Common causes of ST elevation

ATIFUR RAHMAN MB BS, FRACP, FCANZ; **JILANI LATONA** MB ChB, FRACP

ST elevation on an ECG can indicate acute ST elevation myocardial infarction (STEMI) and this should always be considered first. However, there are several other conditions that also elevate the ST segment and other features must be used to distinguish between them.

Case scenario

Olga, a 72-year-old previously well woman, felt lightheaded and experienced severe lower chest pain when at the shopping mall. First medical contact was made within 30 minutes and she was being treated at the nearby peripheral hospital within 45 minutes of onset of pain. On arrival, her blood pressure was 180/103 mmHg and she looked pale. An ECG (Figure) and troponin test were performed as part of her work up.

Olga's history included hypertension and hypercholesterolaemia, and she was a life-long smoker. She had no relevant family history and no previous cardiac events. She had recently travelled from interstate to look after her daughter who had had surgery.

Olga's ECG shows left ventricular hypertrophy (LVH; Box and Table 1).[1-4] She had borderline ST elevation (<2 mm) in the anterior chest leads without any reciprocal ST depression in the inferior leads. A chest x-ray revealed a widened mediastinum and a CT aortogram showed a large dissection of the thoracic aorta. Olga was immediately transferred to the nearest tertiary hospital while continuing blood pressure-lowering treatment and adequate pain relief. Fortunately, Olga did not receive any thrombolysis, antiplatelet agents or heparin.

Commentary

Chest pain and other symptoms suggestive of acute coronary syndrome are among the most common reasons for patients seeking medical attention from their GPs or the hospital emergency department. The underlying cause of these presentations may range from minor disease such as musculoskeletal pain to life-threatening conditions such as acute myocardial infarction (AMI), aortic dissection or pulmonary embolism.

Patients with chest pain require a prompt diagnosis. This can be achieved by taking a focused history and conducting a clinical examination and basic investigations, including an ECG, a chest x-ray and blood tests. Failing to diagnose a life-threatening condition can result in mortality or serious

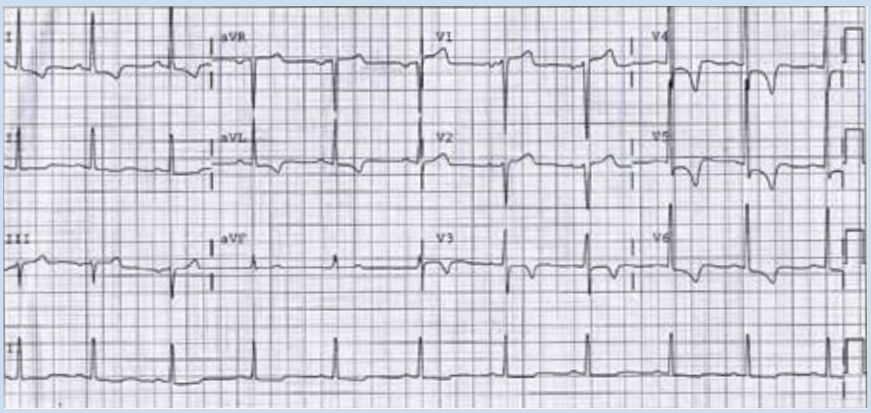

Figure. ECG showing borderline ST elevation in leads V1 and V2.

Key points

- **The 12-lead ECG is an integral part of the diagnostic work up of a patient with acute chest pain.**
- **ST elevation in a 12-lead ECG is an important feature in the diagnosis and treatment of acute myocardial infarction (AMI).**
- **Several other clinical conditions can result in ST elevation.**
- **At times, distinguishing between ST elevation AMI (STEMI) from non-ischaemic causes of elevation of the ST segment is difficult.**
- **STEMI is typically confined to a vascular territory and has a convex upward shape.**

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Associate Professor Rahman is an Interventional Cardiologist at Gold Coast University Hospital, Southport; and Associate Professor at Griffith University, Qld. Dr Latona is a Cardiologist at Logan Hospital, Meadowbrook, Qld.

morbidity for the patient, and also represents a frequent cause of malpractice cases.[5]

A delayed diagnosis of MI can mean that a patient misses the window of opportunity for immediate thrombolysis or primary angioplasty. ECG plays an important role in the early diagnosis of acute ST elevation MI (STEMI) before an immediate reperfusion strategy can be drawn. Although ST elevation in a standard 12-lead ECG is an important factor to consider in the treatment of MI, several clinical conditions result in ST elevation mimicking AMI (Table 2).

