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RESEARCH ARTICLE

SPONTANEOUS CORONARY ARTERY DISSECTION, AN UNDERDIAGNOSED CAUSE OF ACUTE CORONARY SYNDROME: A CASE REPORT

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Abstract

Spontaneous coronary artery dissection (SCAD) is a rare cause of myocardial infarction (MI). It usually strikes young patients, predominantly women, with or without any cardiovascular risk factors. We report the case of a 52-year-old woman with a spontaneous coronary artery dissection revealed during the investigation of unstable angina.

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Introduction:-

Spontaneous coronary artery dissection (SCAD) is a rare cause of myocardial infarction (MI). It usually strikes young patients, predominantly women, with or without any cardiovascular risk factors [1].

SCAD is defined as an epicardial coronary artery dissection that is not associated with atherosclerosis or trauma and not iatrogenic. The main machinery of myocardial damage appearing as a consequence of SCAD is coronary artery obstruction instigated by formation of an intramural hematoma (IMH) or intimal disruption rather than atherosclerotic plaque rupture or intraluminal thrombus. Initially seen as a fatal condition since its first description in 1931 by Pretty [2], it is now clear that it is a different entity from atherosclerosis and its management should be different, also recent reports have refuted these misconceptions.

We report the case of a 52-year-old woman with a spontaneous coronary artery dissection revealed during the investigation of unstable angina.

Case report

A 52-year-old woman presented to the emergency room with severe chest pain triggered by physical exercise and stress. She described mild episodes happening the previous weeks. She had no

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current cardiovascular risk factors, except for smoking cessation 23 years before. She is known hypertensive and diabetic both well controlled under adequate treatment.

Vital signs on admission were blood pressure 119/73 mmHg, pulse 76 beats/min, temperature 36.8 °C, respiratory rate 16 breaths/min, SpO₂ 95% on room air and body mass index (BMI) 27.9 kg/m². Physical examination was unremarkable, and the patient remained hemodynamically stable.

The chest X-ray was normal and the EKG showed a negative T wave in DI-II-III, aVL-aVF, V4-5-6, and QS aspect with large positive T wave in V1-2-3, we also noted an incomplete left bundle block in V5-6 (Figure 1).

High sensitivity troponin T was 500 ng/L compatible with an acute coronary syndrome (ACS).

The transthoracic echocardiography (TTE) showed a non-dilated and non-hypertrophied left ventricle, with a conserved systolic function (systolic ejection fraction estimated at 60%). The study of the ventricular wall contractions showed a hypokinesia in the apex and the anterior wall (lateral and septal). The rest TTE was normal.

Coronary angiography scheduled the same day showed a normal coronary arterial system except a type 1 (of The National Heart, Lung, and Blood Institute (NHLBI) classification scheme for coronary dissection) spontaneous dissection of the second portion of anterior interventricular artery (Figures 2, 3 and 4). We opted for a conservative treatment strategy taking in consideration that the distal flow was normal and the other arteries were normal too.

Discussion:-

SCAD is described as the dissection of an epicardial coronary artery not associated with atherosclerosis, trauma or iatrogenic [1, 3]. SCAD is an uncommon but possibly lethal entity described mostly in young women. It is designated as the cause of myocardial infarction in more than 30 per-cent of women under 50 years of age [1, 2]. The usual age of patients with SCAD when diagnosed fluctuates within an indistinct series from 45 to 55 years, though SCAD cases have been described in patients below 20 and above 80 years [1, 2]. Our patient was 52 years-old when diagnosed with SCAD.

Although commonly SCAD may be atherosclerotic or non-atherosclerotic in origin, modern studies have dedicated their work to the non-atherosclerotic variant, knowing that the pathophysiology, management, and outcomes are distinctive from atherosclerotic coronary artery disease (which causes atherosclerotic SCAD). As such, contemporary usage of the term 'SCAD' is typically synonymous with non-atherosclerotic SCAD [4].

