

ST Elevation - is it always Infarction?

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ABSTRACT

Background: Acute ST elevation MI is a life threatening emergency, recognized on Electrocardiogram as ST Elevation requiring urgent management in the form of reperfusion therapy. But there are causes of ST elevation other than MI needed to be remembered to deliver reperfusion therapy to only to true ischemic events.

Aim: To look for the pseudo-infarction patterns leading to undue delivery of thrombolytic therapy.

Methods: All patients suspected of ST elevation MI given thrombolytic therapy were retrospectively analyzed for true ischemic events and the resultant ST Elevation not indicative of acute coronary event.

Results: There are few causes of pseudo ST Elevation that has to be looked for before giving thrombolytic therapy in suspected ST elevation MI.

Conclusion: Every ST Elevation is not infarction so a careful approach is required to rule in and rule out infarction.

Key words: ST Elevation MI, Pseudoinfarction, Reperfusion.

INTRODUCTION

Acute ST Elevation myocardial infarction (STEMI) constitutes a major public health problem not only in western countries but increasingly in developing countries¹. In United States, there are estimated to be more than half million STEMI events annually and these have provided strong impetus for efforts to improve both the process whereby care is delivered and the treatment elements administered². Acute MI resulting from an occlusive thrombus is recognized on ECG by ST elevation³. Myocardial infarction is the leading cause of death in United States (US) as well as in most industrialized nations throughout the world⁴. Early (60 to 90) and complete (i.e., TIMI flow grade 3) patency of the infarct-related artery is a major determinant of survival after thrombus for acute myocardial infarction^{5,6,7}. Reperfusion therapy has proved to be beneficial in such cases. The earlier the perfusion the better the benefit and time to treatment is now considered to indicate quality of care. Thrombolysis is established treatment in the early management of myocardial infarction and it reduces 35 day mortality by about a quarter⁸. These days when thrombolytic therapy are carried out so readily it is important to remember acute infarction is not the only cause of ST elevation and similar changes of ST Elevation and Q wave may be seen in patients without coronary artery disease ("pseudoinfarction")⁹.

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METHOD

Patients presenting in the emergency department of JHL during the month of July, 2012 to August, 2012 with chest pain and ST elevation in two contiguous leads and they were given thrombolytic therapy. Subsequently they were followed and reviewed with serial ECGs on daily basis, two sets of cardiac enzymes (Troponin), Echocardiography on 3rd or 4th post admission day, and some with coronary angiography within 14 days of discharge to look for evolutionary changes, rise in cardiac enzymes, regional wall motion abnormalities and coronary artery disease respectively to confirm that the patient had true cardiac ischemic event or not. The consent was taken from every patient to include in the study. Total no. of patients was 75 with 28% female and 72% male. The mean ages of the patients taken were 51.4. The mean stay of the patients was 5±1 day. Among these patients 59% were of suspected anterior wall mi, 7% of inferior wall mi, 33% of inferior wall with RV infarct, 1% isolated posterior wall MI. Patients having contraindication to thrombolytic therapy, having co-morbidities, having documented evidence of cardiomyopathies, not giving consent was excluded from the study.

RESULTS

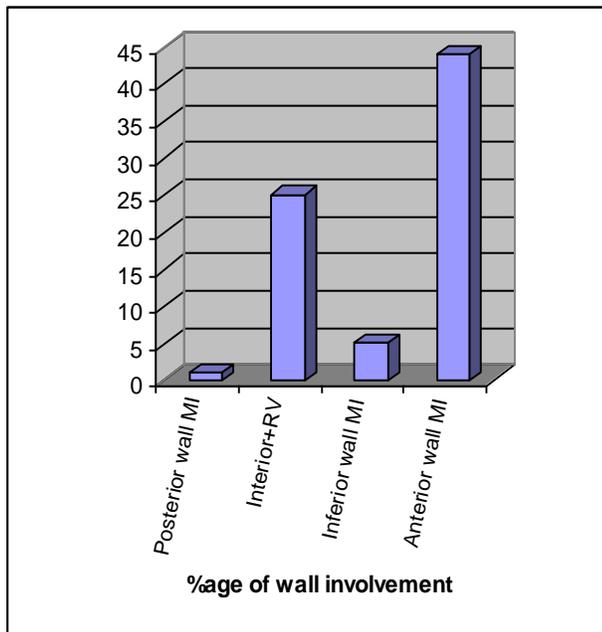
With retrospective analysis of various modalities of ECG, ECHO, Angio, Cardiac enzymes, total of 6 patients were found to have no acute coronary event or in other words not fulfilling the criteria for

streptokinase, received thrombolytic therapy on the basis of chest pain and pseudo ST Elevation in the ECG, these include the following causes

- Male pattern
- Left ventricular hypertrophy
- Early repolarisation therapy
- Cases of left bundle branch block
- Normal variant

So the total 6 out of 75 patients, it came out to be 8% who were having pseudoinfarction pattern miming acute infarction and on that basis were given thrombolytic therapy.

