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Coronary Artery Dissection: A comparison of different entities

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Abstract

Coronary artery dissection is one of the challenging situations routinely faced by clinicians in the cardiac catheterization laboratory. Although underappreciated by angiography alone, dissections occur frequently with intervention and are of varied clinical significance.

The dilemma is in the correct diagnosis and appropriate decision making to ensure an optimal clinical outcome. Coronary artery dissection can be spontaneous (Spontaneous Coronary Artery Dissection, SCAD), secondary to the rupture of an atherosclerotic plaque or iatrogenic (as a result of the interventional procedure). This article presents an overview of the definition, classification, etiology, clinical manifestations, and potential complications associated with coronary artery dissections. An incomplete assessment of the clinical significance of coronary dissections can be associated with suboptimal outcomes. We present a comparative overview of accurate and comprehensive assessment and management strategies for different coronary dissections.

***Key Words**: Dissection, Spontaneous Coronary Artery Dissection (SCAD), Intravascular ultrasound (IVUS), Optical Coherence Tomography (OCT).*

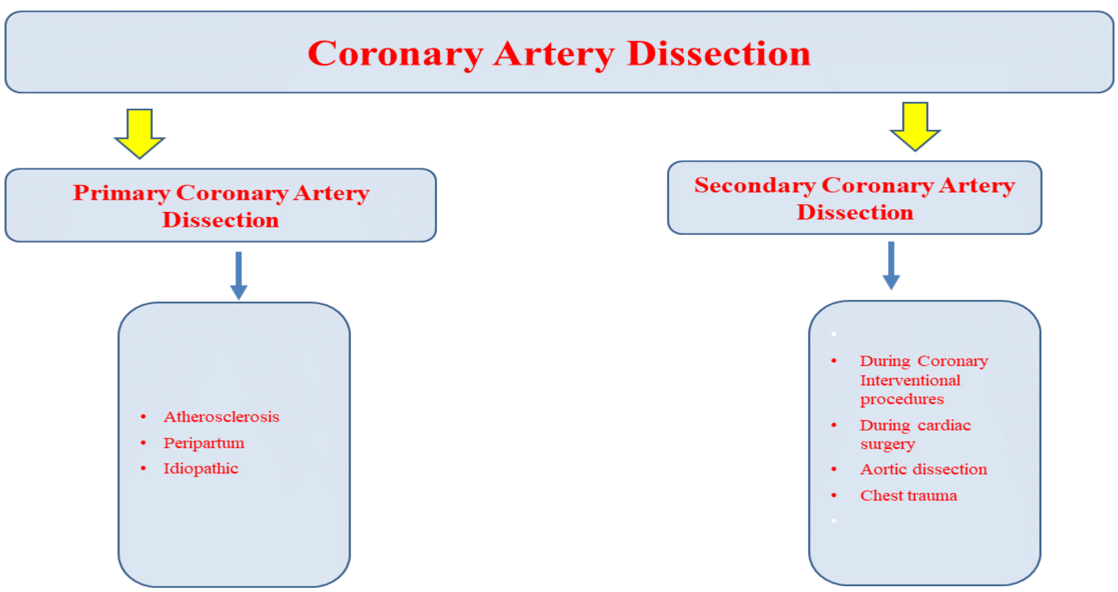
INTRODUCTION

Coronary artery dissection is a condition when a tear occurs in any of the three layers of the arterial wall and in its most severe form, blood can pass through the innermost layer and becomes trapped and bulges inward. This eventually causes narrowing in the lumen of the artery and can lead to a compromise in the blood flow to the myocardium.

Although angiography is routinely performed and considered a gold standard for diagnosis of CAD, [1, 2], coronary artery dissection can be a challenge to diagnose and manage solely based on angiography. [1, 2]

