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Case Report

Unveiling The Diagnosis Pitfall: Complete Heart Block Presenting in Acute Coronary Syndromes Without Chest Pain and Non-Ischemic Electrocardiography in a Young Adult

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ABSTRACT

Background: The majority of studies indicate that the asymptomatic or atypical presentation of acute coronary syndrome (ACS) as a significant manifestation of coronary artery disease (CAD) is associated with a poor prognosis. The purpose of this study was to characterize the clinical characteristics and management of ACS patients who presented without chest pain and non-ischemic electrocardiogram (ECG).
Case Illustration: A 31 years old man with syncope was brought to our hospital. ECG revealed total AV block (TAVB) without ST segment changes. First, he was diagnosed with cardiac syncope because his ECG showed no ischemic sign. He had a strong familial history of CAD, so we examined his cardiac enzymes and found an increase in serial cardiac enzymes. Then, he was diagnosed with non-ST elevation myocardial infarction (NSTEMI). Early invasive strategy with chronic total occlusion (CTO) at osteal right coronary artery (RCA) and acute total occlusion at mid RCA with implantation of 1 DES at osteal-proximal RCA was selected for this patient. After five days of hospitalization, he discharged home with medicines.
Conclusion: In order to significantly lower their morbidity and mortality, this group of high-risk patients needs to receive improved early diagnostic and treatment choices.

1. Introduction

Acute coronary syndrome (ACS) has a heterogeneous clinical presentations, thus, poses a diagnostic, therapeutic, and prognostic challenge.[1] Silent or atypically presenting myocardial infarction (MI) has been linked with a poor prognosis. These undesirable results could be attributed to a failure to appropriately identify the patient and fail to implement efficient treatment plans, in addition to unfavourable patient outcomes.[2,3] Patients with ACS with no chest pain are commonly misdiagnosed and undertreated. These patients had higher rates of morbidity and mortality across the ACS spectrum.[1]

Patients with ACS who reported with a non-ischemic electrocardiogram (ECG) and no chest pain presented with a challenge. An unfavourable result became more likely. 22% of ACS patients in the emergency room had normal ECGs. Clinicians must be aware of these conditions in order to accurately identify patients and determine the best course of treatment for an improved prognosis.[4] Our case highlighted the need to increase awareness of patients with atypical presentation and non-ischemic ECG, which was a diagnostic pitfall in young adult ACS patients, in order to improve prognosis in this patient population.

2. Case Illustration

A 31-year-old man was brought to Saiful Anwar General Hospital on February 26th, 2023, after experiencing syncope 1.5 hours prior to admission. The syncope lasted approximately 20 minutes and was triggered by activity (playing badminton). It was preceded by chest discomfort, dizziness, and vision impairment. There was no convulsion. After spontaneously regaining consciousness, he was fully awake, but he felt lethargic and continued to experience chest discomfort. Chest discomfort did not radiate to the back or left arm and was not accompanied by cold sweating, shortness of breath, nausea, or vomiting. There was no stabbing or tearing like sensation, and the chest discomfort was unrelated to postural changes, respiration movement, or a meal. Prior to and following the onset of syncope, there were no audible noises, flashing lights, paresthesia, seizures, or motoric disorders.

His daily activities as a merchant were not restricted in any way. Previously, he denied similar complaints. He was born aterm without a history of cyanosis or bluishness at birth. His growth and development were comparable to those of his peers. He denied having a history of hypertension, diabetes, and other illnesses. He was an ex-smoker for over a decade, consuming 1-2 packs per day, and ceased smoking six years ago. A history of regular drugs use was denied. His father was 45 years old when he was diagnosed with acute coronary

