

CME Article

ECGs of structural heart disease: Part I

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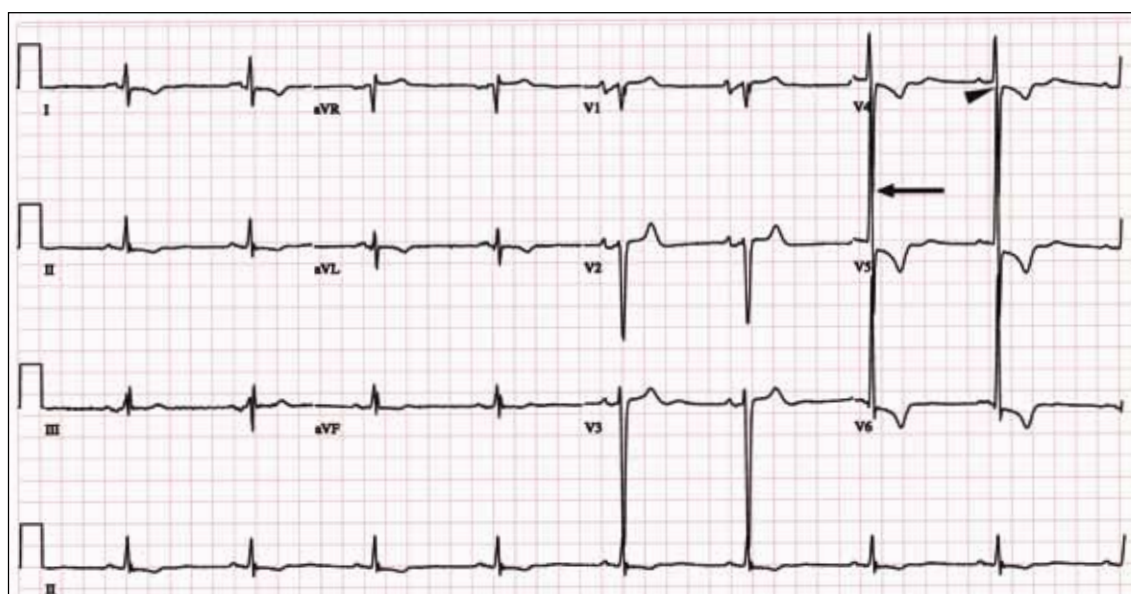


Fig. 1 ECG shows tall R waves in leads V5 and V6, associated with ST segment depression and T wave inversion. Peak of R wave in lead V5 (arrowhead) and the nadir of S wave in lead V4 (arrow) are seen.

CASE I

CLINICAL PRESENTATION

A 78-year-old man was admitted for exertional dyspnoea associated with exertional chest discomfort over the past year. He also had bilateral lower limb swelling over the previous six months. Examination revealed an ejection systolic murmur at the aortic area and a pan-systolic murmur at the apex. Comment on the electrocardiography (ECG) abnormalities in Fig. 1?

ECG INTERPRETATION

Fig. 1 shows left ventricular hypertrophy (LVH). The rhythm is sinus. The R waves are tall in leads V5 and V6. The upper part of the R wave in V5 is superimposed on the lower part of the S wave in V4. The arrowhead indicates the nadir of the R wave in V5, and the arrow indicates the trough of the S wave in V4. The sum of S wave in V1 and R wave in V5 is 41 mm. There are ST segment depression and T wave inversion accompanying the tall R waves in leads V5 and V6. This represents a 'strain pattern'.



Fig. 2 ECG shows normal QRS voltages and resolution of changes documented in Fig. 1.

CLINICAL COURSE

Echocardiography showed severe calcific aortic stenosis. The mean transaortic gradient was 64 mmHg, and the aortic valve area was quantitated to be 0.49 cm² or 0.3 cm²/m². The dimensionless index was low at 0.17. There was also severe mitral and tricuspid valve regurgitation. Both atria and the left ventricle were dilated. The left ventricular ejection fraction was preserved at 50%. The left ventricular mass was markedly increased, at 330 g or 204 g/m². Pulmonary artery systolic pressure was quantitated to be 85 mmHg. Coronary angiography showed minor non-obstructive coronary arteries. In view of the presence of critically severe aortic stenosis and symptoms, the patient underwent transaortic valve implantation (TAVI). Eight months post TAVI, a repeat ECG was performed (Fig. 2), which revealed the absence of LVH. Repeat echocardiography showed a marked regression of LVH, with the left ventricular mass reduced to 188 g or 121 g/m². The left ventricular ejection fraction had increased to 60% and the pulmonary artery systolic pressure reduced to 46 mmHg. The chest radiograph showed the presence of an aortic valve stent post-implantation (Fig. 3).

