

How much do we know about ST elevation?: case report of a patient with acute coronary syndrome in the observation ward

我們對心電圖 ST 節段上升知多少? : 一名在觀察病房內的急性冠狀綜合症病者個案報告

KK Ho 何健基, CM Chan 陳子明, KH Lee 李家興, CH Lit 列就雄

The identification of acute coronary syndrome continues to challenge even experienced clinicians. Emergency physicians have the responsibility to identify, treat and admit those patients with true acute coronary syndrome to the appropriate units. This article described a case of acute coronary syndrome that developed in the observation ward, with discussion on some recent reviews of standard electrocardiogram analysis. It is very important to point out that controversy over the measurement of ST elevation exists which may adversely affect patient management. (*Hong Kong j.emerg.med.* 2004;11:230-235)

識別急性冠狀綜合症仍舊是臨床醫生們（即使是有豐富經驗者）的挑戰。急症科醫生有責任確定、治療及安排那些真正的急性冠狀綜合症病者入住適當的專科病房。本章描述一個在觀察病房顯現急性冠狀綜合症徵狀的個案，並討論最近一些標準心電圖分析的評論。指出一些 ST 節段上升測量法存在的爭議是非常重要的，因其對病者處理可能存在有害的影響。

Keywords: Acute myocardial infarction, male, Prinzmetal's angina, unstable angina, variant angina pectoris

關鍵詞：急性心肌梗塞、男性、普林斯梅圖氏心絞痛、不穩定心絞痛、變異性心絞痛

Case

A 59-year-old gentleman attended the emergency department at 22:23 hour in March 2004 for epigastric discomfort for about one hour. He was deaf and dumb and some information was obtained by using gestures. The epigastric discomfort radiated to the back, but the nature could not be described. He had no

vomiting. He lived alone. He was a chronic smoker and drinker. He had a recent history of endoscopically proven acute gastritis and repeated admissions to the surgical ward for recurrent epigastric pain. He had no history of ischaemic heart disease. Further history taking was difficult because of communication difficulties.

Physical examination showed that the patient was alert and had no diaphoresis. The blood pressure and pulse rate were 152/95 mmHg and 73 beats per minute respectively. The oxygen saturation on room air was 98%. Abdominal examination showed only very mild epigastric tenderness, without guarding or rebound tenderness. Per rectal examination revealed yellow soft stool. Cardiovascular examination was normal, and no gallop rhythm or pulmonary crackles were found.

Correspondence to:

Ho Kin Kei, MBChB(CUHK), MRCSEd(A&E)

Princess Margaret Hospital, Accident & Emergency Department,
Lai Chi Kok, Hong Kong

Email: kkho2002@netvigator.com

Chan Chi Ming, MBChB(CUHK), MRCSEd(A&E)

Lee Ka Hing, MBChB(CUHK), MRCSEd(A&E)

Lit Chau Hung, MRCP(UK), FRCSEd(A&E), FHKAM(Emergency Medicine)

The electrocardiogram (ECG) performed at 22:40 hour was regarded as normal (Figure 1). Chest X-ray (erect) and abdominal X-ray (supine) revealed no abnormality. Blood tests for troponin I, glucose and amylase were normal (taken at 23:05 hour). He was given Gastrocaine (antacid) for symptoms of dyspepsia and admitted to the observation ward for further care. Further examinations and ECG were planned.

He developed severe central chest pain at about 07:15 hour the next morning. An ECG was done showing alarming ST elevation in leads V1-4 (Figure 2). The clinical diagnosis of acute myocardial infarction was made. Because of communication difficulties, consent for thrombolysis could not be obtained. He was treated with oxygen, glyceryl trinitrate, aspirin and admitted to the coronary care unit.