SCAD may be a consequence of an intimal-medial slit that can be visible as numerous radiolucent lumens on angiography, or it may result from spontaneous hemorrhage into the arterial wall that can be angiographically subtle. In fact, <30% of SCAD have an angiographic type I appearance (pathognomonic contrast stain of arterial wall with multiple radiolucent lumens) in the author's large contemporary series [4, 5]. In our case as seen in the figures we can clearly notice the pathognomonic contrast stain of arterial wall with multiple radiolucent lumens.

With its spatial resolution of 10–20 mm superior to IVUS (150 μm) for delineating the lumen-intimal interface and for picturing intraluminal thrombi, false lumen, and intimal slits. Its principal downsides compared with IVUS are the possible risks of hydraulic extension of a dissection with forceful contrast injection, and acute kidney injury due to higher amount of contrast media used [6, 7]. Other risks with intracoronary imaging include extending the coronary dissection with the wire or imaging catheter, catheter-induced occlusion, and guide-catheter iatrogenic dissection [8]. Therefore, intracoronary imaging should only be pursued in adequately sized vessels, when coronary angiographic diagnosis is uncertain [4].

The ideal treatment for SCAD has still to be determined and single-centred retrospective cohort studies are still our best source of evidence [9].

Conservative treatment in a stable patient with normal distal flow is now encouraged as the treatment strategy of choice; follow-up angiography has shown complete spontaneous resolution for such patients [10].

Primary coronary intervention (PCI) has a potentially critical role, but appropriate selection is required based on the presence of ischemia or infarction. Technical failure is frequently due to the passage of the wire into the false lumen and loss of

coronary blood flow from the propagation of dissection following stent placement [11]. Revascularization with PCI, or coronary artery bypass grafting (CABG) contingent to coronary anatomy and operator skill, should be reserved for patients with high-risk features, such as ongoing ischemia, recurrent chest pains, left main artery dissection, ventricular arrhythmias, or hemodynamic instability (Figure 5) [4]. In our case we have chosen a conservative treatment knowing that the distal flow was normal, and the SCAD type was a type 1. We still have to carefully follow up our patient in the next months, a follow-up angiography is scheduled in the next month.

SCAD is a rare condition, but it must be considered in the differential diagnosis of acute myocardial infarction in younger patients with no cardiac risk factors. Although our patient had various cardiovascular risk factors such as diabetes, high blood pressure, and obesity.

Figures

Figure 1:- Patient EKG.