In a US prospective observational study, it was found that in the emergency setting ST segment and T wave abnormalities were frequently misread, with 41% false negatives and 14% false positives.[6] Although AMI needs to be considered first and foremost in the presence of ST elevation, it is not always the cause. A retrospective ECG review of adults with chest pain found only 15% of patients had STEMI and 85% of patients with ST elevation had a non-AMI diagnosis responsible for the ST elevation.[7] Thrombolysis in the wrong clinical setting can result in catastrophic complications such as intracranial haemorrhage and death.

This article discusses common causes of ST elevation in the standard 12-lead ECG and ways to differentiate these conditions.

ECG criteria for diagnosing left ventricular hypertrophy[1-4]

Many different ECG criteria for diagnosing left ventricular hypertrophy have been proposed over the years. Most use the voltage in one or more leads, with or without additional factors such as QRS duration, secondary ST–T wave abnormalities or left atrial abnormalities. The most well known electrocardiographic criteria are:

- Sokolow-Lyon Criteria: S wave in V1 plus the R wave in V5 or V6 is 35 mm[1]
- Cornell criteria: R wave in aVL and the S wave in V3, 28 mm in males or 20 mm in females[2]
- Modified Cornell Criteria: R wave in aVL is 12 mm[3]
- Romhilt-Estes left ventricular hypertrophy point score system (Table 1).[4]

Table 1. Romhilt-Estes left ventricular hypertrophy point score system[4]

Criteria	Points
Voltage criteria (any of)	
R or S in limb leads ≥20 mm	3
S wave in V1 or V2 ≥30 mm	
R wave in V5 or V6 ≥30 mm	
ST–T abnormalities	
ST–T vector opposite to QRS without digitalis	3
ST–T vector opposite to QRS with digitalis	1
Normal ST–T vector	0
P wave abnormalities	
Negative terminal P mode in V1 ≥1 mm in depth or 0.04 s in duration	3
Others	
Left axis deviation (QRS of -30° or more)	2
Delayed intrinsicoid deflection in V5 or V6 (>0.05 s)	1
QRS duration ≥0.09 s	1
Scoring system: 3 points or less = no signs of left ventricular hypertrophy; 4 points = probable left ventricular hypertrophy; 5 or more points = positive for left ventricular hypertrophy.	

Acute myocardial infarction

An ECG is considered to be an essential part of the evaluation of chest pain and diagnosis of AMI. Patients with typical ST elevation or new left bundle branch block (LBBB) are usually referred for immediate reperfusion therapy. An ECG in the setting of AMI can help localise the infarct, identify the coronary artery involved and predict the infarct size and prognosis.

STEMI is typically confined to a vascular territory, and the ST elevation has a convex upward shape (Table 3).[8,9] Often there is reciprocal ST depression in the opposite leads. In the early stages of AMI, ECG may show a tall peaked T wave preceding the ST elevation. As the condition progresses, the ECG shows Q wave formation and T wave inversion.

In the current Australian guidelines, the ECG criteria for the diagnosis of STEMI are development of new LBBB or persistent (>20 minutes) ST elevation in two or more contiguous leads of:[10]

- 2.5 mm ST elevation in leads V2 to V3 in men under 40 years
- 2 mm in V2 to V3 in men over 40 years
- 1.5 mm in V2 to V3 in women
- 1 mm in other leads.

Left ventricular hypertrophy

LVH is frequently associated with secondary ST segment or T wave abnormalities. The ST segment and T wave are directed opposite to the QRS complex: this is called discordance between the QRS complex and the ST–T abnormalities. That means there is typically ST elevation in the precordial leads VI and V2 (Figure), where the QRS complex is predominantly negative. In leads I, II, aVL, V4, V5 and V6, where the QRS complex is upright, the ST segment is often depressed (strain pattern).

ST elevation due to LVH is typically concave and is evident in addition to other features of LVH (Box).[1-4] In a retrospective review of the ECGs from adults with chest pain presenting to an emergency department, LVH was found most often (25%) to be responsible for electrocardiographic ST elevation.[7]

Table 2. Acute myocardial infarction and other conditions that cause ST elevation

