Journal of Enam Medical College
Vol 5 No 1 January 2015

Case Report

A Case Report on Spontaneous Coronary Artery Dissection

Tunaggina Afrin Khan[1], Abdul Wadud Chowdhury[2], Abu Sadique Abdullah[3]
Received: April 12, 2014 Accepted: December 15, 2014 doi: 10.3329/jemc.v5i1.21498

Abstract

Spontaneous coronary artery dissection is an extremely rare cause of acute coronary syndrome. Although it predominantly affects young women in the peripartum period, it can also occur in men. The left coronary artery is most frequently involved. The usual presentation is ST segment elevation myocardial infarction. Although several treatment modalities have been proposed, the optimal treatment options still remain to be established. This current case report focuses on a 40-year-old male presented with acute coronary syndrome and subsequently was found to have coronary artery dissection.

Key words*: Spontaneous coronary artery dissection; Acute coronary syndrome; Peripartum period*

J Enam Med Col 2015; 5(1): 52–57

Introduction

Spontaneous coronary artery dissection (SCAD) is an acute coronary event of uncertain origin and an infrequent cause of acute coronary syndrome (ACS), typically affecting a younger, otherwise healthy population.[1-6] This condition affects young women twice as much as men.[7,8] Clinically it may be asymptomatic or may present with stable angina, acute coronary syndrome, ventricular arrhythmia and even sudden cardiac death.[4,9] SCAD predominantly occurs in peripartum period[10] and usually involves single coronary artery; but multiple coronary arteries may also be involved.[11] SCAD can be due to an intimal tear with medial dissection or as a consequence of primary rupture of the vasa vasorum leading to medial dissection with hematoma formation.[12] The diagnosis of SCAD is made principally with invasive coronary angiography.[1,2] Management options range from conservative medical treatment to percutaneous intervention or a surgical approach. Prognosis of SCAD varies widely.[7]

Case report

A 40-year-old hypertensive and smoker gentleman without any prior cardiac event presented to the Emergency Department with severe crushing type of retrosternal chest pain for 3 hours with accompanying sweating and nausea. He denied any family history of heart disease. Physical examination revealed average body build with pulse 84 beats/min, blood pressure 150/90 mm Hg and no additional sound on auscultation of heart and lung bases. All the peripheral pulses were clearly palpable. His initial ECG showed ST elevation in leads V_1 to V_4 (Fig 1) and troponin I level was raised. His symptoms were relieved after receiving thrombolytics, 300 mg aspirin and clopidogrel, intravenous morphine and nitrate. Subcutaneous heparin, beta blocker, angiotensin converting enzyme inhibitor and statins were added later.

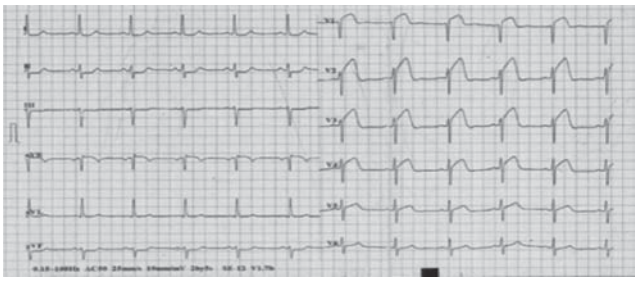

Fig 1. Initial ECG after admission

1. *Assistant Professor, Department of Cardiology, Anwer Khan Modern Medical College & Hospital, Dhaka*
2. *Professor, Department of Cardiology, Dhaka Medical College & Hospital, Dhaka*
3. *Medical Officer, Department of Cardiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka*

Correspondence *Tunaggina Afrin Khan, Email: tunaggina.khan@gmail.com*

His laboratory investigations revealed elevated triglyceride, total cholesterol and LDL cholesterol, normal fasting blood sugar, normal renal and hepatic function, normal complete blood count. Left ventricular septal, anterior wall and apical hypokinesia with 45% ejection fraction, normal ascending aorta and aortic root were found in echocardiography. Coronary angiography revealed a radiolucent linear defect in spiral fashion involving the proximal part of left anterior descending artery suggestive of coronary artery dissection with thrombolysis in myocardial infarction III (TIMI III) flow (Figures 2, 3). The patient became clinically symptom-free and ECG showed complete resolution of ST segment elevation. There was no evidence of compromise in coronary flow. So, we decided to defer any revascularization and continue with medical management including the addition of nifedipine.