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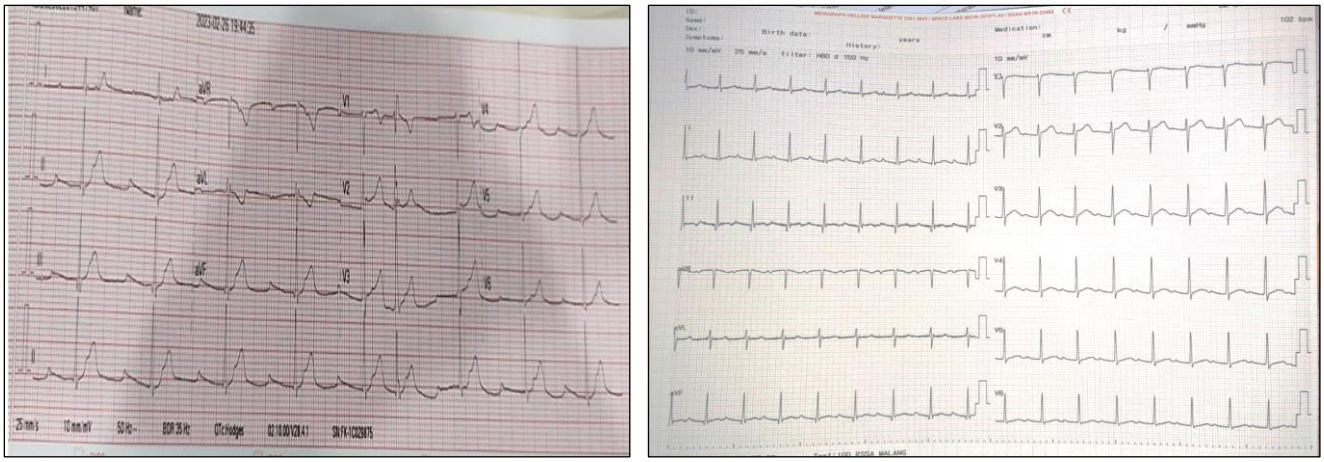


(A) (B)

Figure 1. (A) First ECG performed at our hospital showed total av block (TAVB) with atrial rate 100 bpm, ventricular rate 50 bpm without st segment changes. (B) Second ECG performed at our hospital after be given sulfas atropine showed sinus ryhthm with 1st degree av block without ST-segment changes

syndrome (ACS), and 67 years old when he underwent coronary artery bypass graft (CABG). Young sudden cardiac deaths claimed the lives of his grandmother and uncle. On physical examination, it was determined that the patient had a blood pressure (BP) of 102/60 mmHg without support, a heart rate (HR) of 49 bpm, a respiratory rate (RR) of 20 tpm, a temperature (T) of 36.5 degrees celsius, and a saturation (SpO2) of 99 percent on a nasal cannula at a rate of 3 liters per minute (lpm) with normal urination.