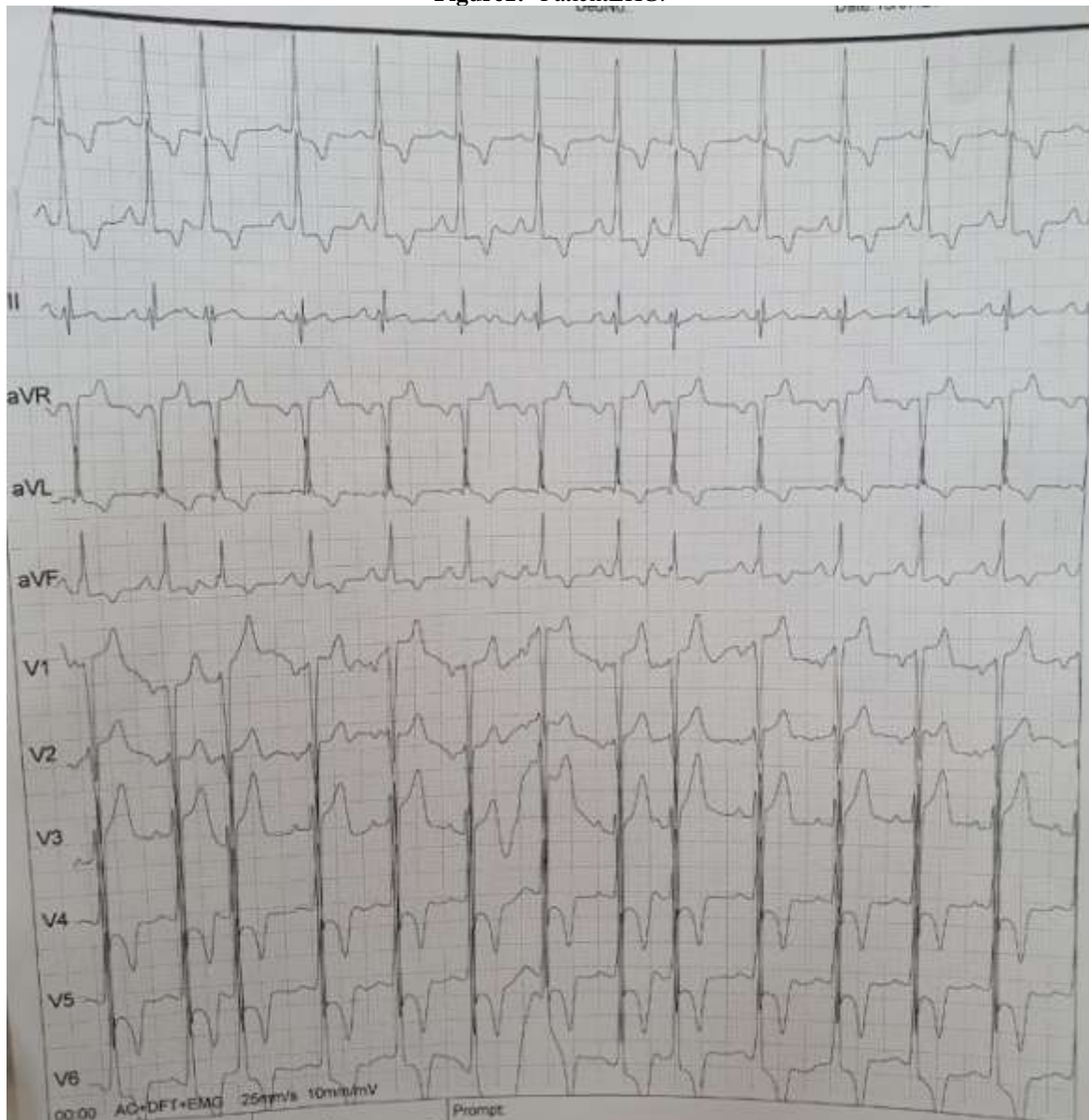


Figure2:- Normalangiographyoftherightcoronaryartery.

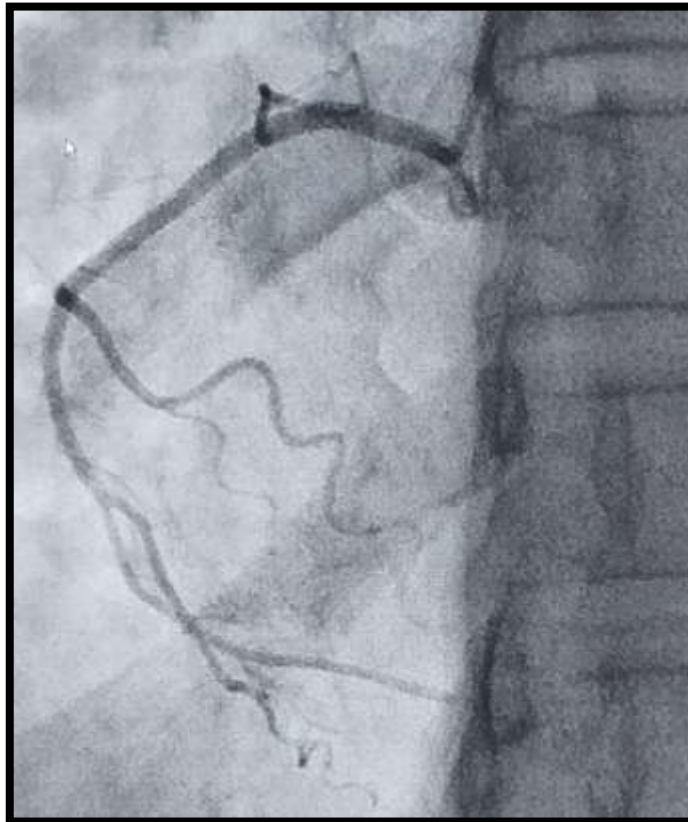


Figure3:- Angiographyoftheleftcoronaryarteryshowingatype I SCADoftheanteriorinterventricular artery(red arrow).