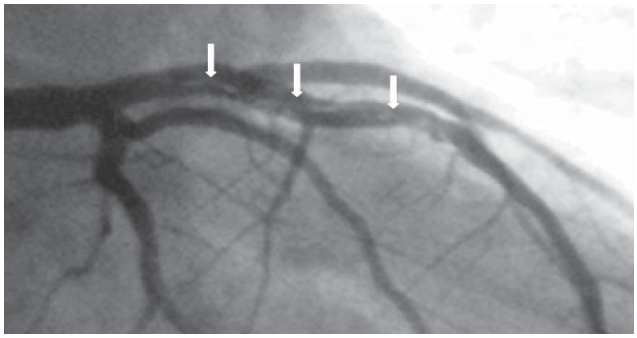

Fig 2. Coronary angiogram showing spiral shaped coronary artery dissection (white arrows) at proximal part of left anterior descending artery

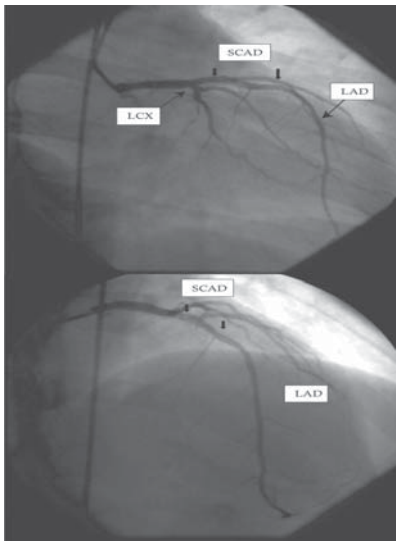

Fig 3. SCAD in proximal LAD (black arrows) with distal TIMI III flow

Before discharge, all the investigations including erythrocyte sedimentation rate, C-reactive protein level, thyroid stimulating hormone level, complement level, anti-nuclear antibody test, anti-ds DNA, anti RNP, anti-Sm, anti Scl-70 antibodies, rheumatoid factor level, perinuclear anti-neutrophil cytoplasmic antibody and centrally accentuated anti-neutrophil tests were found normal. Urine toxicology test did not show the presence of cocaine or amphetamines. He was discharged with advice for cessation of smoking, maintaining healthy lifestyle and taking regular medications. He came after 6, 12 and 24 weeks for further follow-up. He was completely symptom-free and was able to cope up with his daily activities properly.

Discussion

Spontaneous coronary artery dissection (SCAD) is an unusual cause of acute myocardial infarction or sudden cardiac death.[7,13] In 1931 Pretty[14] published the first case and Forker et al[15] reported the first angiographic diagnosis.[16]

It is defined as a primary coronary dissection that occurs in the absence of secondary causes, i.e., propagation of aortic root dissection, cardiac surgery, blunt chest or cardiac trauma, coronary angiography or percutaneous intervention.[7,17]

Majority (75%) of the cases were diagnosed at autopsy.[7,12] False negative results in coronary angiography or spontaneous closure of the dissection also leads to underestimation of true incidence of SCAD.[7,18] Reported angiographic incidence vary between 0.1 and 1.1%.[7,12] This condition affects young women twice as much as men[7,8] and mean age of presentation is 46 years for males and 38 years for females.[7,12]

SCAD usually involves proximal part of a single coronary artery and in most cases (80%) left anterior descending artery is involved. In men, right coronary artery and in women, left coronary artery systems are mainly affected. Left main dissection and multivessel SCAD are more common in females and indicate poor prognosis.[7-9]

The patients who present with SCAD can be divided into three major groups — SCAD occurring during peripartum period, SCAD occurring in association of coronary artery disease (A-SCD), and idiopathic

SCAD.[7,12] About one-third of SCADs occur during peripartum period, but patients presenting as early as 9 weeks of pregnancy and 3 months post-partum had been reported. [7,10,12] A-SCD is not uncommon and in that case intravascular ultrasound (IVUS), optical coherence tomography (OCT) can establish the proper diagnosis.[7,19] The idiopathic SCAD group comprises of heterogenous group of population with some associated predisposing factors, e.g., oral contraceptives, hormone therapy, menstruation, cocaine, cyclosporine, fenfluramine, systemic lupus erythematosus, Ehlers-Danlos syndrome, polyarteritis nodosa, Osler-Weber-Rendu disease, sarcoidosis, fibromuscular dysplasia, polycystic kidney disease, heavy exercise, sleep deprivation, smoking, dyslipidemia. It is weakly associated with hypertension.[1,7]

There are two types of SCADs. One is with intimal tear with propagation of medial dissection, and medial dissection with hematoma formation without intimal tear which occurs due to rupture of vasa vasorum. Intramural hemorrhage is more common, usually involving the outer one-third of the media or between the media and adventitia.[7,8]