CASE 2

CLINICAL PRESENTATION

A 23-year-old woman presented with hoarseness of voice. She had been diagnosed with left vocal cord palsy four years ago, and was later found to have enlarged pulmonary artery and pulmonary hypertension. Subsequently, she underwent a series of cardiac and

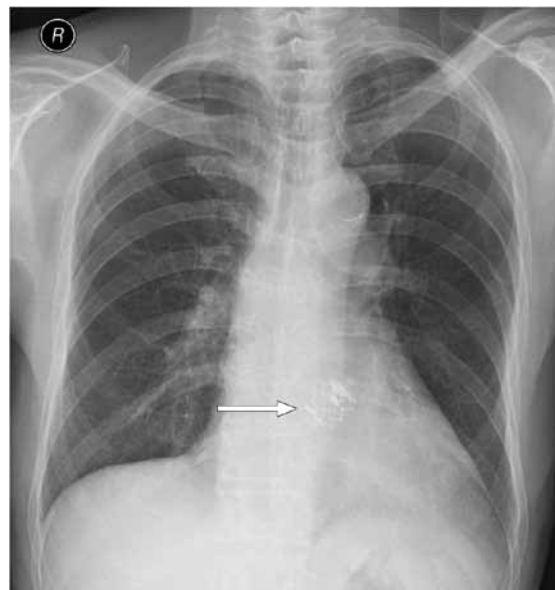


Fig. 3 Chest radiograph shows an aortic valve stent *in situ*.

pulmonary investigations in Malaysia. She presented to us with exertional breathlessness and had been experiencing dizzy spells for several months. What is the ECG diagnosis (Fig. 4)?

ECG INTERPRETATION

There is right axis deviation of approximately 110 degrees. In addition, tall R waves in the right praecordial leads (V1–2) and deep S waves in the left praecordial leads (V4–6) are seen. In lead V1, the amplitude of the R wave is greater than that of the S wave. There are ST depression and T wave inversion accompanying the tall R waves.

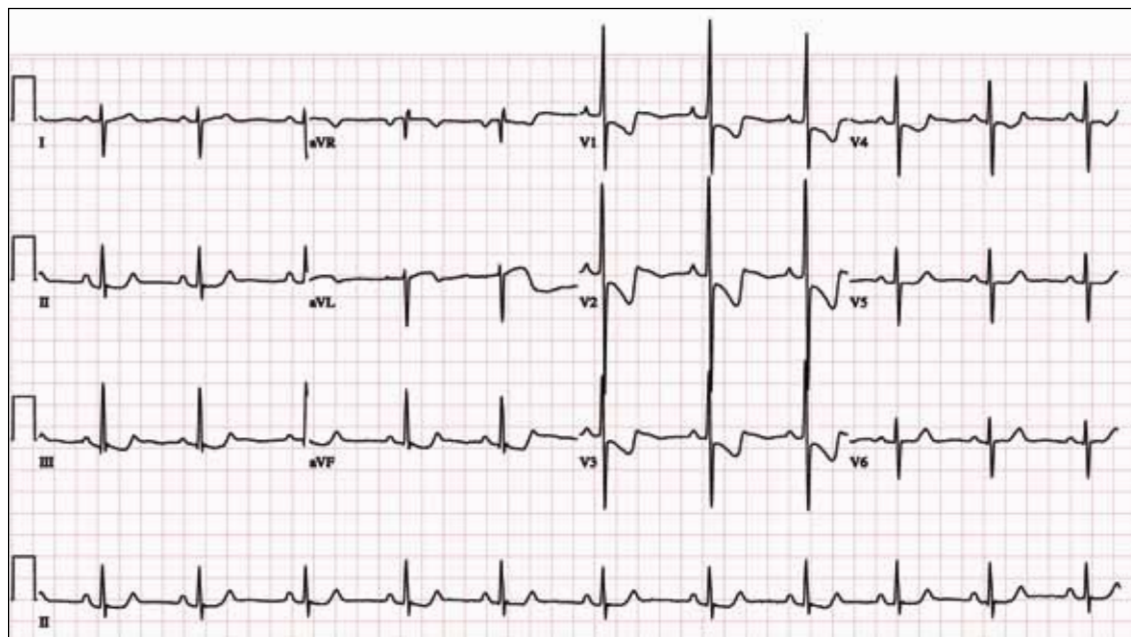


Fig. 4 ECG shows tall R waves in V1–3 and deep S waves in V4–6. There are ST depression and T wave inversion accompanying the tall R waves.

