

15th Sukaman Memorial Lecture**ST Segment Elevation: New Electrocardiographic Insights in 2014**

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**INTRODUCTION**

Of all the non-arrhythmic electrocardiographic (ECG) abnormalities, ST segment elevation (ST elevation) is the most important with regard to diagnosis, prognosis and management.

ST SEGMENT

The **J point** is defined as the junction of the end of the QRS complex and the onset of the ST segment. The ST segment is between the J point and the onset of the T wave. If the J point is elevated or depressed with reference to the isoelectric line, which is defined as the baseline formed by the TP segment, the ST segment is diagnosed to be elevated or depressed respectively (Fig 1). During significant sinus tachycardia, the

ST segment is absent because the P wave is superimposed on the preceding T wave. The horizontal level at the end of the PR segment now becomes the isoelectric line.

ST ELEVATION MYOCARDIAL INFARCTION (STEMI)

Anterior and inferior STEMI is approximately equal in their incidence. In the hyperacute phase of STEMI (first 6-12 hours), concave or straight-upward ST elevation is seen. Tall T waves are also frequently present. The ST elevation is convex and dome shaped in the fully evolved stage when Q waves and deep T wave inversion have evolved about 24 hours after the onset of infarction.

ST elevation in certain leads suggests the occurrence of STEMI in specific areas of the heart. For example, in **anterior STEMI**, ST elevation in leads V₁ to V₄ indicates anteroseptal/

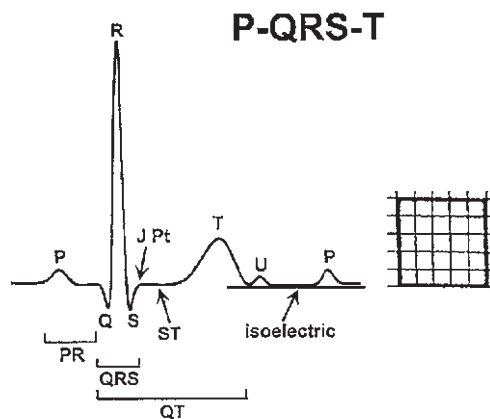
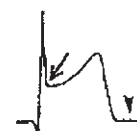


Fig. 1 Diagram showing the various ECG complexes, segments and time intervals. J Pt = J point.

Causes of ST Segment Elevation

- STEMI
- **Other Causes**
 - "Normal male pattern" • Acute pericarditis
 - Early repolarization pattern
 - Type 1 Brugada pattern
 - Acute pulmonary embolism
 - Left bundle branch block
 - **Myocardial Disease**
 - (i) Left ventricular hypertrophy
 - (ii) Takotsubo cardiomyopathy
 - (iii) Acute myocarditis



Arrow indicates J point/
ST segment elevation
Arrowhead indicates
isoelectric line

Table 1. Causes of ST
Segment Elevation

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apical infarction, in leads V_5 and V_6 anterolateral and in leads I and aVL high lateral infarction of the **left ventricle**. In anterior STEMI, the culprit artery is most frequently the left anterior descending artery (*Fig. 2*) and very rarely the left main coronary artery.

In **inferior STEMI**, ST elevation is seen in leads II, III and aVF indicating infarction of the inferior wall of the left ventricle. The right coronary artery (*Fig. 3*) is the culprit artery in about 80% and the left circumflex artery in about 20% of cases (*Fig. 4*). In patients with inferior STEMI, **posterior STEMI** is seen frequently and **right ventricular infarction** in about 40% of cases. In the latter situation, the culprit artery is the right coronary artery and the site of lesion is the proximal segment of the artery before the origin of the right ventricular branch. Right-sided chest leads to diagnose right ventricular infarction must therefore be routinely performed in all cases when the initial 12-lead ECG shows inferior STEMI¹ (*Fig. 3*).

