Original Article

# A Rare Case of Upper Back Pain as The Presenting Complaint of Acute Myocardial Infarction

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Abstract

**Introduction:** Acute upper back pain as one of the atypical symptoms of acute myocardial infarction (AMI) is more frequently encountered in women, elderly, diabetics, and patients with prior stroke or heart failure.1 Failure to recognize atypical clinical presentation of AMI conveys to delayed diagnosis, which are associated with increased morbidity and mortality.[2]

**Abstract :** Acute upper back pain as one of the atypical symptoms of acute myocardial infarction (AMI) is more frequently encountered in women, elderly and diabetics. Failure to recognize atypical clinical presentation of AMI conveys to delayed diagnosis, which are associated with increased morbidity and mortality. Herein we report a case of 46 yearsold male presenting with a sudden onset of severe acute upper back pain 6 hours prior to hospital admission. Diagnosis of AMI was delayed until 12 hours later after typical ischemic chest pain manifested and ECG reading showed evolution of ST-Elevation Myocardial Infarction (STEMI). Due to the atypical clinical presentation, diagnosis of AMI in this patient was delayed. Vigilant observation and low threshold for acute coronary syndrome (ACS) work-up are obligatory to prevent delayed diagnosis and management.

**Keywords: back pain, STEMI, atypical presentation, ACS, myocardial infarction**

**Abstrak :** Nyeri punggung atas adalah salah satu gejala atipikal dari infark miokard akut (IMA) yang lebih sering ditemukan pada perempuan, lanjut usia dan penderita diabetes. Kegagalan untuk mengenali presentasi atipik dari IMA menyebabkan telatnya diagnosis yang dihubungkan dengan meningkatnya mortalitas dan morbiditas. Dalam kasus ini kami melaporkan seorang laki-laki berusia 46 tahun datang dengan keluhan nyeri punggung atas yang berat dan mendadak sejak 6 jam sebelum masuk rumah sakit. Diagnosis IMA tertunda hingga 12 jam kemudian ketika nyeri dada tipikal dirasakan dan EKG menunjukan evolusi dari STEMI. Karena presentasi klinis yang atipikal, diagnosis IMA pada pasien ini tertunda. Pemantauan yang jeli danpemeriksaan lanjutanuntuk sindrom koroner akut (SKA) wajibdilaksanakan untuk mencegah tertundanya diagnosis dan tatalaksana yang sesuai.

**Kata Kunci:** nyeri punggung, STEMI, presentasi atipikal, SKA, infark miokard

## CASE ILLUSTRATION

A 46-year-old male came to Emergency Department with chief complaint of sudden onset of severe upper back pain since 6 hours before admission. Pain was present at rest (Scale 5/10) which worsened with activity and described as squeezing and constant in nature. Patient experienced 2 similar episodes with milder intensity which wasexacerbated with aerobic exertion in the past 5 months. Shortness of breath, cough, nausea, vomiting and fever was denied. Urination and defecation was as usual. Patient has past medical history of poorly controlled hypertension and dyspepsia syndrome. There was no past history of diabetes and heart disease. Patient smokes for around 20 years, 7 years ago patient quit smoking and became returned to active smoking for the past 2 months.

From physical examination Glasgow Coma Scale was 15, patient looked dyspenic with blood pressure of 150/100 mmHg, heart rate 77 beats/minute, respiratory rate 22 times/minute and SaO2 96%. Cardiorespiratory examination was within normal limit.

