# RBBB Configuration Changes Over Years Mimics Myocardial Infarction ECG?: A Case Report

G El Sherbiny

**Citation**

G El Sherbiny. *RBBB Configuration Changes Over Years Mimics Myocardial Infarction ECG?: A Case Report*. The Internet Journal of Emergency Medicine. 2005 Volume 3 Number 1.

**Abstract**

Many patients with acute myocardial infarction have atypical symptoms, and one half of patients with typical symptoms do not have acute myocardial infarction. One half of patients with acute myocardial infarction have non-diagnostic ECGs. 1 This is one among the challenges often encountered by an emergency department physician. We report a case which is more challenging, a patient with diagnostic ECG with atypical symptoms and a completely normal tests and investigations results. The diagnosis as infarction was excluded and the configuration of right bundle branch block (RBBB) as seen in his ECG 10 years ago was suggested to mimic a myocardial infarction ECG pattern. This unusual case was initially managed as AMI.

## INTRODUCTION

Atypical myocardial infarction (MI) has been mentioned in different literatures. Many cases have been reported. Available reports in general however often describe true infarction with vague presentations. MI mimics are highly reported as well. This case report describes a reverse picture of the topics that have been published unveiling the MI ‘atypicality'. To the best of our knowledge no similar case has yet been reported.

## CASE REPORT

A 52-year-old male patient was referred to our hospital from a primary health care clinic as a case of inferio-posterior myocardial infarction, diagnosis made out of his ECG presentation (Figure 1-A and I-B).

**Figure 1**

Figure 1a: (PHCC ECG)

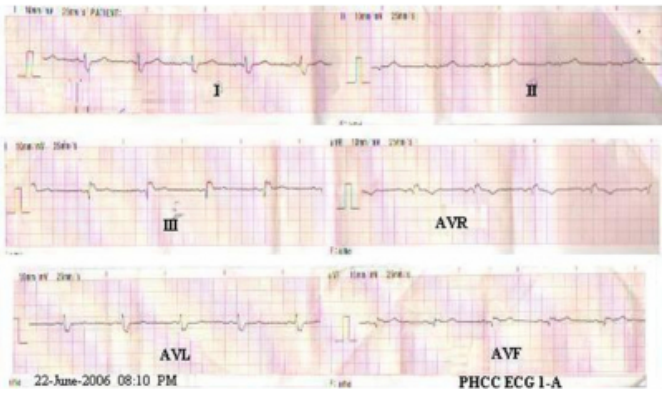
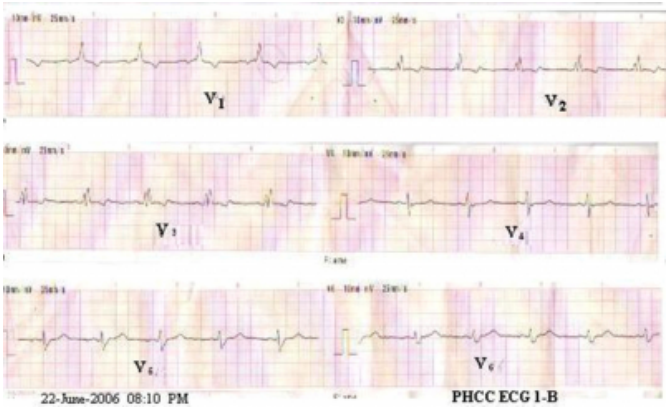



**Figure 2**

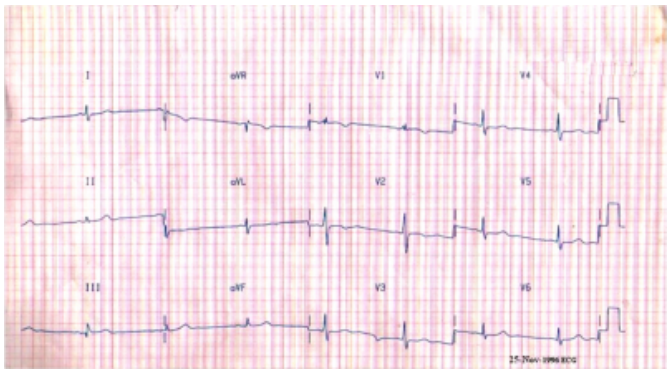
Figure 1b: (PHCC ECG)




This patient presented to our emergency department with complains of fatigue and body malaise for twenty four (24) hours, mild diaphoresis, and feeling of ‘unwellness'. He had two episodes of vomiting in the previous night which he attributed as caused by heavy dinner. This gentleman had an incomplete right bundle branch block (not labeled as ischemic heart disease) noted in his ECG (Figure 2) 10 years ago prior to hypophysectomy for pituitary adenoma . There is no family history of cardiac disease. He quitted smoking since 15 years. He is compliant with the daily maintenance dose of Eltroxin 25 mcg and Prednisolone 5 mg since that pituitary gland surgery. He is physically fit, with good body built, and he is married with children.