“According to the **Third Universal Definition of Myocardial Infarction (2012)**, the following are the cut off values for **ST segment elevation in acute myocardial ischaemia**² New ST segment elevation at the J point in 2 contiguous leads with the following cut off values: ≥ 0.1 mV (1 mm) in all leads other than leads V_2-V_3 where the following cut off values apply: Men < 40 yrs ≥ 0.25 mV (2.5 mm); Men ≥ 40 years ≥ 0.20 mV (2.0 mm). Women ≥ 0.15 mV (1.5 mm)”¹

The criteria for the diagnosis of **right ventricular infarction** is > 0.5 mm ST elevation in right-sided chest leads, especially

in lead V4R but also in leads V_5R and V_6R (except in men who are < 30 years old where the cut off value is > 1 mm).

Reciprocal ST segment depression, which is an electrical phenomenon that is not evidence of additional myocardial ischaemia, is predominantly seen in the limb leads in STEMI. In inferior STEMI due to right coronary artery occlusion, ST elevation is nearly always seen in leads II, III and aVF. This results in obligatory reciprocal ST depression in leads I and aVL (*Fig 3*). In anterior STEMI, ST elevation is seen in leads V_1/V_2 to V_5/V_6 and frequently also in leads I and aVL. It is the ST elevation in leads I and aVL that causes obligatory reciprocal ST depression in leads II, III and aVF (*Fig 2*). In **posterior STEMI**, ST elevation is seen in the posterior leads in V_7 , V_8 and V_9 (which are uncommonly performed in clinical practice), and reciprocal ST depression is seen in leads V_1 to V_3 (*Fig 4*).

In some patients with a typical history of acute myocardial infarction, the initial ECG may show non-specific changes or even be normal. In such situations, repeat ECG recordings every 15-30 minutes is recommended.

In **Prinzmetal’s angina**, which is due to coronary vasospasm, chest pain usually occurs spontaneously in the early hours of the morning and ST elevation is seen, closely simulating STEMI. However unlike STEMI, in this condition, the ECG quickly becomes normal when the chest pain subsides when sublingual glyceryl trinitrate is given. *Table 1* shows the many causes of ST elevation, all of which can mimic STEMI.