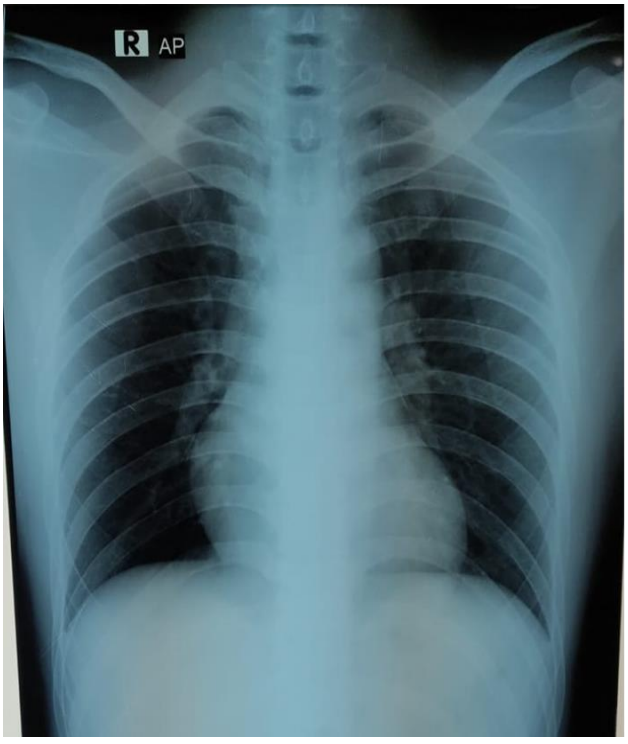


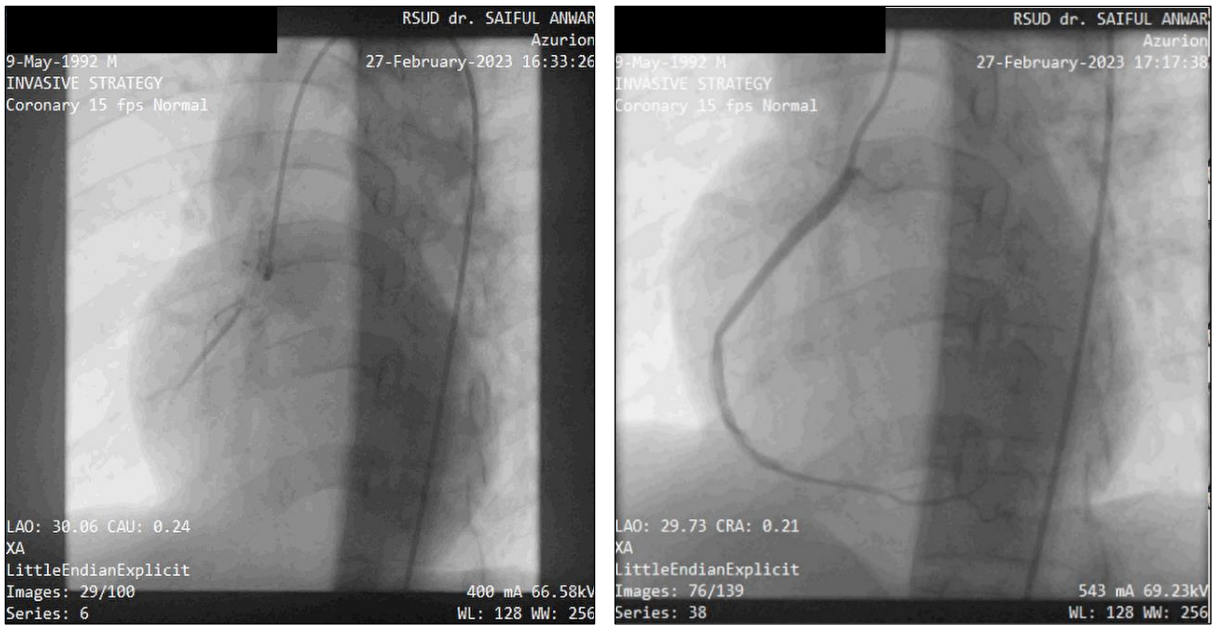
Figure 2. Thoracic antero-posterior (AP) X-ray imaging showed within normal limit

The jugular venous pressure was R+3cm H_20. Auscultation with palpable apex cordis at intercostal space (ICS) V did not reveal evidence of heart enlargement. There were no rhonchi at bilateral pulmonary. Warm acral was discovered in the extremities. As shown in Figure 1A, the electrocardiogram (ECG) revealed a total av block (TAVB) with an atrial rate of 100 bpm and a ventricular rate of 50 bpm without ST segment changes. The patient was then administered 0.5 mg of sulfas atropine intravenously (IV), and an ECG serial revealed sinus rhythm with 1st degree av block and no ST segment changes (Figure 1B). As seen in Figure 2, cor and pulmo were normal upon X-ray examination.

Laboratory examination at our ER showed increase of leucocyte (19880/μL), normal hemoglobin (13.8 g/dL), platelets (214000/uL), ureum (37 mg/dL), increase of creatinine (1.58 mg/dL), sodium (138 mmol/L), potassium (3.91 mmol/L), chloride (114 mmol/L), increase of aspartate transaminase (74 U/L), increase of alanine aminotransferase (59 U/L), slightly increase of 1st troponin I (1.1 Ug/L) and increase of 2nd troponin I (10.4 Ug/L). All cardiac chambers had normal dimensions, normal left ventricular ejection fraction (LVEF) of 69%, dysfunctional left ventricular diastolic function, and global normokinetics, as determined by two-dimensional echocardiography. At first, we diagnosed patient with cardiac syncope due to TAVB because of there was no typical chest pain and ischemia sign from the ECG. Then, we found increase of serial cardiac enzyme and diagnosed patient with non-ST elevation myocardial infarction (NSTEMI). Patient then performed early invasive strategy.

Right femoral artery was used to perform the diagnostic coronary angiography (DCA) which showed normal left main (LM), diffuse stenosis from proximal until mid left anterior descending (LAD) with maximal stenosis 90% at proximal, normal left circumflex (LCx), chronic total occlusion (CTO) 100% at osteal right coronary artery (RCA) which get collateral from ipsilateral, distal LCx and distal LAD, acute total occlusion at mid RCA which thrombus appearance and fistula coronary from RCA into cavum RV. We decided the RCA as the target vessel.

We successfully engaged the guiding catheter amplatz left (AL) 0.75 6F into osteal RCA. We used a Combo Plus 4.0x28mm drug-eluting stent (DES) from the osteal until proximal RCA. Thrombolysis in myocardial infarction (TIMI) flow 3 was seen on cineangiography, with no residual stenosis, and the treatment was completed without complications, as seen in Figure 3. The patient was discharged after 5 days of care with therapy aspirin 80mg once daily, ticagrelor 90mg twice daily, atorvastatin 40mg once daily, ramipril 5mg once daily and bisoprolol 2.5mg once daily.



