Hip fracture and heart attack—a lethal combination

Hip fracture is a devastating condition that occurs out of the blue, most commonly in frail older people with multiple pre-existing medical conditions. It has a high mortality, and often results in long-term disability and dependence. Improving outcomes requires organised care and attention to many details [1], one of which is the recognition and management of concomitant cardiovascular disease and perioperative cardiac complications.

In this issue of Age and Ageing, Ausset and co-workers present interesting observational data on the prognostic value of cardiac troponin release after hip fracture [2]. They measured troponin I on days 1–3 post-operatively in 75 patients, and showed that a troponin rise was predictive not only of in-hospital cardiac events but also of cardiac events and total mortality at 6 months. Twenty-seven per cent of their patients had a raised troponin I.

In a similar study in the March 2009 issue of this journal, Chong and co-workers found an even higher rate of raised troponin I in a series of emergency orthopaedic patients aged over 60, of whom about two-thirds had a fractured neck of femur [3]. Over half of their patients had an abnormal troponin result post-operatively, and multivariate analysis showed that this was strongly predictive of 1 year mortality. Ten of the 53 patients with a raised troponin I had a diagnosis of a myocardial infarction (MI), with 7 of these being dead at 1 year. In addition to a MI, troponin release was associated with atrial fibrillation and heart failure, as well as with non-cardiac conditions including pneumonia, renal failure, delirium and receiving a blood transfusion.

Cardiac troponins are very sensitive markers of any myocardial damage, and rises do not necessarily indicate a MI in the conventional sense. Most of the patients in both studies who had a raised troponin had no clinical evidence of a cardiac event. A new universal definition of a MI, based on sensitive biomarkers, divides the MI into several subtypes [4]. Type 1 is a spontaneous MI, with clinical or ECG evidence as well as raised biomarkers. A type 2 MI refers to ischaemic cardiac damage precipitated by another illness, such as arrhythmia, hypotension and anaemia. However, there are also many causes of non-ischaemic troponin release, particularly in concomitant severe illness, and distinguishing these from a type 2 MI (where there is clinical, ECG or imaging evidence of ischaemia) can be difficult.

It is likely that the majority of cases of troponin rise in these studies were due to non-ischaemic causes, the troponin being released due to physiological stress on a background of frailty. However, a significant number clearly did suffer a MI. These findings raise possibilities for intervention to improve outcomes. For example, using aspirin or beta blockers either perioperatively or long term seems an attractive proposition, given their established roles in cardiovascular prevention. However, we should be cautious before jumping on the obvious therapeutic bandwagons, since the balance between benefits and risks cannot be easily extrapolated from trials in other contexts to this complex patient group.

The Pulmonary Embolism Prevention (PEP) trial was a large randomised controlled trial looking at prevention of vascular complications in hip fracture patients which produced unexpected results [5]. The trial studied the effects of aspirin in a group including over 13,000 hip fracture patients. Although a modest effect on pulmonary embolism was anticipated, it was expected that aspirin would also reduce the risk of other perioperative vascular events, particularly stroke and MI, and thus improve the overall outcomes. In the event, there was a trend towards higher rates of MI and stroke in the aspirin group, despite a 36% reduction in venous thromboembolism. More recently, studies of the use of aspirin in the primary prevention of cardiovascular disease in various high-risk groups have shown negative results, leading some to advise that aspirin should only be prescribed in patients with established, symptomatic cardiovascular disease [6], although a very recent evidence-based guideline still recommends aspirin for primary prevention in selected patients [7].

Beta blockers have been recommended for the prevention of cardiac events in non-cardiac surgery. However, the recent POISE trial showed that, while slow-release metoprolol did reduce the risk of a MI, it increased the overall mortality rate and doubled the risk of stroke [8]. A subsequent meta-analysis of 33 trials of perioperative beta blockade confirmed that non-fatal MI is reduced by beta blockers, but stroke is increased and there is no effect on overall mortality, cardiovascular mortality or heart failure [9]. Beta blockers also significantly increased the risk of perioperative bradycardia and hypotension requiring treatment.

An important consideration in any preventive strategy is the underlying event rate. Amongst the 6,677 hip fracture patients in the PEP trial control group, only 0.45% suffered a non-fatal stroke and 0.34% a non-fatal MI, using old criteria [5]. Most of the beta-blocker trials have studied much younger patients selected for their increased risk of a MI, often undergoing elective surgery, with an overall
non-fatal stroke rate of 0.32%, and a much higher MI rate of 4.4% in the control group [9]. In contrast, using the new criteria, Chong et al. identified 10% of patients suffering a MI [3].

So, how can we apply this knowledge to the practical care of hip fracture patients? Firstly, we should be aware that the rate of ischaemic coronary events by the new criteria is higher than previously expected, and is associated with a high 1 year mortality. Therefore, we should be vigilant for a true MI, with proper investigation of any chest pains or other cardiac symptoms and appropriate intervention and secondary prevention. Secondly, we should realise that many cases of troponin release in this situation are merely a marker of co-morbid physiological stress in frail patients, requiring good holistic care rather than any pre-defined intervention. Finally, we should avoid the temptation to apply any preventive interventions routinely that are not properly evidence based in this type of patient.

**Conflict of interest**

No conflict of interest.

**References**