

An ischaemic leg

What's at the heart of the problem?

AUSTIN N. MAY MB BS(Hons)

ELLEN HARDY BMed

GORDIAN FULDE AO, MB BS, FRACS, FRCS(Ed), FRCS/FRCP, (A&E)Ed, FACEM

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.



Mr NP is a 42-year-old married man who has no children and receives unemployment welfare payments. He has type 2 diabetes and hypercholesterolaemia, for which he has not taken any medication for three weeks. Mr NP has come to see you today with right foot pain that had a sudden onset four days ago. He has not experienced this pain before and has not had any falls or trauma to explain an injury. The pain in his foot is severe, extends to the mid-calf, is constant, waking him from sleep, and is only partially relieved by dependent positioning. He reports no claudication before this event.

As Mr NP enters your room he is writhing in pain and hobbles to your examination table when you ask to examine him. His blood pressure is 125/85 mmHg and his pulse is 70 beats per minute and regular. On examination of his leg, the skin of his foot is pale to the ankle, with mottled, dusky toes. It is cool to the touch. He has a weak right femoral pulse and popliteal pulse, but neither the posterior tibial nor dorsalis pedis pulses are palpable. Toe flexion and extension show that large muscle groups are intact, but weak fanning of his toes suggests poor small muscle group function. Beyond his pre-existing peripheral neuropathy he has decreased sensation in the right foot compared with the left. He has a blood sugar level of 28.9 mmol/L.

What is the next step?

You are concerned that Mr NP has acute limb ischaemia and immediately organise to transfer him to the hospital emergency department. If left untreated, limb ischaemia may cause Mr NP to lose his leg. While waiting for the ambulance, you complete your physical examination. His jugular venous pressure is not elevated. His apex beat is laterally displaced but there are no murmurs or added heart sounds, and his chest is clear on auscultation. The ambulance arrives and transfers Mr NP to hospital.

What happens in the emergency department?

Mr NP is given high priority for review and his physical examination findings are confirmed. He is given intravenous opiate

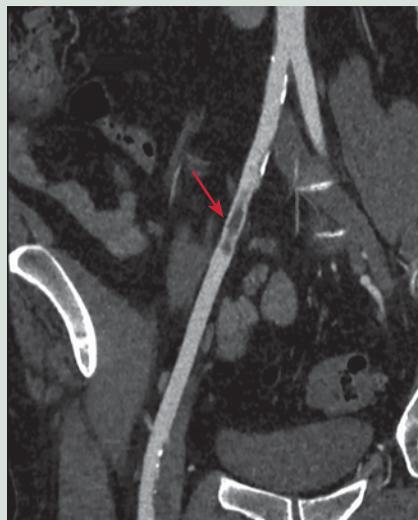
analgesia. His ECG shows sinus rhythm with anterior Q waves and, importantly, he is not in atrial fibrillation. A full blood count, renal function tests, coagulation studies, chest x-ray and lower-limb CT angiogram are requested. His glycosylated haemoglobin (HbA_{1c}) level is 13.8%. Venous blood gases and blood ketones are analysed to exclude concurrent diabetic ketoacidosis. Before the results of these tests become available, the vascular surgery registrar gives instructions for a heparin infusion to be started and says she will attend immediately. The CT angiogram shows a central filling defect of the right common and internal iliac arteries, consistent with an embolus (Figures 1a and b). There is also occlusion of the peroneal and posterior tibial arteries, and no flow is seen below the level of the ankle.

CARDIOLOGY TODAY 2017; 7(2): 23-25

Dr May is a Cardiology Advanced Trainee at Gosford Hospital, Gosford; and a Clinical Associate Lecturer at UNSW Sydney. Dr Hardy is a Vascular Surgery Registrar at Gosford Hospital, Gosford. Professor Fulde is Director of the Emergency Department at St Vincent's Hospital; Professor in Emergency Medicine at UNSW Sydney; and Professor in Emergency Medicine at the University of Notre Dame, Sydney, NSW.



Figures 1a and b. CT angiograms. Axial (a, left) and coronal (b, right) views showing thrombus with subtotal occlusion in the right common iliac artery (arrows).



When the vascular surgery registrar arrives, she organises an urgent right leg embolectomy for Mr NP. She enquires about sources of thromboembolism, and Mr NP reports never having had a clot in the past, including in his limbs and lungs or a stroke. He says he has had no heart problems previously. He felt well before the current episode of foot pain, but recalls an episode of significant breathing difficulty and vomiting while on a rural holiday to visit family about 12 months ago. The symptoms had disappeared by the time he returned home and he did not report them to his GP.

Mr NP proceeds to theatre for an embolectomy the same day. This is performed using embolectomy catheters inserted through a longitudinal groin

incision with a right common femoral artery arteriotomy. When the catheters are passed proximally to the iliac arteries, a white embolus is retrieved and good flow is re-established. The catheters are then passed distally, and a large amount of red thrombus is retrieved; however, there is still poor flow in these vessels. Fluoroscopy is used to assess patency and guide the passage of the embolectomy catheters. Unfortunately, despite removal of the thrombi, no flow is seen below the level of the ankle.

What do these findings suggest?

These intraoperative findings are consistent with an embolic phenomenon with secondary thrombus formation. The proximal clot

is sent for histopathological examination, because rare myxomas can also cause embolus. The result reveals a pale, platelet-rich thrombus. The red clot found is acute thrombus due to the reduced flow distally.