Laboratory examination showed leucocytosis (15,530/uL), elevated aspartate aminotransferase (AST, 70 IU/L) and alanine transaminase (ALT, 71 IU/L). Chest X-Ray (CXR) was within normal limit. Initial electrocardiography (ECG) was inconclusive with possibility of old myocardial infarction (Figure 1). Serial ECG at first and twelve hours after the onset (Figure 2 and 3) revealed evolution of ST segment morphology with increase in height of ST segment elevation and deepening of inverted T wave. With clear ECG evolution and development of typical chest pain 12 hours after onset, loading dose of aspirin 320 mg, clopidogrel 300 mg, statin, ISDN 5 mg sublingual and continuous infusion of unfractionated heparin was administered. Hence, the diagnosis and treatment of anterior ST elevation myocardial infarction (STEMI)Killip class I was delayed by 12 hours. Serial ECG at 44th and 80th (Figure 4 and 5) hours after onset showed deepening T wave inversion which returned closer to isoelectric line at 56th hour while posterior and right lead was within normal limit. ECG at 80$^{th}$ hour after onset showed T wave moves closely to isoelectric line withpersistent ST segment elevation that is atypical of STEMI. CK-MB and Troponin I was supposed to be measured at the initial presentation, however due to technical issues both were measured at 7$^{th}$ day after the onset of back pain, the result was 2.3 ng/mL and 3098 pg/mL respectively with the former was within normal limit and the latter was elevated. Close follow-up was required whether left ventricular aneurysm was formed as a result of anterior MI in this patient.

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## INTRODUCTION

Acute upper back pain as one of the atypical symptoms of acute myocardial infarction (AMI) is more frequently encountered in women, elderly, diabetics, and patients with prior stroke or heart failure.[1] Failure to recognize atypical clinical presentation of AMI conveys to delayed diagnosis, which are associated with increased morbidity and mortality.[2]

## CASE ILLUSTRATION

A 46-year-old male came to Emergency Department with chief complaint of sudden onset of severe upper back pain since 6 hours before admission. Pain was present at rest (Scale 5/10) which worsened with activity and described as squeezing and constant in nature. Patient experienced 2 similar episodes with milder intensity which wasexacerbated with aerobic exertion in the past 5 months. Shortness of breath, cough, nausea, vomiting and fever was denied. Urination and defecation was as usual. Patient has past medical history of poorly controlled hypertension and dyspepsia syndrome. There was no past history of diabetes and heart disease. Patient smokes for around 20 years, 7 years ago patient quit smoking and became returned to active smoking for the past 2 months.

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## DISCUSSION

Diagnosis of AMI in this patient was delayeddue to atypical clinical presentation. Failure in recognizing the symptoms of ACS was due to the atypical symptoms and the absence of established risk factors for atypical presentation of ACS. Acute back pain as the presenting complaint of AMI is rare, especially in those who are not female, elderly, diabetics, and without prior stroke or heart failure (**Table 1**).[1]In addition to rare atypical clinical presentation, uncharacteristic ECG hindered the diagnosis. Vague ECG coupled with suspicious recurrent similar episode leads to a decision to record serial ECG to anticipate possible evolution of ST segment and T wave that confirms myocardial ischemia/infarction. Initial ECG showed possible old anterior myocardial infarction with slight ST segment elevation, however evolution displayed by serial ECG had tendency towards acute condition. Evolution of ECG coupled with manifestation of typical chest pain at 12hours after onset establish the diagnosis of Anterior STEMI Killip Class I. Cardiac biomarkers were supposed to be measured at the initial presentation, however due to technical issues both were measured at 7$^{th}$ day after onset of back pain. Elevated Troponin with normal CK-MB level confirmed that the underlying pathology was AMI. There was possibility of acute aortic dissection as the primary diagnosis with coronary insufficiency as a complication, however the pain from aortic dissection was sharp in natureand will deteriorate with antithrombotic and anticoagulation that this patient received for AMI.

Delay and failure in the recognition of AMI is particularly important because early therapy in these patients is imperative. Studies showed that patients with atypical presentation of acute coronary syndrome (ACS) were less likely to be initially diagnosed with myocardial infarction and consequently, were less likely to receive optimal medical therapy and revascularization with either primary percutaneous coronary intervention (PCI) or thrombolytic therapy.[2,3] A cohort retrospective study by Na JP, et al showed patient with atypical presentation had longer symptom onset to ED arrival with delayed in door-to-ECG time (median 2,0 minutes vs. 5,0 minutes; p<0,001) and door-to-balloon time (median 57,5 minutes vs. 65,0

