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The Constantly Changing Role of the Emergency Physician in the Management of the Patient with an Acute Coronary Syndrome

Peter G Manning

I recall vividly the 92-year-old woman to whom, a decade ago, I administered recombinant Tissue Plasminogen Activator (rTPA). She had presented to our Emergency Medicine Department in Toledo, Ohio with central chest pain and near-syncope. Timely and appropriate processing revealed an anterior wall myocardial infarction. Given the short duration of symptoms and her truly remarkable premorbid status, a mutually agreed decision was reached between the on-call cardiologist and me that this lady was, in spite of her age, a candidate for myocardial salvage therapy. On the evidence available at that time, the decision was made to offer her intravenous thrombolysis with rTPA being the drug of choice. The next hour or so of her stay in the EMD was extremely taxing for both patient and clinician since she demonstrated a catalogue of reperfusion dysrhythmias interspersed with episodes of haemodynamic instability. However, her pain resolved as the ST segments normalized and, happily, she left the hospital six days later to resume her social round of bridge games with her cronies. I learned of this happy ending a few days later when her grand-daughter paid us a visit to thank the staff for their efforts. I finished my shift that day knowing that I had definitely made a difference to that old lady's quality of life and that was accompanied by a feeling of tremendous satisfaction.

Does the potential for that degree of satisfaction persist today given the same clinical scenario? I think it does, but to a lesser degree.

The case I have outlined above occurred at a time when significant mutual trust had developed in our hospital between the then twenty-year-old speciality of emergency medicine and cardiology. In addition, fewer treatment options existed for the management of such cases. Now, a mere ten years later, there has been a veritable explosion of therapeutic modalities for the patient with an acute coronary syndrome. Even our terminology has changed and focuses on the presence or absence of ST segment elevation; and not on the existence or otherwise of Q waves. Further, the term "acute coronary syndrome" is now used to refer to that spectrum of ischaemic processes that demonstrate similar pathophysiology i.e., ST-segment elevation myocardial infarction, non-ST-segment elevation myocardial infarction and unstable angina.

If that wasn't bad enough, we are now confronted with a mind-boggling array of clinical trials the acronyms of which form what our American colleagues have termed as an "alphabet soup". While not quite ranging from A to Z, they do stretch as far as V! While many non-cardiologists, I am sure, have at least heard of the TIMI (Thrombolysis in Myocardial Infarction) and GUSTO (Global Utilization of Streptokinase and T-PA in Occluded arteries)



Cover Picture:
 Pre-operative and
 post-operative
 radiographs.
 (Refer to page 082-084)

Editorial

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studies, who but a cardiologist would have the slightest idea about the aims and results of the ADMIRAL (Abciximab before Direct angioplasty and stenting in acute Myocardial Infarction Regarding Acute and Long-term follow-up) or VANQWISH (Veterans Affairs Non-Q Wave Infarction Strategies in Hospital) studies.

The end-product of these, and multiple other trials, is that clinicians now have available a greater range of therapies for the ST-segment elevation myocardial infarction than ever before. They fall into the categories of drug therapy (antiplatelet drugs, thrombolytics, low molecular weight heparins and glycoprotein IIb/IIIa inhibitors) with numerous variations within those groups, and, a procedural coronary intervention (including coronary artery bypass grafting and angioplasty with or without stenting), or, any combination of the two categories.

Does that mean that we should have a senior cardiologist on duty in the EMDs of Singapore at all times? Clearly that would be impractical from several standpoints. I am glad to say that there is still clearly a role for emergency physicians in the early management of this subset of patients, many of whom are young and productive citizens. An aggressive approach emphasizing cooperation between cardiologists and emergency physicians is mandatory if we are to achieve optimum outcomes through myocardial reperfusion in those patients with ST-segment elevation. Also, it is logical that the cardiologist be the one to make the final decision regarding the precise definitive care to be offered; for example, the relative merits of a procedural coronary intervention versus thrombolytic therapy.

It is incumbent upon heads of emergency departments to establish protocols to permit timely identification of this group of patients even before they are evaluated by a clinician. Triage personnel should be empowered to upgrade to the resuscitation area any patient with the characteristics of acute myocardial ischaemia and to perform a 12-lead ECG immediately. Even better if those staff members are trained to recognize ST-segment elevation on a monitor or 12-lead ECG, and, also, to suspect possible myocardial ischaemia in those patients who have a tendency to atypical presentations viz., diabetics and the elderly.

Heads of departments should encourage the philosophy of very early notification of the emergency physician in the event the nursing staff suspects a patient as having an ongoing myocardial infarction. There are few clinical conditions which should supercede this patient.

Upon recognition of ST-segment elevation myocardial infarction, the onus is on the emergency physician to notify promptly a senior member of the on-call cardiology team as well as simultaneously providing the immediate clinical care. In spite of all the medical advances outlined above in the management of the patient with acute myocardial infarction, it must not be forgotten that certain initial steps are critical and are the responsibility of the emergency physician viz., continuous ECG monitoring, intravenous access and the acquisition of blood samples for the detection of serum cardiac markers, and the time-honoured "MONA" therapy of morphine, oxygen, nitrates and aspirin.

While I have focused principally on the role of the emergency physician in the management of the patient who requires myocardial salvage therapy, our responsibility extends also to detecting patients with non-ST-segment elevation myocardial infarctions. Published in this edition of the Journal is a paper by F. Lateef et al entitled "Comparison of a 6-hour and 9-hour Protocol for Evaluation of Moderate-to-Low Risk Chest Pain Patients in an Emergency Department Diagnostic Unit."

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I could find no major criticisms of the paper other than those offered by the authors, though I thought significant the omission of any mention of the efficacy of serum myoglobin as a serum marker for acute myocardial ischaemia. This was certainly available at the time this study was being run and is reputed to have a sensitivity of 89% at 2 hours. Also, under the discussion section I found it a little confusing as to whether or not the authors were referring to Troponin T or I, since it was the latter that was included in the 6-hour protocol.

The conclusions drawn by the authors are valid in that they reinforce the current belief among emergency physicians that a relatively short period of observation of up to, but not exceeding, 24 hours in an Emergency Department-based Observation / Chest Pain Unit has value in that particular group of patients with acute coronary syndromes. This would provide an ideal setting for the use of bedside serum markers for myocardial ischaemia. The alternative is to admit them to an in-house cardiology bed which may not be as cost-effective.

Long term, a prominent role for the emergency physicians will continue, though, with the complexity of therapeutic options available it behooves us to work closely with our cardiology colleagues to optimize care for our patients.

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