(A) (B)

Figure 3. (A) Diagnostic coroangiography showed CTO 100% at osteal RCA and acute total occlusion at mid RCA which thrombus appearance (B) Cineangiography evaluation after implantation 1 DES at osteal-proximal RCA with TIMI flow 3 and residual stenosis 0%

3. Discussion

This case is intended to illustrate the clinical characteristics of patients with ACS who present without chest pain and non-ischemic electrocardiography (ECG) as well as their management. A 31-year-old male presented with cardiac syncope, chest discomfort, and total AV block (TAVB) without an ischemic pattern on his electrocardiogram. We should be aware of this diagnostic pitfall because a pattern of increasing cardiac enzymes was observed serially. Then, for the final management, he was performed an invasive strategy.

According to Figure 4, the rupture of a lipid-rich plaque triggered by inflammation and followed by a platelet-rich thrombosis accounts for about 64% of ACS cases. A STEMI happens when thrombosis totally obstructs a vessel. NSTEMI typically happens when the thrombus is not occlusive. It is critical to understand that complete vascular occlusion is not necessarily the cause of ST-segment elevation. Particularly, vessels supplying the left ventricular myocardium's lateral wall, like the left circumflex artery or a significant diagonal branch of the left anterior descending artery, may result in less obvious alterations, including ST-segment depression.[5]

Patients frequently present with symptoms other than chest pain when they have ACS. On presentation, approximately 25% of patients were not diagnosed with ACS. Patients with suspected ACS who presented to the hospital were stratified based on whether their predominant presenting symptoms were typical or atypical chest pain.[1] Canto JG et al. defined typical chest pain as (1) pain located substernally in the left or right chest, or (2) pain characterised by squeezing, tightness, arm discomfort, crushing, dullness, aching, heaviness, pressure, fullness or pain exacerbated by exercise and relieved by rest or nitroglycerin.[6]

In ACS, atypical chest pain is the absence of typical chest pain. Dyspnea (69.4%), vertigo (37.7%), diaphoresis (25.2%), discomfort in the arms (11.5%), syncope (10.6%), epigastrium (8.1%), shoulder (7.4%), or neck (5.9%) were the most common symptoms of atypical chest pain.[6] According to National Registry of Myocardial Infarction statistics, one-third of approximately 450 000 individuals with confirmed MI did not report with chest symptoms and were therefore more likely to receive less aggressive medical therapy.[7]

Female gender, advanced age, absence of a history of MI or hypercholesterolemia, dementia, absence of a family history of cardiovascular disease,stroke, history of heart failure, and diabetes mellitus were all important clinical characteristics associated with the absence of chest pain in patients with MI. Delay or failure to recognise ACS might lead to myocardial infarction or death. Improved detection of patients with unusual chest symptoms may lessen the liability associated with a missed or inaccurate diagnosis.[8] Diagnosis and therapy can be accelerated by being aware of the reasons of atypical chest pain. The diagnosis is frequently made too late, and both the initial and follow-up hospital care are insufficient. This patient type needs to receive improved early diagnostic and treatment choices to lower their high morbidity and mortality, therefore emergency department and coronary care staff must be particularly aware of them.[1]

The ECG is essential for assessing any patient suspected of having ACS because it offers a rapid, low-cost, and simple approach to identify patients with ST segment changes who are likely to benefit from hospitalisation.[9] However some patients with ACS and a normal or nondiagnostic ECG can also be at risk for a unfavourable outcome. With a normal or non-diagnostic ECG, biochemical cardiac markers, particularly troponins, can detect high-risk patients. It is practical to perform tests on all individuals who have a normal or non-diagnostic ECG for biochemical cardiac markers. Finding any particular clinical traits that might be utilised to choose patients for further investigation would be beneficial.[9]

The following standards were normal for an ECG: An HR between 55 and 105 beats per minute, a normal QRS interval and ST segment, and a typical T-wave shape or flattening are all characteristics of a normal sinus rhythm. Pathologic Q waves, left ventricular hypertrophy, generalised ST-T wave anomalies, any ST depression, and differences in T wave and QRS axis are disregarded from normal.[10] A normal ECG was paradoxically one of the four characteristics that were independently related with missed ACS in the ED, according to Pope et al.[8] Singer et al. assessed the efficacy of a normal ECG in ruling out AMI in patients with delayed ACS in the ED, concluding that a normal ECG did not rule out AMI in patients with delayed ACS in the ED, regardless of the length of symptoms from onset to presentation.[11]

As previously observed by McCarthy et al., a normal ECG does not rule out ACS, as around 5% of patients discharged from the emergency care with a normal ECG experienced an acute ischemic episode.[4] Risk stratification is a critical step in the therapy and prognosis of patients with Acute Coronary Syndrome (ACS). Over the last decade, various risk scores and factors have been published in the scientific literature in an effort to increase the precision of therapeutic choices. ECG has long been used to gain an electrical understanding of an ACS.[4]