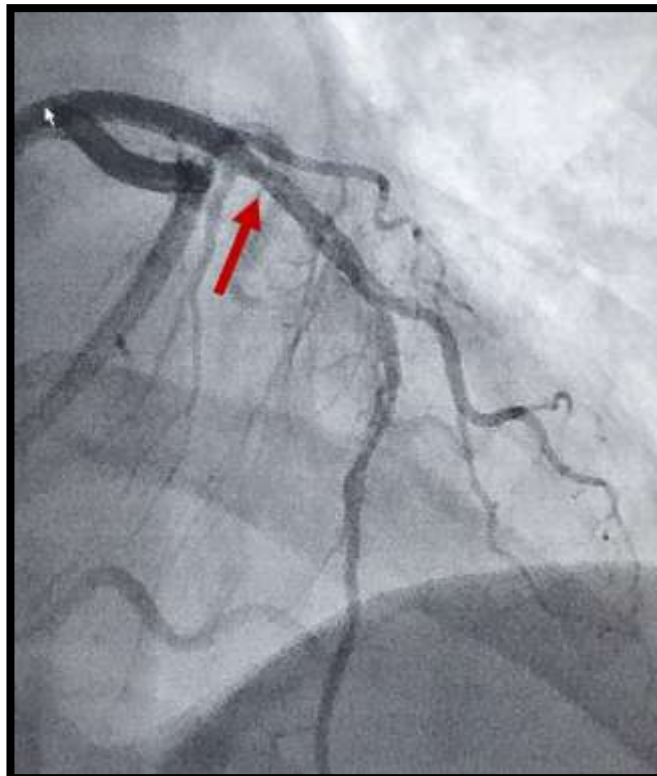


Figure4:- AngiographyoftheleftcoronaryarteryshowingatypelSCADoftheanteriorinterventricular artery(red arrow).