=n	Anterior Wall MI	Simple inferior wall MI	Posterior Wall MI	Complex Inferior Wall MI
75	44	05	01	25



45% were diabetics, 37% were hypertensive, 25% were both diabetic and hypertensive, 15% were having positive family history of IHD.

=n	Diabetics	Hypertensive	HTN + DM	Family history	Smoker
75	34	28	19	11	38

All were given thrombolytic therapy with success except for three patients who became hypotensive and were not completely given thrombolytics. The mean time for SK administration was 47+-5 minutes. Those patients having chest pain, ST elevation in two contiguous leads fulfilling the criteria for streptokinase, giving consent were included in the study.

DISCUSSION

The American College of Cardiology/American Heart Association Guidelines for the Management of Patients with Acute Myocardial Infarction^{10,11} consider the presence of electrocardiographic ST segment elevation of greater than 0.1 mV in two anatomically contiguous leads a class I indication for urgent reperfusion therapy in the patient presumed to have AMI. There are several other causes of ST elevation other than mi which have close resemblance to the infarction pattern so candidate for thrombolytic therapy like LVH, ERP, LBBB, Bugada Syndrome, Male Pattern, Hyperkalemia etc¹².

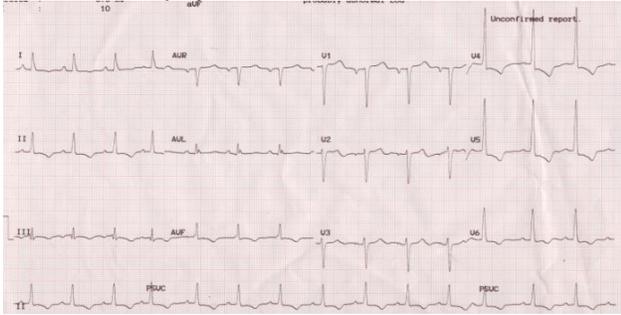
They are misinterpreted as acute infarction and sometimes given undue thrombolytic therapy. Larsen et al¹³ have shown that electrocardiographic LVH is incorrectly interpreted more than 70% of the time in patients thought to have an acute coronary syndrome (ACS). Failure to make this diagnosis is responsible for large financial payments in the medico legal arena involving malpractice cases against emergency physicians (EP)^{14,15}.

Retrospective analysis of a significant minority of these cases has revealed that misinterpretation of the ECG has played a role in as many as 25% of the cases¹⁵.

The thrombolytic therapy has its side effects some of which are life threatening like intra cerebral bleed, major bleeding, hypersensitivity reactions etc. and also in ISIS-2 trial only 4.4% of patients suffered allergic reactions to Streptokinase¹¹ and may encountered more often when Streptokinase is administered a second time¹⁶. Brugemann et al showed that patients with higher levels of antistreptokinase antibodies who received antistreptase (streptokinase analogue) didn't achieve patency of the effected artery¹⁷. One study has showed that neutralizing titres returned to control levels after two years¹⁸. The currently available evidence suggests that it is reasonable to avoid using Streptokinase again in patients requiring Thrombolysis more than 4 days after previous treatment.{{once the streptokinase is given its becomes a contraindication in the future next 6 months so if the patient than develops the acute infarction he will not be benefited from the thrombolytic therapy. because inappropriate fibrinolysis is not without morbidity and mortality^{19,20,21} and any reduction in its occurrence would be beneficial. So recognition of these patterns is very necessary for accurate supply of thrombolytic therapy. In our study we found few of the causes of pseudo infarction pattern.