**Figure 3**

Figure 2: (1996 ECG)



On examinations at the department of emergency medicine (DEM): he was ill-looking with a weak voice, not apprehensive, no distress, no chest pain, and no shortness of breath. Vital signs are as follows: BP 122/74mmHg (equal in both arms); Pulse 54 bpm, regular rhythm, no any special character, with no radio-femoral delay; RR 16/min, afebrile. Bedside Glucose check was 6.3 mmol/L.

The 12 leads ECG recordings at DEM presentation (Figure 3) showed ST segments elevation in the inferior leads II, III, and AVF with reciprocal changes in leads I and AVL, a bifascicular block with right axis deviation led to a diagnosis of Inferior Myocardial Infarction. Right sided ECG showed elevated ST segments in VR5 and VR6 which highly suggested Right Ventricular Infarction (Figure 4). Posterior leads were not significant thus Posterior Myocardial Infarction was excluded. CXR was normal. Laboratory investigations highlighting cardiac enzymes were all within normal limits. CPK was 85 U/L (normal range is 20-195 U/L) while Troponin I was 0 ng/L (normal value is 0-2 ng/L).

**Figure 4**

Figure 3: (Emergency Department presentation)

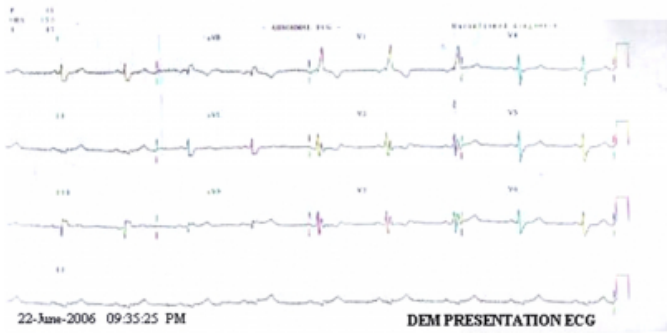
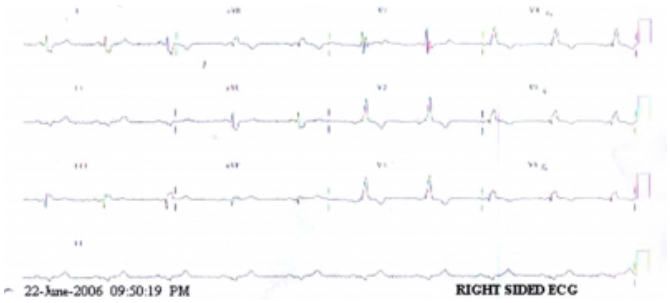



**Figure 5**

Figure 4: (Right-Sided ECG)




This case was managed initially as an acute myocardial infarction (AMI) but not in golden hour. Antithrombotics Aspirin 300 mg p.o. OD and Enoxaparin sodium 60 mg s/c, BID and platelet aggregation inhibitor Plavix (Clopidogrel) 300 mg p.o. OD were started. Such management was supported by the cardiologist “not to miss it” per his opinion despite of a complete normal bedside echocardiography. The patient was admitted to Coronary Care Unit (CCU) for 24 hours observation without any changes noted. He was shifted to the medical ward on the following day. Latter on the third day, with the same ECG tracings, the patient tremendously improved and was discharged with a clinic appointment for follow up. “Persantin Test” after 2 months was scheduled.

## DISCUSSION

The World Health Organization criteria were refined in 2000 to give more prominence to biomarkers. [2] According to the new guidelines in the diagnosis of MI, a cardiac troponin rise accompanied by either typical symptom, pathological Q waves, ST elevation or depression or coronary intervention are diagnostic of MI.

Troponin Rise. Troponin is currently the pearl in myocardial infarction diagnosis. The acute myocardial infarction definition issued in 2000 by the American College of Cardiology and the European Society of Cardiology requires elevation of cardiac biomarkers (preferably troponins). [3] The serum cardiac markers results of the patient were all normal thus excludes this patient entirely out of the troponin criterion.

Accompanying Typical Symptom. The patient denied clinical history of cardiac chest pain. Absence of pain however would not disalarm any health practitioner to exclude the diagnosis. Atypical presentation of myocardial infarction without chest pain is common. Data from the Framingham study [4] (cohort of 5,209 in 30 years follow up)

suggest that 25% of acute myocardial infarcts were only apparent after an examination of the ECG. In almost half of these cases the myocardial infarction was truly 'silent' and the remainder was accompanied by atypical symptoms [4] so atypical that neither the patient nor the attending physician entertained MI as a possible diagnosis. [5] Approximately one third of all myocardial infarctions are silent, without chest pain or other symptoms. [6] Symptoms shown to be associated with unrecognized MI are dyspnea, nonproductive cough, fatigue, abdominal or epigastric pain, nausea and vomiting, syncope, and palpitations. So-called 'soft' clinical features, such as fatigue, weakness, malaise, dizziness, and 'clouding of the mind,' are surprisingly frequent, occurring in 11% to 40% of patients with AMI. [7] With the absence of the chest pain, our patient's complain of fatigue, weakness, and the episodes of vomiting have been given consideration speculating these might give significance in the diagnosis.

