Reminder of important clinical lesson

At the twisted heart of nicotine addiction

Dante A Suffredini,¹ Robert Michael Reed^{1,2}¹Department of Medicine, University of Maryland School of Medicine, Baltimore, Maryland, USA²Division of Pulmonary and Critical Care Medicine, University of Maryland School of Medicine, Baltimore, Maryland, USACorrespondence to Dr Robert Michael Reed, reed@medicine.umaryland.edu

Summary

This case report reviews the ECG manifestations of chronic obstructive pulmonary disease (COPD). Distinct changes of the P-wave axis, QRS axis and morphology are a direct result of the pathophysiological and anatomical changes associated with COPD. These changes are illustrated in a patient with a history of COPD presenting with hypoxaemia and symptoms of shortness of breath.

BACKGROUND

Chronic obstructive pulmonary disease (COPD) is a common disease seen in a myriad of medical settings from outpatient clinics to intensive care units. A diagnosis of COPD is never made by an ECG; however, recognition of commonly associated ECG patterns can help identify uncharacteristic changes that may imply the presence of a secondary disease process. In the evaluation of a patient with shortness of breath this could include disease states such as myocardial ischaemia, infarction or even pulmonary embolus. This article is relevant to not only outpatient providers but also emergency medicine, cardiac and pulmonary physicians.

CASE PRESENTATION

A man in his late 60s presented to the emergency room with complaints of severe shortness of breath. He provided only a limited history as he was irritable, apathetic and mildly confused. He took no medications routinely and had smoked heavily until recently, when he had become so dyspnoeic with walking that he was no longer able to go to the store to buy cigarettes. The patient was

in respiratory distress, cachectic and dishevelled. He was tachycardic and breathing 32 times a minute with an initial oxygen saturation of 74% in room air, which improved to 96% with supplemental oxygen. An ECG was obtained (figure 1).

INVESTIGATIONS

This patient's dyspnoea was due to decompensated severe COPD complicated by pulmonary hypertension. The ECG presented is characteristic for this disease state. Pulmonary function testing during hospitalisation confirmed a severe obstructive ventilatory defect with an FEV₁/FVC ratio of 59.1, FEV₁ of 0.52 litres (18.5% predicted) and FVC of 0.88 litres (24.4% predicted). A transthoracic echocardiogram revealed an elevated estimated right ventricular systolic pressure (RVSP) at 50 mm Hg. Although RVSP estimates are notoriously inaccurate in COPD,¹ the examination provided additional findings consistent with a diagnosis of pulmonary hypertension including leftward septal bowing, right ventricular and right atrial hypertrophy and dilation. The ECG (figure 1) demonstrates several signs that are the direct result of the anatomical and physiological changes

Figure 1 Initial ECG: 25 mm/s, 10 mm/mV.

Figure 2 Chest radiograph illustrating the anatomical changes of chronic obstructive pulmonary disease (COPD) compared to a normal cardiomeastinal silhouette (outlined in blue). Standard placement of ECG leads are shown relative to a normal silhouette. Notice the inferior and clockwise displacement (red arrow) of the COPD heart.

associated with decompensated COPD and these changes are discussed below. Other aspects of this case have been presented previously.²

OUTCOME AND FOLLOW-UP

The patient presented to the hospital with acute hypercarbic respiratory failure and cor pulmonale with volume overload. He was treated with nebulised bronchodilators, antibiotics, intravenous corticosteroids and diuresis with improvement over several days. The patient was

discharged on home oxygen and was encouraged to stop smoking.

DISCUSSION

Severe COPD is associated with lung hyperinflation that depresses the diaphragms into the abdomen, giving them the appearance of being flattened on a chest radiograph³ (figure 2). As the diaphragms descend, the heart shifts inferiorly and medially with clockwise rotation in the coronal plane^{3 4} (figures 2 and 3). Understanding these changes in reference to standard ECG lead placement facilitates understanding of many of the observed changes in vector and voltage (figure 2).

The patient's ECG has a P-wave axis of 86° (normal 0° to 75°). This exemplifies a highly characteristic ECG finding in COPD called 'verticalisation' of the mean frontal P-wave axis with an angle between +70° and +90°. A vertical axis accompanied by an isoelectric P-wave in lead I, negative P-wave in lead aVL and large upright P-waves in leads II and III is considered quite specific for the diagnosis of obstructive lung disease.⁶

Our patient's ECG also shows a P-pulmonale pattern, right-axis QRS deviation and right ventricular hypertrophy (figure 4). P-wave morphology, particularly in the inferior leads, can become tall and peaked (originally described as 'gothic'⁷) with a height exceeding 2.5 mm and is known as 'P-pulmonale'^{5 8 9} when accompanied by a P-wave axis to the right of +70°. These tall P-waves of P-pulmonale are usually attributable to right atrial enlargement and are often associated with right ventricular hypertrophy complicating pulmonary hypertension, COPD or congenital heart disease.¹⁰ The 'lead I sign' refers to an isoelectric P-wave in lead I with QRS and T-wave amplitudes less than 1.5 and 0.5 mm, respectively.¹¹ This pattern of P-wave change is associated with emphysema.