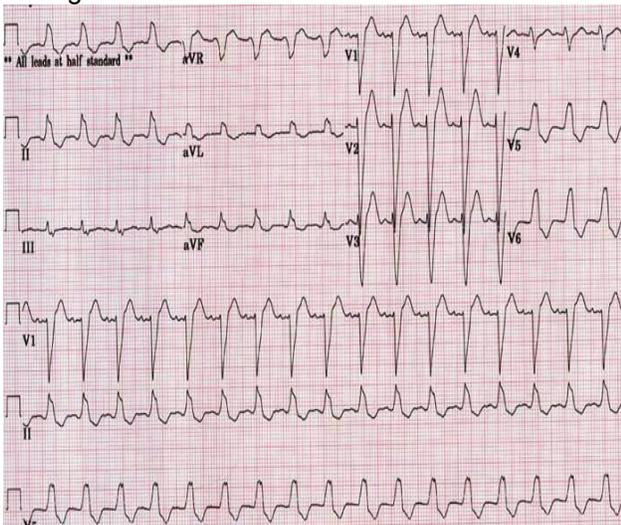
LVH and bundle branch block represent other frequently encountered patterns in three studies^{22,23},

which were either misinterpreted or interpreted with disagreement among the EPs (features of LVH) In LVH there is often a QS deflection or poor R wave progression in the right precordial leads that suggests anterior wall myocardial infarction. The secondary ST segment elevation in these leads may be mistaken as a current of injury.



Tracing is from a patient with left Ventricular Hypertrophy

In 1945, Wilson and associates²⁴ concluded that “in the presence of left bundle branch block, it is seldom possible to make the diagnosis of myocardial infarction on the basis of electrocardiographic findings alone.”



Goldberger et al²⁵ stated that the LBBB pattern is the most frequently misinterpreted pseudo infarct pattern in practice today, with resultant inappropriate management (Features of LBBB). There are number of proposed criteria's which aid in the differentiation of acute LBBB due to acute infarction and chronic LBBB. Several studies have systematically evaluated the value of different ECG findings of acute MI in LBBB. One study by Wackers, for example, correlated ECG changes in LBBB with localization of

the infarct by thallium scintigraphy²⁷. The most useful ECG criteria were:

- Serial ECG changes --- 67% sensitivity
- ST segment elevation — 54% sensitivity
- Abnormal Q waves— 31% sensitivity
- Cabrera's sign — 27% sensitivity, 47% for anteroseptal MI

Initial positivity in V1 with a Q wave in V6 — 20 percent sensitivity but 100% specificity for anteroseptal MI. Cabrera's sign refers to prominent (0.05 sec) notching in the ascending limb of the S wave in leads V3 and V4; a similar finding is prominent notching of the ascending limb of the R wave in lead V5 or V6 (Chapman's sign)²⁷. These signs have a specificity that approaches 90 percent. However, there may be a high degree of inter-observer variability in accurate identification and their sensitivity is quite low.

A large trial of thrombolytic therapy for acute MI (GUSTO-1) provided an opportunity to revisit the issue of the electrocardiographic diagnosis of evolving acute MI in the presence of LBBB²⁸. A scoring system was developed. The three ECG criteria with an independent value in the diagnosis of acute infarction and the score for each were:

- ST segment elevation of 1 mm or more that was in the same direction (concordant) as the QRS complex - score 5.
- ST segment depression of 1 mm or more in lead V1, V2, or V3 - score 3.
- ST segment elevation of 5 mm or more that was discordant with the QRS complex (i.e., associated with a QS or rS complex) — score 2.