The ECGs. The ECG is the most important source for the early diagnosis of an acute myocardial infarction. However, there are many ECG recordings simulating MI and on the other hand some ECGs are vague which make confusion in the diagnosis. Though a picture of typical MI is clearly feasible on this reported patient's ECG, his old ECG recordings ten years back which showed an incomplete RBBB not labelled as an ischemic heart disease was accounted for. Harrigan, et al cited that an incomplete RBBB is a common finding and may be considered a normal variant [8] . Rowlands highlighted that there is no evidence to suggest established RBBB carries other than a normal prognosis [9] . So in general, incomplete bundle branch block should trigger a non-invasive search for underlying heart disease. If none is found, no further tests or treatment are needed. [10] In this patient, echocardiogram confirmed that heart is in normal structure, normal chest x-ray ruled out pulmonary involvement, and the probability of effects of hypophysectomy to such ECG changes deemed irrelevant to have an association based on an evident full control of adrenal physiological functions. Without foreseeable factor to give a suspicion, the possibility of RBBB configuration changes over time which mimic a typical MI ECG tracing was highly considered. We believe that though considered as normal variant, such RBBB is caused by some alteration of a normal process. The same factor altering the normal process causes some configuration changes with an unknown mechanism which results to an ECG morphology transformation, The ECG morphologies may transform from one type to the other or may normalize completely. [7] We clearly emphasize however that it is purely our suggestion.

As there is no any clear explanation to this MI ECG pattern mimic, the resolution remains a question yet to be uncovered in the future studies as there is no specific scientific evidence to support this suggestion for the meantime. Can this be another factor associated with a diagnostic ECG falsely suggesting an acute myocardial infarction? This is just one of the questions waiting for an answer in this case.

Within the context of the WHO guidelines, the reported case did not suffice the criteria to be diagnosed as myocardial infarction. However out of such confusion, the opted initial management was that of an MI aimed to gain the 'benefit of the doubts'. Aspirin should be given to all patients who present with suspected myocardial infarction. This recommendation is based primarily on results of the Second International Study of Infarct Survival. [11] According to Collins; et.al anticoagulant therapy was useful among patients with suspected acute myocardial infarction who, in the past, had received neither aspirin nor fibrinolytic therapy. [12]

## CONCLUSION

It is crucial to make the diagnosis of acute myocardial infarction as quickly as possible but it is equally important however that the interpretation of ST segment elevation should always be made in the light of clinical history, examination findings, and previous ECG recordings (if available) should be given utmost consideration.

## ACKNOWLEDGEMENT

I would like to give a special acknowledgement to my beloved wife who helped in the preparation of the manuscript of this report.

## CORRESPONDENCE TO

Gamal Mohamed Mohamed El Sherbiny, MRCSED (A&E) Main Accident & Emergency Department #65 King Khalid University Hospital P.O. Box 7805, Riyadh 11472 Kingdom of Saudi Arabia E-mail: sherbiny64@hotmail.com

### References

1. Rozenman Y, Gotsman MS. The earliest diagnosis of acute myocardial infarction. Annu Rev Med 1994; 45:31-44.
2. Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined--a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol 2000; 36:959-69.
3. Zahger D, Hod H . A new definition for acute myocardial infarction .Harefuah 2004; 143:270-1, 318.
4. Kannel WB, Abbott RD. Incidence and prognosis of unrecognised myocardial infarction. An update on the Framingham Study. N Engl J Med 1984; 311:1144-1147.
5. Kannel WB. Silent myocardial ischemia and infarction:

insights from the Framingham Study. Cardiol Clin 1986; 4:583-501.
6. Spodick DH. "Decreased recognition of the post-myocardial infarction (Dressler) syndrome in the postinfarct setting: does it masquerade as "idiopathic pericarditis" following silent infarcts?" Chest 2004(5); 126: 1410-1.
7. Pope JH, Selker HP. Diagnosis of acute cardiac ischemia. Emerg Med Clin N Am .2003; 21:27-59.
8. Harrigan RA et al. Electrocardiographic manifestations: bundle branch blocks and fascicular blocks. The Journal of Emergency Medicine 2003(1); 25:67-77.
9. Rowlands DJ.Left and right bundle branch block, left anterior and left posterior hemiblock. Eur Heart J. 1984 Mar; 5 Suppl A: 99-105.
10. Fogoros,RN.URL:http://heartdisease.about.com/cs/arrhythmias/a/BBB_4.htm (July 9, 2006).
11. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. Lancet 1988; 2(8607):349-60.
12. Collins R, McMahon S, Flather M.et.al. Clinical effects of anticoagulant therapy in suspected acute myocardial infarction: systematic overview of randomised trials.BMJ 1996; 313:652-659

## Author Information

**Gamal Mohamed Mohamed El Sherbiny, MBBCH, MRCSED (A&E)**
Registrar, Main Accident and Emergency Department #65, King Khalid University Hospital