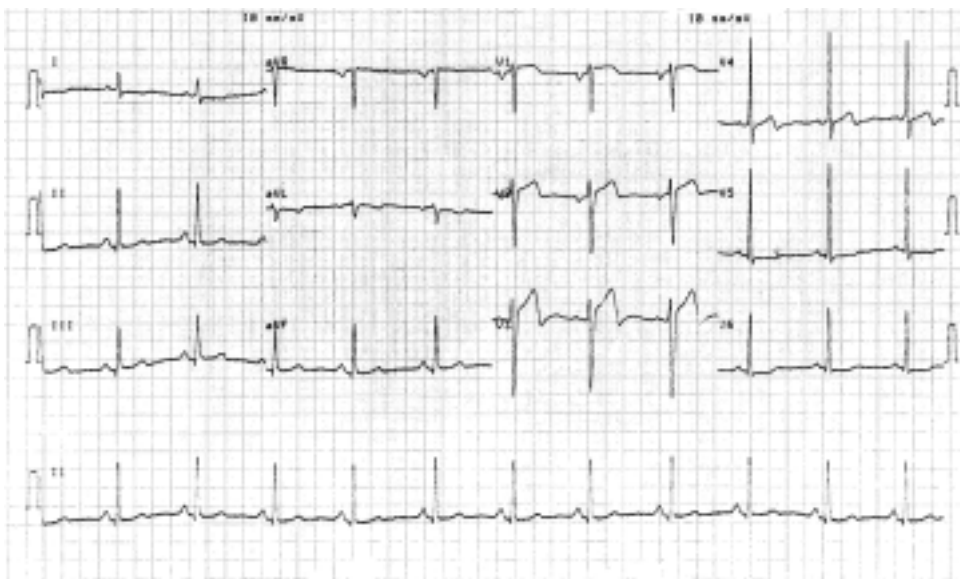


Figure 1. The initial ECG performed in the emergency department at 22:40 hour.



Figure 2. The ECG performed in the observation ward at 07:15 hour.

The initial ECG (Figure 1) was reviewed, and evidence of ST elevation in leads V2-3 was suspected in retrospect.

However, the pain subsided after admission. An ECG (Figure 3) was repeated at 09:14 hour. The findings were similar to the initial ECG. Serial blood tests for troponin I were all normal.

A diagnosis of Prinzmetal's angina was made. He was treated medically with a calcium channel blocker, a long-acting nitrate and low dose aspirin. Unfortunately, he developed another typical attack of Prinzmetal's angina in ward at about 02:30 hour several days later. Again, it subsided with immediate glyceryl trinitrate therapy. The dose of calcium channel blocker was stepped up for the recurrent attacks. Early coronary angiography was performed which showed 70% stenosis in the proximal left anterior descending artery. He did not have further attack thereafter and was discharged 10 days after admission.

Discussion

What is Prinzmetal's angina?

Prinzmetal's angina, described in 1959 by Myron Prinzmetal (an American cardiologist), is characterised by angina at rest associated with transient ST elevation on ECG. Symptoms often occur in the early morning (midnight to 08:00 hour). It is usually brief and very responsive to sublingual nitrates during attacks, with rapid relief of symptoms and ECG normalisation. It is caused by transient coronary artery spasm, usually focal, with normal coronary anatomy or at the site of an atherosclerotic plaque. Smoking is the most important risk factor. The exact incidence and prevalence are unknown. It occurs more often in younger patients than does exertional angina. The average age is 48 years. It affects more women than men.¹

Diagnosis can be made by ECG during pain and after relief of pain. The ECG during attacks shows ST elevation indistinguishable from that of acute myocardial infarction, but generally pathological Q