The QRS complex may also change in COPD as a result of cardiac rotation, right ventricular hypertrophy or strain.^{8 10} This manifests through right-axis deviation

Figure 3 CT scans comparing a normal subject (left) to our patient described in the text (right). Images are aligned at the right pulmonary vein. Note the inferomedial position of the chronic obstructive pulmonary disease (COPD) heart compared with the normal subject. In the normal subject the apex of the heart is visible, whereas it has shifted inferiorly and out of the transverse plane in the patient with COPD.

Figure 4 The patient's ECG demonstrating a vertical P-wave axis, P-pulmonale (P), the $S_1S_2S_3$ syndrome and other changes described in the text.

with prominent R-waves in lead V1. A small percentage of patients exhibit paradoxical left-axis deviation that has been called a 'pseudo left-axis deviation'. This is distinguished from usual left-axis deviation by absence of systemic hypertension, coronary artery disease or other causes of myocardial failure.⁵ One theory to explain this finding is that emphysematous lungs have decreased lateral electrical conductance resulting in primarily vertical vectors to be recorded on an ECG. Thus, forces that are directed to the left arm are recorded as superiorly directed vectors causing the appearance of an axis change.^{12–13} Associated with this is the $S_1S_2S_3$ pattern. It is present when there is an R:S ratio <1 giving prominent S waves in leads I, II and III.¹⁴ The mechanism causing this pattern may be the result of a conduction defect in the crista supraventricularis of the right ventricle.¹² It is an uncommon finding, seen in 23% of patients with severe COPD but can also be observed in normal subjects for unknown reasons.¹⁵

Our patient's ECG includes poor R-wave progression in the precordial leads. In general, poor R-wave progression occurs when the initial anteriorly directed electrical vectors become posteriorly directed or have a decrement in magnitude such as in left or right ventricular hypertrophy

or anterior myocardial infarction.¹⁶ The clockwise rotational changes associated with COPD can alter the transitional zone of R-wave progression in the precordial leads leading to this finding. Cardiac medialisation and rotation in the horizontal plane brings the right ventricle closer to the right precordial leads (V1–V3), whereas the left ventricle moves posteriorly and inferiorly.⁸ Poor R-wave progression is defined by an R-wave magnitude 3 mm or less in V3 when the R-wave in V2 is less than or equal to the R-wave in V3.¹⁷ Another related pattern termed 'reversed R-wave progression' is present when progressive R-wave magnitude decreases such that $V2 < V1$ or $V3 < V2$.¹⁶ In this there is a shift of R-wave progression to the left precordial leads causing a persistent rS pattern as late as V5 or even V6.¹⁰ As a consequence, the right precordial leads may have large QS complexes resembling a pseudo prior Q-wave anterior myocardial infarction.¹⁰

Additional ECG manifestations of COPD are listed in table 1. Several studies have examined the ECG as a diagnostic modality for COPD. The predictive value in using ECG criteria to diagnose COPD has been shown to vary according to the severity of COPD.^{9–18} Of note, a study by Asad *et al*¹⁹ showed that the ECG findings of P-pulmonale in decompensated COPD are dynamic and P-wave amplitude can decrease after treatment.

Table 1 Common ECG changes associated with chronic obstructive pulmonary disease

Normal ECG
Frontal P-wave axis 'verticalisation' between 70° and 90°: negative P-wave in lead aVL and large upright P-waves in leads II and III
Right atrial enlargement or 'P-pulmonale': tall P-waves especially in the inferior leads with heights >2.5 mm
Right axis deviation
Right ventricular hypertrophy
Low limb lead voltage
'Pseudo left-axis deviation': ventricular activation in the setting of a leftward and posterior change in position of the heart
'Poor R-wave progression': R-wave magnitude 3 mm or less in V3 and when the R-wave in V2 is less than or equal to the R-wave in V3
'Reversed R-wave progression': present when progressive R-wave magnitude decreases such that $V2 < V1$ or $V3 < V2$
The ' $S_1S_2S_3$ pattern': R:S wave ratio <1 in leads I, II and III
The 'lead I sign': isoelectric P-wave, QRS amplitude <1.5 mm, T-wave amplitude <0.5 mm in lead I
QRS amplitude in I, II, III, aVR, aVL and aVF equal to or less than 5 mm
R:S wave ratio equal to or less than 1 in leads V5 and or V6