minutes; p<0,001) although door-to-ECG *time* and *door-to-balloon time* was not associated with in hospital mortality.[2,3,4]A study by Na JP, et al demonstrated that non-chest pain presentation was independent predictor of in hospital mortality (odds ratio 2,3; 95% confidence interval 1,1-4,7), a study by El-Menyar added the evidence by stating that absence of typical chest pain was associated with higher mortality rate (Odds Ratio 2,0, 95% confidence intervals 1,29-2,75) and study by Hwang SY, et al reinforced both by stating that AMI patients with atypical symptoms had higher 1-year mortality compared to those with typical symptoms (Hazard Ratio 3,288, 95% confidence interval 1,087-,9,943, p=0,035). Thus, unsurprisingly higher in-hospital mortality, 1- year mortality and rate of complications were seen in the atypical groups.

## CONCLUSION

Physician requires high sense of clinical suspicion in order to recognize atypical clinical presentation and followed by ACS workup to establish diagnosis leading to early treatment, reducing risk of mortality and morbidity. Vigilant observation and low threshold for ACS work-up are obligatory to prevent delayed diagnosis and management.

## REFERENCES


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| Variable | Odds Ratio (95% Confidence Interval) |
|---|---|
| Nonwhite | 1.05 (1.03-1.07) |
| Female | 1.06 (1.04-1.08) |
| Diabetes Mellitus | 1.21 (1.19-1.23) |
| Age (10 years' interval) | 1.28 (1.26-1.28) |
| Prior Stroke | 1.43 (1.40-1.47) |
| Prior Heart Failure | 1.77 (1.74-1.81) |

**Table 1.** Independent risk factors for atypical presentation.Adapted from: Kim HK, Jeong MH.Atypical Presentation in Patients with Acute Coronary Syndrome, Acute Coronary Syndromes, Dr. Mariano Brizzio (Ed.), InTech, 2017.

