Myocardial infarction in a patient with normal coronary arteries

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Articles in this section use cases to illustrate the emergency management of patients presenting in general practice with cardiac problems. They are inspired by, but not based on, real patient situations.

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Mr DM, a 71-year-old retired hospital administrator, telephones his local GP on a weekday morning as he is experiencing an episode of severe central, nonradiating chest pain. His GP advises him to call an ambulance immediately.

When Mr DM arrives in your emergency department (ED), you see that his vital signs are within normal limits except for sinus tachycardia (heart rate, 105 beats per minute). He is transferred to an acute bed.

Mr DM tells you he is currently pain-free but has had four days of intermittent chest pain that he rates as up to 7/10 in severity, which is relieved by rest and use of his wife's glyceryl trinitrate spray. Although his pain sounds typically cardiac, you confirm that he has no risk factors for pulmonary emboli. His ECG shows sinus rhythm with 2 mm of horizontal anterolateral ST depression but no other abnormalities.

Mr DM is married with three adult children and seven grandchildren. He has diet-controlled type 2 diabetes but no other medical problems. He enjoys photography and does not participate in regular physical activity. He consumes two glasses of wine per day but is a lifelong nonsmoker. His blood pressure and cholesterol level have always been normal, and there is no family history of cardiac problems.

What do you look for on clinical examination?

Mr DM has no respiratory distress. His heart rate is now 85 beats per minute and blood pressure is 140/80 mmHg. His jugular venous pressure is not elevated, and his heart sounds are dual without any murmurs. On lung auscultation, there are early inspiratory crackles that clear with coughing but no sign of the coarse, pan-inspiratory crackles of pulmonary oedema. Mr DM has central obesity and a mildly distended, nontender abdomen. This makes palpation difficult, but there is no discernible organomegaly. There is mild bilateral peripheral pitting oedema.

What is the diagnosis?

Mr DM has acute coronary syndrome, which includes unstable angina (defined as new or rapidly progressive chest pain, with or without ECG changes) and myocardial infarction (classified by an elevated level of a cardiac biomarker, usually troponin). Unstable angina requires prompt escalation of medical therapy as it is usually secondary to an unstable coronary plaque. In addition, patients with unstable angina should be considered for coronary angiography if they have ongoing or repeated episodes of symptoms, or if they have other features indicating a high likelihood of subsequent myocardial infarction (such as ECG changes).

1. Types of myocardial infarction*

Type 1: Spontaneous myocardial injury due to coronary artery disease

Type 2: Secondary myocardial injury due to an ischaemic imbalance other than coronary artery disease

Type 3: Cardiac death with symptoms suggestive of myocardial injury but occurring before blood samples could be collected for biomarker assessment

Type 4a: Myocardial injury due to percutaneous coronary intervention

Type 4b: Myocardial injury due to stent thrombosis

Type 5: Myocardial injury due to coronary artery bypass surgery

* According to the 2012 joint task force of the European Society of Cardiology, American College of Cardiology Foundation, American Heart Association and World Health Federation[1]

Conversely, stable angina is very common and is defined as reproducible cardiac-sounding pain that is relieved by rest and/or nitrate therapy; it is usually managed in the outpatient setting.

Mr DM's pain has resolved. His serum troponin T level measured at presentation is elevated at 55 ng/L (normal range, <16 ng/L for men, <26 ng/L for women). His second troponin level, measured six hours after pain onset, is 70 ng/L. The clinical history together with the rise in troponin level is consistent with a diagnosis of non-ST elevation myocardial infarction (NSTEMI).

What do the ED investigations show?

Mr DM's blood tests reveal pancytopenia, with haemoglobin level of 72 g/L (normal range, 120 to 180 g/L), mean cell volume of 90 fL (normal range, 80 to 100 fL), platelet level of 79 x 10^9/L (normal range, 150 to 400 x 10^9/L) and white cell count of 2.8 x 10^9/L (normal range, 3.5 to 11 x 10^9/L). His creatinine level is 83 µmol/L (normal). The chest x-ray shows clear lung fields and no cardiomegaly.

What should be Mr DM's initial management?

Myocardial infarction can be one complication of anaemia, and Mr DM requires urgent blood transfusion (target haemoglobin in this case is over 100 g/L). There are competing priorities affecting the decision whether to use the standard treatments for myocardial infarction of antiplatelet therapy and therapeutic enoxaparin, given his anaemia. As he has clear risk factors for coronary disease combined with ischaemic ECG changes, it is decided to administer some form of antiplatelet therapy. Antiplatelet monotherapy is chosen given the thrombocytopenia, and he is commenced on clopidogrel (without aspirin). Ticagrelor or prasugrel can be used in place of clopidogrel in patients with NSTEMI depending on hospital guidelines. All of these drugs are usually administered with aspirin.

