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Syncope as the Sole Presentation of Multi-Vessel Coronary Artery Disease

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Abstract

Defining the etiology of syncope can be occasionally challenging. We present a case with no history of coronary artery disease (CAD) who presented exclusively with exertional syncope, and was found to have mildly reduced left ventricular systolic dysfunction on echocardiogram and severe multi-vessel CAD with chronic total occlusion (CTO) of the right coronary artery (RCA). Syncope as the initial presentation of advanced CAD in the absence of classic ischemic symptoms is rather an uncommon presentation in clinical practice.

Keywords: Syncope, Coronary artery disease, Arrhythmia

1. Introduction

Syncope is a common medical condition that affects around 20% of the population.[1] Despite recent advancements in diagnostic utility, up to 10% of cases are still labeled to have unknown etiology.[2] Cardiac arrhythmias remain one of the most serious causes that need to be ruled out in the appropriate clinical context.[3] CAD is the leading cause of death in the United States.[4] It has a spectrum of symptoms with chest pain being the most prevalent.[5] Syncope as the sole symptom of CAD is rare.[6]

2. Case report

A 76-year-old female with medical history of hypertension, peripheral artery disease, chronic obstructive pulmonary disease (COPD), tobacco use disorder, and anemia presented to the emergency department after exertional syncope. She described feeling suddenly lightheaded, and lost consciousness for about 30 s. She did not have any chest pain, shortness of breath, or palpitations prior, during, or after the episode.

There was no abnormal movements, tongue bite, or loss of bladder/bowel control. After the episode, she was not confused or disoriented. She had no history of prior syncope in the past. She does not take any regular medications at home and has no known history of CAD. She is an active smoker, does not consume alcohol, nor has a history of illicit drug use. She had no previous surgeries and non-contributory family history.

Patient had normal vital signs upon arrival to the Emergency Department including normal orthostatic vital signs. Physical exam was essentially normal with euvolemic volume status, no audible murmurs, strong and equal pulses bilaterally, and no focal neurological deficits.

2.1. Diagnostic work-up

ECG: Normal sinus rhythm, normal PR and QT intervals, no acute ischemic changes, and non-specific ST-T wave changes (Fig. 1). Basic laboratory workup is shown in Table 1.

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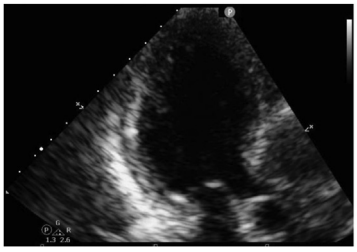
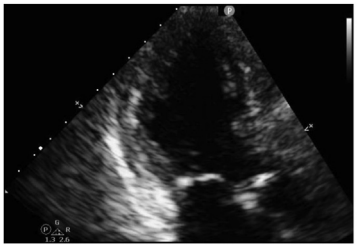
Chest X-ray was unremarkable. CT head showed no acute intracranial pathology with evidence of chronic small vessel disease and remote infracts in the right Posterior Cerebral Artery distribution, right and left cerebellar hemispheres, thalamus, right caudate head, and inferior right frontal lobe.

Carotid ultrasound showed mild bilateral carotid atherosclerotic plaque with <50% stenosis in the right internal carotid artery, left common carotid artery, and left internal carotid artery.

Echocardiogram revealed left ventricular hypertrophy, mildly reduced left ventricular systolic function, estimated left ventricular ejection fraction (LVEF) of 45%, and regional wall motion abnormalities in the RCA territory (Hypokinesia in the basal inferior, basal inferoseptal, and the entire inferolateral wall).



End-diastolic frame of a left ventricular 3-chamber view on echocardiogram.

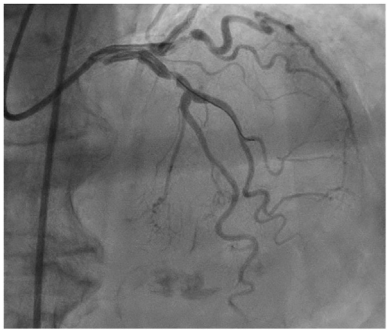


End-systolic frame of the same clip showing hypokinesis of the inferolateral wall with a hinge point.

2.2. Hospital course

Given the regional wall motion abnormalities in the RCA distribution and the mildly reduced LVEF, cardiology team proceeded with coronary angiography. This was done keeping in mind the absence of classic anginal symptoms, ischemic ECG changes, and negative cardiac enzyme profile, and was based solely on the presentation of exertional syncope.

Coronary angiogram revealed severe multi-vessel CAD with CTO of the RCA with left to right collaterals, 80% stenosis in the proximal left anterior descending (LAD) artery, and 90% stenosis in the left circumflex (LCx) artery.



Coronary angiogram showing severe LAD and LCx disease.

Patient underwent successful intravascular ultrasound (IVUS)-guided percutaneous coronary intervention (PCI) of the LAD and LCx with two Xience Skypoint drug-eluting stents (DES).

She had an uneventful 7-day hospital stay apart from bradycardia on telemetry monitoring. Patient was discharged home on aspirin, ticagrelor, high-intensity statin, carvedilol, entresto, an implantable loop recorder (ILR) to evaluate for potential arrhythmias, and outpatient follow-up for possible addition of sodium-glucose co-transporter 2 (SGLT2) inhibitor and spironolactone. She was counseled regarding the importance of smoking cessation and was prescribed nicotine replacement therapy.

3. Discussion

We present a case of exertional syncope as the sole presentation of severe multi-vessel CAD. Cardiac causes of syncope remain the most worrisome and carry a higher mortality rate compared to non-cardiac causes. Syncope in the setting of CAD increases the risk of sudden cardiac arrest by approximately three-folds even in individuals with normal LVEF.[7] Kapoor et al. has conducted a prospective cohort study of 204 patients with syncope to investigate different prognostication factors and it was validated that patients with cardiovascular causes are at higher risk for mortality (18).