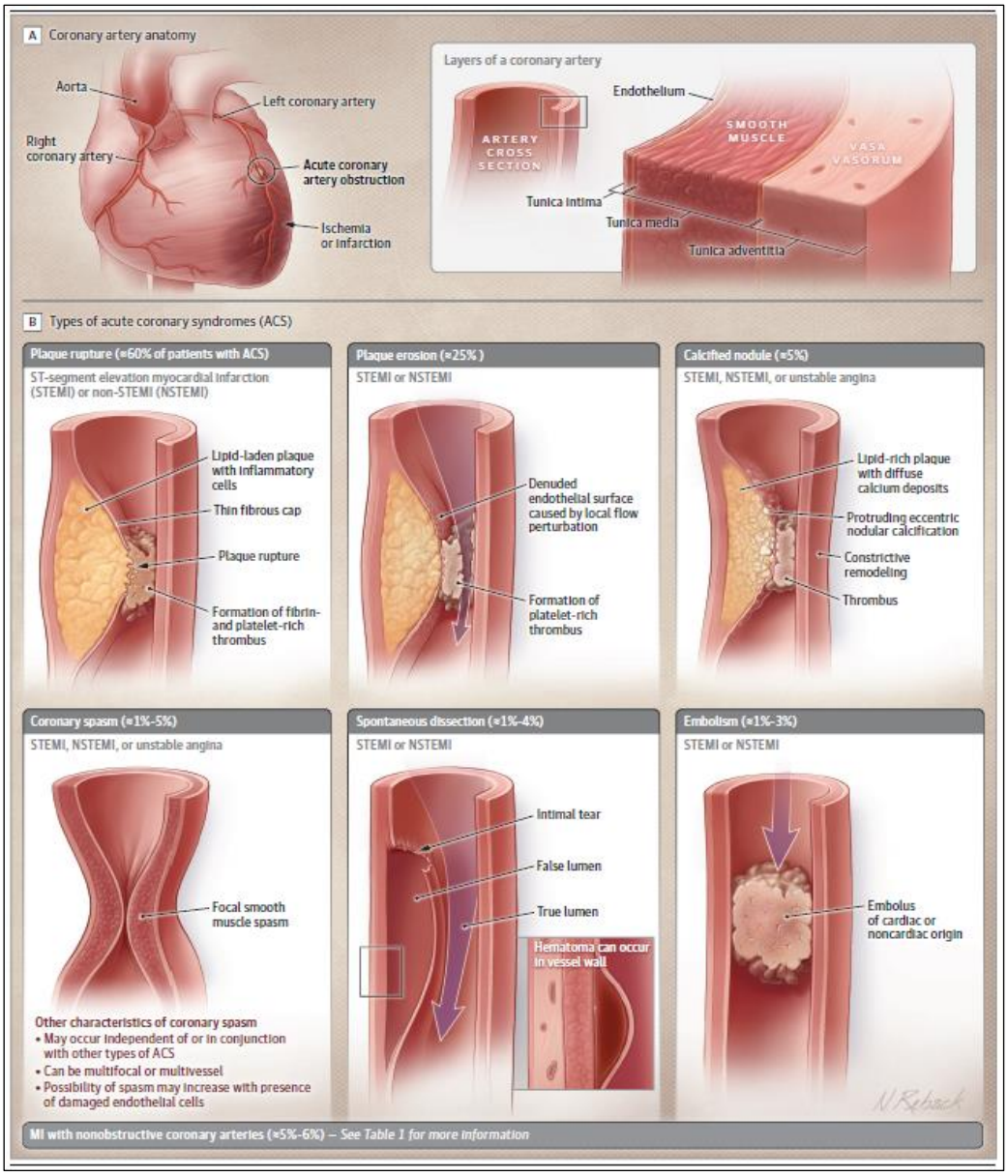


Figure 4. Etiologies of Acute Coronary Syndromes[5]

STEMI = ST-segment elevation myocardial infarction; NSTEMI = non ST-segment elevation myocardial infarction; ACS = Acute coronary syndromes

22% of emergency department patients with a normal ECG will have ACS, necessitating a careful clinical and biochemical characterization, since a normal ECG may not be such a benign finding after all. We consider that ischemia changes are dynamic and that, for logistical reasons, they may have been ignored in our group of patients with normal ECGs; hence, the 22% of patients may be overrepresented.[4]