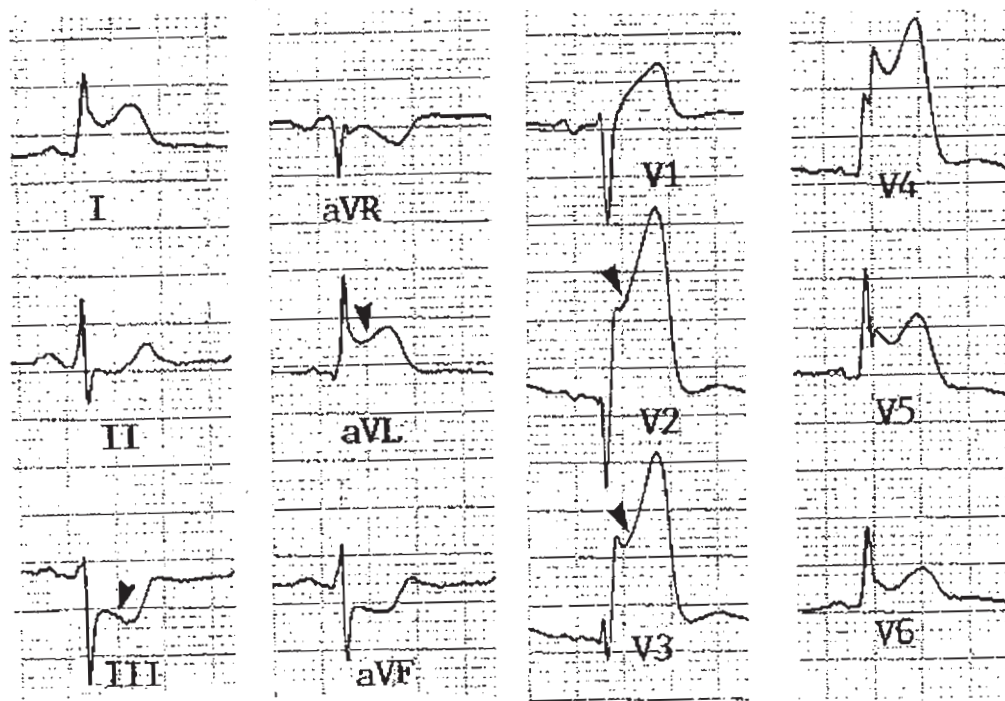


Fig. 2 “Hyperacute phase” of anterior STEMI in a 51-year-old man. Note: (1) Markedly elevated ST segments (concave upwards) in V_2 to V_6 , I and aVL (arrowheads in V_2 , V_3 and aVL) merging with tall T waves in V_2 to V_4 (ST segment elevation and T wave in V_2 = 10 mm and 20 mm respectively). (2) Reciprocal ST segment depression in II, III and AVF (arrowhead in III). Intravenous streptokinase therapy was given. Subsequent coronary angiography revealed a 90% stenosis of the **proximal left anterior descending artery**. (Reproduced with permission. Poh KK, HC Tan, Teo SG. ECG ST segment elevation in patients with chest pain. *Sing Med J* 2011; 5(1): 3 [with adaptation]).

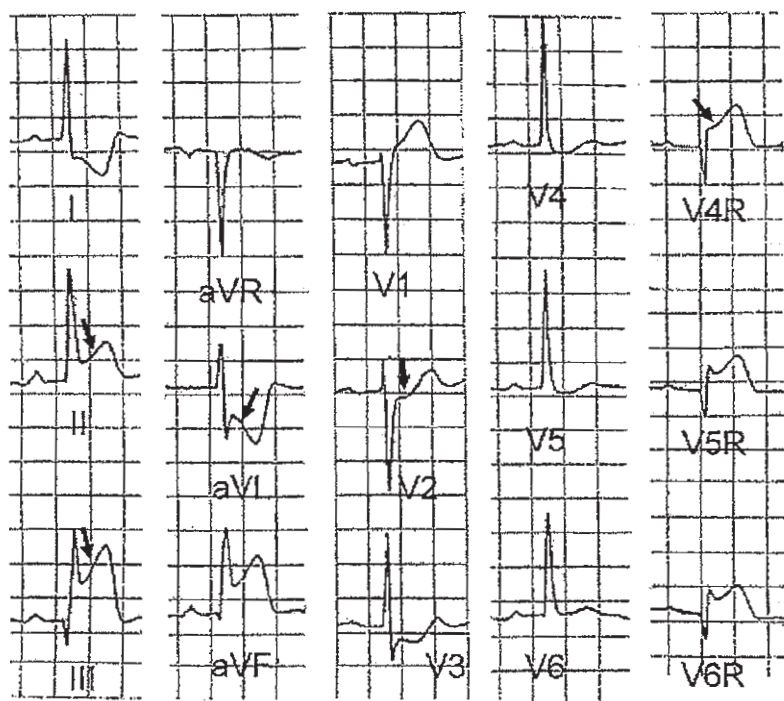


Fig. 3 ECG showing (i) ST elevation in II, III (arrows in both) and aVF and reciprocal ST depression in I and aVL (arrow in aVL) indicating **inferior STEMI** (ii) ST depression in V₂ (arrow) and V₃ indicating posterior STEMI. In patients presenting with inferior STEMI, if the ST elevation in III is greater than that in II (as is seen in this ECG), the culprit artery is most likely the **right coronary artery** (Reference 3) and less frequently a **dominant circumflex artery**. However, the presence of right ventricular infarction in this patient as indicated by ST elevation in V_{4R} (arrow), V_{5R} and V_{6R} confirms that the culprit artery is the right coronary artery (proximal segment).

