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Case Report

A 28-year-old male with spontaneous coronary artery dissection complicated by apical thrombus and acute ischemic stroke: An interesting case ☆,☆☆

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ABSTRACT

Spontaneous coronary artery dissection is described as the intramural bleeding that separates the layers of an epicardial coronary artery wall, either with or without an intimal tear. Atherosclerosis, iatrogenic damage, or trauma are not linked to this syndrome. Here we present a 28-year-old male with 1 month history stroke but no any chronic disease as well family history of heart disease who presented with 2 days' duration of typical cardiac chest pain. Based on an emergency electrocardiogram that showed biphasic T-wave inversion with ST-elevation myocardial infarction, the patient was taken to the a catheterization laboratory(cath-lab), with the result of spontaneous coronary artery dissection of the left anterior dissenting artery with thrombolysis in myocardial infarction flow grade 0 and normal of other vessels. Then we successfully did angioplasty, and the patient was discharged with aspirin 100 mg 1 × 1, clopidogrel 75 mg 1 × 1, and rivaroxaban 20 mg 1 × 1.

Introduction

Myocardial ischemia and subsequent infarction can result from reduced blood flow produced by the constricted lumen. The last decade has seen a rise in clinical evidence that suggests the phrase "acute risk factors" can be used to describe activities and events that temporarily and abruptly increase the likelihood of acute cardiac diseases. The most important acute risk factors are thought to be vigorous exercise, emotional stress, and exposure to cold or heat, drinking coffee or alcohol, using drugs like cocaine or marijuana, and having sexual relations [1].

Spontaneous coronary artery dissection (SCAD) is described as the intramural bleeding that separates the layers of an epicardial coronary artery wall, either with or without

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an intimal tear. As a result, an intramural hematoma (IMH) forms, compressing the artery lumen and, to varying degrees, impairing antegrade blood flow, which can lead to myocardial ischemia or infarction [2].

Non-atherosclerotic SCAD often results from a combination of disease pathways that make artery beds vulnerable to injury. These include connective tissue disorders like Marfan's syndrome, Ehler Danlos, and cystic medial necrosis, multiple pregnancies, fibromuscular dysplasia, systemic inflammation like lupus erythematosus, Crohn's disease, polyarteritis nodosa, and sarcoidosis, hormonal therapy, and coronary artery spasm [3].

It has different presentations, ranging from asymptomatic, unstable angina, acute myocardial infarction, or even ventricular arrhythmias that result in sudden cardiac death.

Any young person suffering from an acute myocardial infarction without any risk factors should have SCAD taken into consideration in the differential diagnosis.

It is more in female following postpartum and rare in male compared to female. Here, we discuss a 28-year-old male with no known risk factor other than smoking and khat chew who diagnosed with spontaneous coronary artery disease with thrombolysis in myocardial infarction 0 following presentation with signs and symptoms consistent with acute coronary syndrome.

Case presentation

A 28-year-old male presents to the emergency room with heavy chest pain for 2 days' duration radiating to his left arm. He had 1 month history of ischemic stroke but no other chronic diseases as well as a family history of chronic diseases. He was on clopedigrol 75mg for his newly diagnosed stroke but no any other history of alcohol or smoking, except he that he was a heavy khat user.

In an emergency, vital signs were as follows: blood pressure (BP) of 110 mmHg, pulse 90bpm, SpO2 95, and temperature of 36.5c. The cardiac examination as well as systemic review were unremarkable except right side weakness and slured speech .