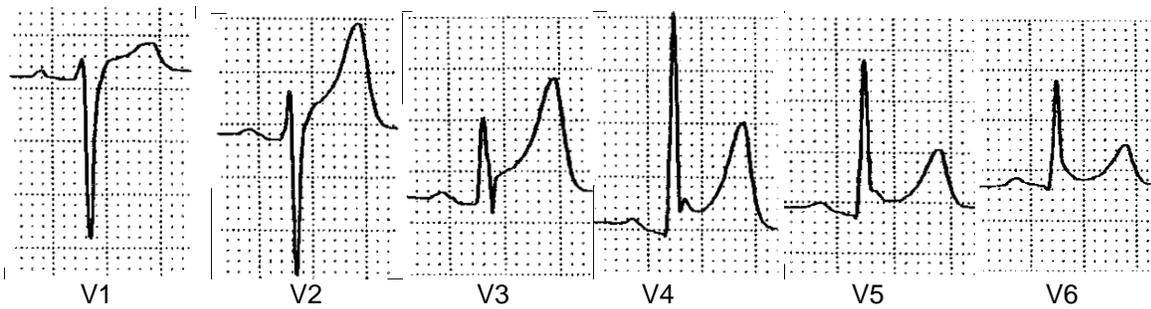
A minimal score of three was required for a specificity of 90%. Normal male pattern was seen in 90% of healthy young men. The concave upwards ST elevation usually 1-3mm and most marked in v2¹². In a study of 6014 healthy men in US Air Force who were 16 to 58 years old, 91% had ST-segment elevation of 1-3mm in one or more precordial leads²⁹. The elevation was most common and marked in lead V2. In a recent study of normal electrocardiograms from 529 men, the prevalence of ST-segment elevation of at least 1 mm in one or more of leads V1 through V4 was 93% in the men who were 17 to 24 years old³⁰. The prevalence declined gradually with increasing age, reaching 30% in men who were 76 years of age or older. In contrast, about 20% of normal electrocardiograms from women had ST-segment elevation of 1mm or more, and this prevalence remained unchanged regardless of the women ages.



Since the majority of men have ST-segment elevation of 1 mm or more in precordial leads, it is a normal finding, not a normal variant, and is designated as a male pattern; in these patterns the ST elevation is concave. The deeper the S wave, the greater the ST elevation.

Early repolarisation of the ST segment is an electrocardiographic variant with a benign long-term prognosis³¹. Its prevalence has been estimated at between 1 and 5% of healthy adults^{32,33} and it is more

common in young athletic men³⁴ but this distribution is not rare in women, older persons, whites and inactive persons³⁵. The mechanism of early repolarisation is not completely understood; although accumulating evidence suggests a vagal origin^{32,36} seen in young black men. This pattern consists of a prominent notch or slur on the down sloping portion of QRS complex, followed by a diffuse upward ST Segment concavity concordant with the QRS and a positive T wave in the same leads³⁷.

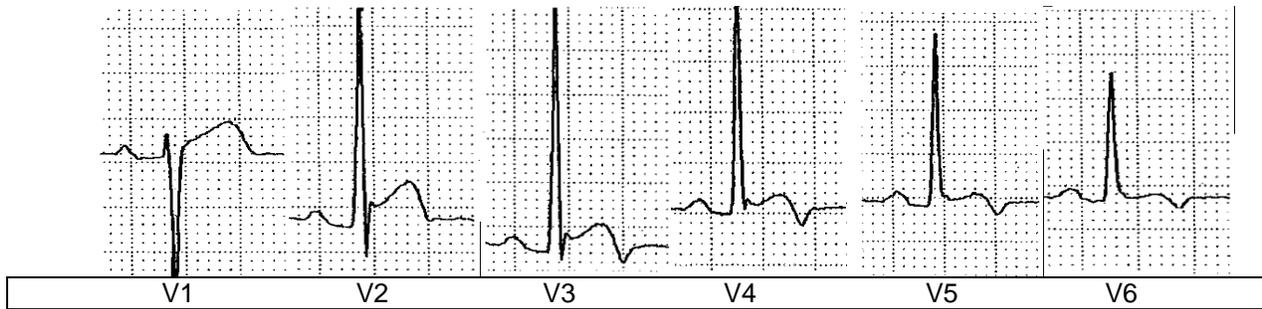


The degree of ST-segment elevation is usually less than 2mm but may be great as 5mm. another ECG feature that defines this ECG phenomena is the localization of the ST Segment elevation. Mid to lateral precordial leads (V2-V5) generally show the greatest ST segment elevation. Similar but usually less prominent changes may appear in the other leads. It is rare to find ST segment elevation in limb leads alone. Slower heart rates tend to increase the degree of ST segment elevation and the amplitude of the T waves. Fast heart rates (>100/min) have the opposite effect with normalization of ST Segment and reduction of T wave amplitude. Additional ECG criteria for the ERP include reciprocal ST segment elevation in AVR and waxing and waning of the ST Segment over time³⁸.

It has been called by a variety of names including "unusual RT Segment deviation", "premature repolarisation", "normal RS-T elevation variant", and "early repolarisation syndrome"³⁵. It was first described by Shipley and Halloran in 1936.³⁹ The rationale is that this "abnormal" pattern doesn't

represent organic disease of the heart, is not associated with symptoms, has no effect on longevity and doesn't require treatment. The benign nature of this ERP has been established by longitudinal follow up studies some as long as 26 years. ST Segment elevation persists for decades but tends to decrease with decreasing age. All studies have consistently shown no evidence of increased likelihood of fatal or nonfatal cardiovascular events^{35,40}.