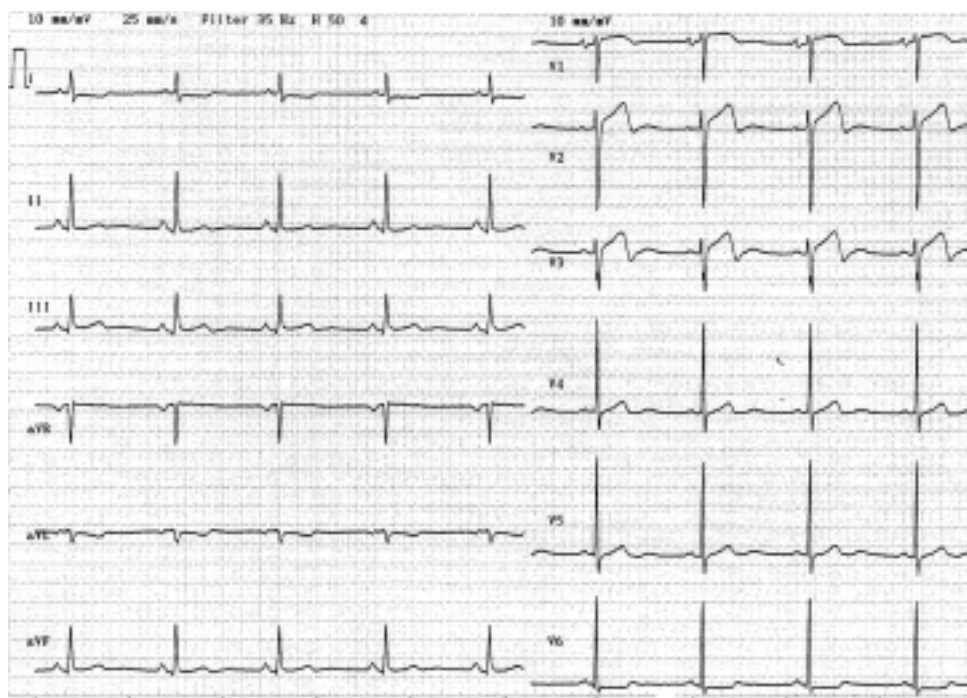


Figure 3. The ECG performed in the coronary care unit at 09:14 hour (after taking one tablet of sublingual glyceryl trinitrate).

waves or reciprocal changes should not be present. Correlation with the rapid clinical and electrocardiographic response to sublingual nitrate is also very helpful. Ambulatory ECG monitoring may be considered if the diagnosis is uncertain.

The treatment goal is to prevent coronary spasm. It is very important to stop smoking. Calcium channel blocker therapy is the mainstay of treatment. More than 50% of patients become asymptomatic with this therapy. Long-acting nitrates are also effective, but patients may develop tolerance. Sublingual nitrate therapy is indicated for acute attacks. Thrombolysis therapy is not indicated. Recognition of this condition will save patients from the potentially life-threatening complications of thrombolysis. Percutaneous transluminal coronary angioplasty with stenting has also been used successfully in patients refractory to medical therapy.¹

The acute active phase usually lasts three to six months, then symptoms often remit. Sudden death and acute myocardial infarction occur most often during this phase. Once the patient has passed this phase, the chance of long-term survival is excellent (89-97%).¹

Was the initial ECG abnormal? (Figure 1)

The ECG findings are sinus rhythm, ST elevation V1-3, heights of ST segments 2-4 mm (measured at 0.04 second past the J point), (the heights become 1.5-3 mm if measured at the J point), ST-segments concave upwards with no pathological Q waves nor reciprocal changes. This is a normal ECG called the male pattern.

A study was conducted among 6,014 healthy men in the US Air Force who were 16-58 years old. The result showed that 91% had ST elevation of 1-3 mm in one or more precordial leads and the elevation was most common and marked in lead V2.²

In another recent study of normal ECGs from 529 men, the prevalence of ST elevation of at least 1 mm in one or more of leads V1-4 was 93% in those 17-24 years old (measurement at the J point). The prevalence

decreased gradually with increasing age, reaching 30% in those 76 years of age or older.^{3,4}

As the majority of men have ST elevation of 1 mm or more (1-3 mm) in precordial leads, it is a normal finding, not a normal variant, and is designated as the male pattern.³ In this pattern, the ST-segment is concave upwards. The deeper the S wave, the greater the ST-segment elevation – a relation that is often found in patients with left ventricular hypertrophy. Since the QRS vector loop is swung posteriorly in these patients, often resulting in a QS pattern in leads V1-3, ST elevation in these leads can be deceptive.⁴

How do you measure the height of ST elevation?

