

Atrial fibrillation: A kiss of death?

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Atrial fibrillation (AF) is frequently encountered in acute ST-elevation myocardial infarction (STEMI) as well as in elderly patients, and is the most common cardiac arrhythmia in general. Its incidence increases with age and above 80 years it affects approximately 8 % of patients [1]. However, the clinical and prognostic significance depends on the setting in which it occurs. The most likely pathophysiological reasons to develop AF in STEMI are acute ischaemia of the atria and conduction system resulting in electrophysiological changes and vulnerability, and haemodynamic deterioration as a result of more advanced coronary artery disease in elderly patients. Other accompanying phenomena such as acute ischaemic mitral regurgitation, left ventricular hypertrophy and left atrial distension together with fibrosis are also likely to play a role. These factors, mostly in combination, create an ideal substrate for atrial fibrillation, but are also major determinants of a worse prognosis. Whether atrial fibrillation itself contributes to deterioration of the clinical status and thus may be the cause and not only the sign of a grim prognosis remains unclear.

As Martínez-Sellés and co-workers nicely demonstrate in their small series of very old (≥ 89 years) patients with STEMI in the current issue of the Netherlands Heart Journal, it simply means bad news, especially if the patient was in sinus rhythm before (new-onset AF) [2]. This is in accordance with the findings of another study, where age was an independent variable (together with haemodynamic state) for the occurrence of new-onset AF after primary angioplasty, and was associated with increased mortality [3]. The same finding was reported from other STEMI cohorts and patients treated with different reperfusion modalities [4, 5]. Interestingly enough, already in 1961 Thomas James noticed that in the pre-reperfusion era, the overall incidence of AF in STEMI was approximately 10 %, and if it occurred mortality was

extremely high [6]. In the current study, about 70 % of the patients did not receive reperfusion therapy and only 20 % underwent primary angioplasty, which means that the studied nonagenarians are at extreme risk of dying after their STEMI if they develop AF. Part of the bad news is the fact that the majority did not have reperfusion therapy at all, but whether timely reperfusion would have led to better survival is highly speculative. In the Zwolle database of all patients undergoing primary angioplasty for STEMI between 1998 and 2008, out of almost 8,000 patients, 1.6 % were between 85 and 90 years of age, and only 0.6 % were nonagenarian (unpublished data) but these numbers have been increasing rapidly over the last few years [4]. Survival shows a sharp decline in these very old patients, irrespective of additional treatment or the occurrence of atrial fibrillation [7].

It is remarkable that in the current study, new-onset AF did not influence in-hospital mortality, and only during longer follow-up was the extreme mortality observed in the new-onset AF patients, which makes us wonder and speculate about the possible pathophysiological mechanism. One possible cause of death might be related to the high risk of stroke, but by presenting us the CHA₂DS₂-VASc scores the authors suggest that this may not explain the difference, which is an important finding. This may also have implications for therapy at discharge and follow-up, because the general evidence-based medication has to be scrutinised against possible alternatives, considering increased bleeding risk and complex pharmacological interactions in very old persons. Older patients with STEMI do in general have a different profile than the younger group and usually have longer ischaemic time delays, are more likely to be women and diabetics, often have a history of previous revascularisation and MI, and a higher incidence of left ventricular failure on admission. This makes them high-risk patients anyway, but atrial fibrillation and very old age seem to be ruthless harbingers of death. However, the elderly are in general subjected to more conservative treatment strategies. As elderly STEMI patients are less likely to receive evidence-based treatment, including reperfusion therapy, there

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may be a hidden message in this observation for everyone in charge of treating patients with STEMI: consider primary angioplasty in every old patient with new-onset AF, as this is the best treatment option available [8, 9]. This is even more important as the population ages and the number of elderly patients presenting with acute myocardial infarction will continue to increase. The decision to treat or not treat an elderly individual with primary angioplasty needs good clinical discernment, but on the other hand gives little time for judgment or deliberation because of the critical ischaemic time delays. Will timely reperfusion improve prognosis in these very old patients? We do not know the answer but the door to the cathlab should also be wide open, even for very old STEMI patients with comorbidities and age-related problems, regardless of whether they present with new-onset AF. This unique patient group will never be suitable for randomised clinical trials for obvious reasons and we have to rely on observational data as derived from this small study.

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