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The ECG "Lead I Sign" in Cardiac Disease – An Indicator of Coexisting Obstructive Pulmonary Disease

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ABSTRACT

Two patients with co-existing cardiac disease and chronic obstructive pulmonary disease are described. The first patient had Wolff-Parkinson-White syndrome and the second patient had extensive anterior Q wave myocardial infarction. In addition to the distinctive ECG patterns of their cardiac abnormalities, both patients also showed the "lead I sign" which is a highly specific marker of chronic obstructive pulmonary disease. These two patients suggest that even in the presence of cardiac disease, the diagnosis of chronic obstructive pulmonary disease should be strongly suspected when the "lead I sign" is present.

Keywords: "lead I sign", cardiac and chronic obstructive pulmonary disease

INTRODUCTION

The "lead I sign" is a highly specific ECG marker of chronic obstructive pulmonary disease (COPD) and has been very rarely documented in any other condition[1-3]. We describe the ECG findings in two patients with COPD co-existing with cardiac disease ie. Wolff-Parkinson-White (WPW) syndrome and extensive anterior myocardial infarction. In addition to the abnormalities attributable to their cardiac anomalies, both these patients also showed the "lead I sign" in their 12-lead ECG due to the co-existing COPD. To the best of our knowledge, such unique ECG patterns have so far not been reported in the literature in patients with COPD and cardiac disease.

CASE REPORTS

Case 1

A 58-year-old man was admitted to hospital for palpitations. Clinical examination revealed a regular heart rate of about 180/minute and a blood pressure of 110/70 mmHg. It was noted that the patient, who was a heavy smoker for the past 20 years, had severe dyspnoea on minimal exertion and had been admitted several times to hospital for acute attacks of breathlessness. Clinical examination revealed persistent pursed lip breathing and a barrel shaped chest. Lung function test showed severe irreversible airflow limitation: FEV1 35% predicted, FVC 74% predicted, FEV1/FVC = 30%. Arterial blood gases analysis showed moderate hypoxaemia with mild hypercapnoea (PaO_2 = 69 mmHg, $PaCO_2$ = 48 mmHg). Chest radiograph showed a normal sized heart but hyperinflated lungs and flattened diaphragms. High resolution computerised tomography confirmed severe diffuse emphysema.

The 12-lead ECG which was recorded on admission to hospital showed a regular narrow complex tachycardia of around 220/minute and a diagnosis of supraventricular tachycardia was made. Intravenous verapamil was given resulting in reversion of the cardiac arrhythmia. Subsequent clinical examination of the heart revealed no cardiac murmurs or other abnormalities. A repeat ECG was then performed. It showed sinus rhythm with no evidence of conduction abnormalities. A subsequent ECG which was done a few days later showed intermittent WPW conduction (Fig 1). Wolff-Parkinson White conduction was seen in beats 2, 3 and 4 in leads I, II, III, beats 1, 3 and 5 in leads aVR, aVL, aVF, beats 1 and 3 in leads V1, V2, V3, beats 1, 2 and 4 in leads V4, V5, V6 and beats 2 to 5, 7, 9, 10 and 12 in the long rhythm strip II. In the beats which were conducted normally, the following classical features of COPD were present: (1) tall peaked P waves measuring 3 mm ("P pulmonale") in leads II, III and aVF and (2) poor R wave progression in the praecordial leads resulting in a RS complex in lead V4. We believe that the "lead I sign" was also present because of the following reasons. The P and T waves in lead I were isoelectric, thus fulfilling 2 of the 3 criteria for a "lead I sign". In addition, the predominantly positive QRS complex in this lead is very small in amplitude measuring only 3 mm. In support of the hypothesis that this low QRS amplitude is truly a reflection of the "lead I sign", is the finding that the predominantly negative QRS amplitude in lead aVL is even smaller measuring only 2 mm, thus suggesting that the QRS axis is somewhere between +60° to +90°. In the beats with WPW conduction, the classical ECG pattern of this conduction abnormality is seen as indicated by a short PR interval (< 0.12 second) and wide QRS complexes due to delta waves in all the leads with an upright R wave or Rs wave[4]. In addition, "P pulmonale" was seen in leads II, III and aVF. Lastly, the "lead I sign" was also seen as indicated by an absent P wave and very low