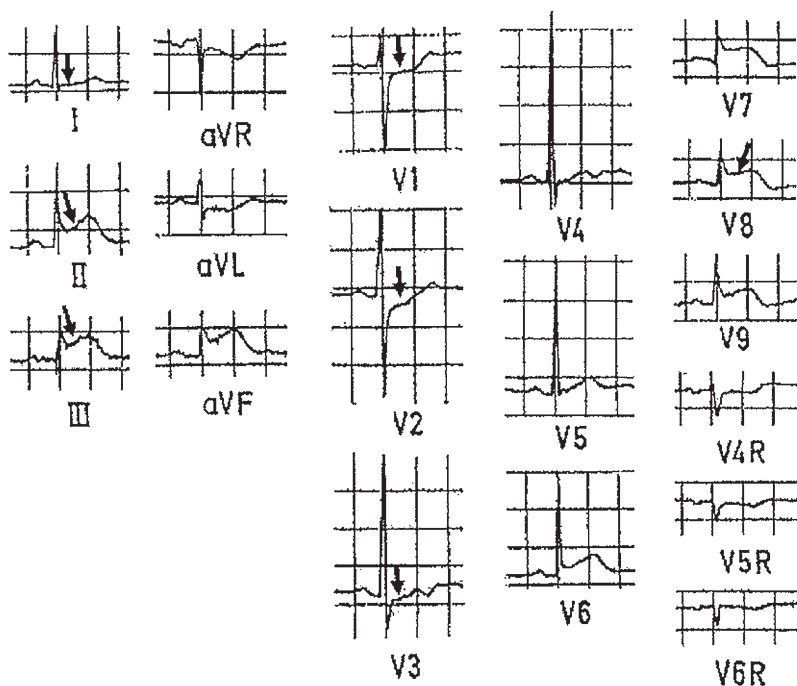


Fig. 4. ECG showing inferior STEMI indicated by ST elevation in II, III (arrows in both) and aVF and posterior STEMI (indicated by ST elevation in V₇, V₈ (arrow) and V₉ and ST depression in V₁-V₂ [arrows]). The ST segment in I (arrow) is isoelectric. In inferior STEMI, if the ST elevation II is equal to III (as seen in this patient) or less frequently greater than the ST elevation in III, the culprit artery is a **non-dominant circumflex artery**.

NORMAL MALE PATTERN

In a study by Surawicz and Parikh, 91% of 529 normal males between the age of 17-24 years showed ≥ 1 mm ST elevation in 1 or more of the leads V1 to V4. This prevalence however decreased to 14% in men who were 76 years and older. This very high prevalence of ST elevation in young normal

males has been termed “**normal male pattern**”^{4,5} (Fig. 5). In contrast, the prevalence of this similar ST elevation in females was much lower (about 20% or less) and remained constant throughout the different age groups.

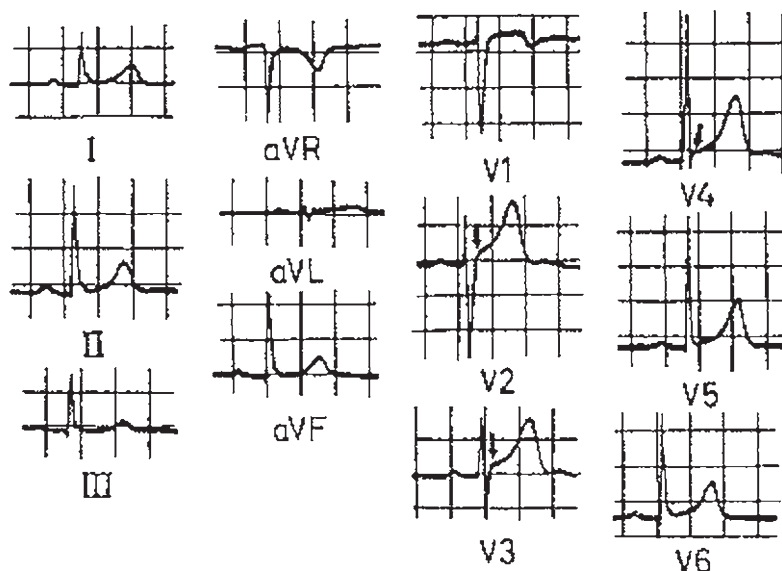


Fig. 5 12-lead ECG in a normal young man. Note ST elevation in V₂ to V₄ (arrows) reflecting the **normal male pattern**. (Reproduced from Clinical Cardiology 4th Edition by BL Chia, World Scientific Publishing).

