

Diagnostic and therapeutic dilemma due to new pattern electrocardiographic change in myocardial infarction: a case report from a remote island

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Abstract

Acute myocardial infarction results due to acute disruption of coronary blood flow, commonly presented with chest pain. Electrocardiogram showing ST-segment elevation in consecutive leads is a classical ST-elevation myocardial infarction; however, atypical ST-segment elevation also can occur. We report an elderly lady who presented to the island health centre with intractable central chest pain. Electrocardiogram showed ST-segment elevation in V1 and aVR; whereas ST-depression in V4-6. With primary management, she was referred to a regional hospital. Repeat electrocardiogram showed ST-segment elevation in V1, aVR, and III; whereas ST-depression in V4-6, aVL, and I. Therefore, considering Aslanger's pattern of ECG change myocardial infarction, she was referred to the cardiac centre where coronary angiography revealed severe diffuse triple vessel disease. Primary PCI was performed on the right coronary artery initially as it is the primary culprit vessel with stenosis of other vessels. Therefore, staged angioplasty was performed on the left coronary system. Uncommon and atypical electrocardiographic presentations may be encountered in daily practice, associated with severe multiple-vessel diseases. So, it can be challenging for a cardiologist as well during interventions.

Keywords: Acute coronary syndrome, Aslanger pattern, Electrocardiography, Myocardial infarction

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Introduction

Acute coronary syndrome (ACS) which includes the spectrum of ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina (UA), usually results from the acute disruption of coronary blood flow, commonly presented to the emergency department with classical chest pain or sometimes with atypical symptoms. Electrocardiography revealing ST segments elevation on consecutive leads is a classical ST elevation myocardial infarction.^{1,2} Discordance ST-segment changes leads to diagnostic dilemma whether to consider acute myocardial infarction or not; and it is challenging in low resources setting like in primary health centre (island) or where there is unavailability of cardiologist service.

Aslanger et al (2020)³ revealed a specific ECG pattern ST-segment elevation in lead III that does not display contiguous ST-segment elevation in lead II and aVF, and ST-elevation in aVR and V1 >V2 with ST-depression in V4-6 indicates acute inferior myocardial infarction associated with concomitant critical stenosis on the other coronary arteries and has a multi-vessel disease and higher mortality.

We are reporting a 64-year-old female case with discordance ST elevation on ECG, challenging for a doctor working on a remote

island where there is an unavailability cardiologist or even an internist.

Case Report

We report a 64 years old lady who presented to the health centre (HC) with the chief complaints of severe central chest pain, associated with numbness and radiation to the left shoulder and arm for 20 minutes durations. She gives a history of similar chest pain non-radiating for one month, aggravated by exertion and relieved by rest. She also complained of chest discomfort, belching, burping, and taking pantoprazole daily for it. She was a known case of diabetes mellitus, hypertension, and dyslipidemia on regular medications with an optimum dose of glimepiride, metformin, sitagliptin, atorvastatin, amlodipine, and aspirin 75mg daily. She had a history of hypothyroidism, currently not on medication. Her blood pressure was 160/98mmHg, pulse rate was 100 beats/min, otherwise normal respiratory rate and oxygen saturation of 98% at room air. Initially, intravenous pantoprazole 40mg and paracetamol 1gm were given stat as acute pain management.

Electrocardiography showed sinus rhythm of heart rate 91per minute, normal axis, ST-segment elevation in V1-2 and aVR, and ST-segment depression in leads- V4-6, aVL, I, II, III, aVF (Figure

1). The patient was suspected of ACS and a loading dose of aspirin 300mg, clopidogrel 300mg, atorvastatin 80mg, ramipril 2.5mg and metoprolol 25mg oral stat were given after consultation with an internist in the nearby referring hospital and processed for transfer. CK-MB and troponin I and T were negative in HC. At reaching the nearby regional hospital, her chest pain was minimal and non-radiating, and her vitals were stable with the normal systemic examination; however, mild tenderness was present over the upper sternum and 2nd and 3rd ribs. Repeat ECG revealed inconsecutive ST-segment elevation in leads aVR, V1, and III; whereas ST-segment depression in leads V4-6, aVL, and I; and normal in aVF and II (Figure 2). Cardiac enzymes were repeated and demonstrated weakly positive troponin I (0.58ng/ml) and positive CK-MB (31 U/L), CK of 312 IU/L, random blood sugar of 245mg/dl and HbA1C of 8.8%; otherwise, normal renal function test, AST and LDH. Therefore, the case was discussed with an on-call cardiologist- at the cardiac centre (National cardiac centre, Male) regarding ECG changes and further management, and then the case was transferred to the coronary care unit of the National cardiac centre for coronary

