LETTERS TO THE EDITOR

EVALUATION OF THERAPIES IN THE TREATMENT OF *HELICOBACTER PYLORI* ASSOCIATED NON-ULCER DYSPEPSIA

Dear Sir,

The above paper by Nafeeza et al[1] purports to study the effectiveness of various regimes in 'elimination' of *H pylori* from the gastric antrum. The end-point of treatment was based on a repeat biopsy two weeks after a course of treatment and a negative result was said to show elimination. Failure to find *H pylori* soon after a course of therapy may not signify eradication of the infection since the latter frequently would return, presumably having regrown from sanctuary sites. It is generally agreed that assessment of therapy of *H pylori* should be performed at least 4 weeks after cessation of treatment[2]. Failure to demonstrate infection within 4 weeks of cessation of treatment should be considered only as suppression. The use of the term elimination in this report is therefore misleading.

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REPLY FROM AUTHOR

Dear Sir

It was stated in our abstract that the suppression rate of *H. pylori* was highest in patients on combination therapy. We regret if the term "elimination" was misleading. What we really meant by the term elimination was suppression and not eradication. In our conclusion, we also mentioned that a short term combination therapy was successful in suppressing *H. pylori* with associated improvement of gastritis but these effects may not be long lasting (as *H. pylori* can 'regrow ' from sanctuary sites).

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ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: Acute pulmonary embolism

DISCUSSION

The initial ECG shows sinus tachycardia with ST elevation in lead V1 and ST depression in leads V4 to V5. On the second ECG, there is development of a complete right bundle branch block with prolongation of the QRS duration, a tall rSR' complex in V1 and a deep S wave in leads V5 and V6. In addition, there is an S wave in lead I, and a Q wave with T wave inversion in lead III; the S1, Q3, T3 complex. These findings were strongly suggestive of pulmonary embolism, which was confirmed on subsequent ventilation-perfusion imaging and pulmonary angiography.

The S1, Q3,T3 pattern was originally described by McGinn and White in 1935[1] and has become the traditional electrocardiographic sign associated with pulmonary embolism. However, it occurs in only a minority of cases (12-30%)[1,2]. Transient right bundle branch block (9%) has also been described in pulmonary embolism[2,3]. Other ECG features include right (7%) or left (7%) axis deviation, tall peaked P waves (P pulmonale, 6%) and an S1, S2, S3 pattern (7%). Note that these features are relatively uncommon. Indeed the ECG may be completely normal (13%) with only sinus tachycardia. The absence of sinus tachycardia is believed to be a point against the diagnosis of pulmonary embolism[4]. Other common findings include ST and T wave changes such as T inversion (42%) from V1 to V3 or ST depression (26%). These non-specific features are present in many other conditions such as ischaemic heart disease. Thus the ECG in many cases of pulmonary embolism may not contribute to the diagnosis. Atrial arrhythmias such as atrial fibrillation and flutter have also been described, but these are uncommon.

The differential diagnosis to consider in this case is that of inferior myocardial infarction in view of the presence of a Q wave in lead III and T inversion in leads III and aVF. However, in inferior infarction, Q waves and T wave changes, together with ST elevation tend to involve leads II, III and avF rather than lead III mainly[5] as in this example. Pulmonary embolism may also simulate an anteroseptal infarction with a QR pattern in leads V1 and V2, and ST or T wave abnormalities in leads V1 to V3 as a result of right ventricular strain[4]. However, these changes do not involve leads V4 to V6. Additional helpful features (not seen in this case) that may occur with pulmonary embolism but are less likely with myocardial infarction are right axis deviation, clockwise rotation, right atrial abnormalities (tall peaked P waves) and right ventricular strain.

Right bundle branch block may occur in normal hearts as well as a wide range of cardiac conditions, including coronary artery disease, hypertension, rheumatic heart disease, cor pulmonale, cardiomyopathies, Ebstein's anomaly, and atrial septal defect. In a survey of healthy air force personnel aged 20 to 40 years, right bundle branch block occurred in 1.5 per thousand[6]. Transient right bundle branch block may occur in acute pulmonary embolism, as a result of elevated right ventricular pressures giving rise to compression of the right bundle. Acute myocardial infarction may also give rise to an acute right bundle branch block, a poor prognostic feature. However, it usually occurs in association with anterior myocardial infarction, rather than inferior myocardial infarction[7]. The right bundle is supplied by septal branches from the left anterior descending artery; thus the development of a new right bundle branch block suggests proximal left anterior occlusion, before the origin of the first septal perforator.

In general, the diagnosis of pulmonary embolism is dependent on a constellation of clinical features such as pleuritic chest pain (74%), dyspnoea (84%), tachypnoca (92%) and sinus tachycardia (43%)[8], often in the setting of a predisposing condition, with confirmation by ventilation-perfusion lung scan or pulmonary angiography. A high index of suspicion in the appropriate clinical situation is important, since pulmonary embolism may mimic many other conditions such as congestive cardiac failure, pneumonia and myocardial infarction; thus it has been called "the great masquerader"[9].

In this patient the ECG is very suggestive of the diagnosis. However, it is important to re-emphasize that in most cases of pulmonary embolism, the electrocardiographic findings are non-specific. A normal looking ECG does not exclude the possibility of embolism. In the Urokinase-Streptokinase Pulmonary Embolism Trial study, typical ECG features such as S1Q3T3, right bundle branch block, P pulmonale, or right axis deviation were present in only 26% of cases[1].

In this patient, the ventilation-perfusion scan showed moderately high probability of pulmonary embolism and pulmonary angiography showed a massive pulmonary embolus at the main pulmonary artery. He was given intra-pulmonary streptokinase and anticoagulated. His general condition improved.

This patient had no prior history of illness or recent immobilisation. Subsequent investigations showed no evidence of deep vein thrombosis, nor was there any abnormality of coagulation such as antithrombin III deficiency or Protein S deficiency that might have predisposed him to develop pulmonary embolism. A CT scan of the abdomen, however, revealed renal cell carcinoma with involvement of the inferior vena cava. In the management of pulmonary embolism, it is important to consider the possibility of an underlying pathology, especially if no obvious predisposing cause (eg recent confinement to bed due to surgery) is present.

References

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