CLINICAL COURSE

The patient underwent cardiopulmonary exercise, which showed dead space ventilation that was consistent with pulmonary hypertension. Echocardiography revealed dilated right heart chambers, right ventricular hypertrophy (RVH) with a pressure overload pattern and moderate RV systolic dysfunction. No other abnormalities (e.g. valvular dysfunction, shunts) were detected. The pulmonary artery systolic pressure was quantitated to be 119 mmHg. A diagnosis of idiopathic pulmonary hypertension was made. She is currently on medical therapy, and five years after her first presentation to us, she is in New York Heart Association (NYHA) Class I despite her persistent severe pulmonary hypertension.

DISCUSSION

In the first case, LVH was observed in the ECG. Pathophysiologically, there is an increase in the left ventricular myocardial fibre size, in response to chronic volume or pressure load. It is reflected by an increase in the left ventricular thickness and mass on echocardiography. Some important causes of LVH include systemic hypertension, aortic stenosis (pressure overload) and aortic or mitral valve regurgitation (volume overload). Another important cause of LVH is hypertrophic cardiomyopathy. LVH usually occurs over months or years, and patients are at increased risk for adverse cardiovascular events, including heart failure and arrhythmias.⁽¹⁾

The ECG is a useful tool for LVH screening, as it is relatively inexpensive and widely available. However, it is imperfect due to its limited sensitivity and specificity.^(2,3)

In contrast, echocardiography is the procedure of choice, as LV mass can be quantitated and the pattern of LVH (concentric, eccentric or concentric remodeling) and its aetiology ascertained.

LVH can result in increased QRS voltages and duration, left axis deviation, P wave abnormalities and ST-T 'strain' pattern in pressure overload state. In contrast, in volume overload state, LVH may be associated with positive T waves in the lateral leads. As such, many different ECG criteria have been proposed for the diagnosis of LVH. The most commonly used are the Sokolow-Lyon and Cornell voltage criteria. False negatives and positives are not uncommon. Mild hypertrophy, body habitus (obesity), age, gender, the presence of pulmonary conditions and other conduction abnormalities such as right bundle branch block and left anterior fascicular block may all affect the sensitivity and specificity of the ECG in the diagnosis of LVH.⁽⁴⁾

In the Sokolow-Lyon criterion, the sum of S wave in V1 and R wave in either V5 or V6 (whichever is taller) is > 35 mm. In Case 1, this criterion was satisfied. Another criterion is the amplitude of R wave in aVL > 1.1 mV (11 mm) or 1.3 mV (13 mm). The Cornell voltage criteria are based on correlation with echocardiographic studies to detect LV mass index of > 132 g/m² in men and > 109 g/m² in women.⁽⁵⁾ In men, it is S in V3 + R in aVL > 2.8 mV (28 mm) and in women, S in V3 + R in aVL > 2.0 mV (20 mm).

In our first case, there is also LVH with a strain pattern. Here, the T wave inversion was asymmetrical (unlike in ischaemic heart disease), where the distal limb was

steeper than the proximal limb. Using echocardiography, the presence of LVH was confirmed, and the LV mass was measured and indexed to the body surface area. The cause of LVH was also ascertained – the aortic valve was critically stenosed, resulting in elevated afterload. Serial monitoring of ECG voltage over time may reflect changes in LV mass and correlate with cardiovascular risk. In Case 1, the ECG evidence of LVH was resolved on relieving the pressure afterload through replacement of the aortic valve by TAVI. This is evidenced in both the repeat echocardiography and ECG after the surgical procedure.