The relationship between CAD and syncope has been previously studied in literature. Mcdermott et al. prospectively followed a cohort of 1474 patients to determine the incidence of acute myocardial infarction (AMI) in patients presenting to the ED with syncope. Of this cohort, only 46 (3.1%) patients were diagnosed with AMI as the cause of syncope. Moreover, they further investigated the utility of ECGs in diagnosing AMI in this subset of population and concluded that initial ECGs have high negative predictive value given low incidence of AMI in patients presenting with syncope.[8] Same

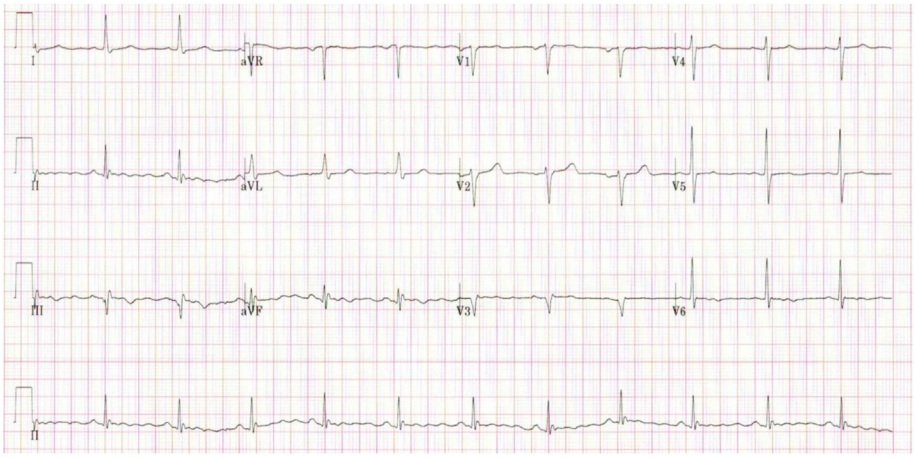


Fig. 1. ECG showing normal sinus rhythm and non-specific ST-T wave changes.

findings were obtained by Georgeson et al. who followed a cohort of 5762 patients who presented to the ED with syncope. It was noteworthy that the incidence of AMI in the setting of syncope was as low as 7%.[9,10] These results confirm the rarity of such presentation in patients with AMI. Moreover, syncope has been related to specific coronary artery occlusions. For instance, Aniruddha, P. et al. reported a case of syncope caused by LAD artery occlusion.[11]

Table 1. Laboratory tests.

Laboratory test	Patient's values	Normal reference range
General hematology		
White blood cells (10^3/μL)	**2.3 (at baseline)**	4–10
Hemoglobin (g/dL)	**10.1 (at baseline)**	11.5–16
Platelets (10^3/μL)	266	140–400
General chemistry		
Urea (mg/dL)	19	9–23
Creatinine (μmol/L)	1.06	0.5–1.10
Sodium (mmol/L)	137	136–145
Potassium (mmol/L)	4.2	3.5–5.1
Chloride (mmol/L)	105	99–109
Magnesium (mmol/L)	2.2	1.6–2.6
Glucose (mg/dL)	125	60–99
Bicarbonate (mmol/L)	26	20–31
Albumin (g/dL)	3.6	3.5–5.2
Calcium (mg/dL)	8.7	8.6–10.2
Alkaline phosphatase (U/L)	66	53–141
Bilirubin total (mg/dL)	0.4	0.3–1.2
ALT (U/L)	11	1–33
AST (U/L)	22	14–34
Cholesterol (mg/dL)	188	1–199
Triglycerides (mg/mL)	155	0–149
HDL (mg/mL)	36	40–60
Non-HDL-c (mg/mL)	152	0–129
LDL calc (mg/mL)	121	0–99
Iron (μg/dL)	33	35–160
TIBC (μg/dL)	367	250–400
Iron saturation (%)	9	15–50
Ferritin (ng/mL)	20	10–291
Cardiac Markers		
Troponin I highly sensitive (pg/mL)	4 (at 0 HR) 6 (at 1 HR) +2 (Delta 0–1 HR)	0–50
Endocrine Chemistry		
Hemoglobin A1c (%)	5.7	4.2–5.6
TSH (μIU/mL)	1.57	0.35–5.5

The cause of syncope in our case remains unclear. Severe ischemia rarely depresses the cardiac function to the degree of causing syncope due to hypotension.[12] This is supported by the fact that our patient had only a mildly reduced LVEF. One plausible explanation is Bezold–Jarisch reflex which entails hypotension and bradycardia that might eventually lead to syncope in the setting of inferior wall ischemia. Our case has RCA CTO with left to right collaterals which suggests that there might have been some demand-perfusion mismatch with exertion resulting in syncope. Interestingly, with such advanced CAD, our patient did not complaint of ischemic symptoms. Another reasonable explanation is that ischemia might have triggered cardiac arrhythmias which subsequently resulted in syncope.[13] Although, we could not detect any arrhythmia with the inpatient telemetry monitoring, it still stands as a potential factor. This hypothesis can be nourished by the evidence of widespread remote brain infarcts which might be of embolic source. Patient had ILR placement prior to discharge to check for arrhythmia. Additionally, close follow-up with cardiology as an outpatient is necessary for optimization of guideline directed medical therapy, and interrogation of the ILR.

4. Conclusion

Our case points out a rare presentation of severe multi-vessel CAD that should not be overlooked in dealing with similar patients in the future.

Disclosure

All the authors have nothing to disclose. No funds or grafts were received by the authors.

Conflict of interest

None.

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