Biphasic T-wave inversion with V1-V4 ST-elevation compatible with anterior-septal myocardial infraction was seen on a bedside electrocardiogram (Fig. 1). Urgently, the patient was taken to catheterization laboratory (cath-lab), after loading with aspirin 300 mg and clopidogrel 600 mg. The result of angiography was intersiting as SCAD of left anterior descending (LAD) with thrombolysis in myocardial infarction 0 flow with normal circumflex (CX) and right coronary arteries (Figs. 2A-C). The LAD was pre-dilated with a 1.5 × 15 mm balloon, the mid LAD was implanted using 2.75 × 24 mm, the proximal LAD was implanted using 3.0 × 16 mm DES, and finally, final angiography views were taken (Figs. 3A-D). Basic lab tests such as renal, liver function test, lipid profile, Glucose, HbA1c, CK-MB, troponin were sent with normal results.

The patient was taken to the coronary intensive care unit (CIU) for 24 follow-up. Thereafter, the patient's general condition improved, and he was moved to the inpatient department. Post PCI Echocardiography revealed apical hypokinesia, aneurysmatic with a 0.63 × 1.24 cm thrombus, an ejection fraction (EF) of 40%-45% and mild mitral regurgitation (Figs. 4A & B).

Due to the history of stroke the patient was sent for carotid Doppler ultrasound and brain CT scan (Fig. 5). Three days later, the patient was discharged with aspirin 100 mg 1 × 1, clopidogrel 75 mg 1 × 1 and rivaroxaban 20 mg 1 × 1 and recommended physiotherapy, and khat cessation. At 10 days postfollow, the patient's general condition was good except mild weakness, and of ejection fraction improved up to 50% with no thrombus.

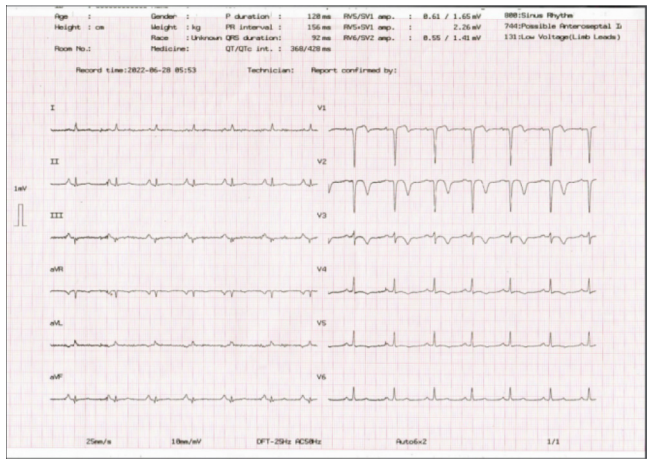

Fig. 1 – Electrocardiography: normal sinus rhythm, T-wave inversion and V1-V4 ST elevation with Q wave compatible with apical aneurism.

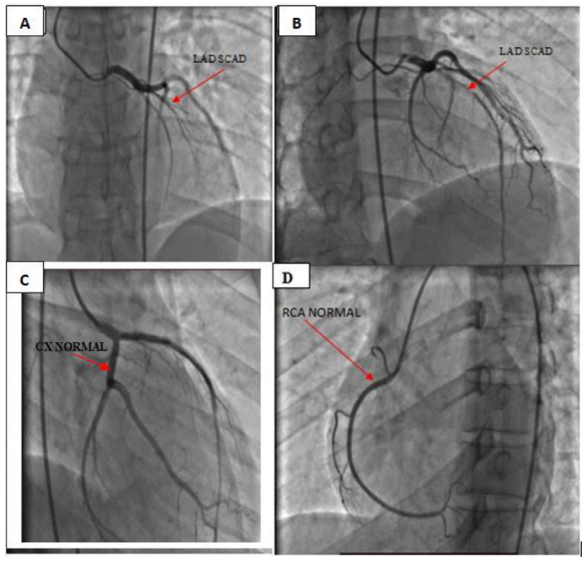

Fig. 2 – Panel A: shows SCAD of LAD in AP cranial. Panel B: shows SCAD of LAD in LAO cranial. Panel C: shows normal CX. Panel D: shows normal right coronary arteries.