EARLY REPOLARIZATION PATTERN

“The Early Repolarization Pattern has been recognized for the past few decades. The J point is elevated ≥ 1 mm in ≥ 2 contiguous leads in leads V4 to V6. Less commonly, this pattern can also be seen in the limb leads. At the elevated J point, there is frequently either a notch or a slurr, followed by a concave elevation of the ST segment which slopes upwards, merging with a tall T wave. In addition, the left praecordial R waves are also usually tall (Fig. 6). This pattern is seen in

about 5% of the general population and for decades, it has been regarded as totally benign. However, in the past few years, there has been a controversy whether some patients with this pattern carry a very small risk of ventricular fibrillation. Most likely, the reported cases of Early Repolarization Pattern and ventricular fibrillation belong to a different entity. This is because many of these patients do not show ST elevation, but instead exhibit a horizontal or downsloping ST segment.”¹

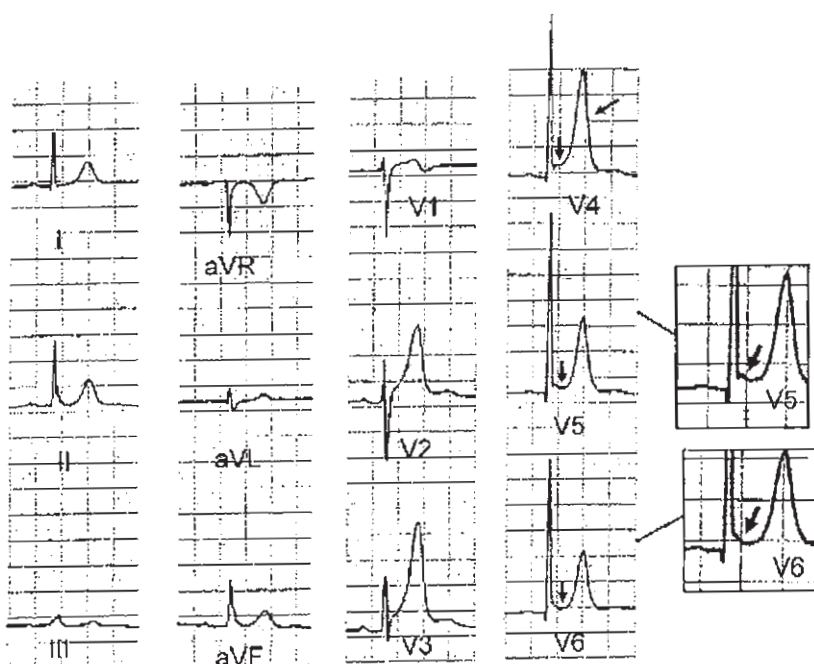


Fig. 6 Early Repolarization Pattern. Note: (1) ST segments which are elevated ≥ 1 mm concave upwards in V₂-V₆ (vertical arrows in V₄-V₆) merging with tall T waves. The T wave in V₄ (oblique arrow) is 21 mm. In the enlarged images of V₅ and V₆, the oblique arrows indicate a notch in V₅ and a slurr in V₆, (2) The R waves in V₄ to V₆ are prominent and the amplitudes are increased. R in V₅ = 35 mm. (Reproduced from Clinical Cardiology 4th Edition by BL Chia, World Scientific Publishing).