Condition	Other ECG features	Clinical features
Acute myocardial infarction	• ST elevation in one vascular territory • Convex ST elevation • Reciprocal ST depression in the opposite leads	• Typical/atypical chest pain or discomfort • Risk factors for coronary artery disease • Positive cardiac biomarkers
Left ventricular hypertrophy	• High QRS voltage • ST and T wave changes • Left atrial enlargement • Left axis deviation	• Patient may have left ventricular enlargement, hypertension, aortic regurgitation/stenosis or other valvular heart diseases
Left bundle branch block (LBBB)	• Typically, ST and T waves are opposite in direction to the terminal QRS (appropriate discordance) • In myocardial infarction in the presence of previous LBBB, ST and T waves go to the same direction of the QRS complex (inappropriate concordance) • Extreme ST elevation in leads V1 and V2 may at times indicate ischaemia • QRS duration >0.12 s	• Commonly associated with hypertension, ischaemic heart disease and cardiomyopathy • Comparison with the old ECG is helpful
Pericarditis	• Widespread PR depression • Diffuse ST segment elevations with upward concavity • Reciprocal PR segment elevation and ST segment depression in leads aVR and occasionally V1	• Characteristic clinical findings in pericarditis include pleuritic chest pain and a pericardial friction rub on auscultation • The most common aetiologies of pericarditis are idiopathic and viral
Hyperkalaemia	• ST elevation can be striking at times • Elevated ST segment often slopes down • Other ECG features of hyperkalaemia are often present including wide QRS complex, tall pointed tented T wave and low amplitude or no P wave	• More common with increasing age, in patients with diabetes and chronic kidney diseases who are taking medications including ACE inhibitors, angiotensin receptor blockers, spironolactone or eplerenone
Early repolarisation	• Notch at the 'J' point (at the junction of ST segment and T wave) • Concave ST elevation • Most marked in lead V4	• Absent cardiac biomarker • Present in about 3% of healthy individuals
Takotsubo cardiomyopathy	• ST segment elevation, most commonly in precordial leads • Diffuse deep symmetric T wave inversion	• Classic patient is postmenopausal and presents with chest pain following an intense emotional or physical stress
Pulmonary embolism	• Sinus tachycardia • Complete or incomplete right bundle branch block • S1 Q3 T3 pattern • ST elevation or T wave inversion in inferior or septal leads	• Typically patient presents with dyspnoea, pleuritic chest pain, syncope following surgery or immobilisation
Left ventricular aneurysm	• Persistent ST segment elevation • Presence of Q wave	• May have history of previous myocardial infarction
Normal ST elevation	• ST elevation in the praecordial leads • The ST elevation is usually upwardly concave and is most marked in V2	• Common in younger men
Brugada syndrome	• Typical ECG findings include a right bundle branch block pattern • ST elevation in the right precordial leads (V1 and V3)	• Family history of Brugada syndrome or sudden cardiac death may be present
Coronary artery spasm	• Often dramatic, transient ST elevation	• May present with early morning chest pain • May have history of substance abuse including cocaine and amphetamine use

Table 3. Localisation of infarct, artery involved and ECG findings in patients with acute myocardial infarction[8,9]

Location	Artery involved	ECG features
Inferior	Right coronary artery (RCA)	• Greater ST elevation in III compared with II • ST depression in I and aVL
	Left circumflex (LCx)	• Isoelectric or elevated ST segment in I, aVL, V5 and V6 • (in addition to ST elevation in II, III and aVF)
Right ventricle infarction	Proximal RCA	• ST elevation in V4R (right-sided chest lead) • ST elevation in V1 associated with ST elevation in II, III and aVF
Posterior	Usually posterior descending branch of the RCA	• ECG changes are mirror image of an anteroseptal MI: – R/S ratio in V1 or V2 >1 – hyperacute ST–T wave changes: i.e. ST depression and large, inverted T waves in V1 to V3 • Posterior infarction is confirmed by the presence of ST elevation and Q waves in the posterior leads (V7 to V9)
Anterior	Left anterior descending artery (LAD)	• Anterior = V2 to V5 • Anteroseptal = V1 to V3 • Extensive anterior = V1 to V6, I and aVL
	Proximal LAD	• Prominent ST elevation in I and aVL and inferior ST depression is consistent with proximal LAD occlusion
	Distal LAD	• ST depression in aVL particularly if combined with isoelectric ST segments in the inferior leads suggests distal LAD occlusion
Lateral	Diagonal branch of the LAD Obtuse marginal branch of LCx Ramus intermedius	• ST elevation in the lateral leads (I, aVL,V5 and V6) • Reciprocal ST depression in the inferior leads (III and aVF)

Takotsubo cardiomyopathy

Takotsubo cardiomyopathy mimics AMI in presentation and should be considered in all postmenopausal women who present with chest pain after intense emotional or physical stress; however, about 20 to 35% of cases may not reveal any obvious precipitant. The patient may also present with dyspnoea, palpitations, syncope, cardiac arrest or ECG changes. Although a chest x-ray may be normal, the patient may present with acute pulmonary oedema and cardiomegaly.