SCAD remains an unclear etiopathologic entity and several mechanisms have been proposed. In pregnancy, hormonal and hemodynamic changes, weakening of the vessel wall media because of collagen damage, periadventitial eosinophilic infiltrates may explain the increased susceptibility to SCAD. Multiparity and advanced age may be predisposing factors for SCAD. Nevertheless, peripartum SCAD is an infrequent cause of acute myocardial infarction, complicating around 1 in 100,000 pregnancies.[7,10, 20]

Clinical presentation of SCAD may be simple angina pectoris, unstable angina, non-ST elevation myocardial infarction, ST elevation myocardial infarction, cardiogenic shock, ventricular arrhythmia, heart failure, even sudden cardiac death. Asymptomatic SCAD is extremely rare and only 3 cases had been reported. SCAD may unusually present as ischemic stroke; only 2 cases had been diagnosed till date.[4,5,7,21]

Coronary angiography (CAG) can detect intimal tear but can give false impression of intramural hematoma as coronary atherosclerosis.[7,8,13] Angiographically coronary dissections can be graded as types A to F. Type A dissections represent radiolucent areas within the coronary lumen with no persistence of contrast. Type B dissections are parallel tracts separated by a radiolucent area with no persistence after dye clearance. Type C dissections appear as contrast outside the coronary lumen, with persistence of contrast. Type D dissections represent spiral luminal filling defects, frequently with extensive contrast staining. Type E dissections appear as new, persistent filling defects. Type F dissections represent total occlusion of the coronary artery without distal flow.[22]

IVUS can diagnose SCAD, especially intramural hematoma. It guides correct catheter wire placement, accurate positioning and optimal deployment of coronary stents. It can also differentiate SCAD from atherosclerosis.[23,24] Optical coherence tomography (OCT) is a new technology for visualization of intracoronary details as it has better resolution power compared to IVUS. It prevents excessive or inadequate stent coverage, avoids premature sealing of the intimal flap and propagation of hematoma.[24,25] Multidetector row CT coronary angiography allows detection of intimal tear and intramural hematoma but with lower spatial resolution and somewhat less accuracy than invasive CAG. It may be the optimal imaging method for the follow-up of patients with SCAD in whom a conservative management is initially chosen.[24] Magnetic resonance coronary angiography can also help in diagnosis. Trans-esophageal echocardiography with pulsed and color Doppler flow can detect left main dissection.[7]

There is no guideline regarding optimal treatment of SCAD. The decision is dictated primarily by clinical presentation, extent of dissection and amount of ischemic myocardium at risk. Spontaneous resolution is rare but possible if obstruction of the lumen is incomplete or fatal complications do not occur.[7,8] A conservative medical approach is considered when patients present with clinical and hemodynamic stability showing limited single-vessel involvement without affecting the left main coronary artery. Antiplatelet agents (aspirin, clopidogrel and glycoprotein IIb/IIIa inhibitors) reduce thrombus size in the false lumen and thereby minimize true lumen compression.[7,11] Antiplatelet therapy is given until resolution of the dissection (ranging from four to twelve months).[7] Vasodilator drugs (nitrates, calcium blockers), beta blockers, angiotensin converting enzyme inhibitors, anticoagulants are also added to the therapy.[7,20]

Immunosuppressive agents (prednisone, cytoxan) can be used where the coronary involvement is too extensiv to revascularization considering periadventitial inflammatory cellular infiltration.[7,26]

The use of thrombolytics in SCAD is controversial. Some authors report effectiveness of thrombolytics for lysis of thrombi in false lumen[27] whereas some have reported clinical deterioration after thrombolysis[28] as it favored further extension of the dissection. However, given the extreme rarity of spontaneous dissection, it is not justified to withhold thrombolysis and deny the benefit of this therapy.[7,28]

Intracoronary intervention is recommended when there is evidence of ongoing ischemia and involvement of the left main.[7] Intracoronary stenting is chosen for well-localized dissections, involving single-vessel or (in selected cases) multi-vessel disease, with complete coverage of the lesion.[7-9] Risks involved during PCI include wiring the false lumen, acute closure of the vessel and propagation of the dissection or intramural hematoma and inappropriate stent deployment. A successful 'conservatively invasive' approach is often used. It consists of implantation of a stent only at the proximal edge of the dissection, allowing resolution of acute ischemia, preventing further proximal progression of the dissection but with high risk of in-stent restenosis. Spontaneous left main dissection has mainly been managed surgically, but some propose that stenting may be a safe and effective alternative.[7,29]