Many doctors usually measure above the PT baseline at a point 0.04 second (1 mm) past the J point (the angle between the end of the QRS complex and the start of the ST-segment), following the American Heart Association. At the same time, many others measure at the J point.⁵

In fact, the inconsistent practices are well observed by Carley et al. They noted that the precise point at which ST elevation should be measured in acute myocardial infarction is unclear. They found that many textbooks on general medicine⁶⁻¹⁰ or electrocardiology¹¹⁻¹³ do not specify the point at which ST elevation should be measured. Among the major clinical trials of thrombolysis, most fail to specify where ST elevation should be measured.¹⁴⁻²² Of the few trials that have specified a point, inconsistency is noted. Koren et al²³ specified an ST segment of >0.2 mV persisting for more than 0.08 second beyond the J point, while Verstraete et al²⁴ used a point 0.06 second past the J point.⁵

In clinical practice, doctors may get additional evidence from the ECG to diagnose acute myocardial infarction (e.g. reciprocal changes in other leads or pathological Q waves). Moreover, the morphology of the ST segment itself changes with time in acute myocardial infarction. It is also very useful to compare it with old ECGs. Experienced doctors may therefore rely more on pattern recognition rather than on an absolute measurement of the ST segment.⁵

The future

Thus, ST elevation in the precordial leads can be regarded as normal for male pattern or benign early-repolarisation pattern. These ST elevations unfortunately meet the ECG criterion for thrombolytic therapy according to the guidelines of the American College of Cardiology and American Heart Association: ST elevation greater than 0.1 mV in 2 or more contiguous leads. Since this criterion can be misleading, the Clinical Policies Subcommittee of the American College of Emergency Physicians refined the guidelines by adding the qualifier ST elevations that are not characteristic of early repolarisation or pericarditis, nor of a repolarisation abnormality from left ventricular hypertrophy or bundle branch block. Nonetheless, these guidelines still fail to address the normal ST elevation in the right precordial leads that is present in many healthy persons.⁴

In our locality, the ECG criterion for thrombolysis (in the precordial leads) is usually ST elevation 2 mm or more (i.e. 0.2 mV or more) in two or more contiguous leads.^{16,25-27} It is already better in this aspect but is still not good enough to elucidate the confusion.

Besides, there is no consensus about the point at which ST elevation should be measured in acute myocardial infarction. In fact, there is wide variation in the point at which doctors measure ST elevation. As a result, there is a significant variation in the observed magnitude of ST elevation. Such variation may result in an inappropriate prescription, or a failure to prescribe thrombolytic.⁵ Worse still, the J point may not be easily defined in quite a number of ECGs.

Carley et al stated that, "If the aim of early thrombolysis in acute myocardial infarction is to be achieved, it is important that clear instructions are given to junior staff with regard to ST measurement".⁵

Logically, it is probable that intravenous thrombolytics have been prescribed to patients having a male pattern in ECGs presenting with non-Q-wave acute myocardial infarction/unstable angina (clinically very similar to ST-segment elevation acute myocardial

infarction but for whom intravenous thrombolysis is not helpful or even potentially harmful).

More effort in further studies and drawing up of new guidelines is necessary and expected.

Conclusion

Practically, clinical correlation is always important. Persistent chest pain compatible with acute myocardial infarction should always be present before thrombolysis is considered in emergency rooms. For Prinzmetal's angina, the pain and the ECG changes are usually very transient. It typically occurs in the early morning (midnight to 08:00 hour). Night shift duty emergency physicians should be alert to this possibility.

Acknowledgement

We thank Dr Hui Tze Leung and Dr Chan Kwok Hei for their advice and support.

References

1. Hott BJ, Wenger NK. Angina, Prinzmetal's, variant angina. In: Alpert JS, editor. The AHA clinical cardiac consult (5-minute). Philadelphia: Lippincott Williams & Wilkins; 2001. p.12-3.
2. Hiss RG, Lamb LE, Allen MF. Electrocardiographic findings in 67,375 asymptomatic subjects. *Am J Cardiol* 1960;6:200-31.
3. Surawicz B, Parikh SR. 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* 2002;40(10):1870-6.
4. Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. *N Engl J Med* 2003;349(22):2128-35.
5. Carley SD, Gamon R, Driscoll PA, Brown G, Wallman P. What's the point of ST elevation? *Emerg Med J* 2002; 19(2):126-8.
6. Kumar P, Clark M. Clinical medicine. 3rd edn. London: Saunders; 1997.
7. Souhami RC, Moxham J. Textbook of medicine. 3rd edn. Edinburgh: Churchill Livingstone; 1997.

