Acute chest and abdominal pain

GORDIAN FULDE
MB BS, FRACS, FRCS(Ed), FRCS/RCP(A&E)Ed

Emergencies can spring up at any time and in many incarnations. Are you adequately equipped to deal with them? Here is a case study in emergency medicine that is based on a real case. Would you have been able to help this patient?

Both in your busy group general practice and during the shifts you also do as a general practitioner in the local emergency department, you have the impression that patients are not only getting older but many of them are becoming more complex medically.

Urgent hospital attendance advised

During one of your regular Monday morning shifts in the emergency department you attend a patient with chest pain who has been allocated a triage category of 1 (to be seen immediately). The patient was expected as his wife had telephoned. You had overheard the surprisingly common conversation where it was made clear to the wife that she and her husband should call an ambulance and come in immediately. They, however, had decided to pack a bag and drive themselves in, arriving over an hour after the phone call and advice.

The 74-year-old patient has severe chest pain (8/10), is breathless and sweaty and looks pale. His history reveals several hours of typical cardiac chest pain and he has an extensive cardiac history that includes a permanent pacemaker, atrial fibrillation and coronary artery graft surgery some 18 months ago, which had a 'stormy' postoperative course that included reoperation.

You wonder briefly why an intelligent and pleasant man who knew that he had cardiac pain and should present to hospital quickly did not do so. Many studies have shown delayed presentation is still a major problem for improving cardiac outcomes.

Professor Fulde is Director, Emergency Department, St Vincent's Hospital, and Associate Professor in Emergency Medicine at the University of New South Wales, Sydney, NSW.

Further investigations

An oxygen mask is put on the patient, an intravenous cannula is placed and bloods are taken. Aspirin (salicylic acid 300 mg) and sublingual nitrate has already been given, on the patient's arrival at the department.

His blood pressure is 140/80 mmHg, with a pulse rate of 60 beats per minute (paced). His ECG is far from normal (left bundle block and depressed ST changes) but shows no acute changes, especially when compared with a previous ECG he has brought with him. His cardiac pain has now eased to 2/10.

Although this patient has classic cardiac pain with radiation to both arms and jaw, a more detailed assessment will allow you to exclude other significant pathology, such as aortic dissection or lung pathology. Portable bedside chest x-ray reveals cardiomegaly with left ventricular failure. Since there is no test that can be relied on to exclude infarction and the patient already fulfils the criteria for unstable angina (now called acute coronary syndrome) and possible infarction, the cardiologists respond quickly with a view to an expeditious cardiac catheter study. Cardiac troponins are now the serum markers of choice for detecting myocardial damage, and a risk stratification approach to unstable angina is promoted by the current National Heart Foundation of Australia guidelines for the management of unstable angina (see the box on this page).[1-3]

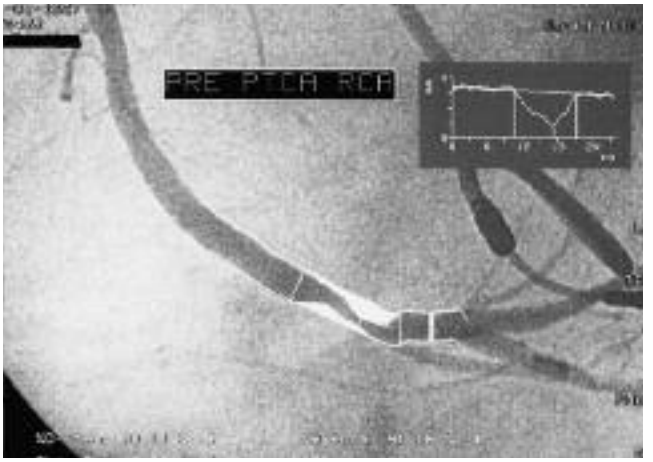
Angiography, however, shows mild left coronary disease

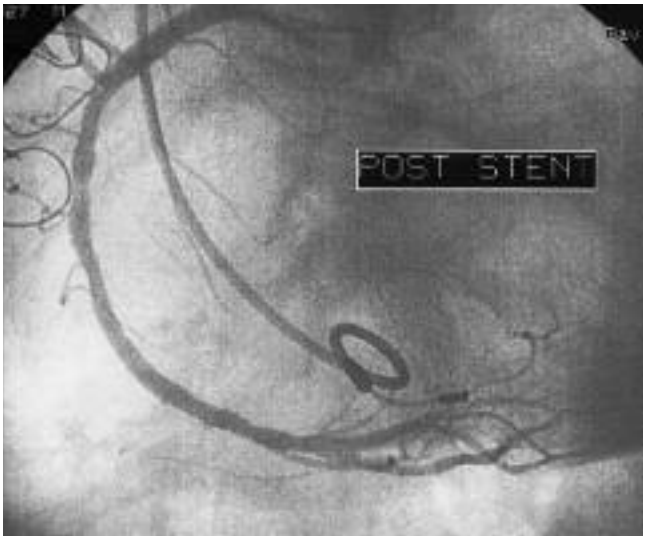
Unstable angina: NHF guidelines

The recently revised National Heart Foundation of Australia guidelines for the management of unstable angina promote a risk stratification approach to unstable angina, now known as acute coronary syndrome.[1-3]