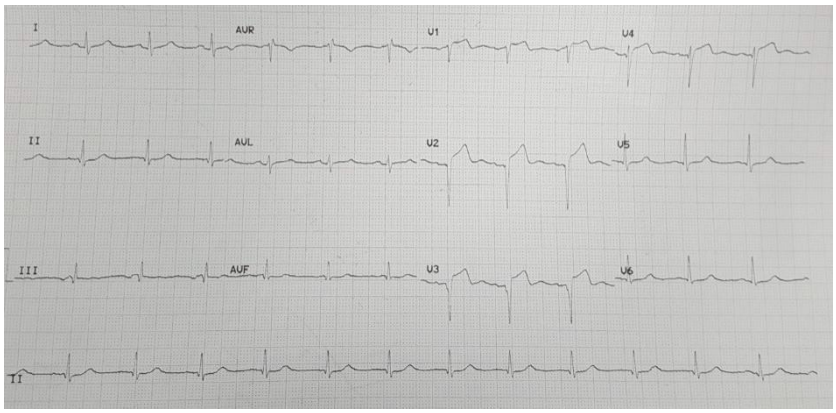


**Figure 1**.Initial ECG

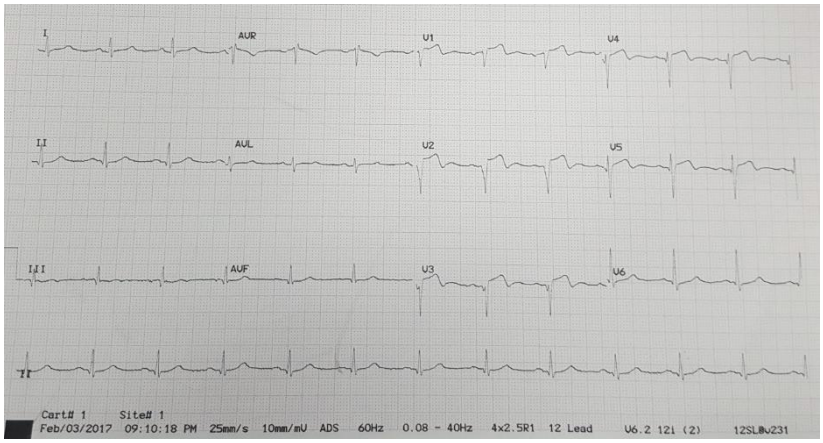


**Figure 2**.One-hour ECG

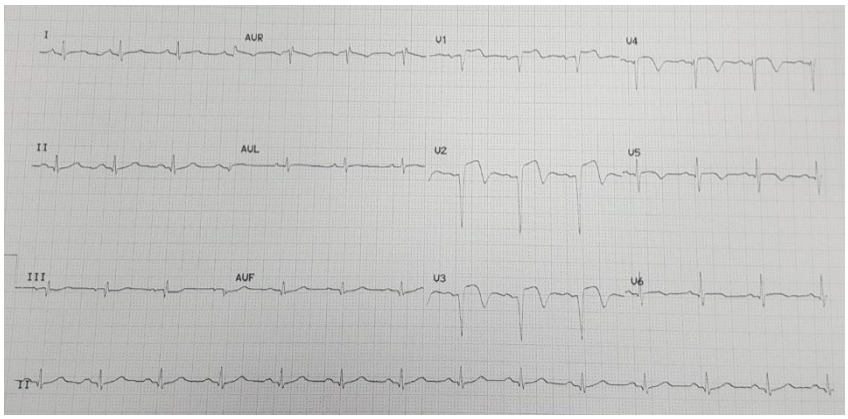


**Figure 3**.12-hour ECG

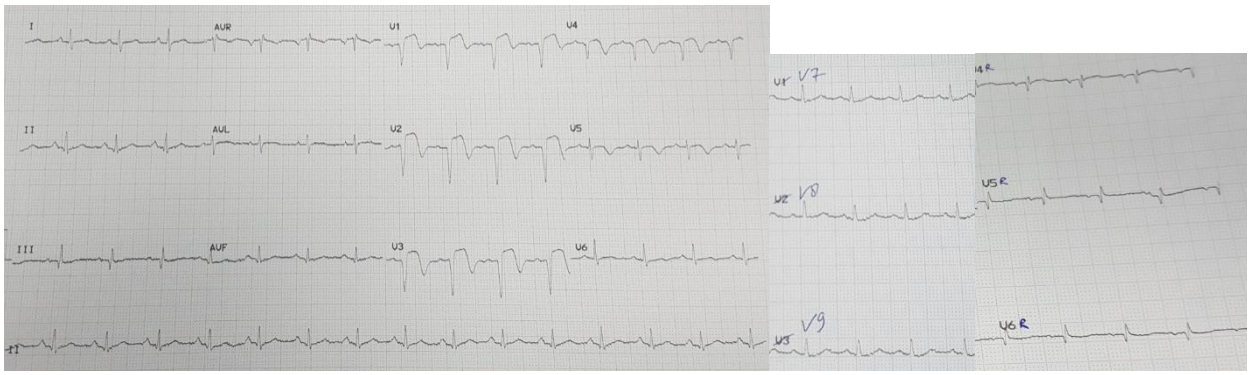


**Figure 4.**44-hour ECG (Includes right leads and Posterior leads)

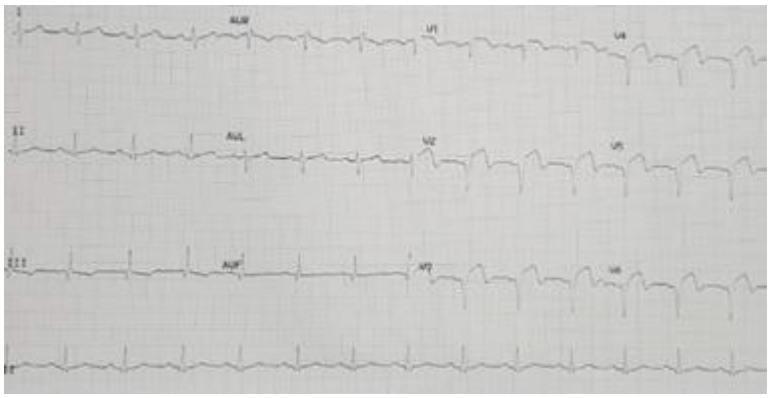

**Figure 5**.80-hour ECG