ACUTE PERICARDITIS

Figure 7 shows the classical ECG findings in acute pericarditis

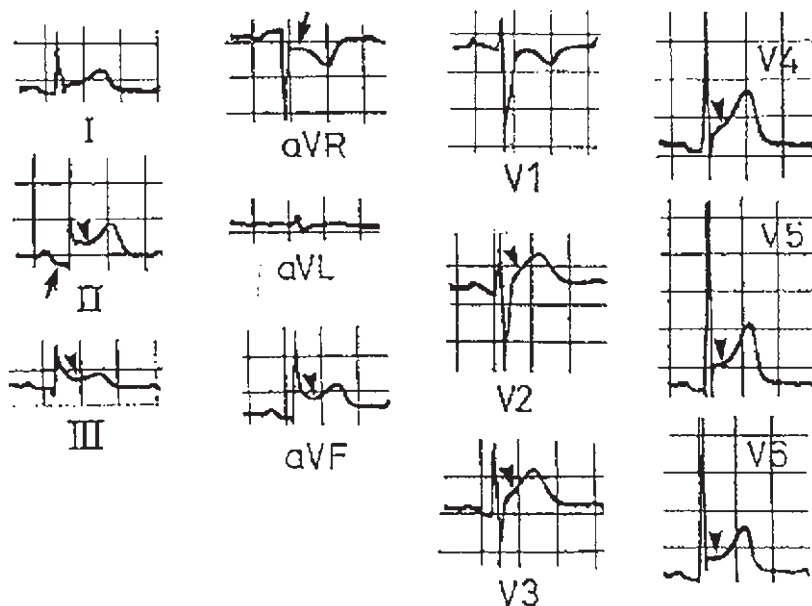


Fig. 7 Acute pericarditis. Note: (1) Elevated ST segments (concave upwards) in V₂ to V₆, II, III and aVF (arrowheads). The ST segment elevation in the limb leads is maximal in II resulting in reciprocal ST segment depression in aVR (arrow). Depressed PR segment in II (arrow). Normal QRS and T wave voltages. (Reproduced from Clinical Cardiology 4th Edition by BL Chia, World Scientific Publishing).

LEFT VENTRICULAR HYPERTROPHY

Left ventricular hypertrophy is well known to cause ST elevation in leads V₁-V₃. The deeper the S wave, the higher the ST elevation.

Left ventricular hypertrophy and Early Repolarization Pattern are 2 of the most common causes of non-ischaemic ST elevation which may be misdiagnosed as STEMI.

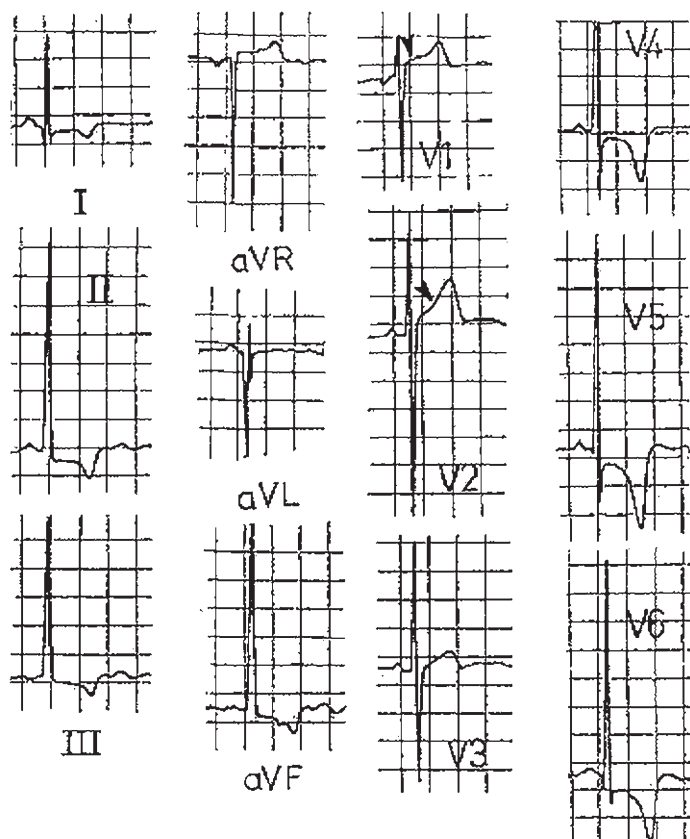


Fig. 8 Left ventricular hypertrophy with strain pattern in a patient with hypertrophic cardiomyopathy. Note 3 mm ST elevation in V₁ and V₂ (arrowheads). Figure reproduced from Clinical Cardiology 4th Edition by BL Chia, World Scientific Publishing. [with adaptation].

