

Things are not always what they seem

Dr Ken McDonald

As in many areas in medicine, the apparently straightforward presentation in cardiology may hold clues to a potentially life-threatening condition. The following brief clinical histories are recent presentations to St Vincent's University Hospital underlining the need for us to be always vigilant, even in the routine presentation.

Case 1

A 60-year old male presented to the A+E department with a two-week history of palpitation. He also reported similar duration of fatigue but no other symptoms. There was no significant past medical or family history and no risk for cardiovascular disease. The patient reported nothing unusual about the weeks leading up to this presentation. Examination revealed atrial fibrillation with a rapid ventricular response. There was no evidence of heart failure. His admission ECG and CXR were normal.

While rate control, anticoagulation and further investigation can be done at outpatient level in this setting, the patient was admitted. Digoxin and warfarin were prescribed. Further investigations (including serial cardiac enzymes and thyroid function) were normal. Cardiac consultation was requested.

The author reviewed the patient, agreed that examination was normal and that investigations suggested isolated atrial fibrillation without clear explanation. An echocardiogram was requested with the advice that, if possible, this should be done before discharge. To our surprise, echocardiography demonstrated a significantly dilated left and right ventricle with marked reduction in systolic function. The patient was kept in hospital for a further 10 days for coronary angiography (normal), viral studies and rhythm observation. He was discharged home on digoxin, converting enzyme inhibition and warfarin with instructions to rest.

The presumptive diagnosis was myocarditis or idiopathic cardiomyopathy complicated by atrial fibrillation. Follow up echocardiography at two months revealed a return to normal ventricular structure and function, further supporting the diagnosis of myocarditis.

Discussion

Most cases of new onset atrial fibrillation relate to hypertensive or ischaemic heart disease, and do not require

admission to hospital. Rate control is initiated, consideration is given to anticoagulation and cardioversion and other investigations ordered on an elective outpatient basis.

The above case does not suggest that we alter this approach, but does underline the need to obtain a prompt echocardiogram on patients presenting with atrial fibrillation for the first time (further support for open access echocardiography).

The occasional discovery of an underlying cardiomyopathy does significantly alter management decisions, and can in particular influence the choice of rate-slowing agents (avoid calcium channel blockade) and the need for anticoagulation. This case also emphasises that we should not accept a normal CXR as a surrogate for a normal heart, and furthermore that the absence of signs of heart failure when patients are in rapid atrial fibrillation does not exclude a cardiomyopathy.