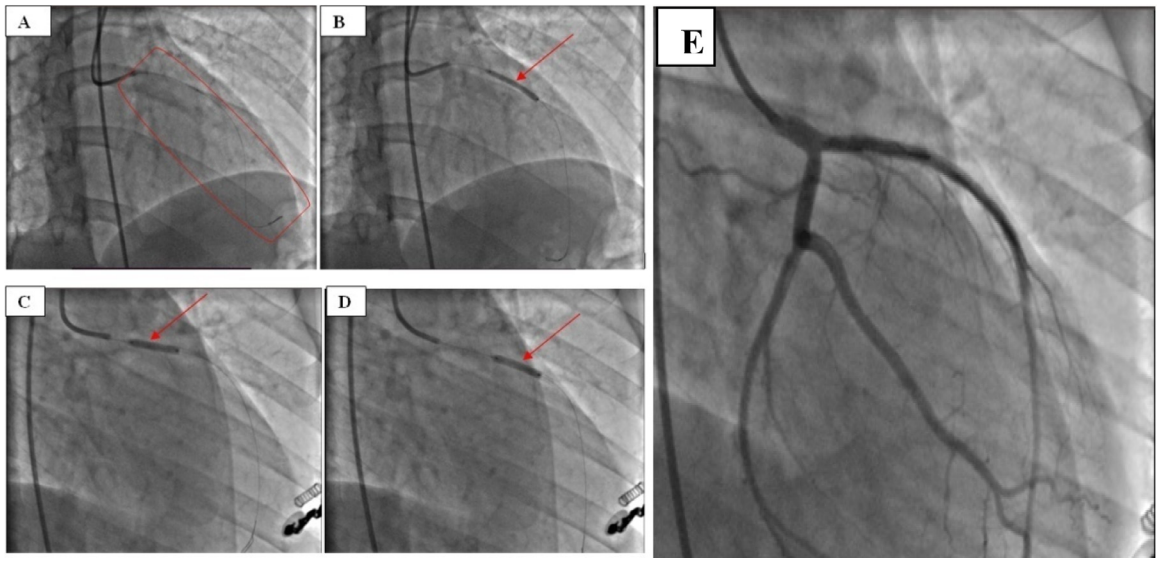

Fig. 3 – Panel A: LAD wiring and 1.5 x 15 mm balloon pre-dilatation. Panel B: Mid LAD 2.75 x 24 mm DES ımplantatıon. Panel C: Proximal LAD 3.0 x 16 mm DES implantation. Panel D: post-dilation with 3.5 x 20 non-compliance (NC) balloon. Panel E: final view.

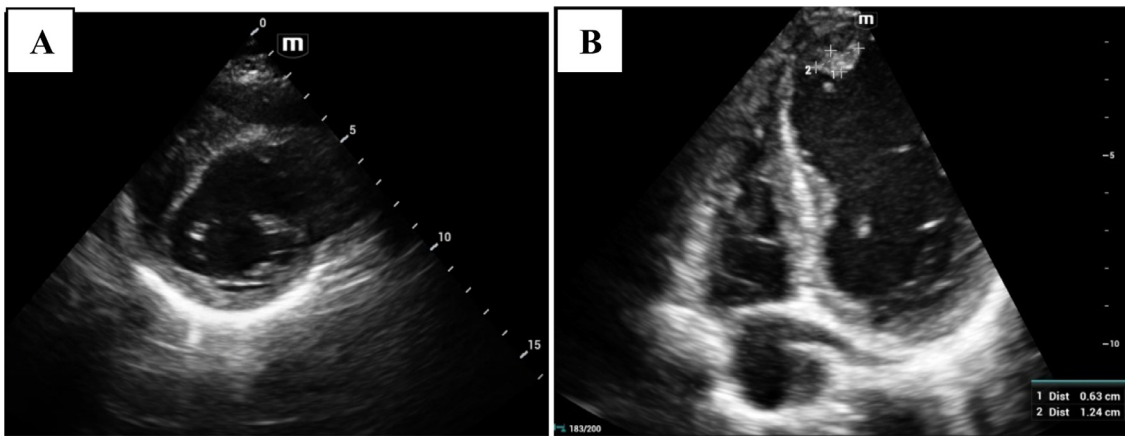

Fig. 4 – Panel A: Echo: parasternal short axis of the left ventricle with mild aneurysm. Panel B: apical 4 chamber view with apical thrombus of 0.63 x 1.24 cm.