Decisions on risk are made during admission, hospitalisation, and even in the catheterization lab. Risk assessment involves a multi-step temporal analysis. One of the most important decision-making stages occurs in the emergency room, influencing not only the diagnostic evaluation but also aggressive medical treatment and the decision to employ an invasive strategy. A patient should be rationally "understood" on a global scale.[4]

Patients with AMI who had a normal or nonspecific ECG typically had evaluation later, showed less symptoms diagnostic of AMI, and had to wait longer for an initial ECG. The researchers came to the conclusion that AMI patients do not necessarily have a good hospital outcome even if their initial ECG is benign.[12] In a recent study using cardiac troponin, Chase et al. found a 2.8% incidence of AMI in emergency department patients with a normal or non-specifically changed ECG.[13] The prevalence of ACS has increased among younger age groups. For instance, between 1995 and 2014, there was a 27% to 32% increase in the proportion of patients aged 35 to 54 who were hospitalised for an acute MI (30% to 33% for men and 21% to 31% for women).[14]

Current clinical practise guidelines that electrocardiography be performed within 10 minutes of arrival to the emergency room when ACS is suspected, as shown in Figure 5. To confirm the diagnosis of STEMI and begin PCI treatment, urgent invasive coronary angiography should be done within two hours if ST segment elevation is observed. In the absence of significant ST-segment elevations on the ECG, T-wave inversions (11.7%), ST-segment depressions (31.3%), both (15.7%), or neither (41.2%) are linked to ACS.[5]

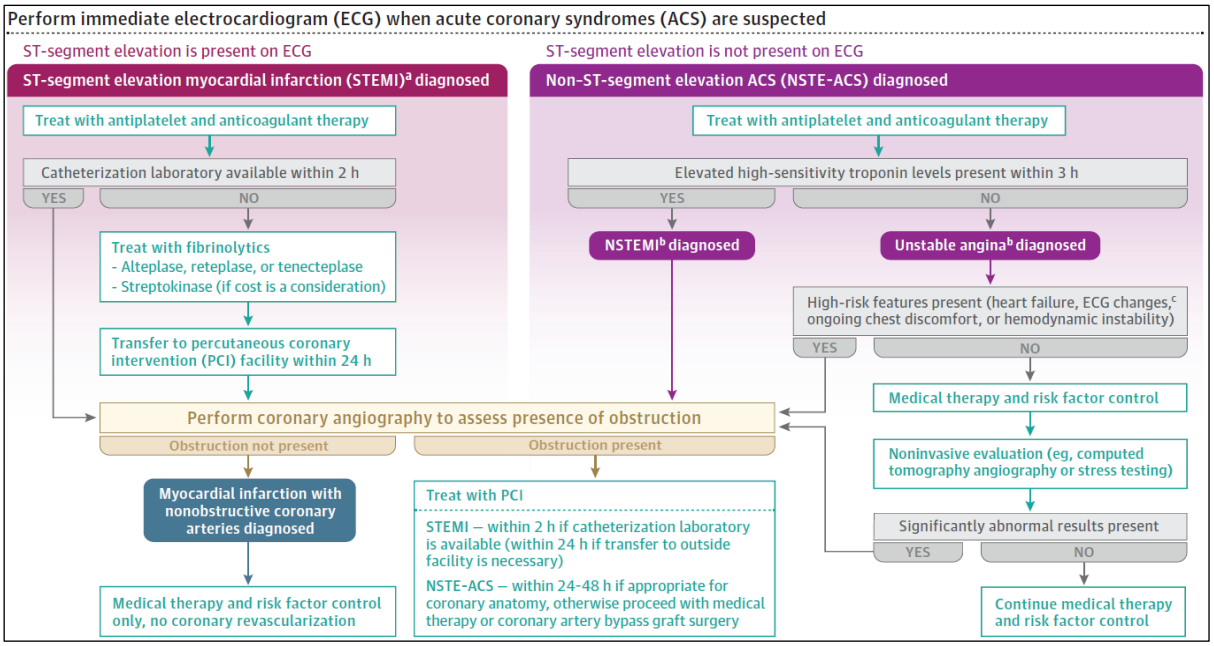


Figure 5. Initial Diagnosis and Management of Acute Coronary Syndromes[5]
STEMI = ST-segment elevation myocardial infarction; NSTEACS = non ST-segment elevation acute coronary syndromes; ACS = Acute coronary syndromes; ECG = Electrocardiography, PCI = Percutaneous coronary intervention

Cardiac troponin T or I testing needs to be done promptly when a patient exhibits symptoms of ACS but no ST-segment elevation. With a negative predictive value of about 99%, a second normal high-sensitivity troponin measurement within three hours can rule out ACS if the first one is normal. However, algorithms that suggest testing at presentation and again one or two hours later could be recommended.[5]

Our patient presents with cardiac syncope. Syncope is defined as a transitory loss of consciousness (TLOC) with a rapid start, short duration, and spontaneous recovery that is brought on by cerebral hypoperfusion. There are many differential diagnoses for syncope because it shares many clinical features with various diseases.