Case 2

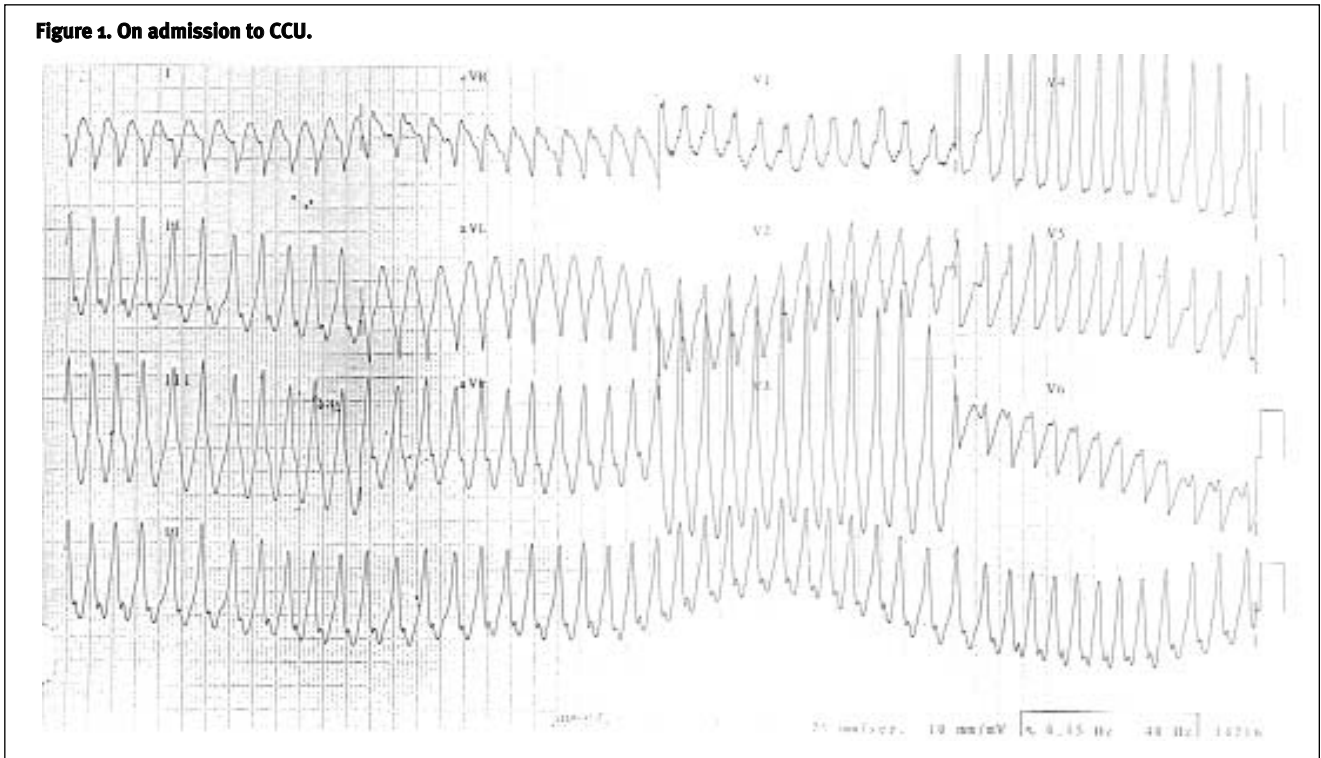
A 55-year old male presented to A+E with a several month history of palpitation, which had gradually become more frequent. Occasionally he felt light-headed but had never fainted. No prior history of note, and nothing to suggest that the patient should be predisposed to arrhythmia. Physical examination revealed a heart rate of 260/minute with a low normal blood pressure and no evidence of heart failure. ECG demonstrated a rapid broad-complex tachycardia, with definite periods of irregularity (see Figure 1). No prior ECG records were available for comparison.

The patient was prescribed intravenous amiodarone and transferred to the CCU. On arrival at CCU, the rhythm had converted to sinus with clear demonstration of delta waves, indicating pre-excitation (see Figure 2). He subsequently underwent full electrophysiological work-up with successful radio frequency ablation of an accessory tract (see Figure 3).

Discussion

The importance of this case is that it underlines the need to always consider a pre-excitation syndrome (WPW) in the presentation of a tachycardia with fast ventricular response (i.e. >200/min). While uncommon, the application of standard rate-slowing agents in this setting can precipitate ventricular fibrillation. Verapamil, digoxin and lidocaine

Figure 1. On admission to CCU.



can all accelerate ventricular response in this condition and result in degeneration of the rhythm to ventricular fibrillation.

In the setting of no known prior history of WPW, clues to its presence are a particularly fast tachycardia or, of course, a prior ECG demonstrating a delta wave which had not been recognised. Where doubt exists in the acute management of a rapid tachycardia intravenous adenosine, beta blockade or cardioversion are all reasonable and safe options.