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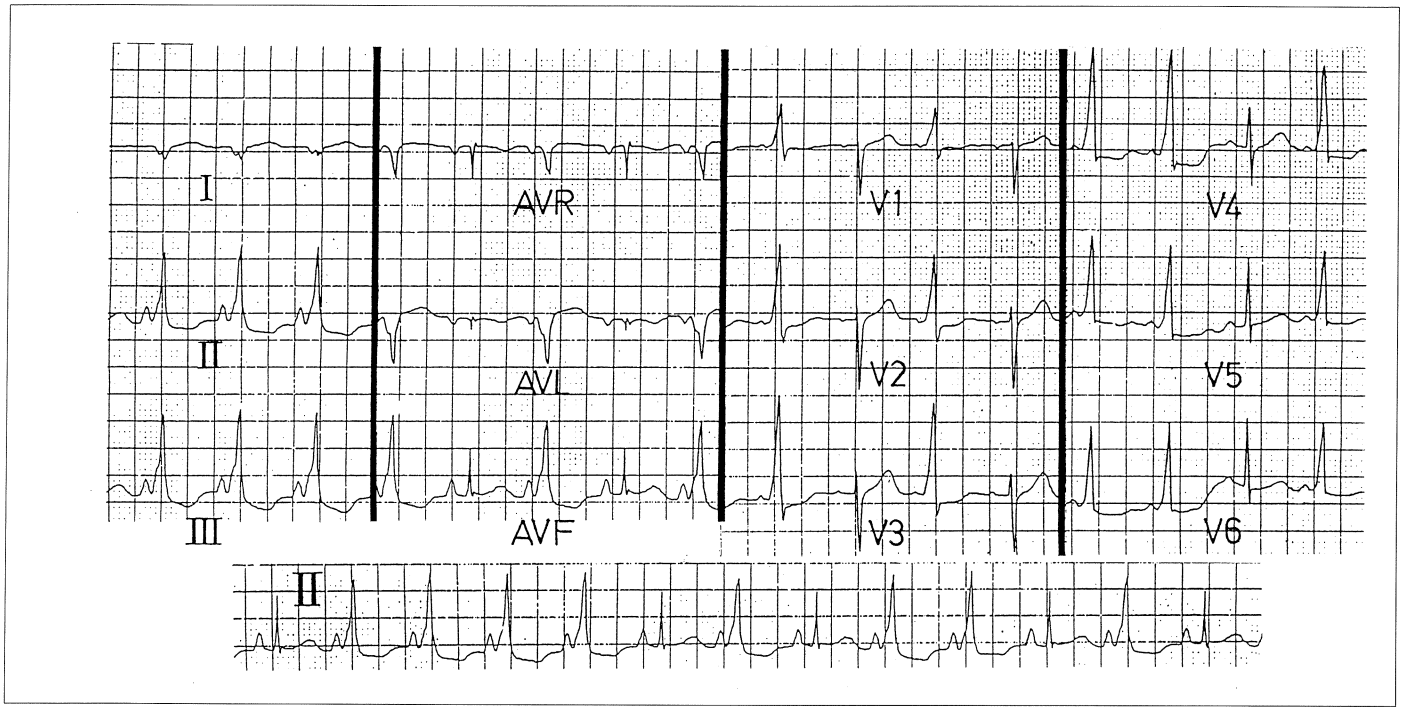

Fig 1 – The ECG shows intermittent WPW conduction. During both normal and WPW conduction, the "lead I sign" was present as reflected by absent P wave and very low amplitudes of the QRS and T wave complexes in lead I. "P pulmonale" was seen in leads II, III and a VF (see text).

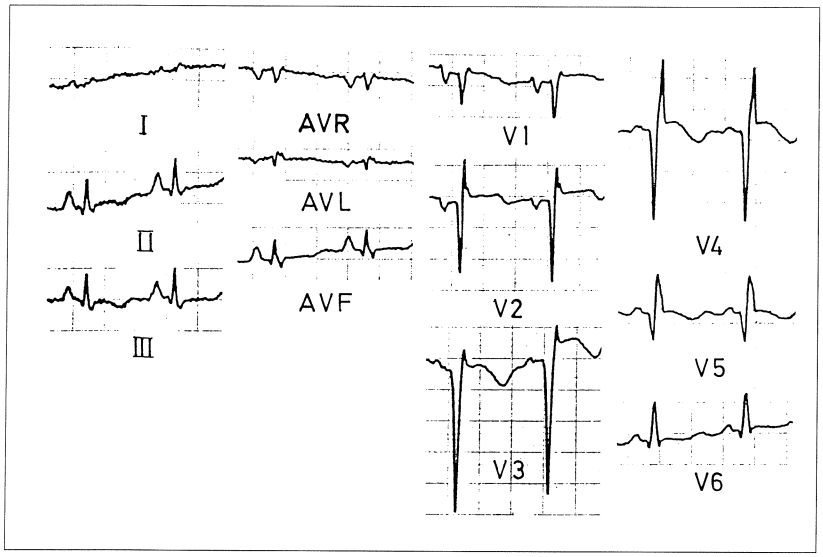

Fig 1 – The ECG shows (1) Q waves myocardial infarction in leads V2 to V5. (2) "Pulmonale pilmonale" in lead II and (3) "lead I sign" (see text).

amplitudes of the QRS and T wave complexes in lead I. Both the latter two ECG abnormalities strongly indicate COPD. It is interesting to note that in this patient, the "lead I sign" was seen even in the presence of WPW conduction.