BRUGADA SYNDROME

Fig. 9 shows the findings of Type 1 Brugada pattern. Patients having this ECG pattern, who in addition present

with polymorphic ventricular tachycardia and ventricular fibrillation are diagnosed to have the “Brugada syndrome”.

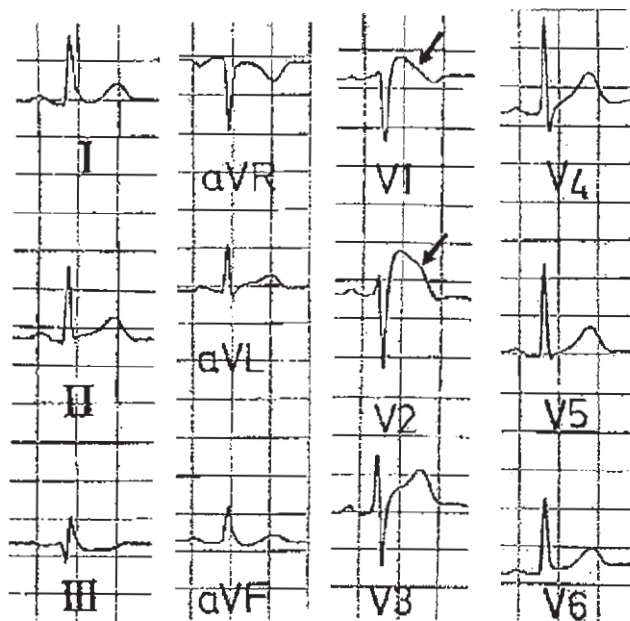


Fig. 9 Type 1 Brugada pattern. In this ECG, the J point is elevated ≥ 2 mm in V_1 and V_2 . The elevated ST segment is covered shape (arrows in V_1 and V_2) and downsloping, terminating in an inverted T wave. To qualify for the diagnosis of Type 1 Brugada pattern, the above ECG pattern must be seen in either V_1 or V_2 or in both these leads as is seen in this patient. (Reproduced from Clinical Cardiology 4th Edition by BL Chia, World Scientific Publishing).

TAKOTSUBO (STRESS) CARDIOMYOPATHY

In this condition, either ST elevation (Fig. 10) or deep T wave inversion, simulating closely STEMI or

non-STEMI respectively, is seen in the acute phase.

