Cardiology

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Abstract

Cardiac disease remains a significant threat to both local and deployed military populations. In this article we present several cardiac case reports which may be of educational use to the military clinician.

Case 1

A 39 year old soldier, serving in Afghanistan, presents with a five hour history of acute central chest pain with associated shortness of breath and nausea. He denies any recent trauma. He has an unremarkable family and past medical history and smokes 20 cigarettes per day. On further enquiry he admits to antecedent minor flu like symptoms over five days prior to presentation. On examination his heart rate is 116/minute with a blood pressure of 136/80 mmHg and a low grade pyrexia of 37.8ºC. His 12 lead ECG and initial blood tests in the Emergency department are shown in Figure 1 and Table 1 respectively.

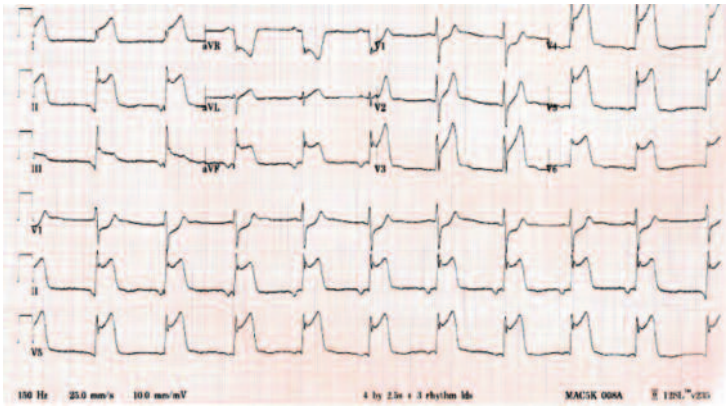

Figure 1: 12 lead ECG at presentation

Hb (g/dL)	15.4 ↑	CK MB (IU/l)	260 ↑
WCC (x109/L)	16.2 ↑	Albumin (g/l)	38
Neutrophils (x109/L)	13.6 ↑	Protein (g/l)	72
Platelets (x109/L)	450 ↑	ALP (U/l)	250 ↑
CRP (mg/L)	190 ↑	AST (U/l)	46 ↑
Cardiac troponin (ng/ml)	6.8 ↑	LDH (U/l)	680 ↑
CK (IU/l)	1200 ↑	Bilirubin (µmol/l)	36 ↑

Table 1: His initial blood tests

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a. What does this ECG show?
b. What is your differential diagnosis at this stage?
c. What further investigations should be arranged?
d. How would you treat this patient?

Case 2

An 18 year old soldier presents to the Emergency Department having 'collapsed' on a run. He admits to intermittent light headedness occasionally with heavy exercise particularly in warmer weather. He has an otherwise unremarkable past medical history and on examination is clinically well and apyrexial. Clinical examination is unremarkable. His Regimental Medical Officer (RMO) had arranged a 12 lead ECG (Figure 2).

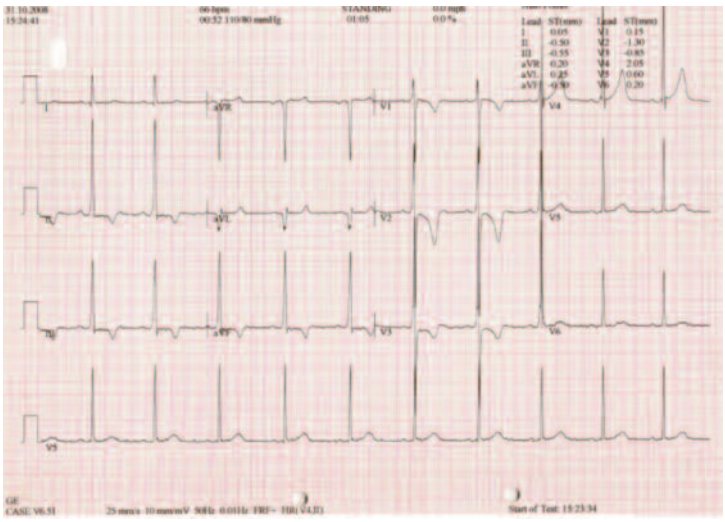

Figure 2: 12 lead ECG at presentation

a. What does this ECG show?
b. What is your differential diagnosis?
c. What further investigations are required?
d. What further question might you ask?

