A sinus tachycardia and chest pain

Introduction

This patient presented with severe central chest pain and the electrocardiogram (ECG) in *Figure 1*. What are the principal findings and which specific diagnoses should be promptly evaluated?

The electrocardiogram

The ECG in *Figure 1* demonstrates sinus tachycardia at 120 beats/min. There is evidence of T wave flattening in lead aVL. The PR interval is normal and there is a suggestion of ST elevation in leads II and aVF, although the baseline is wandering making interpretation difficult. There are no significant repolarization abnormalities and the QRS axis is within normal limits. The finding of a resting sinus tachycardia is the most common and often sole electrocardiographic manifestation of acute pulmonary embolism.

Specific diagnoses

In any patient presenting with chest pain and sinus tachycardia three key life-threatening diagnoses should be considered and excluded: acute pulmonary embolism, myocardial infarction and aortic dissection. In an elderly patient presenting with severe central chest pain occurring after vomiting one should also consider oesophaegeal rupture which will be most apparent on the chest radiograph as a pneumo-mediastinum and possible pleural effusion. This article will focus on the ECG features of acute pulmonary embolism and aortic dissection. The electrocardiographic manifestations of acute coronary syndromes and myocardial infarction were delineated in the previous article in this series (Pandya and Strike, 2006).

Pulmonary embolism

Pulmonary embolism is a common and acutely life-threatening condition with a wide clinical spectrum of severity and chronicity. Its prompt diagnosis and treatment may be life-saving, although it can be a difficult diagnosis to establish. There are many aspects to making the diagnosis and the importance of a comprehensive history and examination cannot be underestimated. Several sophisticated imaging techniques are available to the clinician to make a definitive diagnosis, but the ECG can give simple, rapid and important information at the bedside or in the emergency department.

There are a variety of changes which may be seen and one of the diagnostic difficulties is that these tend to be non-specific and the sensitivity is poor. As one of the important differential diagnoses may be primary myocardial ischaemia or infarction, then the absence of findings such as ST segment elevation or depression may be helpful. However, one must be wary as myocardial ischaemia may be a phenomenon secondary to the occurrence of pulmonary embolism. Therefore, one should keep an open mind about findings such as ST segment depression that could represent either pathology. Clearly, ECG findings should never be interpreted in isolation from entire clinical picture.

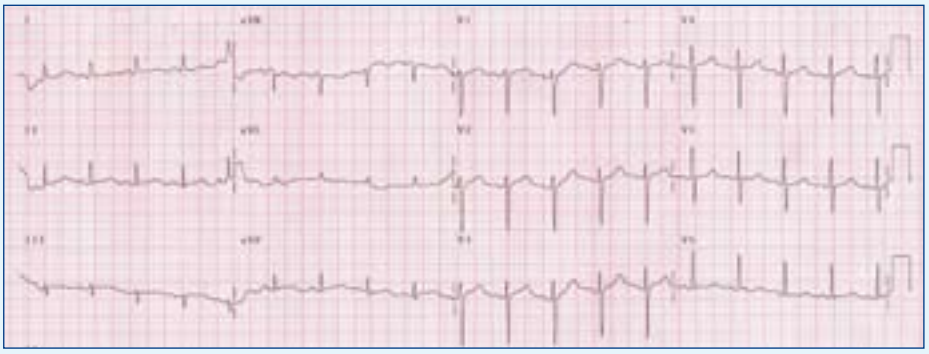

Figure 1. Electrocardiogram showing sinus tachycardia.

Pathophysiology of ECG changes in pulmonary embolism

The central pathological event of pulmonary embolism is physical pulmonary arterial obstruction resulting in increased right heart pressures and ventilation:perfusion mismatch. There is no consensus as to the precise causes of ECG changes but they are likely to represent a combination of mechanical right heart strain, subsequent right ventricle (RV) ischaemia, acute right ventricular and right atrial dilatation, and global myocardial ischaemia. The latter arises because of a combination of hypotension, decreased RV output, reduced left ventricle (LV) preload and relative hypoxia.