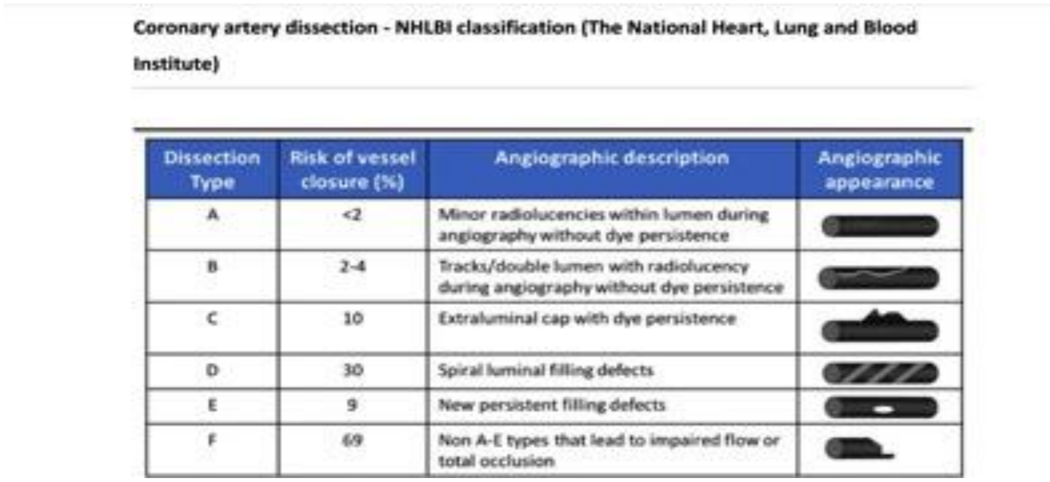
In this article, we provide an overview of how to comprehensively assess a coronary dissection and how best to make a treatment decision by using adjunctive modalities. [3, 4]. Despite the potentially harmful implications of visual estimation techniques, it remains the most commonly used form of evaluation for coronary lesions as well as the coronary artery dissections and is still widely practiced [5]. The limitations of angiography are evident with regards to assessment and decision making for the management of coronary artery dissections.

The differentiation between various etiologies and severity of dissection is important, as the treatment strategies are different in various clinical settings. [Table].



Table

Coronary artery dissection has been categorized from type A to F in the NHLBI classification with risk of vessel closure increases with the complexity of dissection from type A to F as shown in figure 1below.



Coronary artery dissection - NHLBI classification (The National Heart, Lung and Blood Institute)

Dissection Type	Risk of vessel closure (%)	Angiographic description	Angiographic appearance
A	<2	Minor radiolucencies within lumen during angiography without dye persistence	
B	2-4	Tracks/double lumen with radiolucency during angiography without dye persistence	
C	10	Extraluminal cap with dye persistence	
D	30	Spiral luminal filling defects	
E	9	New persistent filling defects	
F	69	Non A-E types that lead to impaired flow or total occlusion	

Figure 1

Plaque Rupture as a Cause of Coronary Artery Dissection:

Pathophysiology:

Plaque rupture, usually of a precursor lesion known as a 'vulnerable plaque' or 'thin-cap fibro atheroma' (TCFA), is the leading cause of thrombosis and can present as a dissection on the angiogram.

A CASE OF PLAQUE RUPTURE CAUSING DISSECTION:

A male patient with a history of hypertension, diabetes mellitus, and hyperlipidemia presented with NSTEMI-ACS. His echocardiogram showed mild left ventricular systolic dysfunction. His cardiac catheterization showed no significant stenosis in the left coronary system, however, a dissection flap was found in a large caliber, dominant right coronary artery. Angiogram image is shown below in

Figure 2.

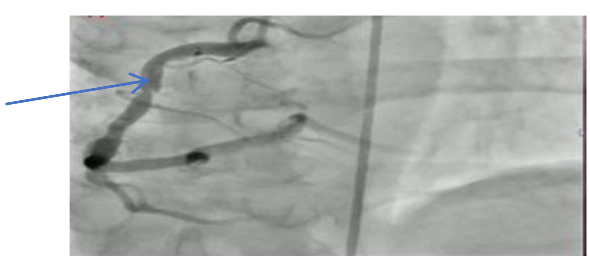
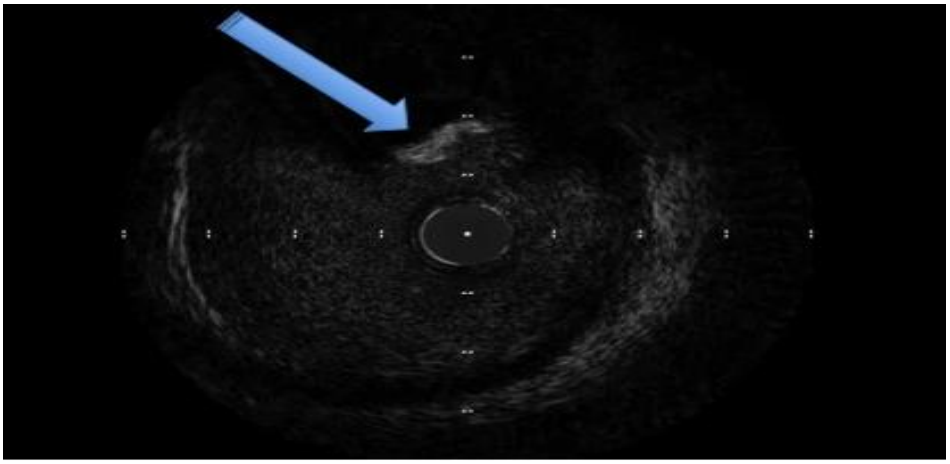


Figure 2: A dissection flap (arrow) in a large caliber, dominant right coronary artery.