Therapeutic enoxaparin is avoided at this stage. If Mr DM were to develop unstable symptoms or evolving ECG changes, he would be commenced on an unfractionated heparin infusion as its reversibility is superior to that of enoxaparin.

The remaining therapy for patients with NSTEMI comprises a potent statin (atorvastatin or rosuvastatin), a cardioselective beta-blocker (usually metoprolol) and a low-dose ACE inhibitor. Mr DM receives these drugs and is admitted to a monitored bed, so as to identify any ventricular arrhythmias, which can occur in the acute phase of myocardial infarction. He undergoes the two common investigations in patients with myocardial infarction: echocardiography to assess cardiac function and coronary angiography to assess coronary disease.

What do echocardiography and coronary angiography show?

The transthoracic echocardiogram shows normal biventricular function with no significant valve disease. Pulmonary pressures are also normal. Mr DM undergoes coronary angiography, which surprisingly shows normal left and right coronary systems (Figure 1). This suggests that the myocardial infarction was not secondary to acute plaque rupture but rather to an imbalance between myocardial oxygen supply and demand secondary to the anaemia.

This type of infarct is termed a type 2 myocardial infarction (Box 1).[1] Type 2 myocardial infarction is secondary to a precipitant, commonly pneumonia, sepsis, anaemia, pulmonary embolism or left ventricular hypertrophy. Some practice points on type 2 myocardial infarction are summarised in Box 2.

Figure 1. Coronary angiograms in a 71-year-old patient with NSTEMI showing (a, left) normal left coronary arteries and (b, right) a normal right coronary artery.

2. Practice points on type 2 myocardial infarction

- Troponin assays are very sensitive and will detect any type of myocardial injury.
- Elevated troponin levels may result from imbalances in oxygenation, blood delivery, myocardial demand and/or troponin excretion.
- Type 2 myocardial infarction is due to an imbalance between myocardial oxygen supply and demand caused by a condition other than coronary artery disease.
- Type 2 myocardial infarction is most common in patients with pneumonia, sepsis, anaemia, pulmonary embolism or left ventricular hypertrophy.
- In patients with type 2 myocardial infarction, the decision to proceed with coronary angiography depends on symptoms, ECG changes and the individual risk factor profile.

With the information from the coronary angiography, Mr DM's cardiac medications, including clopidogrel, are ceased.

What further investigations are required?

Diagnosing the cause of Mr DM's pancytopenia will explain his presentation. The causes of pancytopenia can be categorised as decreased blood cell production, haemodilution or blood cell sequestration. Blood tests in Mr DM show normal vitamin B_{12} and folate levels and normal results of iron studies and haemolysis screening. Upper abdominal ultrasound and CT scanning reveals a hepatobiliary mass, suggestive of a localised intrahepatic cholangiocarcinoma, complicated by portal vein thrombus and splenomegaly (Figure 2). Consequently, hypersplenism is considered responsible for Mr DM's pancytopenia. Treatment consists of therapeutic enoxaparin to recanalise his portal vein. Eligibility for surgical resection of cholangiocarcinoma must be considered, as it provides the only cure for this rare malignancy that is often complicated by early metastases.

Outcome: Mr DM underwent partial hepatectomy for cholangiocarcinoma. Histopathological examination showed that the margins were clear. To the relief of his family, he had a good postoperative recovery. His three-week hospital admission prompted him to improve his physical activity and cease drinking alcohol. He did not require cardiology follow up. **CT**

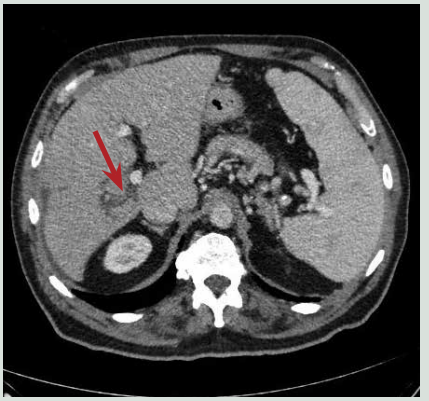

Figure 2. CT axial view of the abdomen in a 71-year-old patient with NSTEMI showing a right portal vein thrombus (arrow).

References

1. Thygesen K, Alpert JS, Jaffe AS, et al; Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction. Third universal definition of myocardial infarction. Circulation 2012; 126: 2020-2035.

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