Journal in 2003 using novel analytical techniques actually recommended a dose of 300—325 mg. Thus together with the lack of evidence that 150 mg of aspirin causes a higher incidence of gastrointestinal complications compared to 75 mg and also with second level evidence of aspirin resistance in some patients that we considered, but did not include in the final review, we concluded that 150 mg would be our final minimum dosage.

With regard to recommending clopidogrel for postoperative cardiac surgical patients, we again fully reviewed the evidence and published this in the ICVTS in two papers prior to publication of the guideline. We summarised the evidence from 11 papers and guidelines, and we in fact referenced and endorsed the 2004 ACCP recommendation that states that ‘clopidogrel should be started in addition to aspirin and continued for 9—12 months after CABG for non-ST segment elevation acute coronary syndrome’. This was given a grade 1C recommendation by the ACCP.

Thank you once again for your interest in our guideline process and for your active research in this area to resolve the important unanswered issues of antiplatelet therapy in bioprosthetic valvular heart disease.

References


Letter to the Editor

T2 weighted images as a useful tool in determining myocardial viability in patients performing cardiac magnetic resonance imaging

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Keywords: Cardiac MRI; Imaging; Myocardial viability

We read the article published by Sozzi et al. [1] with great interest and we must thank the authors for such a descriptive study on this case which emphasises the importance of cardiac magnetic resonance imaging (MRI) as an evolving tool that is capable of delineating nonviable or infarcted myocardium from potentially salvageable myocardium, with the additional advantages over the usual nuclear and PET methods of having high spatial resolution and shorter examination time. We would like to add another point specific to the case mentioned by the authors.

The patient presented with an inferior STEMI but had a previous history of an anterior myocardial infarction. Those groups of patients can be particularly challenging in interpretation of their cardiac MRI. Areas of acute or chronic infarction may be difficult to distinguish using cine or delayed enhancement MR images. Both acute and chronic infarctions show bright areas on delayed enhancement MRI. If the infarction is transmural and chronic, the myocardium will show thinning as mentioned in the case presented by the authors.

However, enhancement of the myocardium on MRI can be non-specific and requires knowledge with respect to the clinical setting. We advise combining knowledge from coronary catheterisation, which may aid interpretation of the MRI results. For example, patients with large acute myocardial infarctions usually have micro-vascular obstruction with delayed first pass enhancement on MRI. However, a patient with chronic infarction and total occlusion of a coronary territory may also show delayed first pass enhancement in that coronary distribution. Both types of patients will have delayed enhancement on MR images obtained 10—20 min after injection of the gadolinium agent.

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If the clinical setting is ambiguous, we advise the use of T2 weighted images (rather than T1 in this case) as these may prove very useful in increasing specificity for acute versus chronic infarction. T2 weighted imaging relies upon local dephasing of spins following the application of the transverse energy pulse. The contrast of a T2 weighted image is predominantly dependent on T2 and using a long echo time will increase the T2 dependence. Therefore T2 weighted image contrast state is approached by imaging with a TR long compared to tissue T1 (to reduce T1 contribution to image contrast) and a TE between the longest and shortest tissue T2s of interest. Water has a very high T2 constant, therefore has very high T2 signal and thus appears bright on a T2 contrast image. Consequently, T2 weighted MR images depict more clearly the distribution of oedema in acute myocardial infarction, which is not present in scar/fibrosis.

References


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Reply to the Letter to the Editor

Reply to Elsayed and Poullis

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Keywords: Magnetic resonance imaging; Myocardial infarction; No-reflow

We thank the Editor for giving us the opportunity to reply to Elsayed and Poullis’ letter to the Editor [1]. We would like to thank also Elsayed and Poullis for their interest on our case report. We agree with their comments on T2-weighted images as a useful tool in characterization of myocardial tissue. T2-weighted cardiovascular magnetic resonance imaging (MRI) depicts infarct-related myocardial edema as a marker of acute myocardial infarction (MI) [2]. Water has a very high T2 constant, therefore it gives a very strong T2 signal. Consequently, T2-weighted images differentiate acute from chronic MI. In our case we used conventional T2-weighted imaging of edema with a turbo spin-echo readout with dark-blood preparation [3] (T1 was a typing mistake).

Historically, non-invasive assessment of myocardial viability has been problematic. Myocardial injury can be broadly characterized as either reversible or irreversible. Within irreversibly injured (infarcted) regions microvascular perfusion can vary from nearly normal to nearly zero, even in the presence of an open infarct-related artery (no-reflow). Advances in imaging modalities have improved visualization of no-reflow, showing its frequency to be higher than was estimated by clinical judgment alone. MRI, by means of cine-MRI, T2-weighted image and contrast-enhanced MRI, has emerged as a promising approach to the examination of these regions in patients with MI. After acute coronary event MRI can readily define several different zones of myocardium: normal tissue, non-necrotic stunned tissue, and necrotic tissue with or without microvascular damage. These zones are defined by examining contractility using cine-MRI and tissue characteristics using a contrast-enhanced technique, after administering a gadolinium-based contrast agent. Normal tissue is represented as a tissue with normal contractility, without late enhancement; stunned tissue is characterized by a reduced contractility, without late enhancement. Infarct pattern is defined as an area of hyperenhancement alone or as noticeable hypoenhanced area within a region of hyperenhancement. These different areas of enhancement occur secondary to differences in the wash-in kinetics of the gadolinium contrast agent. Areas of hypoenhancement ('dark') have reduced signal intensity after contrast administration secondary to a delay in fill-in of the contrast agent. Hyperenhanced ('bright') areas reflect necrotic tissue with intact microvasculature. Hyperenhanced areas within areas of hyperenhancement reflect necrotic tissue with damaged microvasculature (no-reflow zones). It has been reported that no-reflow is associated with reduced left ventricular ejection fraction, left ventricular remodeling, and poor clinical outcomes, placing patients with this effect in a high-risk group among reperfused patients [4].

Areas of acute or chronic infarction may be difficult to distinguish using cine or delayed enhancement MR images. Both acute and chronic infarctions are shown as akinetic zones with bright areas on delayed enhancement MRI. In the case of no-reflow presence T2-weighted images provide a means of differentiating acute and chronic MI by the edema visualization.

References


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