Coronary artery bypass grafting (CABG) has been commonly reserved for triple vessel disease, left main involvement or recurrent ischemia in patients who remain unstable despite optimal non-surgical management.[7,8,12] Potential limitations are difficulty in identifying the true lumen, grafting of the false lumen and propagation of dissection.[7,8] Further surgical treatments, such as ventricular assist devices or cardiac transplantation, should be considered when ventricular function is severely impaired and/or there is extensive dissection in multiple vessels, particularly in young patients who suffered from SCAD and previously treated unsuccessfully by CABG.[7,8]

For those who survive the first event, prognosis is favorable, with survival rates averaging 80% at 25–30 months. Nonetheless, short-term mortality following SCAD is higher than with ACS.[7,9,12] Men have higher survival rates than women (93% vs 74%) and women presenting in the peripartum period have a better outcome (15% vs 34%).[9] Prognosis was found to be more favorable in A-SCD due to improved collateral circulation that develops with chronic atherosclerosis.[17]Atherosclerosis also leads to medial atrophy and scarring which protects against propagation of the dissection.[8] In up to half of the patients with SCAD, a second dissection in the same vessel or in another territory may develop, stressing the importance of close follow-up.[9]

Naughton et al[30] reported a case of a 43-year-old female who presented with NSTEMI having SCAD involving mid LAD with total luminal occlusion. She was treated surgically. Monodeep et al[21] reported a case of SCAD of mid LAD with distal TIMI III flow, who presented with ischemic stroke and had severe left ventricular systolic dysfunction and LV apical thrombus. He was alright on conservative management alone. Sudeep et al[31] reported a case of 53-year-old lady who presented with TIA and angiographic evidence of SCAD in proximal LAD and LCX with 90% ostial stenosis of LAD. Revascularization was not done considering severely impaired LV systolic function. Conservative therapy led to remission of SCAD in follow-up angiography. Martinez[32] reported a case of 41-year-old lady who presented with acute STEMI and angiography revealed extensive dissection of LAD. She was treated with PCI with stenting.

SCAD is an unusual cause of coronary occlusion. Without proper diagnostic evaluation it can be easily missed and patients may suffer from serious consequences. Treatment options also differ according to angiographic findings. Though common in female in peripartum period, it can affect male and older female. With timely treatment prognosis is good with complete recovery. Given the rare incidence of SCAD in middle aged man, there is a relative dearth of information regarding the clinical presentation and management. We hope that this case report and the associated literature review will serve to provide valuable insight into this rare yet intriguing disease process.

References

1. Marysia ST, Sharonne NH, Sridevi RP, Robert DS, Amir Lerman, Ryan JL et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation 2012; 126: 579–588.

2. Adlam D, Cuculi F, Lim C, Banning A. Management of spontaneous coronary artery dissection in the primary percutaneous coronary intervention era. J Invasive Cardiol 2010; 22: 549–553.

3. Fontanelli A, Olivari Z, La Vecchia L, Basso C, Pagliani L, Marzocchi A et al. Spontaneous dissections of coronary arteries and acute coronary syndromes: rationale and design of the DISCOVERY, a multicenter prospective registry with a case-control group. J Cardiovasc Med 2009; 10: 94–99.

4. Vanzetto G, Berger-Coz E, Barone-Rochette G, Chavanon O, Bouvaist H, Hacini R et al. Prevalence, therapeutic management and medium-term prognosis of spontaneous coronary artery dissection: results from a database of 11,605 patients. Eur J Cardiothorac Surg 2009; 35: 250–254.

5. Mortensen KH, Thuesen L, Kristensen IB, Christiansen EH. Spontaneous coronary artery dissection: a Western Denmark Heart Registry Study. Catheter Cardiovasc Interv 2009; 74: 710–717.

6. Thompson EA, Ferraris S, Gress T, Ferraris V. Gender differences and predictors of mortality in spontaneous coronary artery dissection: a review of reported cases. J Invasive Cardiol 2005; 17: 59–61.

7. Sílvia MO, Alexandra G, Paula D, Maria JM. Spontaneous coronary artery dissection: a diagnosis to consider in acute coronary syndromes. Rev Port Cardiol 2009; 28(6): 707–713.

8. Khan NU, Miller MJ, Babb JD, Ahmed S, Saha PK, Shammas RL et al. Spontaneous coronary artery dissection. Acute Card Care 2006; 8: 162–171.

9. Kamineni R, Sadhu A, Alpert JS. Spontaneous coronary artery dissection: report of two cases and a 50-year review of the literature. Cardiol Rev 2002; 10: 279–284.

10. Goland S, Schwarz ER, Siegel RJ, Czer LS. Pregnancy associated spontaneous coronary artery dissection. Am J Obstet Gynecol 2007; 197: e11–e13.