Learning points

- ▶ The ECG manifestations can be characteristic but are not specific for chronic obstructive pulmonary disease (COPD) and may be seen in other disease states such as pulmonary hypertension and congenital heart disease.
- ▶ Distinct ECG changes seen with COPD are a result of lung hyperinflation causing rotation and medialisation of the heart.
- ▶ Recognising how these changes evolve may provide a better understanding of the presentation of simultaneous disease processes such as myocardial ischaemia in a patient with COPD and dyspnoea.

Competing interests None.

Patient consent Not obtained.

REFERENCES

1. **Arcasoy SM**, Christie JD, Ferrari VA, *et al*. Echocardiographic assessment of pulmonary hypertension in patients with advanced lung disease. *Am J Respir Crit Care Med* 2003;**167**:735–40.
2. **Reed RM**. Captain Ignose to the rescue. *Am J Med* 2010;**123**:704–6.
3. **Thomas AJ**, Apiyasawat S, Spodick DH. Electrocardiographic detection of emphysema. *Am J Cardiol* 2011;**107**:1090–2.
4. **Shah NS**, Koller SM, Janower ML, *et al*. Diaphragm levels as determinants of P axis in restrictive vs obstructive pulmonary disease. *Chest* 1995;**107**:697–700.
5. **Spodick DH**. Electrocardiographic studies in pulmonary disease. I. Electrocardiographic abnormalities in diffuse lung disease. *Circulation* 1959;**20**:1067–72.
6. **Baljeppally R**, Spodick DH. Electrocardiographic screening for emphysema: the frontal plane P axis. *Clin Cardiol* 1999;**22**:226–8.
7. **Zuckerman RCE**, Fishleder BL, Sodi-Pallares D. The electrocardiogram in chronic cor pulmonale. *Am Heart J* 1948;**35**:421–37.
8. **Rodman DM**, Lowenstein SR, Rodman T. The electrocardiogram in chronic obstructive pulmonary disease. *J Emerg Med* 1990;**8**:607–15.
9. **Wasserburger RH**, Kelly JR, Rasmussen HK, *et al*. The electrocardiographic pentalogy of pulmonary emphysema. A correlation of roentgenographic findings and pulmonary function studies. *Circulation* 1959;**20**: 831–41.
10. **Harrigan RA**, Jones K. ABC of clinical electrocardiography. Conditions affecting the right side of the heart. *BMJ* 2002;**324**:1201–4.
11. **Fowler NO**, Daniels C, Scott RC, *et al*. The electrocardiogram in cor pulmonale with and without emphysema. *Am J Cardiol* 1965;**16**:500–5.
12. **Grant RP**. Left axis deviation; an electrocardiographic-pathologic correlation study. *Circulation* 1956;**14**:233–49.
13. **Perloff JK**, Roberts NK, Cabeen WR Jr. Left axis deviation: a reassessment. *Circulation* 1979;**60**:12–21.
14. **Kamper D**, Chou TC, Fowler NO, *et al*. The reliability of electrocardiographic criteria of chronic obstructive lung disease. *Am Heart J* 1970;**80**:445–52.
15. **Schaeffer JW**, Pryor R. Pseudo left axis deviation and the S₁S₂S₃ syndrome in chronic airway obstruction. *Chest* 1977;**71**:453–5.
16. **Zema MJ**, Kligfield P. ECG poor R-wave progression: review and synthesis. *Arch Intern Med* 1982;**142**:1145–8.
17. **Zema MJ**, Kligfield P. Electrocardiographic poor R wave progression. II: correlation with angiography. *J Electrocardiol* 1979;**12**:11–15.
18. **Holtzman D**, Aronow WS, Mellana WM, *et al*. Electrocardiographic abnormalities in patients with severe versus mild or moderate chronic obstructive pulmonary disease followed in an academic outpatient pulmonary clinic. *Ann Noninvasive Electrocardiol* 2011;**16**:30–2.
19. **Asad N**, Johnson VM, Spodick DH. Acute right atrial strain: regression in normal as well as abnormal P-wave amplitudes with treatment of obstructive pulmonary disease. *Chest* 2003;**124**:560–4.

This pdf has been created automatically from the final edited text and images.

Copyright 2012 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Suffredini DA, Reed RM. At the twisted heart of nicotine addiction. *BMJ Case Reports* 2012;10.1136/bcr-2012-006240, Published XXX

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow