CLINICAL PRESENTATION

A 58-year-old man presented to the emergency department with a one-week history of left-sided weakness. Computed tomography (CT) of his brain showed an infarct involving the right putamen and internal capsule. He was otherwise asymptomatic. What does the electrocardiogram (ECG) in Fig. 1 show?

Fig. 1 ECG shows bifid P waves in leads II, III and aVF.
ECG INTERPRETATION
ECG shows the presence of ‘P mitrale’ in multiple leads – inferior leads II, III and aVF, and also leads V2, V3 and V4. The duration of the P wave is 0.12 s or longer, and it has a bifid appearance. In lead V1, a biphasic P wave with a wide and deep terminal negative deflection is seen. The rhythm is sinus. The abnormalities are consistent with left atrial enlargement.

CLINICAL COURSE
Transthoracic echocardiogram showed moderately severe mitral stenosis (Fig. 2a), and the maximal orifice area of the mitral valve during diastole was reduced (Fig. 2b). Although the left atrium was dilated, there was no obvious thrombus. The patient was subsequently found to have paroxysmal atrial fibrillation, and anticoagulation therapy was initiated.

CASE 2
CLINICAL PRESENTATION
A 26-year-old woman presented to the emergency department with a three-day history of bilateral lower limb swelling and reduced exercise tolerance. On examination, dual heart sounds with a loud P2 was heard. Her lungs were clear on auscultation, and there was bilateral lower limb oedema up to the level of her knees. What does the ECG in Fig. 3 show?
ECG INTERPRETATION

ECG in Fig. 3 shows the presence of ‘P pulmonale’. The P waves are tall and peaked, with a height of 3 mm in lead II. The rhythm is sinus. The findings are consistent with the presence of right atrial enlargement.

CLINICAL COURSE

An initial transthoracic echocardiogram showed severe pulmonary hypertension with dilated right heart chambers. The right ventricular systolic function was moderately reduced and there was severe tricuspid valve regurgitation. The left ventricular systolic function, however, was normal. A right heart catheterisation revealed severe pulmonary hypertension with partial response to inhaled iloprost. Right atrium pressure was normal, indicating that the patient was unlikely to be in fluid overload. Pulmonary capillary wedge pressure was also normal, suggesting normal left ventricular filling and relaxation. There was no evidence of the presence of an intracardiac shunt. The results of further investigations to rule out secondary causes of pulmonary hypertension were negative.

The patient was eventually diagnosed with idiopathic pulmonary hypertension, and was commenced on diuretics, vasodilator therapy (tadalafil) and warfarin. Bosentan was subsequently added.

A follow-up transthoracic echocardiogram revealed dilated right heart chambers (Fig. 4a). Consequently, the tricuspid annulus was dilated, resulting in malcoaptation of the tricuspid leaflets and severe tricuspid regurgitation. The left ventricle, normally round, was D-shaped as a result of increased pressure in the right ventricle flattening the ventricular septum (Fig. 4b), in both systole and diastole. Continuous wave Doppler across the tricuspid valve showed the profile to be V-shaped, which was consistent with significant tricuspid regurgitation. Using the modified Bernoulli’s equation, the transtricuspid valve gradient was quantitated to be 85 mmHg (Fig. 4c). Considering the right atrial pressure of 25 mmHg, the pulmonary artery systolic pressure was quantitated to be 110 mmHg, which was at the systemic level.

DISCUSSION

The P wave represents atrial depolarisation and is reflected by a low amplitude positive deflection before the QRS complex. It is normally upright in leads II and aVF, and is always inverted in lead aVR. Since the sinus node is in the right atrium, right atrial depolarisation occurs before left atrial depolarisation. Hence, the P wave is often notched in the limb leads and may appear biphasic in lead V1. The initial positive deflection in V1 is a result of right atrial depolarisation that is directed anteriorly. The second negative deflection, which is due to left atrial depolarisation, is directed posteriorly.(1)

The duration of a P wave reflects the duration of atrial activation, and this is normally < 0.12 s. The amplitude is < 2.5 mm in the limb leads and < 1.5 mm in the precordial leads. The standard ECG should be calibrated to a voltage sensitivity of 10 mm/mV on the vertical axis and with a time scale of 25 mm/s on the horizontal axis.(2)

There are many kinds of P wave abnormalities. They can generally be classified into three main groups: left atrial abnormalities, right atrial abnormalities and abnormal patterns of activation.(3) However, there is a wide range of normal variations in the P wave duration, amplitude and shape. This is due to variables such as the position of the heart, placement of the ECG leads and extracellular factors, which will all influence ECG deflections on the body surface. As a result, this limits the sensitivity of P wave abnormalities in reflecting atrial abnormalities.(4)

Left atrial abnormality

Left atrial abnormality is more commonly associated with conduction delay than right atrial abnormality.(5) The delay in left atrial activation results in the classic appearance of P wave in the limb leads and lead V1. In the limb leads (especially the inferior leads), the P wave appears to be broad (> 0.12 s) and significantly notched and bifold, with an interpeak interval of > 0.04 s. This is otherwise known as ‘P mitrale’. For lead V1, the terminal negative deflection of the P wave appears wider (> 0.04 s) and deeper (> 1 mm), with an area larger than that of the initial positive deflection of the P wave.(6)

Left atrial abnormality is brought about by pathophysiologic processes that result in an increase in left atrial pressure, left atrial volume and delayed conduction. The causes of left atrial abnormality include valvular heart disease (mitral stenosis, mitral...
regurgitation and aortic stenosis), systemic hypertension and hypertrophic cardiomyopathy. There is also a higher incidence of atrial fibrillation in patients with left atrial abnormalities.

In the first case, the patient has left atrial enlargement due to significant mitral valve stenosis. The most common aetiology of mitral stenosis is rheumatic heart disease. Often, the valve leaflets thicken and the chordae tendinae shorten, and become fused together. A pressure gradient develops between the left atrium and left ventricle during diastole, and pulmonary hypertension can eventually ensue. ECG is not sensitive in detecting atrial abnormalities secondary to mild mitral stenosis, but it is useful in moderate or severe mitral stenosis where ECG features of left atrial enlargement are commonly seen.\(^{25}\)

**Right atrial abnormality**

In right atrial abnormality, there is delayed activation of the right atrium, resulting in simultaneous depolarisation of both the right and left atria. The summation of the two waveforms from right and left atrial depolarisation gives rise to a tall, upright and relatively narrow P wave in lead II (\(>2.5\) mm). This tall P wave often has a peaked appearance and is also referred to as 'P pulmonale'. In lead V\(_1\), right atrial abnormality is present when there is a prominent initial positive deflection (\(>1.5\) mm) of the P wave. The main cause of right atrial abnormality is pulmonary hypertension. The causes of pulmonary hypertension are listed in Table I.\(^{26}\) To work up the causes of pulmonary hypertension, besides taking a detailed history and carefully examining the patient, investigations such as transthoracic echocardiography, high-resolution CT of the lung, pulmonary ventilation-perfusion imaging and checking of serum for selected autoimmune markers may also be performed. Apart from right atrial abnormality, patients with pulmonary hypertension may also have right axis deviation, right ventricular hypertrophy or strain, and right bundle branch block on ECG. These ECG features have low sensitivity but high specificity for detecting right ventricular disease.\(^{26}\) In addition, the ECG does not correlate with clinical progression of the disease.\(^{26}\)

**ACKNOWLEDGEMENT**

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**REFERENCES**


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**Table I. Classifications and causes of pulmonary hypertension.**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Group 1</td>
<td>Pulmonary arterial hypertension</td>
</tr>
<tr>
<td>- Idiopathic</td>
<td></td>
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<tr>
<td>- Heritable</td>
<td></td>
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<tr>
<td>- Drug- and toxin-induced</td>
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<tr>
<td>- Associated with HIV infection, connective tissue disease, portal hypertension</td>
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<tr>
<td>Group 2</td>
<td>Pulmonary hypertension secondary to left heart disease</td>
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<tr>
<td>- Systolic dysfunction</td>
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<tr>
<td>- Diastolic dysfunction</td>
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<tr>
<td>- Valvular heart disease</td>
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<tr>
<td>Group 3</td>
<td>Pulmonary hypertension associated with lung disease and/or hypoxaemia</td>
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<tr>
<td>- Chronic obstructive pulmonary disease</td>
<td></td>
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<tr>
<td>- Interstitial lung disease</td>
<td></td>
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<tr>
<td>- Sleep-disordered breathing</td>
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<tr>
<td>Group 4</td>
<td>Chronic thromboembolic pulmonary hypertension</td>
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<tr>
<td>Group 5</td>
<td>Pulmonary hypertension due to unclear multifactorial mechanisms</td>
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<tr>
<td>- Systemic disorders (e.g. sarcoidosis)</td>
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<tr>
<td>- Haematological disorders (e.g. myeloproliferative disease)</td>
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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
(Code SMJ 201301A)

Question 1. The following statements regarding normal P waves are true:
(a) In lead V1, the initial positive deflection of a P wave is due to left atrial depolarisation.
(b) The P wave is always inverted in lead aVR.
(c) The duration of a P wave is less than 0.12 s.
(d) The amplitude of a P wave in the precordial lead is more than 2.5 mm.

Question 2. The following statements regarding ‘P mitrale’ are true:
(a) It is best visualised in the inferior limb leads.
(b) It is bifid with an interpeak interval of less than 0.04 s.
(c) Patients with ‘P mitrale’ on ECG may have a mid-diastolic rumbling murmur on auscultation.
(d) It is associated with pulmonary stenosis.

Question 3. The following statements regarding the presence of ECG evidence of right atrial abnormalities are true:
(a) ‘P pulmonale’ has a tall and relatively narrow appearance.
(b) In lead V1, the second negative deflection of the P wave is greater than the initial positive deflection.
(c) The presence of right atrial abnormalities has a low specificity for detecting right ventricular disease.
(d) There may be the presence of right bundle branch block on the ECG.

Question 4. The following investigations are essential in the workup of pulmonary hypertension:
(a) Echocardiogram.
(b) High-resolution computed tomography of the lung.
(c) Pulmonary ventilation-perfusion imaging.
(d) Autoimmune markers.

Question 5. Causes of pulmonary hypertension include:
(a) Obstructive sleep apnoea.
(b) Systemic sclerosis.
(c) Pneumonia.
(d) Mitral stenosis.

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