An IVUS was also performed which revealed a ruptured plaque and was responsible for the acute presentation of the patient. [Figure 3].



[Figure 3]: IVUS showing a dissection flap (arrow)

The lesion was successfully treated with a Self-apposing stent, which resulted in complete sealing of the plaque and dissection flap. [Figure 4 a,b. Below].

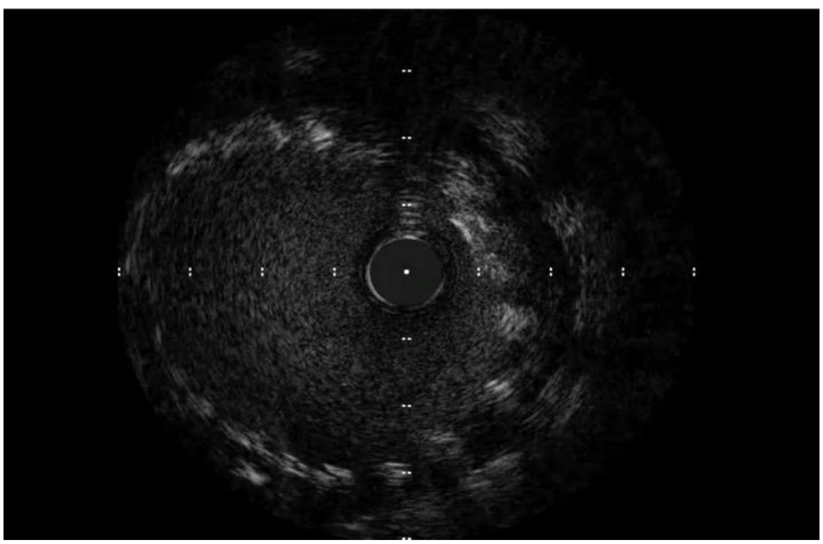


Figure 4a

Figure 4b

FACTORS ASSOCIATED WITH PLAQUE RUPTURE:

A variety of factors have been attributed to the mechanisms leading to plaque rupture and the appearance of dissection on the angiogram.

These include increased shear stress injury,[6] turbulent plaque injury,[7] transient collapse of the stenosis,[8] rupture ofvasa vasorum,[9] and circumferential stress within the plaque.[10] Contemporary literature supports that an inflammatory process plays a crucial role in the mechanism of plaque rupture.[11]

IATROGENIC DISSECTION

ETIOLOGY:

Iatrogenic dissection occurs during a coronary angiogram or percutaneous coronary intervention. Normally this is hardware induced and could be due to catheter, wire, balloon, or stents or various other adjunctive equipment used during coronary interventions including guide extension catheters, imaging probes, 'mother-and-child catheters, and atherectomy devices.

PATHOPHYSIOLOGY:

Guide catheters may induce dissections in the ostio-proximal segment of the vessel and may occur with increased incidence with the use of guide extension catheters. These may occur when the guide catheters are not coaxial to the vessel. Small aortic root dimensions and aggressive manipulations of the catheters can increase the chances of these dissections. These dissections can extend proximally into the aortic root or propagate down the coronary artery.

INCIDENCE:

Guide catheter induced dissection is common in the cases where there is ostial disease and aggressive, large size, supportive guide catheters are used.[12] The exact incidence remains unknown, with some suggesting it may be underreported. [13]

A CASE EXAMPLE OF IATROGENIC DISSECTION:

A young female patient presented with ongoing symptoms of chest pain and was evaluated by coronary angiography. (Figure 5).

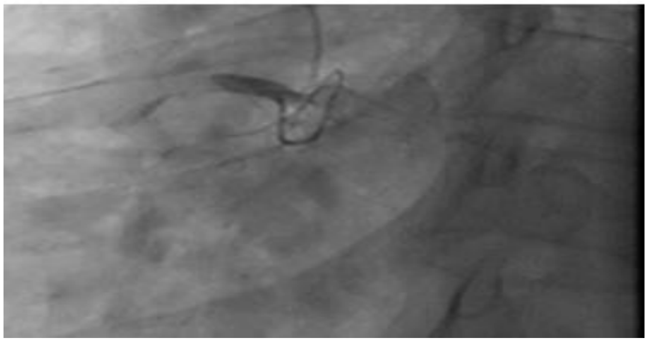


Figure 5: A case of catheter induced RCA dissection and complete occlusion of artery.