angiography and primary coronary intervention within 24 hours duration of the presentation. Echocardiography showed regional wall motion abnormality segment and mild left ventricular systolic dysfunction, with an ejection fraction of 47%. Further, coronary angiography revealed severe diffuse triple vessels disease: (i) right coronary artery (RCA)- dominant vessel, proximal to mid-RCA diffuse disease with maximum 80-90% stenosis), (ii) left anterior descending artery (LAD)- diffuse disease, proximal to mid LAD diffuse stenosis maximum of 80-90%, major diagonal and major septal arise from diseased segments, mid to distal LAD had diffuse disease with a maximum of 80% stenosis, diffuse disease of distal LAD with maximum 90% stenosis), and (iii) left circumflex artery (LCX)- non-dominant vessel, proximal to distal LCx is diffusely disease with maximum 80% stenosis, major OM1 and OM2 arises from diseased segments with ectasia. Initially, primary coronary intervention (PCI) to RCA was performed with 3.0 x 48mm Xience Xpedition 48 (Figure 3). Followed by staged PCI after 3 weeks to LCX with 2.75 x 23mm Xience Xpedition, and LAD with 2.75 x 33mm Xience Xpedition LL after 3 weeks. The patient was asymptomatic on 3 monthly follow-ups to the regional centre.

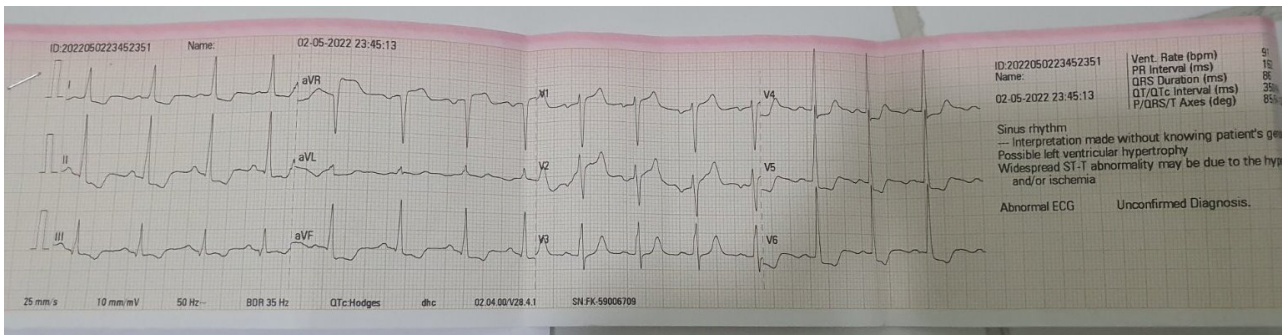


Figure 1. Electrocardiogram at presentation in the health centre showing ST-segment elevation in aVR and V1; and ST-segment depression in precordial leads V4-6; and leads II, III, aVF, and I.

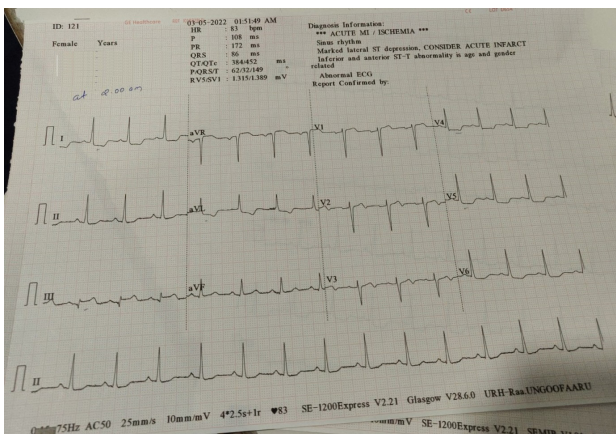


Figure 2. Repeat electrocardiogram at the referral regional hospital after 2.30 hours of chest pain showing ST-segment elevation in lead III, aVR, and V1; and ST-segment depression in V4-6, I, and aVL which was similar to Aslanger's pattern of ECG.



Figure 3: A and B: Angiography showing proximal to mid-RCA diffuse disease with maximum 80-90% stenosis; C: stenting in RCA with 3.0 x 48mm Xience Xpedition 48, & D: showing RCA post angioplasty result.