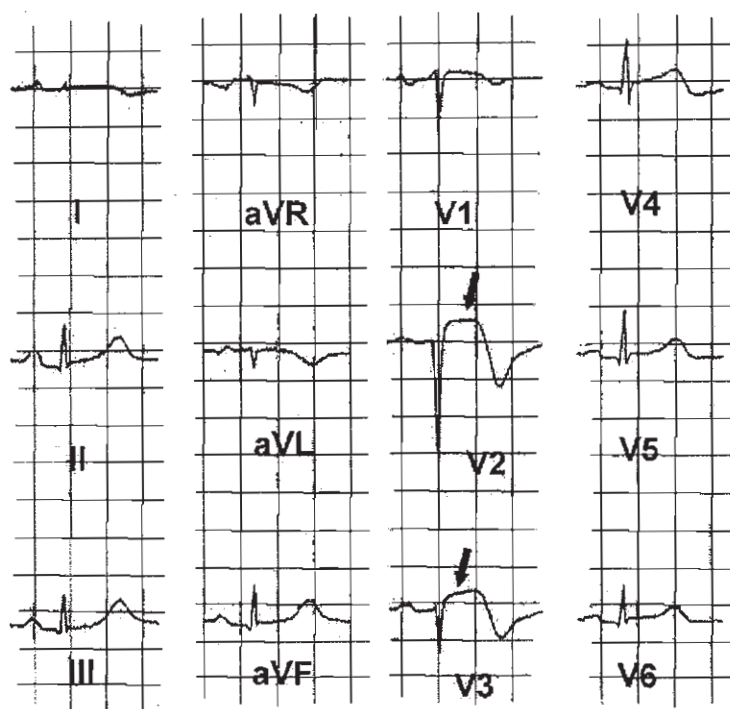


Fig. 10 ECG of a patient with Takotsubo cardiomyopathy showing convex upwards ST elevation (arrows in V_2 and V_3) together with biphasic T wave inversion in V_2 - V_4 . This ECG closely simulates anterior STEMI.

CONCLUSION

ST segment elevation is an extremely important finding in modern day electrocardiography. It has many causes, of which the most important is ST elevation myocardial infarction (STEMI). Although there are many pitfalls in the ECG evaluation of STEMI, with our current ECG

knowledge and expertise coupled with clinical correlation as well as the present availability of cardiac biomarkers, echocardiography, computed tomography and percutaneous coronary angiography, accurate diagnosis today can be achieved in the majority of cases.

REFERENCES

1. **Chia BL**, *Clinical Electrocardiography 4th Edition 2015*, World Scientific Publishing.
2. **Thygesen K**, Alpert JS, Jaffe AS, et al. Joint ESC/ACC/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction (2012). Third universal definition of myocardial infarction. *Circulation* 126: 2020-35.
3. **Chia BL**, Yip JW, Tan HC, Lim YT (2000). Usefulness of ST Elevation II/III Ratio and ST Deviation in Lead 1 for identifying the Culprit artery in inferior wall acute myocardial infarction. *Am J Cardiol* 86: 341-43.
4. **Surawicz B**, Parikh SR (2002). Prevalence of male and female patterns of early ventricular repolarization in the normal ECG of males and females from childhood to old age. *J Am Coll Cardiol* 40: 1870-6.
5. **Wang K** (2013). *Atlas of Electrocardiography*, page 223. Jaypee Brothers Medical Publishers (P) Ltd.

Citation for Professor Boon-Lock Chia, Republic of Singapore

It is with great pleasure for me to deliver the citation of our 15th Sukaman Memorial lecturer, Professor Boon-Lock Chia from Singapore. Prof Chia graduated in Medicine in 1963 from the University of Singapore (now the National University of Singapore [NUS]). His initial training in Cardiology was in Sydney, Australia and later at the Division of Cardiology, Stanford University Medical Centre in America. He joined the NUS as a Senior Lecturer in Medicine in 1972 and was promoted to full Professor in 1981 till 1999.

He is currently an Emeritus Professor at the NUS and an Emeritus Consultant at the National University Heart Centre, Singapore. His former appointments, achievements and awards include the following: (1) Head, Division of Cardiology and Chief, Cardiac Department at the National University Hospital, Singapore (2) President of the Singapore Cardiac Society (3) President of the Singapore Hypertension Society (4) Secretary-Treasurer, Asean Federation of Cardiology (5) Chairman of the Workgroup for Singapore Ministry of Health's Clinical Practice Guidelines in Lipids (2001, 2006) (6) Life Time Achievement Award from: (i) The Singapore Cardiac Society and (ii) The Lee Foundation – National Healthcare Group (7) Honorary Member of the Singapore Medical Association (8) Dato Paduka Mahkota Brunei (9) Fellow of the Royal Australasian College of Physicians and 3 other colleges (10) International Editor of "Journal of Electrocardiology" (11) Delivered the 9th Antonio Samia lecture at the 14th Asia Pacific Congress of Cardiology in 2004.

Prof Chia was one of the pioneers of echocardiography and 24-hour ambulatory BP monitoring in Singapore. He has published more than 160 papers in international and regional journals and authored 2 books – (i) "Clinical Electrocardiology" and (ii) "An Atlas of Two dimensional and Doppler Echocardiography".

Dato' Dr Azhari Rosman

President National Heart Association of Malaysia