Discussion

A separate pathophysiological etiology of acute coronary syndromes is SCAD [4] which is a rare cause of an acute coronary syndrome.

SCAD patients had ST-elevation myocardial infarction at a higher rate (47.6% vs 39.3%) than non-SCAD MI patients [5].

A database from an interventional cardiology laboratory shows that SCAD now has a prevalence rate of 0.2% in a population of 11,605 participants. Men account for 0.7% of SCAD participants, while women account for 0.6% [6].

In young individuals with a low risk of atherosclerotic CAD, SCAD is an uncommon cause of myocardial infarction. Although there are no recognized causes, it has been linked to connective tissue disorders, renal fibro muscular-dysplasia, and the postpartum period [7]. Due to CTCA's lower spatial resolution, which restricts assessment of the more distal coronary regions that are frequently impacted by SCAD, routine noninvasive diagnosis by CTCA is not recommended [8]. As a result, the diagnosis still depends on identifying distinctive characteristics during invasive angiography [4]. Its association with ischemic stroke is rare as reported by some case reports [9]. Like-wise this young patient had stroke following apical thrombus due SCAD.

The majority of SCAD patients have a favorable prognosis [10]. No studies have compared different treatment techniques; however, reports have shown that conservative care (with documented angiographic resolution), fibrinolysis, percutaneous coronary intervention (PCI), and coronary artery bypass grafting (CABG) had favorable outcomes [11]. Conservative management is indicated if the patient is stable and

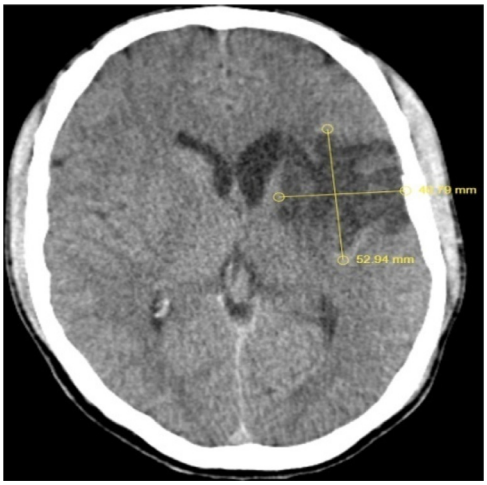

Fig. 5 – Brain CT scan: In the left frontal lobe, a 4 cm sized hypo dense lesion.

asymptomatic. Successful percutaneous coronary intervention (PCI) revascularization, as it was in the case of our patient, is for critically ill patients [12].

Conclusion

Although the main cause of ischemic strokes is carotid artery disease, it's crucial to rule out an embolic phenomenon in younger patients. A left ventricular thrombus or a paradoxical embolus through a patent foramen ovale could cause an embolus.

Data sharing statement

We declared that we had full access to all of the data in this study, and we take complete responsibility for the integrity of the data. All original data are available in the Mogadishu Somali Turkish Training and Research Hospital, Mogadishu, Somalia and all authors.

Ethics approval

Based on the regulations of the review board of the Mogadishu Somali Turkish Training and Research Hospital, institutional review board approval is not required for case reports.

Author contributions

All authors performed substantial contributions to the case sections. Took part in drafting the cas or revising it critically for important intellectual content and gave final approval of the version to be published.

Provenance and peer review

Not commissioned, externally peer reviewed.

Patient consent

Written informed consent had obtained by the patient to have the case details and any accompanying images published.

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