These guidelines now pay more attention to cardiac enzyme changes in the diagnosis of the condition and to stratifying patients into risk groups for management, with earlier intervention advised for the higher risk patients. The cardiac troponins I and T are the serum markers of choice, the presence of either one indicating myocardial damage and the levels correlating with early risk of cardiac death or myocardial infarction. Creatine kinase levels are also considered. The acute coronary syndromes are divided into:

- low risk unstable angina – all cardiac enzymes normal
- high risk unstable angina (or minor myocardial damage) – mild elevation of serum troponin I or T, with normal creatine kinase levels
- non-ST elevation or ST elevation myocardial infarction – elevation of both troponin and creatine kinase levels with evolving ECG changes.





Figures 1a and b. Coronary angiograms before (a, above) and after (b, right) stenting of a tight stenosis of the right coronary artery.

with patent grafts to the diagonal and its branch. A tight 90% stenosis of the right coronary artery was dilated by balloon and stented, resulting in good flow distally (Figure 1).

A week later

The patient re-presents to the emergency department during your Monday shift the following week. He is distressed and in obvious pain and you quickly examine him.

The pain is mainly right upper quadrant, and you are concerned that, even though he is taking anticoagulants (warfarin), aspirin 300 mg and clopidrogel 75 mg (routine after stenting), the upper abdominal pain could be due to severe inferior cardiac ischaemia from blockage of his right coronary stent. (It is worth noting that these three medications are not usually prescribed concurrently.) Almost unbelievably, the patient has had the discomfort and pain since Saturday night after a meal.

It soon becomes evident that this presentation is clinically quite different from the previous presentation as the patient is complaining of a bloated abdomen, bowels not opened for two days, marked flatulence and anorexia. He has no fever and examination of his abdomen reveals a tender mobile right upper quadrant muscle guarding.

An abdominal ultrasound shows a mobile large gallstone (Figure 2), no dilation of the common bile duct, two echogenic well-defined lesions in the liver (most likely haemangiomata) and a horseshoe kidney. The surgeons successfully treat the patient conservatively for his cholecystitis and discharge him so that the physicians can get him fit (coagulation, cardiac) for a semielective laparoscopic cholecystectomy.

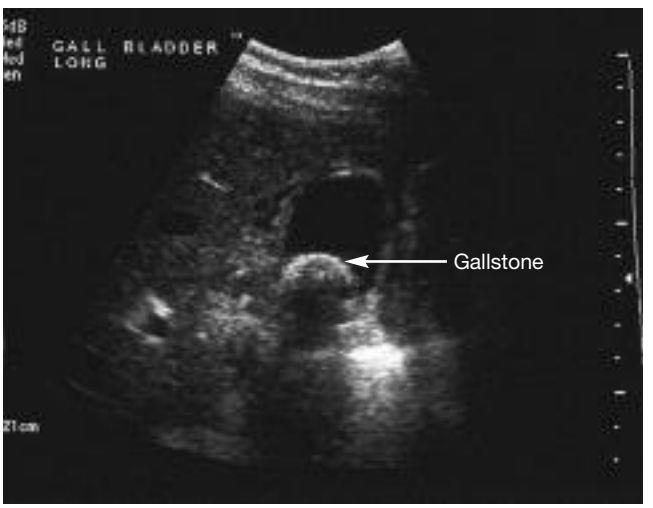


Figure 2. Abdominal ultrasound showing a large gallstone (arrowed).

On reflection

You muse that chasing the diagnosis, especially if it is different from what you expect, is part of the fun of clinical medicine – as long as all goes well. MT

References

1. National Heart Foundation of Australia. Unstable angina guidelines. www.heartfoundation.com.au/prof/index_fr.html
2. Aroney C, Boyden AN, Jelinek MV, Thompson P, Tonkin AM, White H. The National Heart Foundation of Australia/The Cardiac Society of Australia and New Zealand. Management of unstable angina guidelines – 2000. Med J Aust 2000; 179(Suppl): S65-S88.
3. Federman J. Unstable angina: assessment and management. Medicine Today 2003; 4(1): 37-45.