An electrophysiological study which was performed several months later confirmed a single accessory pathway localised to the distal left ventricular free wall. Echocardiography was not performed in this patient. Following his discharge from the hospital, the patient has been followed up in the outpatient clinic and has been maintained on oral prednisolone, frusemide and inhaled salbutamol and ipratropium bromide from metered dose inhalers.

Case 2

A 60-year-old man presented with congestive cardiac failure. From his medical history, he had been a known case of COPD and was also a heavy cigarette smoker (40 sticks per day) for many years. One month previously, he was admitted to another hospital for acute myocardial infarction.

Clinical examination on admission revealed signs of congestive heart failure as indicated by elevated jugular venous pulse and pedal oedema. Examination of the heart revealed normal heart sounds and no cardiac murmurs. The blood pressure was 140/80 mmHg. The patient was also noted to have a barrel shaped chest. Examination of the lungs showed evidence of emphysema such as hyperresonance to percussion, loss of liver and cardiac dullness, poor lung expansion and poor air entry in both the lungs.

The chest radiograph showed hyperinflated lungs and flattened diagrams suggesting COPD. No lung function tests were performed.

The 12-lead ECG (Fig 2) showed sinus rhythm and deep Q waves associated with elevated ST segment elevation and T wave inversion in leads V2 to V5 indicating anterior Q wave myocardial infarction. In addition, tall 3 mm P waves were seen in lead II ("P pulmonale"). The "lead I sign" was also present as reflected by very low amplitudes of the P, QRS, T wave complexes in lead I resulting in almost a straight line. The presence of the "lead I sign" and "P pulmonale" in this patient strongly suggest COPD. The co-existence of a large anterior myocardial infarct as described above was subsequently confirmed by two-dimensional echocardiography which showed akinesia of the ventricular septum and apex of the left ventricle.

The patient improved with diuretic therapy which was given and was discharged from the hospital. However, during the next few weeks, he was repeatedly readmitted to hospital for intractable heart failure and finally passed away.

DISCUSSION

The typical ECG changes in COPD are: (1) prominent P waves in leads II, III and aVF; (2) rightward shift of the QRS axis in the frontal plane; (3) poor progression of the R wave in the praecordial leads; (4) low voltage of the QRS complexes especially in the left praecordial leads, and (5) the "lead I sign"[2].

In patients with COPD, the frontal plane P, QRS and T wave axes are not infrequently all directed at around +90°. These three vectors are therefore directed either precisely or almost perpendicular to the standard lead I axis. As a result of this, lead I reflects either absent or very low amplitude P, QRS,T wave complexes giving the appearance of a minimally disturbed baseline. This ECG phenomenon is known as the "lead I sign".

In 1965, Fowler and co-workers reported 15 patients with severe pulmonary emphysema with cor pulmonale. They found that 5 patients (33%) showed the "lead I sign"[1]. In 13 control patients with pulmonary thromboembolism or idiopathic pulmonary hypertension, only one patient showed the "lead I sign". This patient had normal lung function tests. These authors proposed very strict arbitrary criteria for the diagnosis of the "lead I sign" consisting of isoelectric P wave in lead I combined with a very small QRS complex of less than 1.5 mm total deflection and a T wave of less than 0.5 mm in lead I. Since then, there has been no other studies on the prevalence of the "lead I sign" in COPD. However, more recently, Schamroth described the "lead I sign" as being reflected by "absent or very low amplitude P, QRS, T wave complexes giving the appearance of a minimally disturbed baseline" without any specification of the cut-off values for the amplitudes of these three waveforms[2]. In our experience, the "lead I sign" is a highly specific indicator of COPD, being rarely, if ever seen, in any other conditions[5]. In our two cases, the amplitudes of the P, QRS and T waves were exceedingly small, especially when compared to the corresponding amplitudes of these three waves in most of the other leads. As such, we believe that they are truly a reflection of the "lead I sign".

The "lead I sign" so far has been described only in patients presenting with COPD alone. To the best of our knowledge, it has not been described in COPD patients with co-existing cardiac disease. Therefore, our two cases are unique, because they are the first examples of the "lead I sign" co-existing with specific ECG changes of WPW conduction pattern and anterior Q wave myocardial infarction.

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