The most common acute ECG findings of takotsubo cardiomyopathy are ST elevation in the precordial leads and T wave inversions in almost all leads.[11] Unlike in AMI, ECG changes in takotsubo cardiomyopathy are not limited to one vascular territory. ECG findings are often dramatic and not in proportion with the changes in the patient's troponin levels. Patients also may develop pathological Q waves that typically resolve before hospital discharge with restoration of normal R wave progression and prolonged QT interval (beginning of Q wave to end of T wave), which usually normalises in one to two days and prolonged PR (beginning of P wave to beginning of QRS complex) interval.[12]

The diffuse ST elevations that do not follow any vascular territory and the absence of reciprocal ST changes makes the diagnosis of STEMI unlikely. ST elevation in patients with myocardial infarction is reciprocal between leads III and aVL.

Pericarditis

In patients with acute pericarditis, the ST segment is elevated diffusely with upward concavity in the precordial and the limb leads. The ST elevation usually involves more than one coronary vascular territory and there is an absence of reciprocal ST changes between leads III and aVL. In patients with pericarditis, the PR segment is depressed but this is not specific for acute pericarditis as early repolarisation or atrial infarction can also cause the depression.[13,14]

Acute myocarditis can cause diffuse ST elevation similar to that seen in pericarditis. In Olga's case, the absence of any systemic symptoms and absence of prior illness made the diagnosis of pericarditis or myocarditis unlikely.

Early repolarisation

Early repolarisation is characterised by ST elevation with a concave morphology and notching of the J point (the junction where the QRS complex ends and the ST segment begins). It is seen in leads with a tall R wave and is most marked in V4 (V2 to V5). Rarely, it can involve inferior leads.

It occurs in 2 to 5% of the population, predominantly in young men. Reciprocal changes typically seen in patients with AMI are generally absent. Unless associated with an acute coronary syndrome, the cardiac biomarker troponin should be negative.

Although sometimes called 'benign early repolarisation', whether it is a totally benign condition or carries slightly increased risk of sudden cardiac death remains controversial. A Finnish study found that patients with J-point elevation of more than 0.1 mV in inferior or lateral leads were about 1.3 times more likely to die from sudden cardiac death, whereas those who had J-point elevation of more than 0.2 mV were three times more likely to have sudden cardiac death.[15] On the other hand, no significant association between any components of early repolarisation and cardiac mortality has been found.[16]

Normal ST elevation

A study of 6014 asymptomatic men, conducted to delineate the range of variation in a normal ECG, found that ST elevation in the precordial leads is very common (91.2%). ST elevation was most marked in the anterior leads (elevation in V2 to V4; 43.8%), followed by the lateral leads (elevation in V3 to V6; 24%), anterolateral leads (elevation in all precordial leads; 16.8%) and rightward (elevation in V1 to V2 only; 6.8%). Only 8.8% of men had no ST elevation in any precordial leads, 44.3% had 0.1 mV ST elevation, 38.4% had 0.2 mV ST elevation and 0.1% had ST elevation of 0.5 mV. The ST elevation is usually upwardly concave and is most marked in V2. ST elevation is common in younger men and the prevalence decreases with age.[17]

Left bundle branch block

The guidelines for the management of patients with AMI recommend immediate revascularisation with thrombolysis or primary angioplasty for patients with chest pain and new LBBB.[10] ECG changes in the presence of pre-existing LBBB is slightly more complex because ST segment and T wave changes including ST elevation and ST depression in the presence of LBBB are common.[10]

Normally in LBBB, the ST segments and T waves are opposite in direction to the terminal QRS deflection. These changes are secondary to the bundle branch block (secondary ST segment and T wave changes). This means that in the presence of LBBB the septal leads (V1 to V3) quite often show ST elevation and an upright T wave where the QRS complex is predominantly negative. There is also association with ST depression and T wave inversion in the lateral leads (V5 and V6) where the QRS complex is predominantly positive. This is called 'appropriate discordance'.

ST elevation in association with a positive QRS complex (in V4 to V6) or ST depression in leads that have predominantly negative QRS complexes (V1 to V3) is not expected in patients with uncomplicated LBBB and is termed 'inappropriate concordance', which strongly indicates the presence of acute ischaemia.

In patients with LBBB, the modified Sgarbossa criteria is useful in identifying STEMI: ST elevation of more than 1 mm concordant with QRS (five points); ST depression of more than 1 mm in lead V1 to V3 (three points); ST elevation more than 5 mm discordant with QRS (two points).[10,18] More than three points is associated with a 98% chance of having MI, but a score of 0 does not rule out STEMI.