This group of circumstances is known as TLOC.[15] The most important factor in predicting outcomes and risk stratifying patients who experience syncope has been found to be the presence of cardiac disease. To lower the risk of mortality and sudden death, patients experiencing syncope should undergo examination, be given a diagnosis, and receive treatment for any underlying structural heart problems.[16]

Figure 6 shows a current recommendation for the evaluation of syncope from the European Society of Cardiology (ESC) Guidelines on Management of Syncope. The initial clinical assessment is essential for the therapy of syncope patients. Patients who are at a high risk of cardiac syncope can be classified using their existence of structural heart disease or an irregular ECG. Patients with structural heart disease or an abnormal ECG should have a hearts examination. Syncope patients are managed according to the underlying cause of the condition.[16]

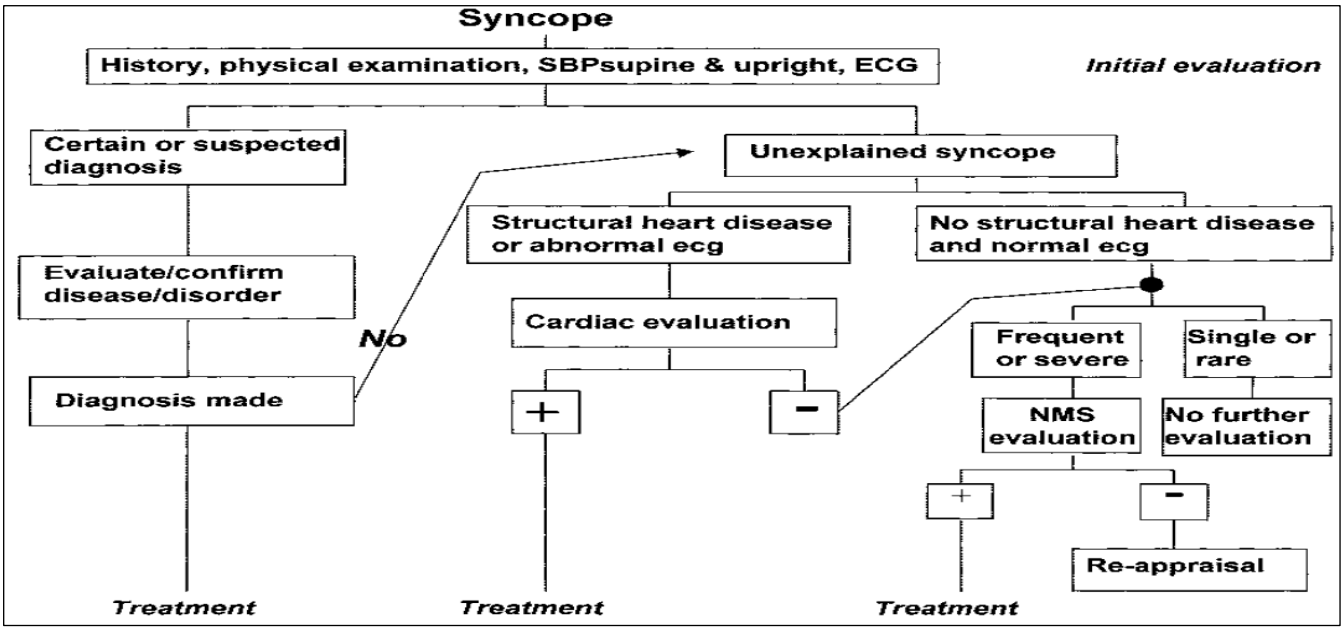


Figure 6. An Approach to the Evaluation of Patients with Syncope[16]
SBP = Systolic blood pressure; ECG = Electrocardiography; NMS = Neurally mediated syncope

4. Conclusion

Patients with ACS who present with a non-ischemic ECG and no chest pain commonly are frequently misdiagnosed and undertreated. Clinicians, especially those working in coronary care, need to be more cognizant of this patient population. To lower their high morbidity and mortality, this patient population should receive better prompt diagnoses and improved treatment options.