Case 3

A 38 year old sergeant major presents to his local medical centre with a two month history of increasing shortness of breath coupled with ankle swelling and weight gain of 5 kg over the previous six weeks. He denies any significant past medical history. On examination he is tachycardic with a heart rate of 112/minute and a blood pressure of 96/48 mmHg with an audible third heart

sound and mild basal crepitations. A chest x-ray is performed on admission to hospital (Figure 3).

Figure 3: AP chest radiograph on admission to hospital

a. What is the likely diagnosis?
b. What does the chest x ray show?
c. What further investigations are required?
d. How would you treat this patient?

Case 4

A 36 year old soldier presents to the Emergency Department with sudden-onset central chest pain (like 'being punched in the chest') whilst lifting weights. Pain radiates to his throat and is associated with nausea. He has no previous medical history. He is a body-builder and admits to intermittently self injecting with the androgenic anabolic steroid (AS) Nandrolone for seven years. He has a fifteen pack year smoking history. His father suffered from a myocardial infarction at age 50. On examination he is pale and clammy with an unremarkable respiratory and cardiovascular examination. An ECG recorded is below (Figure 4).

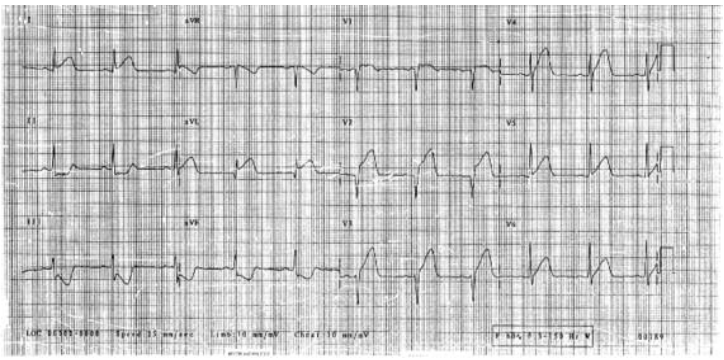

Figure 4: 12 Lead ECG recorded in the emergency department

a. What does the ECG show?
b. What is the most likely diagnosis?
c. Is the history of androgenic anabolic steroid abuse relevant?
d. What is your immediate management plan?

Answers to Self Assessment Questions

Case 1

a. The ECG shows concave saddle-shaped ST elevation in I, II, III, aVF, V3-6.
b. The differential diagnoses include acute widespread ST elevation myocardial infarction (STEMI) and acute myopericardits.
b. A chest radiograph should be arranged as this will provide information on the presence of cardiomegaly, pulmonary oedema or an associated pneumonic process. The patient will require an echocardiogram and consideration made for coronary angiography following medical evacuation from theatre and transfer to a fully resourced cardiac unit.
c. In an operational deployment setting you will probably be compelled, at least initially, to consider treating this as an acute myocardial infarction, given the patient's age and smoking history. However, the widespread pattern of the patients ST elevation, and significant derangement in liver function and CRP makes acute STEMI a less likely diagnosis. However, if there is uncertainty it may be appropriate to commence standard treatment for a presumed myocardial infarction (MI) and withdraw this later when the diagnosis is confirmed occasionally following exclusion of MI. This patient had an acute myopericarditis, which is differentiated from acute pericarditis by the presence of myocardial injury as evidenced by an increase in his cardiac enzymes [1]. Acute pericarditis is often accompanied by some degree of myocarditis and may lead to an increase in cardiac enzymes. The pain of acute pericarditis is often positional (eased by sitting upright) and may be associated with PR depression on the resting 12 lead ECG. In clinical practice both pericarditis and myocarditis often coexist as they share common aetiologic agents, mainly cardiotropic viruses. The term myopericarditis indicates a primarily pericarditic syndrome and it is responsible for the majority of cases [1]. The clinical presentation is varied, reflecting the variability of myocardial involvement, which may be focal or diffuse, affecting any or all cardiac chambers. The management of this condition includes analgesia, the identification of a possible cause and treating any associated heart failure if present. Patients should be encouraged to refrain from any more than light exercise until fully recovered.