Discussion

Acute coronary syndrome (ACS) results from acute disruption of coronary blood flow due to occlusion or near occlusion of coronary arteries leading to myocardial ischemia or infarction.⁴ 12 leads ECG is a very important investigation to rule out myocardial infarction and can be categorized as ST-segment elevation MI (STEMI) or non-STEMI based on the presence or absence of ST elevation in 2 or more contiguous. STEMI is defined as new ST-elevation at the J-point in contiguous leads with a cut-point ≥ 1 mm in all leads other than V_2 - V_3 where the following cut-points apply ≥ 2 mm in men ≥ 40 years; ≥ 2.5 mm in men < 40 years, or ≥ 1.5 mm in women regardless of age.^{5,6} However, there is not always a typical presentation that follows standard criteria for ST-segment elevation myocardial infarction. Among them, the Aslanger pattern ECG change is the one. Aslanger et al (2020)³ revealed a specific ECG pattern that did not display contiguous ST-segment elevation attributing acute inferior myocardial infarction with concomitant critical stenosis in the other coronary arteries as well, and the pattern was associated with multi-vessel disease and higher mortality compared to inferior myocardial infarction only. This pattern was observed in 6.3% of the non-STEMI cohort whereas 0.5% of no-MI patients. It has 3 criteria: (i) unconsecutive ST-segment elevation in lead III among inferior leads, and others in aVR, and V1; (ii) concomitant ST-segment depression in any of V4-6 with positive/terminally positive T wave; and (iii) ST-segment elevation in V1 more than V2. Isolated ST-segment elevation in lead III may associate with inferior wall infarction with severe stenosis in non-infarct related arteries.^{2,3,5,7} In our case, initially, ST depression was noticed in lead III whereas ST-segment elevation in aVR, V2 more than V1, ST-segment depression in lateral leads. Repeat ECG showed ST-segment elevation in lead III, aVR, and V1-2 along with positive cardiac troponin I and CK-MB. Subsequently, coronary angiography revealed severe triple vessel disease- occlusion of the right coronary artery, left anterior descending and left circumflex artery.

The strength of our case report is that we were able to pick the case early once presented in the health centre at midnight as the case was consulted to a nearby referral hospital (URH) for physician opinion regarding the probability of ACS and referred for further management despite of negative cardiac enzymes (CK-MB and troponin I and T) levels. Moreover, raised cardiac enzymes (CK-MB and troponin I) with ST elevation in lead III along with V1 and aVR compared to earlier ECG, besides ST depression in lateral leads, indicated the possibility of acute STE myocardial infarction, and the case was discussed with the on-call cardiologist of cardiac centre (National cardiac centre- IGMH). Then, the case was transferred to the coronary care unit and subsequently coronary angiography and PCI were performed within the window period. Additionally, the primary culprit vessel was identified and opened; otherwise, a dilemma may lie in which vessels to open first.

There were some limitations though we were able to perform angioplasty within 24hrs of the presentation. The case could have been directly transferred to a cardiology centre for early angiography and angioplasty within 12hrs for a better outcome; however, dilemma in ACS versus no ACS and the insurance system of the country, the case was initially transferred to a nearby regional hospital and then finally transferred to the cardiology centre.

Conclusion

Acute coronary syndrome cases may present with atypical ECG changes, sometimes discordance ST-segment elevation as described as Aslanger ECG pattern changes in our daily practice. So, the primary physicians need to get updated regarding atypical ECG presentation, and the new pattern of changes which has to be correlated clinically with cardiac enzymes. Furthermore, this might be challenging to an interventional cardiologist as well regarding decision-making in which vessel is the primary culprit vessel and to be open first.

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Conflict of Interest

The Authors declare no conflicts of interest regarding the publication of this paper

References

1. Di Toro M, Weissbacher S, Wakeling J, et al. The de Winter electrocardiogram pattern in a 52-year-old-male: a case report. *Eur Heart J Case Rep.* 2020;4:1. <https://doi.org/10.1093/ehjcr/ytaa321>
2. Zhang Q, Zhao Y, Huang X. Acute inferior occlusion myocardial infarction with a solitary ST-elevation in lead III: A case report. *J Electrocardiol.* 2022;72:35-38. <https://doi.org/10.1016/j.jelectrocard.2022.02.010>
3. Aslanger E, Yıldırım Türk Ö, Şimşek B, Sungur A, Cabber AT, Bozbeyoğlu E, Karabay CY, Smith SW, Degertekin M. A new electrocardiographic pattern indicating inferior myocardial infarction. *J Electrocardiol* 2020;61:41-46. <https://doi.org/10.1016/j.jelectrocard.2020.04.008>
4. Aslanger EK, Meyers HP, Smith SW. Time for a new paradigm shift in myocardial infarction. *Anatol J Cardiol.* 2021;25:156. DOI: 10.5152/AnatolJCardiol.2021.89304
5. <https://clincasequest.hospital/aslangers-pattern/>.
6. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, White HD, Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth universal definition of myocardial infarction (2018). *Circulation* 2018;138:e618-e651. <https://doi.org/10.1161/CIR.0000000000000617>
7. Aslanger EK, Meyers PH, Smith SW. STEMI: A transitional fossil in MI classification? *J Electrocardiol.* 2021;65:163-169. <https://doi.org/10.1016/j.jelectrocard.2021.02.001>