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Original Article

Spontaneous coronary artery dissection: Case series and review of literature

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ABSTRACT

Aim: Spontaneous coronary artery dissection (SCAD) is a less recognized cause of ST elevation myocardial infarction (STEMI) in clinical practice. The aim of this communication is to describe a case series in South Asian population and highlight on the long-term clinical outcomes on conservative management.

Methods: A retrospective analysis of data of five patients (6 instances) of SCAD managed in a tertiary care center during January 1994 to June 2015 was done. Clinical, angiographic, therapeutic, and follow-up data till end of June 2015 are analyzed.

Results: All patients were young (mean – 33 years) and predominantly male. Etiology of SCAD was diverse and included peripartum state, vigorous activity and atherosclerosis. Left anterior descending (LAD) coronary artery was predominantly involved and the majority had angiographic type 1 SCAD. Medical treatment provides excellent long-term benefits. Coronary stenting provided symptomatic benefit in a patient with favorable anatomy.

Conclusions: Clinical recognition of SCAD is difficult. It should be suspected in peripartum state, young females and in presence of other precipitating factors. Coronary angiography is essential for establishing the diagnosis. Medical treatment provides favorable long-term survival.

Implications and practice: The awareness of SCAD is important for all clinicians involved in STEMI care. A prompt suspicion can avoid administration of thrombolytic therapy. Early coronary angiography will provide an accurate diagnosis and help in deciding appropriate therapy. Percutaneous intervention can be challenging.

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1. Introduction

Spontaneous coronary artery dissection (SCAD) is a rare clinical entity that frequently presents as an acute coronary syndrome (ACS).[1,2] Dissection of the coronary artery results in separation of the layers of the arterial wall, creating a false lumen. The separation may be between the intima and media, or between the media and adventitia. Hemorrhage into the false lumen can impinge upon the true lumen of the coronary artery, impairing the blood flow and causing myocardial ischemia, infarction, sudden death, cardiogenic shock, or pericardial tamponade.[1,3,4]

SCAD is often classified according to the associated predisposing condition and has been broadly divided into atherosclerotic and nonatherosclerotic. Nonatherosclerotic associations have included peripartum state, connective tissue disorders, systemic inflammatory conditions, and coronary artery spasm.[5,6] Fibromuscular dysplasia (FMD) as a possible predisposing factor particularly in females has received a lot of attention recently.[5–9] Screening for FMD of renal, iliac, and cerebrovascular arteries by computed tomography angiography and magnetic resonance angiography can be helpful. Association of extracoronary vascular abnormalities like dissections, aneurysms, and dilatation in chest, abdomen, pelvis, and neck has also been described.[7] Those without identifiable predisposition have been labeled as idiopathic. Vigorous exercise with increased cardio-circulatory stress and shear forces against the coronary arterial wall can precipitate SCAD among patients with or without these predisposing conditions.[3,4]

Diagnosis of SCAD is made at coronary angiography.[3,4,6,8,9] Recent reports suggest the value of intravascular ultrasound (IVUS) and optical coherence tomography (OCT) for diagnosis and management.[6,8–12] Various treatment options are available including medical therapy, percutaneous coronary intervention (PCI), and coronary artery bypass graft (CABG) surgery.[3,4,6]

There are isolated case reports on this entity from South Asia.[12,13] This communication reports a series of 5 cases from South Asia with diagnostic, therapeutic, and follow-up data along with brief review of the literature.

2. Methods

The material for this study is obtained from records of academically interesting patients maintained by the first author in a tertiary care referral postgraduate teaching institute. During January 1994 to June 2015, five cases of SCAD were diagnosed, treated, and followed up. The records of these cases form the material for this analysis.

The diagnosis of SCAD was made on the basis of the following angiographic features[1,3,4,6,8,9]:

(1) Visualization of thin radiolucent intimal flap and the presence of extraluminal contrast after wash out of dye from the remainder of the vessel.
(2) Presence of haziness and irregularity of the vessel. Haziness and intraluminal filling defects indicate possibility of a thrombus.

Patients were classified into types 1, 2, and 3, as per the angiographic classification proposed by Yip[6] and Saw.[14] Type 1 is pathognomic angiographic appearance of SCAD with contrast dye staining of arterial wall with multiple radiolucent lumen. Type 2 includes lesions with long, diffuse, and smooth stenosis representing intramural hematoma. Type 3 mimics atherosclerosis.

History of pregnancy, vigorous exercise, connective tissue disorder, substance abuse, diabetes and hypertension was noted. Clinical data, electrocardiogram (ECG), two-dimensional echocardiography (2DE), management details, and follow-up information were analyzed. In four patients, full blood analysis, including blood count, total body profile, coagulation workup, lipoprotein assay, thyroid function test, and a workup for autoimmune disorder, was available. One patient had repeat coronary angiography at one year and a multidetector computed tomography (MDCT) coronary angiography at 2 years follow-up.

Patients with coronary dissection who had any history of cardiac surgery, coronary intervention, trauma, or aortic dissection were excluded.

3. Results

The patient characteristics are summarized in Table 1. The patients were from different states of India. Cases 1 and 5 were from Maharashtra, whereas cases 2 and 3 were referred from Madhya Pradesh and case 4 from Rajasthan. Four patients were male and ages ranged from 18 to 56 years, mean – 33 years. There were 6 instances of SCAD in 5 patients. All patients presented with ST elevation on ECG. Case 1 was treated during both the episodes at our center whereas the

Table 1 – Clinical data.

Case number	Year	Age (years)	Sex	Presentation	Follow-up (years)	Predisposing/Precipitating factor
1[a]	1994; 2011	39; 56	F	AW-STEMI; IW-STEMI	19	Pregnancy; DM, HTN
2	1998	18	M	AW-STEMI	5	No obvious cause
3	1998	19	M	AW-STEMI	5	Vigorous activity
4	2011	40	M	IW-STEMI	4	DM, HTN, ↑homocysteine
5	2013	31	M	AW-STEMI	2	Tobacco, ↑homocysteine

Abbreviations: AW, anterior wall; DM, diabetes mellitus; F, Female; HTN, hypertension; IW, Inferior wall; M, Male; STEMI, ST elevation myocardial infarction.

[a] Case 1 had an anterior wall STEMI in 1994 and inferior wall in 2011.

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