CLINICAL SIGNIFICANCE:

During balloon angioplasty, coronary dissection is inevitable and occurs nearly universally. This however often is limited to small intimal dissections that are not evident angiographically and of no clinical significance when covered with a stent. This has been recognized more recently with high-resolution intravascular imaging such as optical coherence tomography. Increased sensitivity for the detection of dissections with OCT has allowed for stratification based on severity to determine management strategy.

Invasive physiologic assessment often involves coronary wires with reduced steerable dexterity and there is a risk of dissection. For instance, in the ORBITA study 4% of patients undergoing physiologic assessment that were randomized to undergo conservative management, crossed over to PCI due to wire-related dissections.[14] Following stent implantation, edge dissections may frequently occur.[15] The overall incidence of edge dissections following stent implantation as assessed by OCT has been reported as 37.8%, with 84% not detectable by angiography.[16] There is a relationship between plaque-type and risk for edge dissection. Edge dissections may be minimized when a stent edge is implanted in normal or fibrous tissue and by avoiding implanting the edge of a stent within a region of lipid plaque with TCFA.

PREVENTION:

During the engagement of guide catheters, extra care needs to be taken to ensure the coaxial engagements and an operator should always monitor the arterial waveform to ensure that there is no ventricularization or damping in the aortic pressure waveform. The tracing below is to show a case of Coronary Ventricularization which was identified and corrected by careful catheter removal. (Courtesy Dr. Raufi)

Dissection associated with a guide catheter can be asymptomatic but it can lead to disastrous consequences and sometimes it necessitates urgent revascularization.

MANAGEMENT:

Coronary dissections caused by balloon angioplasty, intracoronary imaging probes, and other equipment into the vessel can similarly have varied consequences. Stents can be used to easily seal dissection flaps to prevent acute or abrupt vessel closure. If dissection involves the ostio-proximal segment of a vessel close to the aortic root, adequate sealing of the ostium with an appropriately expanded stent is important to minimize the risk of retrograde propagation. Small asymptomatic dissections (types A and B) may settle conservatively.

The dissections related to balloons or stents may be seen at both ends of the device. The stent-related edge dissection is a known phenomenon. Unless there is less than 20 degrees edge dissection on IVUS, most of these edge dissections need to be treated by further stents as if untreated, may progress to the reduction of flow.

The dissections related to wire almost unanimously related to the distal segments and may complicate with perforations. If it happens, the diagnosis needs to be made immediately by echocardiogram. Due to the small size of the segments, treatment may be challenging and if the severity has extended to a coronary perforation, may require implantation of coils. From the precautionary standpoint, a continuous check on the wire position while performing angioplasty is mandatory. This becomes more important while maneuvering balloons or stents in complex anatomy, particularly in tortuous vessels.

SPONTANEOUS CORONARY ARTERY DISSECTION:

DEFINITION:

Spontaneous Coronary Artery Dissection (SCAD) is a non-traumatic and non-iatrogenic separation of the coronary arterial walls, creating a false lumen [2, 17].

INCIDENCE:

SCAD has a possible incidence of up to 1% to 4% of acute coronary syndrome (ACS) cases. [18-20]. This incidence can be high particularly in the cases of pregnancy-associated myocardial infarction (MI) [21-23].

PATHOPHYSIOLOGY:

The separation of the coronary arterial walls in SCAD can occur between the intima and media or between the media and adventitia, with intramural hematoma (IMH) formation within the arterial wall that compresses the arterial lumen, decreasing antegrade blood flow with subsequent myocardial ischemia or infarction [2, 17]. The dissecting hematoma is located in the outer third of the tunica media and produces luminal occlusion by forcing the inner media against the opposing wall. Blood filling the false lumen may simulate a coronary thrombosis to the naked eye. Thus, the real incidence of this entity may be underestimated at autopsy, unless a careful histologic examination of the coronary artery is done. In comparison with aortic dissections, an intimal tear is difficult to observe in spontaneous cases, suggesting that the source of blood may be through vasa vasorum bleeding. Multivessel SCAD may occur in up to 23% of cases [18, 24, 25].