8. Hope RA, Longmore JM, Hodgetts TJ, et al. Oxford handbook of clinical medicine. 3rd edn. Oxford: Oxford University Press; 1993.
9. Moulton C, Yates D. Emergency medicine. 2nd edn. Oxford: Blackwell Science; 1999.
10. Weatherall DJ, Ledingham JGG, Warrell DA. Oxford textbook of medicine. 2nd edn. Oxford: Oxford Medical Publications; 1998.
11. Rowlands DJ. Understanding the electrocardiogram. Section 2. Morphological abnormalities. Cheshire: Imperial Chemical Company; 1982.
12. Hampton JR. The ECG made easy. 4th edn. Edinburgh: Churchill Livingstone; 1992.
13. Hampton JR. The ECG in practice. Edinburgh: Churchill Livingstone; 1989.
14. Tandberg D, Kastendieck KD, Meskin S. Observer variation in measured ST-segment elevation. *Ann Emerg Med* 1999;34(4 Pt 1):448-52.
15. AIMS Trial Study Group. Effect of intravenous APSAC on mortality after acute myocardial infarction: preliminary report of a placebo-controlled clinical trial. *Lancet* 1988;1(8585):545-9.
16. The GUSTO investigators. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. *N Engl J Med* 1993;329(10):673-82.
17. The I.S.A.M. Study Group. A prospective trial of intravenous streptokinase in acute myocardial infarction (I.S.A.M.). Mortality, morbidity, and infarct size at 21 days. *N Engl J Med* 1986;314(23):1465-71.
18. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986;1(8478):397-402.
19. Bates ER, Califf RM, Stack RS, et al. Thrombolysis and Angioplasty in Myocardial Infarction (TAMI-1) trial: influence of infarct location on arterial patency, left ventricular function and mortality. *J Am Coll Cardiol* 1989;13(1):12-8.
20. Chesebro JH, Knatterud G, Roberts R, et al. Thrombolysis in Myocardial Infarction (TIMI) Trial, Phase I: A comparison between intravenous tissue plasminogen activator and intravenous streptokinase. Clinical findings through hospital discharge. *Circulation* 1987;76(1):142-54.
21. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. *Lancet* 1988;2(8607):349-60.
22. Wilcox RG, von der Lippe G, Olsson CG, Jensen G, Skene AM, Hampton JR. Trial of tissue plasminogen activator for mortality reduction in acute myocardial infarction. Anglo-Scandinavian Study of Early Thrombolysis (ASSET). *Lancet* 1988;2(8610):525-30.
23. Koren G, Weiss AT, Hasin Y, et al. Prevention of myocardial damage in acute myocardial ischemia by early treatment with intravenous streptokinase. *N Engl J Med* 1985;313(22):1384-9.
24. Verstraete M, Bernard R, Bory M, et al. Randomised trial of intravenous recombinant tissue-type plasminogen activator versus intravenous streptokinase in acute myocardial infarction. Report from the European Cooperative Study Group for Recombinant Tissue-type Plasminogen Activator. *Lancet* 1985;1(8433):842-7.
25. The Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO III) Investigators. A comparison of reteplase with alteplase for acute myocardial infarction. *N Engl J Med* 1997;337(16):1118-23.
26. Assessment of the Safety and Efficacy of a New Thrombolytic Investigators. Single-bolus tenecteplase compared with front-loaded alteplase in acute myocardial infarction: the ASSENT-2 double-blind randomised trial. *Lancet* 1999;354(9180):716-22.
27. Ball C, Shenker N. Myocardial infarction. In: Ball CM, Philips RS, editors. Evidence-based on-call acute medicine. Edinburgh: Churchill Livingstone; 2001.