5. Declaration

5.1 Ethics Approval and Consent to participate
Not applicable.

5.2. Consent for publication
Not applicable.

5.3 Availibility of data and materials
Data used in our study were presented in the main text.

5.4 Competing interests
Not applicable.

5.5 Funding Source
Not applicable.

5.6 Authors contributions
Idea/concept: IMS. Design: IMS, AR. Control/supervision: AR, SW, AFR. Data collection/processing: IMS. Analysis/interpretation: IMS, AR. Literature review: IMS. Writing the article: IMS. Critical review: AR, SW, AFR. All authors have critically reviewed and approved the final draft and are possible for the content and similarity index of the manuscript.

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References

1. Brieger D, Eagle KA, Goodman SG, et al. Acute Coronary Syndromes Without Chest Pain, An Underdiagnosed and Undertreated High-Risk Group. *Chest*. 2004;126(2):461-469. doi:10.1378/chest.126.2.461

2. Dorsch MF. Poor prognosis of patients presenting with symptomatic myocardial infarction but without chest pain. *Heart*. 2001;86(5):494-498. doi:10.1136/heart.86.5.494

3. Jayawardana S, Salas-Vega S, Cornehl F, Krumholz HM, Mossialos E. The relationship between off-hours admissions for primary percutaneous coronary intervention, door-to-balloon time and mortality for patients with ST-elevation myocardial infarction in England: a registry-based prospective national cohort study. *BMJ Qual Saf*. 2020;29(7):541-549. doi:10.1136/bmjqs-2019-010067

4. Teixeira R, Lourenço C, António N, et al. The Importance of a Normal ECG in non-ST Elevation Acute Coronary Syndromes. *Arq Bras Cardiol*. 2010;94(1):25-33. doi:10.1590/S0066-782X2010000100006

5. Bhatt DL, Lopes RD, Harrington RA. Diagnosis and Treatment of Acute Coronary Syndromes. *JAMA*. 2022;327(7):662. doi:10.1001/jama.2022.0358

6. Canto JG, Fincher C, Kiefe CI, et al. Atypical presentations among medicare beneficiaries with unstable angina pectoris. *Am J Cardiol*. 2002;90(3):248-253. doi:10.1016/S0002-9149(02)02463-3

7. Canto JG. Prevalence, Clinical Characteristics, and Mortality Among Patients With Myocardial Infarction Presenting Without Chest Pain. *JAMA*. 2000;283(24):3223. doi:10.1001/jama.283.24.3223

8. Pope JH, Aufderheide TP, Ruthazer R, et al. Missed Diagnoses of Acute Cardiac Ischemia in the Emergency Department. *N Engl J Med*. 2000;342(16):1163-1170. doi:10.1056/NEJM200004203421603

9. Goodacre S, Pett P, Arnold J, et al. Clinical diagnosis of acute coronary syndrome in patients with chest pain and a normal or non-diagnostic electrocardiogram. *Emerg Med J*. 2009;26(12):866-870. doi:10.1136/emj.2008.064428

10. Turnipseed SD, Trythall WS, Diercks DB, et al. Frequency of Acute Coronary Syndrome in Patients with Normal Electrocardiogram Performed during Presence or Absence of Chest Pain. *Acad Emerg Med*. 2009;16(6):495-499. doi:10.1111/j.1553-2712.2009.00420.x

11. Singer AJ, Brogan GX, Valentine SM, McCuskey C, Khan S, Hollander JE. Effect of Duration From Symptom Onset on the Negative Predictive Value of a Normal ECG for Exclusion of Acute Myocardial Infarction. *Ann Emerg Med*. 1997;29(5):575-579. doi:10.1016/S0196-0644(97)70243-9

12. Welch RD. Prognostic Value of a Normal or Nonspecific Initial Electrocardiogram in Acute Myocardial Infarction. *JAMA*. 2001;286(16):1977. doi:10.1001/jama.286.16.1977

13. Chase M, Brown AM, Robey JL, Pollack C V., Shofer FS, Hollander JE. Prognostic Value of Symptoms during a Normal or Nonspecific Electrocardiogram in Emergency Department Patients with Potential Acute Coronary Syndrome. *Acad Emerg Med*. 2006;13(10):1034-1039. doi:10.1197/j.aem.2006.06.051

14. Arora S, Stouffer GA, Kucharska-Newton AM, et al. Twenty Year Trends and Sex Differences in Young Adults Hospitalized With Acute Myocardial Infarction. *Circulation*. 2019;139(8):1047-1056. doi:10.1161/CIRCULATIONAHA.118.037137

15. Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. *Eur Heart J*. 2018;39(21):1883-1948. doi:10.1093/eurheartj/ehy037

16. Kapoor WN. Current Evaluation and Management of Syncope. *Circulation*. 2002;106(13):1606-1609. doi:10.1161/01.CIR.0000031168.96232.BA