Case 2

a. His 12 lead ECG shows voltage criteria of left ventricular hypertrophy with a lateral strain pattern.
b. The main differential diagnosis in an 18 year old soldier without hypertension includes an athletic heart or hypertrophic cardiomyopathy (HCM). This patient had HCM.
c. Further investigations should be performed by a cardiologist and should include transthoracic echocardiography, a 24 hour tape (to look for evidence of arrhythmias), a supervised exercise test and occasionally a cardiac MRI. His transthoracic echocardiogram (Figure 5) and Cardiac MRI (Figure 6) both showed marked septal hypertrophy, with systolic anterior motion of the mitral valve. This was leading to dynamic left ventricular outflow tract obstruction and was the likely explanation of his blackouts.

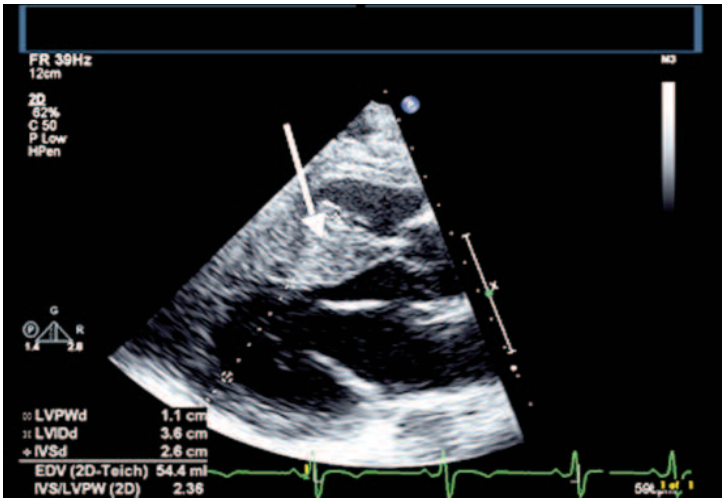

Figure 5. Transthoracic echocardiogram showing marked asymetrical septal left ventricular hypertrophy (white arrow).

Figure 6. Cardiac MRI demonstrating again marked septal hypertrophy (white arrow) with later gadolinium enhancement (patchy white areas within the septum) in keeping with extensive fibrosis

d. It is always worth asking about a family history of sudden cardiac death or unexplained syncope among close family members. This soldier's father had been complaining of intermittent pre-syncope with meals and was subsequently investigated following his son's diagnosis and was found to have also have HCM. HCM is the commonest inherited primary heart muscle disorder affecting 1 in 500 of the general population and its inheritance is autosomal dominant [2]. Individuals with HCM demonstrate a degree of left ventricular hypertrophy which is classically asymetrical affecting predominantly the septum but a number of differing patterns of hypertrophy hare recognised. About 30% of individuals with HCM demonstrate an obstruction to the outflow of blood from the left ventricle during rest, but this increases to up to two thirds of cases with exercise hence the term dynamic obstruction. HCM is the commonest cause of sudden cardiac death in athletes and probably in young younger persons [3]. This patient has three possible risk factors for sudden cardiac death: a positive family history, marked ventricular hyperrtrophy (septal thickness >3 cm) and a previous history of syncope. Consequently, he underwent the insertion of an implantable cardioverter defibrillator and was commenced on beta blocker therapy. He was discharged from military service on medical grounds.

Case 3

a. The likely diagnosis is heart failure
b. The chest x ray shows cardiomegaly (even after allowing for the fact that it is an AP film) with basal and mid zone interstitial shadowing and upper lobe blood diversion in keeping with pulmonary oedema.
c. This patient needs an echocardiogram which is crucial to the diagnosis of heart failure. This showed a dilated heart with severely impaired left ventricular systolic function. His coronary angiogram was normal. He underwent a cardiac MRI (Figure 7) which confirmed a likely diagnosis of a dilated cardiomyopathy.