Hyperkalaemia

ST elevation in hyperkalaemia can be striking at times.[19] Other ECG features of hyperkalaemia that can be present are widened QRS complexes; tall, pointed T waves and low amplitude or no P waves. In hyperkalaemia, the ST segment often slopes downward.

Hyperkalaemia is more common with increasing age and in patients with diabetes and chronic kidney disease who take medications that block the renin-angiotensin-aldosterone system including ACE inhibitors, angiotensin receptor blockers, direct renin inhibitors and aldosterone receptor antagonists such as spironolactone or eplerenone.

Left ventricular aneurysm

A left ventricular aneurysm can be diagnosed on ECG when there is persistent ST elevation with Q wave after a transmural myocardial infarction. In a patient with an anterior or apical aneurysm, the persistent ST elevation is most marked in lead V1 and V2. In a patient with an aneurysm after an inferior MI, changes are marked in leads II, III and aVF.[20]

The patient's history of a previous AMI and an echocardiography is helpful to document the presence of an aneurysm. The shape of the ST elevation is also relatively unique and has been described as 'coving' (over Q waves).

Brugada syndrome

The Brugada syndrome is a familial disease with an autosomal dominant mode of transmission. Family history is often positive for sudden cardiac death at a young age, most commonly occurring during sleep, in particular during the early morning hours.[21]

Typical ECG findings of Brugada syndrome include a right bundle branch block (RBBB) pattern and ST elevation in the right praecordial leads (V1 and V3) in the absence of long QT intervals and any structural disease.[22] The ST elevation can have a saddleback shape but in typical cases the ST segment is down sloping and ends with an inverted T wave. ST elevation in Brugada syndrome may be present intermittently. Challenging the patient with a sodium channel blocker such as flecainide can unmask a typical electrocardiographic pattern.[23]

Pulmonary embolism

ECG features of pulmonary embolism include sinus tachycardia, simultaneous T wave inversions in the inferior (II, III and aVF) and right precordial (V1 to V4) leads, ST elevation, S1 Q3 T3 pattern (deep S wave in lead I, Q wave in III, inverted T wave in III) and complete or incomplete RBBB. ST elevation in the presence of massive pulmonary embolism can be striking at times.[24]

Consider pulmonary embolism as a differential diagnosis in any patient who presents with chest pain, dyspnoea and syncope in the postoperative setting or after prolonged immobilisation. Patients may have an elevated jugular venous pressure and other signs of congestive cardiac failure. A D-dimer test is very helpful in excluding pulmonary embolism as it is considered a highly sensitive test for pulmonary embolism and a negative test makes diagnosis of pulmonary embolism unlikely. Unfortunately, D-dimer lacks specificity and has low positive predictive value. When clinical suspicion of pulmonary embolism is high, a CT pulmonary angiogram may be needed to confirm the diagnosis.

Coronary artery spasm

Spasm of an epicardial coronary artery can produce dramatic, transient ST elevation in patients with chest pain. Although coronary spasm is usually brief and the ST segment returns to normal without myocardial injury, prolonged spasm can result in MI.[25] Vagal withdrawal is most often the mechanism leading to spontaneous spasm.

Patients with coronary artery spasm are usually younger than those that present with unstable angina or chronic stable angina and tend to present with early morning chest pain. An important associated risk factor is substance abuse including tobacco and marijuana smoking, alcohol consumption and cocaine and amphetamine use.

Conclusion

The 12-lead ECG is an integral part of the diagnostic work up of a patient with acute chest pain. It is the recommended bedside test to confirm or exclude the diagnosis of STEMI and AMI needs to be considered first and foremost.

ST elevation caused by conditions other than acute ischaemia is common. At times, distinguishing between STEMI from non-ischaemic causes of elevation of the ST segment is difficult, especially in patients with atypical presenting symptoms. Understanding common patterns of ST elevation that are not caused by ischaemia is crucial for rapid and accurate diagnosis of STEMI and subsequent reperfusion strategy.

In a certain clinical context, when equivocal ECG abnormalities are detected, other differential diagnoses should be taken into account in order to avoid unnecessary, potentially dangerous therapies. Serial ECG, comparison to old ECGs, ST elevation confined to single vascular territories with or without reciprocal ST depression, expert help and at times remote ECG review can assist in the diagnosis of STEMI. **CT**

References

A list of references is included in the online version of this article (www.cardiologytoday.com.au).

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