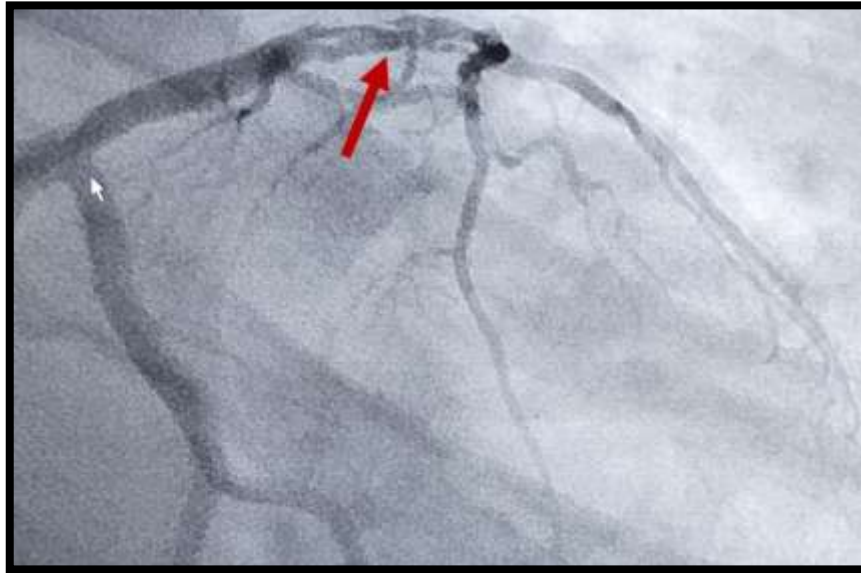
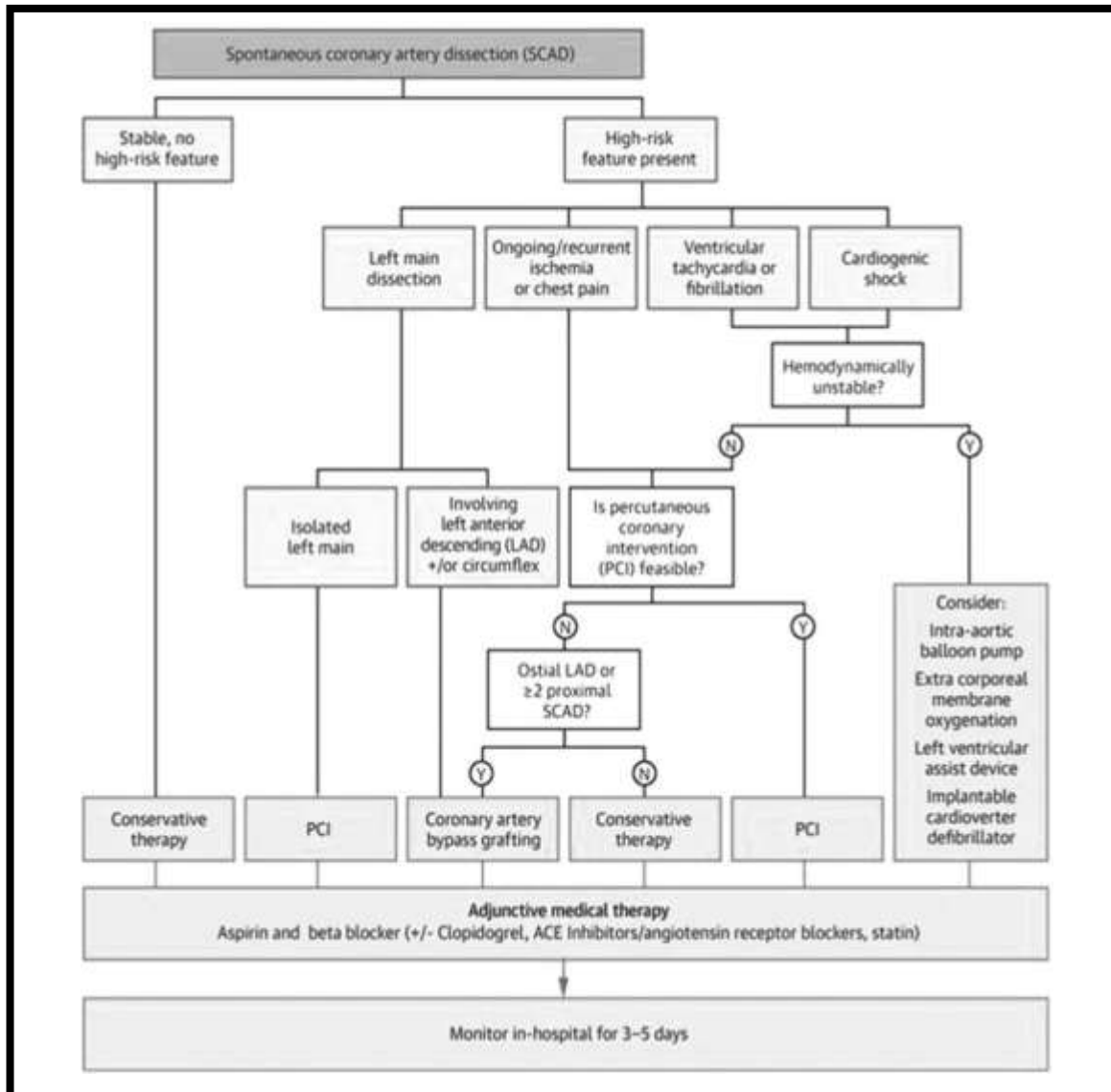


Figure5:- Recommendedtreatmentalgorithmwithspontaneouscoronaryarterydissection[12]



Conclusion:-

Understanding the nuances of SCAD, a nonatherosclerotic etiology of ACS, is critical to improving delivery of care to women. It is indeed a rare condition, but it must be considered in the differential diagnosis of acute myocardial infarction in younger patients with no cardiac risk factors.

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