A total of 28 different ECG abnormalities have been described in pulmonary embolism (Rodger et al, 2000). These include incomplete or complete right bundle–branch block, low limb lead voltage, T wave inversion in leads V1–V4, T wave inversion in III and aVF, Q waves in III and aVF, ST elevation in leads V1–V4, delayed transition (i.e. the S wave becoming the dominant deflection more laterally in the chest leads) and p-pulmonale (Steeram et al, 1994; Chan et al, 2001; Falterman et al, 2001) (*Table 1*). Many of the studies investigating ECG changes in pulmonary embolism have been lacking in statistical power because of inadequate numbers of controls or inappropriate matching of patient groups.

One of the commonest findings is that

Table 1. Electrocardiogram features in acute pulmonary embolism

Sinus tachycardia
Incomplete or complete right bundle–branch block
T wave inversion in leads III and aVF
ST elevation in leads V1–V4
T wave inversion in V1–V4
Q waves in III and aVF
SI QIII TIII

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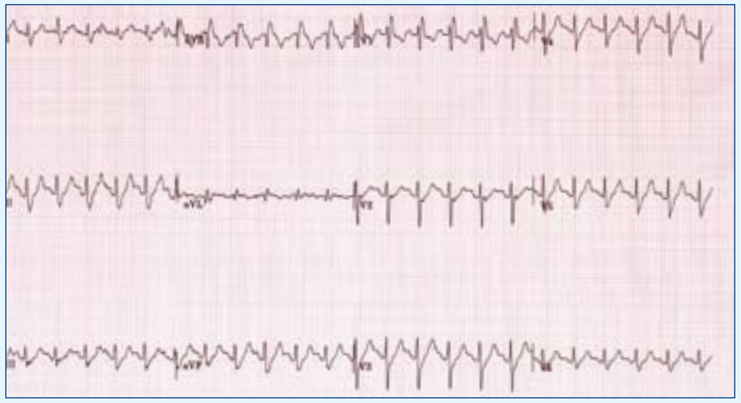


Figure 2. Right bundle–branch block in pulmonary embolism.

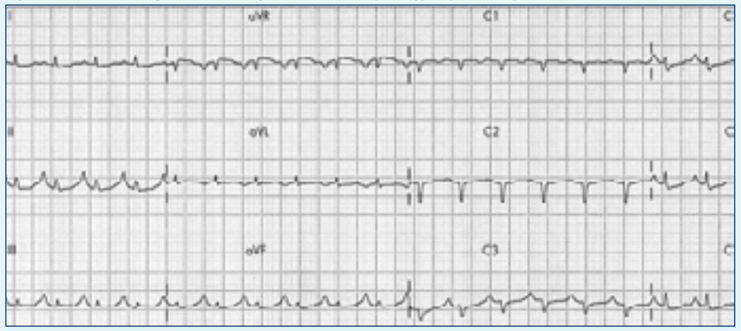
of an isolated sinus tachycardia which should always be treated as suspicious (39–67% of confirmed pulmonary embolism cases). The so-called classical ECG triad of S wave in lead I, Q wave in lead III, T wave inversion in lead III are in fact very uncommon. However, using case controls, the specificity of ECG findings for pulmonary embolism is low (Richman et al, 2004). A normal ECG does not rule out the diagnosis of pulmonary embolism and has been reported in as many as a quarter of patients with acute pulmonary embolism. Similarly, other findings such as axis shift are non-specific. Some authors advocate the use of a right-sided ECG in the diagnosis of pulmonary embolism. This may show ST elevation in the chest leads not detected by the routine left-sided ECG, although it lacks specificity similar to the conventional left-sided ECG (Akula et al, 2003).

Figure 2 shows right bundle–branch block and right heart strain. Note the ST depression seen in leads V1 to V3. *Figure 3* shows peaked broad P waves particularly in the inferior leads which lie closest to the right atrium as a result of right atrial strain in cases of pulmonary embolism.

It should always be remembered that pulmonary embolism is also a cause of atrial fibrillation. Atrial tachyarrhythmias have been seen in approximately 15% of cases of angiographically proven pulmonary embolus and are probably related to acute atrial wall stretch and myocardial ischaemia. Ventricular arrhythmias may also result from sudden pulmonary arterial obstruction as a result of a combination of hypoxia and RV wall stretch in pulmonary embolism causing substantial acute increases in pulmonary arterial pressure.