ST elevation of normal variant seen in V3-V5 with T Wave inversion, short QT interval and high QRS voltage. Usually combination of early repolarisation pattern and persistent juvenile T Wave pattern¹² often the findings are so suggestive of myocardial infarction that an echocardiogram is often necessary to differentiate them, especially if one is not aware of this normal variant. In most of these cases the QT interval is short, whereas it is not short in acute myocardial infarction or pericarditis. This normal variant differs from the early-repolarisation pattern in that the T waves are inverted and the ST segment tends to be coved.



REFERENCES

1. Yusuf S, Reddy S, Onpuu S, Anand S. Global Burden of Cardiovascular Diseases : part I: General consideration, the Epidemiological Transition, Risk Factors, and Impact of Urbanisation. *Circulation* 2001; 104: 2746-2753.
2. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology / American Heart Association Task Force Committee to Revise the 1999 Guidelines for the Management of patients with acute myocardial infarction. *J Am Cardiol.* 2004 ; E1-E211.
3. Thygesen, K, Alpert, JS, White, HD, et al. Universal definition of myocardial infarction: Kristian Thygesen, Joseph S. Alpert and Harvey D. White on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. *Eur Heart J* 2007; 28:2525.
4. Ryan TJ, Antman EM, Brooks NH, et al. 1999 update: ACC/AHA guidelines for the management of patients with acute myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines [Committee on Management of Acute Myocardial infarction]. *J Am Coll Cardiol* 1999;34:890-911 and *Circulation* 1999; 100: 1016-1030.
5. The GUSTO Angiography Investigators, the effects of tP, Streptokinase, or both on coronary angiography patency, ventricular function, and survival after acute myocardial infarction. *N Engl J Med* 329 (1993), pp. 1615-1622.
6. ML. Stadius, Angiographic monitoring of repolarisation therapy for acute myocardial infarction: TIMI grade 3 perfusion is the goal. *Circulation* 87 (1993), pp. 2055-2057.
7. JW Kennedy, Optimal management of acute myocardial infarction requires early and complete reperfusion. *Circulation* 91 (1995), pp. 1905-1907.
8. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group, Randomized trial of Intravenous Streptokinase, oral aspirin, both or neither among 17187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet* 1988 ; ii : 349-60.
9. Cheng TO. The International Textbook of cardiology. New York: Per-gamon; 1987: 72-73.
10. A.L. Goldberger, Myocardial Infarction: Electrocardiographic Differential Diagnosis. (4th ed.), Mosby, St. Louis (1991).
11. E.B. Sgarbossa, S.L. Pinski, A. Barbagelata *et al.*, Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. *N Engl J Med* 334 (1996), pp. 481-487.
12. WANG K, Asinger RW, Marriot HJL.ST-Segment elevation in conditions other than acute mi. *N Eng J Med* 2003; 349: 2128-35.)
13. J.H. Pope, T.P. Auferheide, R. Rutzhaizer *et al.*, Missed diagnoses of acute cardiac ischemia in the emergency department. *N Engl J Med* 342 (2000), pp. 1163-1170.
14. Karcz, J. Holbrook, M.C. Burke *et al.*, Massachusetts emergency medicine closed malpractice claims: 1988-1990. *Ann Emerg Med* 22 (1993), pp. 553-559
15. R.A. Rusnak, T.O. Stair, K. Hansen *et al.*, Litigation against the emergency physician: common features in cases of missed myocardial infarction. *Ann Emerg Med* 18 (1989), pp. 1029-1034.
16. Lee HS, Yule S, Mckenzie A, Cross S, Reid T, Davidson R. Hypersensitivity reactions to Streptokinase in patients with high pretreatment antistreptokinase antibody and neutralization titres. *Eur Heart J* 1993; 14: 1640-3
17. Brugemann J, Van der Meer J, Born VJ, Van der Schaff W, de Graeff PA, Lie KI. Antistreptokinase Antibodies inhibit fibrinogen effects of antistreplase in acute myocardial infarction. *Am J Cardiol* 1993; 72: 462-4.
18. McGrath K, Hogen C, Hant D, O Malley C, Green N, Dauer R, Dalli A. Neutralising antibodies after Streptokinase treatment for myocardial infarction: a persistent puzzle. *Br Heart J* 1995; 74: 122-3.
19. W.J. Rogers, L.I. Bowlby, N.C. Chandra *et al.*, Treatment of myocardial infarction in the United States (1990 to 1993). Observations from the National Registry of Myocardial Infarction. *Circulation* 90 (1994), pp. 2103-2114.
20. Committee on the Management of Acute Myocardial Infarction, ACC/AHA Guidelines for the Management for Patients with Acute Myocardial Infarction. *J Am Coll Cardiol* 28 (1996), pp. 1328-1428.
21. Committee on the Management of Acute Myocardial Infarction, 1999 update: ACC/AHA Guidelines for the Management for Patients with Acute Myocardial Infarction. *J Am Coll Cardiol* 34 (1999), pp. 1890-1911.
22. R.E. Rude, W.K. Poole, J.E. Muller *et al.*, Electrocardiographic and clinical criteria for recognition of acute myocardial infarction based on analysis of 3697 patients. *Am J Cardiol* 52 (1983), pp. 936-942.