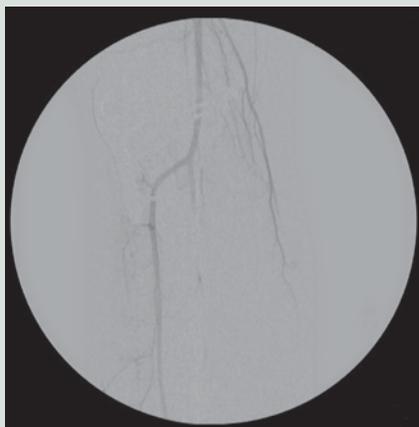
Mr NP's delayed presentation makes this thrombus particularly adherent and ultimately it causes irreversible endothelial damage. This is a 'no-reflow' phenomenon, whereby despite adequate macrovascular flow, there is no perfusion through the damaged microvasculature (Figures 2a and b). The role of heparin in acute ischaemia is to reduce the propagation of this secondary clot.

Embolism is a particularly devastating cause of acute limb ischaemia because it strikes suddenly at otherwise normal vessels. This is in contrast to acute or chronic ischaemia that develops in patients with progressive atherosclerotic disease. These patients often have collaterals that allow for some perfusion of tissues.

Mr NP undergoes transthoracic echocardiography to find the source of the thrombus. It reveals a normal-sized left ventricle with moderate segmental impairment and a left ejection fraction of 40%. There is apical akinesis and a 3 cm apical thrombus (Figure 3). The right ventricle is of normal size and has normal function and there is no significant valve disease.

What further investigations are required?

After a left ventricular thrombus has been identified, the indication for performing a thrombophilia screen depends on the presence or absence of left ventricle wall motion abnormalities. If a patient has wall motion abnormalities sufficient to explain the development of thrombus, such as in the case of Mr NP, then a thrombophilia screen is an expensive test that carries a low yield. However, if a patient has normal wall motion, recurrent thrombi or mixed arterial and venous thrombi, then it is necessary to perform a thrombophilia screen to investigate the patient's propensity to form clots.



Figures 2a and b. Intraoperative angiograms showing flow before (a, left) and after (b, right) distal thrombectomy. The anterior tibial artery is patent from the beginning.



Figure 3. Transthoracic echocardiograms showing left ventricular thrombus in a patient with diabetes and apical akinesis.

In addition to ongoing telemetry to examine for paroxysmal atrial fibrillation, the next investigation for Mr NP is a coronary angiogram to identify the source of his left ventricle wall motion abnormalities.

Mr NP undergoes coronary angiography via a right radial approach to avoid his lower limbs. Left ventriculography is not performed, as it would require passing a catheter into the left ventricle with the potential to dislodge further thrombus. The angiogram reveals a high-grade stenosis in the proximal left anterior

descending artery (Figure 4). The remaining arteries have minor stenosis only.

What is the diagnosis?

The conclusion is that Mr NP suffered a myocardial infarction in the left anterior descending artery 12 months previously. He did not experience any chest pain at the time because of the altered or atypical pain sensation that occurs with diabetic neuropathy.

Mr NP undergoes successful percutaneous coronary stenting and commences warfarin in addition to the heparin infusion and antiplatelet therapy.

Can the leg be salvaged?

Given the findings at embolectomy there is concern about the viability of Mr NP's foot. In particular, the inability to establish flow below the ankle makes it unlikely that the foot would survive. Over the following days, the foot is observed for signs of demarcation (Figure 5). Eight days postoperatively, a distinct line appears between dusky, non-viable skin distally and pink perfused skin proximally. This line of demarcation is at the level of the heel on the plantar surface of the foot.

All of this nonviable tissue needs to be removed. Selection of the amputation level is a balance between creating a stump proximal enough to ensure a well-healing wound, but also distal enough to preserve functional length. Although Mr NP has a well-perfused ankle, this joint would be an unstable base for a prosthesis. In this situation, a below-knee amputation provides the most appropriate point for the stable fitting of a functional prosthesis.

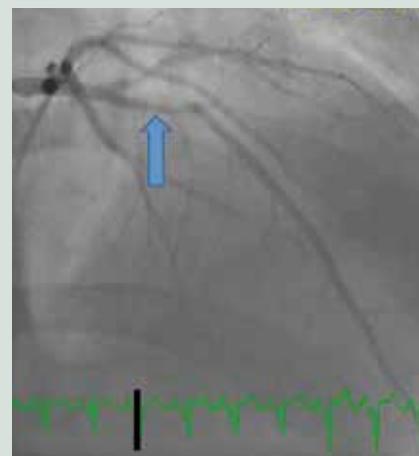


Figure 4. Coronary angiogram showing an eccentric high-grade carotid stenosis (arrow) in the proximal left anterior descending artery in the patient with left ventricular thrombus.

Outcome: After undergoing a below-knee amputation, Mr NP recovers smoothly and the wound heals well. Of particular note, he becomes highly motivated in looking after himself after this episode, including adherence to antiplatelet, warfarin and statin therapy. He feels concerned about how his previous poor health decisions have affected the lives of those around him.

Mr NP successfully undergoes rehabilitation and becomes independent with his activities of daily living. He mobilises using a custom-fitted lower limb prosthesis. Following review with an endocrinologist, he starts insulin during his inpatient stay and achieves glycaemic control. He aspires to return to the workforce in an office environment.

You see him regularly for INR checks and refer him for surgical follow up and surveillance transthoracic echocardiography of the left ventricle thrombus. You also initiate regular ophthalmic, renal and podiatry reviews and encourage Mr NP to attend his dentist. He and his wife are grateful that you recognised the urgency of his initial presentation.

CT



Figure 5. Demarcation of Mr NP's right foot. Note the extravasation through damaged capillaries, causing the fixed colour change. There is a glyceryl trinitrate patch on the foot to assist local vasodilation.

COMPETING INTERESTS: None.