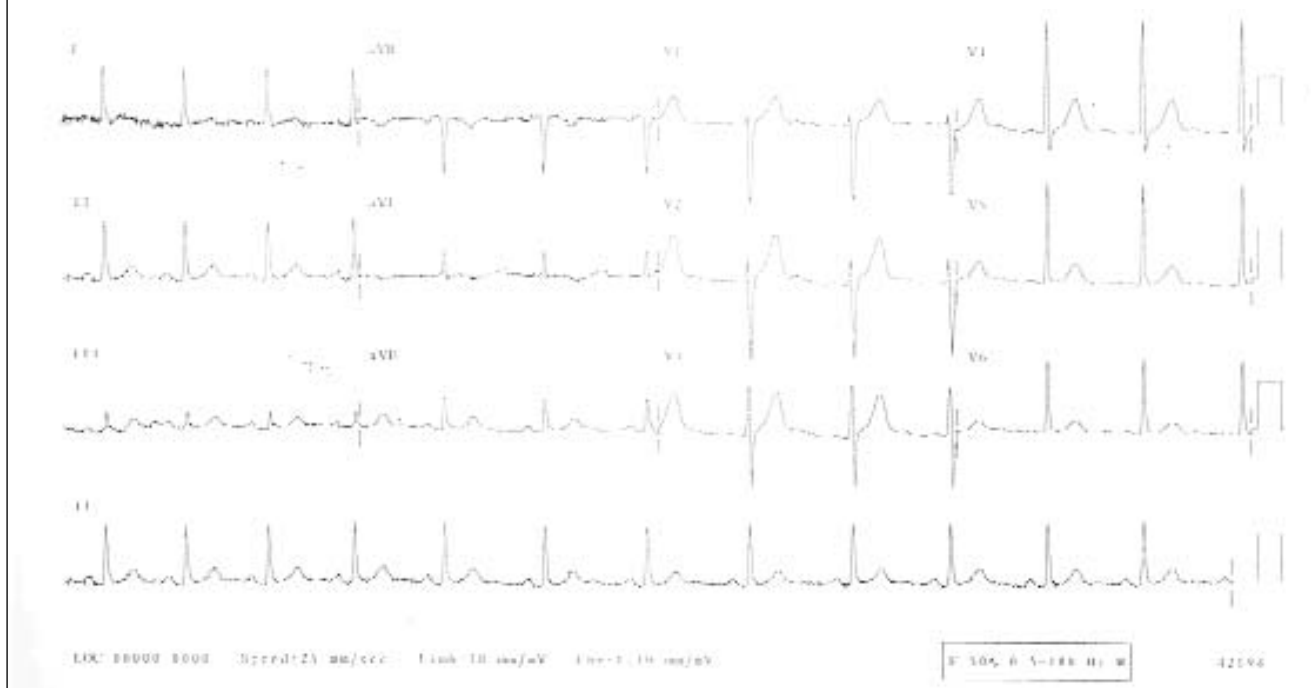
Case 3

A 72-year old Englishman arrived in Ireland for a holiday in his usual state of health. He woke in the early hours of his first morning in this country feeling acutely breathless. Urgent admission to hospital was arranged where initial assessment demonstrated a picture consistent with either a diffuse pulmonary process or pulmonary oedema. Physical examination revealed tachycardia, diffuse lung crackles with no added sounds or murmurs while listening to his heart.

Figure 2.



Figure 3. Post ablation.



ECG showed sinus tachycardia with minor repolarisation changes. CXR demonstrated widespread infiltrates. The man was prescribed antibiotics, lasix and oxygen and admitted to a general medical ward. His condition improved only modestly over the next two days. Serial cardiac data suggested possible ischaemic injury (minor elevation in cardiac enzymes) but no electrocardiographic evidence of a transmural infarct.

Cardiac consultation was requested. Significant change had occurred on physical examination, with progressive hypotension (BP 80mmHg systolic), hypoxaemia and the presence of a short ejection systolic murmur in the mitral area. While the murmur was not dramatic, suspicion turned to ischaemic dysfunction of the mitral valve apparatus, associated with a small subendocardial infarct producing refractory pulmonary oedema. Doppler echocardiography demonstrated well-preserved systolic function of the left ventricle, ruptured chordae tendineae and a flail posterior leaflet of the mitral valve producing wide open mitral regurgitation.

Immediate coronary angiography was performed which demonstrated significant three-vessel disease. An

intra-aortic balloon pump was inserted and the patient was transferred to the Mater Hospital for successful coronary artery bypass surgery and replacement of the mitral valve.

Discussion

Acute pulmonary oedema is a syndrome, not a diagnosis. It has many causes, and, when present, the cause should be actively and immediately sought. If the CXR picture shows diffuse infiltrates, immediate cardiac assessment with Doppler echocardiography and possible haemodynamic evaluation should be performed. Acute mitral valve dysfunction should always be considered in refractory pulmonary oedema, and the absence of a murmur or the presence of a quiet murmur does not exclude the diagnosis. Acute mitral valve dysfunction most often occurs in the setting of an ischaemic event (not always a large infarction), but can occur without ischaemia in the setting of a myxomatous valve.

Dr Ken McDonald, consultant cardiologist, St Vincent's University Hospital, Dublin.