The ECG in the second case shows classical RVH. This occurs in the presence of increase RV mass and may be seen in the presence of pulmonary, tricuspid or mitral stenosis with pulmonary hypertension or cor pulmonale from severe lung disease. Clues to the diagnosis of RVH include: (1) R/S ratio in V1 > 1; (2) R/S ratio in V6 < 1; (3) right axis deviation (> 90 degrees); (4) RV1 > 7 mm; (5) R in V1 + S in V5 > 10 mm; (6) ST-T wave 'strain' pattern; (7) right atrial hypertrophy (prominent, narrow and peak P waves – P pulmonale, especially in the inferior leads).⁽⁶⁾

In the presence of increased R/S ratio in V1, other differentials that should be considered are posterior wall myocardial infarction, Wolff-Parkinson-White syndrome, hypertrophic cardiomyopathy, counterclockwise rotation of the heart and normal variant. In Case 2, despite multiple investigations, the cause of RVH was not found, and it is most likely to be secondary to idiopathic pulmonary hypertension.

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ABSTRACT

Electrocardiogram (ECG) is a useful but imperfect investigation in the diagnosis and possible follow-up of structural heart disease such as ventricular hypertrophy. Different ECG criteria with different sensitivity and specificity are available to aid the detection of left or right ventricular hypertrophy. Subsequent echocardiography can help in the quantification of ventricular mass and identification of the aetiology.

Keywords: ECG, echocardiography, left ventricular hypertrophy, right ventricular hypertrophy

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 201112A)

- | | True | False |
|--|--------------------------|--------------------------|
| Question 1. The following ECG criterion for left ventricular hypertrophy is true: | | |
| (a) In the Sokolow-Lyon criterion, S wave in V1 + R wave in either V5 or V6 is > 35 mm. | <input type="checkbox"/> | <input type="checkbox"/> |
| (b) The amplitude of R wave in aVL is < 1.1 mV (11 mm). | <input type="checkbox"/> | <input type="checkbox"/> |
| (c) In the Cornell voltage criterion, S in V3 + R in aVL is > 2.8 mV (28 mm) in men. | <input type="checkbox"/> | <input type="checkbox"/> |
| (d) In the Cornell voltage criterion, S in V3 + R in aVL is < 2.0 mV (20 mm) in women. | <input type="checkbox"/> | <input type="checkbox"/> |
| Question 2. Left ventricular hypertrophy may result in the following ECG changes: | | |
| (a) Decreased QRS voltages. | <input type="checkbox"/> | <input type="checkbox"/> |
| (b) Increased QRS duration. | <input type="checkbox"/> | <input type="checkbox"/> |
| (c) Left axis deviation. | <input type="checkbox"/> | <input type="checkbox"/> |
| (d) ST depression and T wave inversion. | <input type="checkbox"/> | <input type="checkbox"/> |
| Question 3. The causes of left ventricular hypertrophy include: | | |
| (a) Systemic hypertension. | <input type="checkbox"/> | <input type="checkbox"/> |
| (b) Aortic valve stenosis. | <input type="checkbox"/> | <input type="checkbox"/> |
| (c) Aortic valve regurgitation. | <input type="checkbox"/> | <input type="checkbox"/> |
| (d) Mitral valve stenosis. | <input type="checkbox"/> | <input type="checkbox"/> |
| Question 4. The following are consistent with features of right ventricular hypertrophy : | | |
| (a) R/S ratio in V1 > 1. | <input type="checkbox"/> | <input type="checkbox"/> |
| (b) S/R ratio in V6 > 1. | <input type="checkbox"/> | <input type="checkbox"/> |
| (c) Incomplete left bundle branch block. | <input type="checkbox"/> | <input type="checkbox"/> |
| (d) Left axis deviation. | <input type="checkbox"/> | <input type="checkbox"/> |
| Question 5. The following are causes of increased R/S ratio in V1: | | |
| (a) Wolff-Parkinson-White syndrome. | <input type="checkbox"/> | <input type="checkbox"/> |
| (b) Posterior wall myocardial infarction. | <input type="checkbox"/> | <input type="checkbox"/> |
| (c) Left ventricular hypertrophy. | <input type="checkbox"/> | <input type="checkbox"/> |
| (d) Counterclockwise rotation of the heart. | <input type="checkbox"/> | <input type="checkbox"/> |

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(1) Answers will be published in the SMJ February 2012 issue. (2) The MCR numbers of successful candidates will be posted online at www.sma.org.sg/cme/smj by 13 January 2012. (3) All online submissions will receive an automatic email acknowledgment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council. (6) One CME point is awarded for successful candidates.

Deadline for submission: (December 2011 SMJ 3B CME programme): 12 noon, 06 January 2012.