11. Choi J, Davidson C. Spontaneous multivessel coronary dissection in a long-distance runner successfully treated with oral antiplatelet therapy: a case report and review of the literature. J Invasive Cardiol 2002; 14: 675–678.

12. DeMaio SJ, Kinsella SH, Silverman ME. Clinical course and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol 1989; 64: 471–474.

13. Basso C, Morgagni GL, Thiene G. Spontaneous coronary artery dissection: a neglected cause of acute myocardial ischaemia and sudden death. Heart 1996; 75: 451–454.

14. Pretty H. Dissecting aneurysms of coronary artery in woman aged 42: rupture. BMJ 1931; 1: 667.

15. Forker AD, Rosenlof RC, Weaver WF, Carveth SW, Reese HE. Primary dissecting aneurysm of the right coronary artery with survival. Chest 1973; 64: 656–658.

16. Fernando A, Manuel P, Vera L, Jaime D, Esther B, Pilar JQ et al. Spontaneous coronary artery dissection: long-term follow-up of a large series of patients prospectively managed with a "conservative" therapeutic strategy. JACC: Cardiovascular intervention 2012; 5(10): 1062–1070.

17. Celik SK, Sagcan A, Altintig A, Yuksel M, Akin M, Kultursay H. Primary spontaneous coronary artery dissections in atherosclerotic patients. Report of nine cases with review of the pertinent literature. Eur J Cardiothorac Surg 2001; 20: 573–576.

18. Hurtado-Martinez JA, Manzano-Fernandez S, Pinar-Bermudez E, Valdes-Chavarri M. Conservative management of spontaneous left main coronary artery dissection. Rev Esp Cardiol 2007; 60: 1103–1104.

19. Hering D, Piper C, Hohmann C, Schultheiss HP, Horstkotte D. Prospective study of the incidence, pathogenesis and therapy of spontaneous, by coronary angiography diagnosed coronary artery dissection. Z Kardiol 1998; 87: 961–970.

20. Koul AK, Hollander G, Moskovits N, Frankel R, Herrera L, Shani J. Coronary artery dissection during pregnancy and the postpartum period: two case reports and review of literature. Catheter Cardiovasc Interv 2001; 52: 88–94.

21. Monodeep B, Arjinder S, Stephen JV. Spontaneous coronary artery dissection: case report and review of literature. Heart Views 2012; 4(12): 149–153.

22. Huber MS, Mooney JF, Madison J, Mooney MR. Use of a morphologic classification to predict clinical outcome after dissection from coronary angioplasty. Am J Cardiol 1991; 68: 467–471.

23. Maehara A. Intravascular ultrasound assessment of spontaneous coronary artery dissection. Am J Cardiol 2002; 89: 466–468.

24. Vrints CJM. Spontaneous coronary artery dissection. Heart 2010; 96: 801–808.

25. Karl P, Brendan B, Owen CR, Darren LW, Ik-Kyung J. Spontaneous coronary artery dissection: utility of intravascular ultrasound and optical coherence tomography during percutaneous coronary intervention. Circ Cardiovasc Interv 2011; 4: e5–e7.

26. Koller PT, Cliffe CM, Ridley DJ. Immunosuppressive therapy for peripartum-type spontaneous coronary artery dissection: case report and review. Clin Cardiol 1998; 21: 40–46.

27. Leclercq F, Messner-Pellenc P, Carabasse D, Lucke N, Rivalland F, Grolleau R. Successful thrombolysis treatment

of a spontaneous left main coronary artery dissection without subsequent surgery. Eur Heart J 1996; 17: 320–321.

28. Zupan I, Noc M, Trinkaus D, Popovic M. Double vessel extension of spontaneous left main coronary artery dissection in young women treated with thrombolytics. Catheter Cardiovasc Interv 2001; 52: 226–230.

29. Petronio AS, De Carlo M, Gistri R, Ciabatti N, Mazzoni A, De Viti D. Successful treatment of a spontaneous coronary dissection causing acute myocardial infarction with stenting of the proximal edge of the dissection: a case documented with intravascular ultrasound. Int J Cardiol 2008; 128(2): e74 –76.

30. Naughton P, Nolke L, Veerasingam D, McCarthy J. Spontaneous coronary artery dissection. Emerg Med J 2005; 22: 910–912.

31. Sudeep T, Jaya B, Satish M, David HS, Prasad M. An unusual case presentation of spontaneous coronary artery dissection. World Journal of Cardiovascular Diseases 2012; 2: 313–315.

32. Martinez LG. Spontaneous coronary artery dissection: a rare threat to young women. Critical Care Nurse 2012; 32(4): e19–e26.