23. L.A. Otto and T.P. Aufderheide, Evaluation of ST segment elevation criteria for the prehospital electrocardiographic diagnosis of acute myocardial infarction. *Ann Emerg Med* 23 (1994), pp. 17–24.
24. Wilson FN, Rosenbaum FF, Johnston FD, et al: The electrocardiographic diagnosis of myocardial infarction complicated by left bundle branch block. *Arch Inst Cardiol Mexico* 14:201-224, 1945
25. S. Schor, S. Behar, B. Modan *et al.*, Disposition of presumed coronary patients from an emergency room: a follow-up study. *JAMA* 236 (1976), pp. 941–943.
26. Laham, CL, Hammill, SC, Gibbons, RJ. New criteria for the diagnosis of healed inferior wall myocardial infarction in patients with left bundle branch block. *Am J Cardiol* 1997; 79:19.
27. Wackers, FJ. The diagnosis of myocardial infarction in the presence of left bundle branch block. *Cardiol Clin* 1987; 5:393.
28. Sgarbossa, EB, Pinski, SL, Barbagelata, A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. *N Engl J Med* 1996; 334:481.
29. Hiss RG, Lamb LE, Allen MF. Electrocardiographic findings in 67,375 asymptomatic subjects. *Am J Cardiol* 1960;6:200-31.
30. 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;40:1870-6.
31. Klatsky AL, Oehm R, Cooper RA, Udaltsova N, Armstrong MA. The early repolarization normal variant electrocardiogram: correlates and consequences. *Am J Med* 2003;115:171-7.
32. Mehta MC, Jain AC. Early repolarization on scalar electrocardiogram. *Am J Med Sci* 1995;309:305-11.
33. Mehta M, Jain A, Mehta A. Early repolarisation. *Clin Cardiol.* 1999; 22: 59-65.
34. Bjornstad H, Storstein L, Meen HD, Hals O. Electrocardiographic findings of repolarization in athletic students and control subjects. *Cardiology* 1994;84:51-60.
35. Klatsky A, Oehm R, Cooper R, Udattsova N, Armstrong M. the ERP normal variant electrocardiogram: Correlates and consequences. *Am J Med.* 2003; 115: 171-177.
36. Haydar ZR, Brantley DA, Gittings NS, Wright JG, Fleg JL. Early repolarization: an electrocardiographic predictor of enhanced aerobic fitness. *Am J Cardiol* 2000;85:264-6.
37. Wasserburger R, Alt W, Lloyd C. the normal RS-T segment variant. *Am J Cardiol.* 1961;8:184-192.
38. Gussak I, Antzelevitch C. Early repolarisation syndrome: clinical characteristics and possible cellular and ionic mechanism. *J Electrocardiol.* 2000; 33: 299-309.
39. Alimurung B, Gilbert C, Felner J, Schlant R. The influence of ERP variant on the exercise Electrocardiograph: A correlation with coronary angiogram. *Am Heart J.* 1980; 99:739-745.
40. Kambara H, Phillips J. Long term Evaluation of ERP syndrome(normal variant RS-T segment elevation). *Am J Cardiol.* 1976; 38: 157-166.