A variety of conditions have been observed to predispose the patients to SCAD and one of the important considerations to note is that fibromuscular dysplasia (FMD) and extra-coronary vascular abnormalities may be commonly found in patients with SCAD. [17, 18, 23, 26, 27]

Pregnancy and sex hormones are similarly associated with predisposition to SCAD [23, 28, 29]. In this context, we present an example of a post-pregnancy patient who presented with acute coronary syndrome; however, her cardiac catheterization revealed SCAD. (Figure 6).

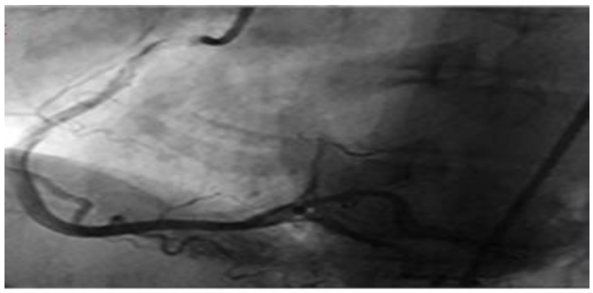


Figure 6: An example of Spontaneous Coronary Artery Dissection post pregnancy presenting as Acute Coronary Syndrome

A case of widespread Spontaneous Coronary Artery Dissection is mentioned below.

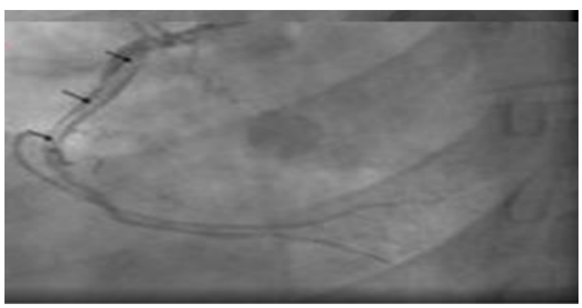


Figure 7: A case of widespread Spontaneous Coronary Artery Dissection

Similarly, there is an association reported between SCAD and sarcoidosis (23,30). Genetic predisposition has also been reported. [18, 23] The term SCAD is synonymous with non-atherosclerotic SCAD to differentiate from the atherosclerotic SCAD due to its peculiar pathophysiology features.[23] [33, 34].

ANGIOGRAPHIC AND OTHER IMAGING DIAGNOSIS OF SCAD:

Despite the inherent limitations of conventional coronary angiography that reduces the diagnostic capability for coronary dissection, this is still the most common test performed for the diagnosis of

SCAD. The limiting factor is 2-dimensional “luminography” making the operator not fully able to image the arterial wall.

Advances in interventional cardiology and particular intracoronary imaging modalities including intravascular ultrasonography and optical coherence tomography, provide detailed visualization of the arterial wall that aids the diagnosis of various types of coronary dissection. Furthermore, intravascular imaging goes beyond simple diagnosis and provides detailed information on the severity that can be deterministic of the treatment approach. Despite the advantages of intracoronary imaging, there are limitations due to additional risks of propagating the dissection, costs, expertise in interpretation and they are not readily available in all catheterization laboratories worldwide. As a consequence, conventional coronary angiography remains instrumental in diagnosing coronary artery dissections and in particular the SCAD, and cardiologists should become proficient at recognizing its various angiographic patterns. The pathognomonic appearance of SCAD as a radiolucent lumen with extraluminal contrast staining is not seen in all cases.

Several studies have reported that the diffuse smooth stenosis pattern (i.e., type 2) is the most common angiographic manifestation of SCAD, occurring in up to 67.0% of dissected arteries, followed by type 1 in 29.1% and type 3 in 3.9%.[31]

Thus, reliance solely on the traditional multiple lumens or contrast staining of arterial walls to diagnose SCAD might result in missing a diagnosis in more than 70% of SCAD cases. Therefore, familiarity with the diffuse narrowed-lumen appearance of IMH and the use of intracoronary imaging are important steps to improve the diagnosis of SCAD. An association of SCAD with Takotsubo syndrome has been reported in literature. [26, 32, 33]

VARIOUS PATTERNS OF SCAD:

Three different patterns of SCAD have been characterized in the literature. [26]

(I) Type 1 is a condition where an obvious arterial wall strain is seen on angiography. This was considered as pathognomonic in the SCAD series where multiple radiolucent staining of the arterial wall is seen.

(II) Type 2 depicts a condition where diffuse stenosis of varying severity is seen.

This appearance can be misleading and can get confused with normal coronary stenosis.