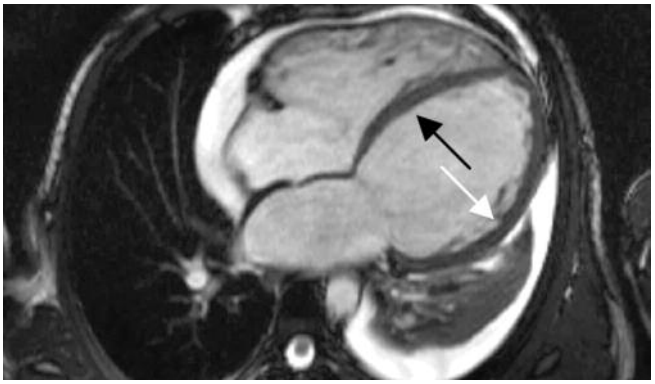

Figure 7: Cardiac MRI showing a severely dilated left ventricle (arrows outlining the septal [black] and lateral walls [white]) with an ejection fraction of 31% in keeping with severe left ventricular systolic dysfunction. On the delayed contrast images there were no features of infarction, scar or active inflammation.

d. The patient should be initially treated with intravenous diuretics, morphine, a GTN infusion and oxygen. He should undergo strict bed rest and nursed sitting up. Further medical treatment should include the use of an oral Angiotensin Converting Enzyme inhibitor, such as Ramipril (2.5 mg OD), a cardioselective beta blocker, such as Bisoprol (2.5 mg OD), both of which improve prognosis. The doses should be doubled at two weekly intervals aiming for the maximal dose. The patient should be provided with strict lifestyle advice and education on the personal management of his condition. On average 25% of cases of DCM may be genetic hence, if no other cause is identified the soldiers children may need to be screened also.

Case 4

a. The ECG demonstrates sinus rhythm with ST elevation in leads aVL, V1-V4 and reciprocal ST depression in leads II, III and aVF.
b. The history and ECG findings are strongly suggestive of an anteroseptal STEMI.
c. The use of anabolic steroids (AS) is highly relevant especially given the very young age of presentation. AS have been linked to a number of deleterious cardiovascular effects such as accelerated atherosclerosis, acute myocardial infarction, life threatening cardiac arrhythmias and even sudden cardiac death [4]. The mechanisms are highly complex and include elevation of blood pressure, dylipidaemia, polycythaemia and activation of the coagulation cascade.
d. The patient should be nursed upright and given high flow oxygen and sublingual or buccal glyceryl trinitrate to reduce myocardial ischaemia. He should also receive immediate treatment with oral aspirin 300mg (chewed) and clopidogrel 300-600mg. He should also be given analgesia with intravenous morphine (5-10mg) and an appropriate intravenous antiemetic, such as 10 mg of metoclopramide.

Patients with suspected MI should be transferred to a hospital with a coronary care unit and preferably an interventional cardaic catheterisation laboratory. The patient should either be thrombolysed or undergo primary percutaneous coronary intervention (PCI) to open the occluded coronary artery depending on the timely and local availability of each resource [5]. Both treatments have been shown to improve survival. Primary PCI is preferable, if available as it allows definite treatment of the culprit coronary lesion/s. This patient underwent primary PCI and was found to have an occluded ostial Left anterior descending coronary artery (Figure 8) which was effectively treated using a 3.0 x 16 mm drug eluting stent with return of flow and a good angiographic result.

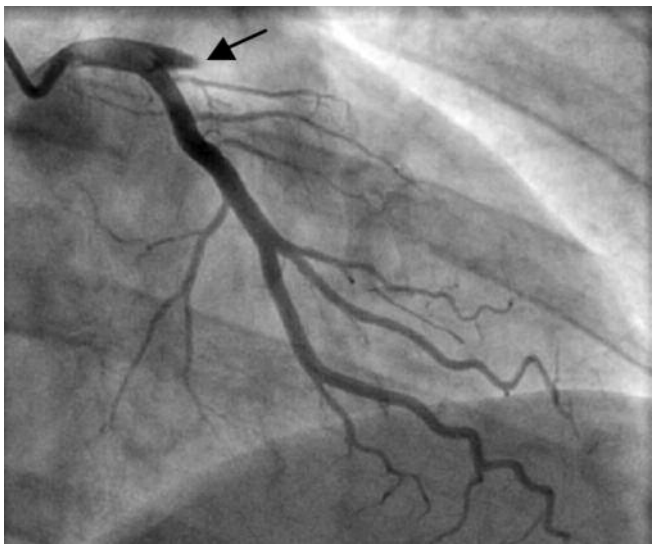

Figure 8: Angiographic image with an occluded ostial LAD (arrow)

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