Several authors have attempted to combine various ECG criteria to increase the diagnostic yield and specificity but none have reliably differentiated between pulmonary embolism and other cardiac or respiratory pathologies. Rodger et al (2000) studied 212 patients with suspected pulmonary embolism in whom the diagnosis was proven in only one third. They found that many of the classical ECG findings associated with pulmonary embolism were equally prevalent in patients suspected of pulmonary embolism in whom the diagnosis is ultimately excluded by a ventilation:perfusion scan or pulmonary angiography.

Figure 3. Electrocardiogram illustrating the P wave morphology in pulmonary embolism.



Aortic dissection

The diagnosis of aortic dissection is important to make as rapidly as possible because good medical treatment and consideration of early surgery may be life-saving. Factors in the history such as the onset and nature of the pain are of the utmost importance but there are several abnormalities of the ECG that can either help or hinder the diagnostic process.

Again, one of the chief differential diagnoses is that of myocardial infarction or ischaemia, and the ECG may be helpful by displaying its absence. However, it is possible for complications of aortic dissection to mimic an acute coronary syndrome secondary to coronary atherosclerotic plaque disease. The dissected aorta can dissect the coronary artery ostia casing ST elevation, or can occlude the coronary ostium with the dissection flap.

Both inferior and anterior ST elevation are well described, but inferior elevation is allegedly more common. Mechanistically, ECG changes can also develop as a result of the presence of shock and as a result of pre-existing coronary artery disease. Other changes such as ST depression, T wave changes and bundle–branch block have been described (Hirata et al, 1995). If the dissection tracks down into the pericardial space then it may be possible to also have the pericarditic ECG changes of concave ST elevation, which can be confused with acute coronary syndrome unless care is taken (*Figure 4*).

In the majority of cases, as with pulmonary embolism, the ECG findings are non-specific. In one third of cases, the ECG is normal and in one third of cases there is left ventricular hypertrophy, usually secondary to hypertensive heart disease (Isselbacher et al, 1997). The presence of severe chest pain and an ECG showing left ventricular hypertrophy should always raise the suspicion of aortic dissection. Ultimately, however, the firm diagnosis of aortic dissection requires further imaging (such as computed tomography, magnetic resonance imaging or transoesophageal echocardiography) and this should be performed as urgently as possible.

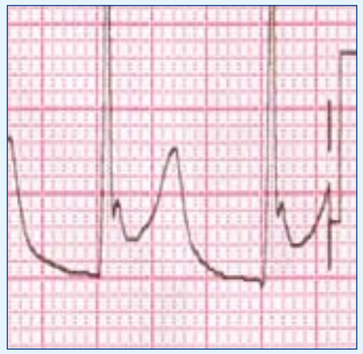


Figure 4. Pericarditic ST segment elevation and first-degree heart block in a patient with extensive aortic dissection involving the aortic root.

Conclusions

The presence of chest pain with an abnormal ECG does not always indicate acute coronary syndrome. It is important to recognize the wide variety of ECG changes which may accompany conditions such as pulmonary embolism and aortic dissection, although one must bear in mind that these ECG changes have low diagnostic specificity. Comprehensive history taking and examination are necessary in addition to a high index of clinical suspicion to ensure that these potentially life-threatening conditions are rapidly recognized and appropriately treated. **BJHM**

Figure 3 *is reproduced from Hirata et al (1995) by kind permission of Elsevier Press.*
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KEY POINTS

- If a patient presents with acute chest pain and sinus tachycardia, exclude three life-threatening diagnoses: myocardial infarction, aortic dissection and pulmonary embolism.
- The clinical history and examination are critical in making a correct diagnosis.
- A normal electrocardiogram does not rule out pulmonary embolism in up to 25% of cases.
- Pulmonary embolism may present with atrial arrhythmia including atrial fibrillation.
- Aortic dissection may cause ST elevation as a result of coronary ostial occlusion or electrocardiogram changes of pericarditis as a result of pericardial bleeding.