SCAD can involve the mid to distal segments and can be so extensive that it can involve the entire coronary tree

(III) Type 3 can present as atherosclerosis of the artery and can be very challenging to diagnose and treat.

ANGIOGRAPHIC FEATURES FAVORING SCAD:

(i) Lack of atherosclerotic changes in other coronary arteries;

(ii) Long lesions (11-20 mm);

(iii) Hazy stenosis; and

(iv) Linear stenosis.

There are no apparent differences in clinical presentations among the three angiographic classifications of SCAD. Type 1 SCAD should be easily diagnosed with angiography. Type 2 SCAD can be distinctive enough for diagnosis by angiography due to the familiar classic pattern. However, some type 2 cases (e.g., shorter lengths 20-30 mm) will require intracoronary imaging or repeat angiography to make the diagnosis. Type 3 SCAD is often indistinguishable from atheroma and requires intracoronary imaging for diagnosis.

ROLE OF INTRACORONARY IMAGING IN DIAGNOSIS OF VARIOUS TYPES OF DISSECTIONS:

The utility of intracoronary imaging has greatly helped us in the detailed understanding of various dissections and strategies for the best management of these conditions.[34]

In this regard, IVUS has a very high penetration power and can be used for the differentiation of various dissections.[35] One of the advantages of using IVUS is not using contrast material which may be helpful in preventing the propagation and extension of dissection.[36] Due to the higher penetration power of IVUS, the extent of the dissection process may be studied better. However, due to the low resolution of IVUS systems, IVUS can help to differentiate between the false and true lumen and can help localize the intimal tear. (Figure 8)

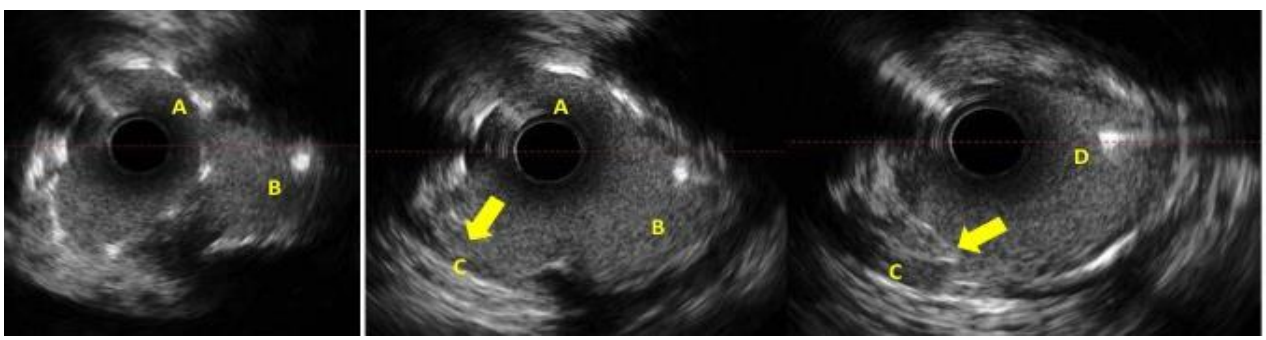
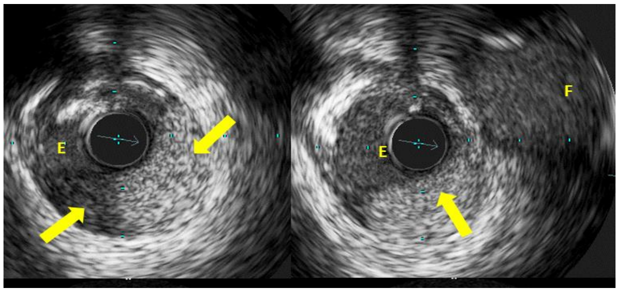


Figure 8: IVUS showing dissection flap of an ostial LAD. (A) Ostial LAD, (B) Ostial LCx, (C) Dissection with Flap, (D) Left Main.

In clinical settings whenever there is suspicion of dissection on angiography, IVUS can be used to locate the false lumen and it can help facilitate directing the wire into the true lumen. IVUS is superior in many cases of dissection including left main stem (LMS) dissection.**[**35**]** **[**1**]** In these cases, IVUS does not require the use of contrast and it avoids the hydraulic extension of dissection to other arterial trees.**[**36**]**

IVUS can give detailed information about the dissected plaques **[**37**].** (Figure 9)



(Figure 9) Intramural Hematoma, compression of the lumen by the echogenic hematoma. Lumen (E). Branch (F)

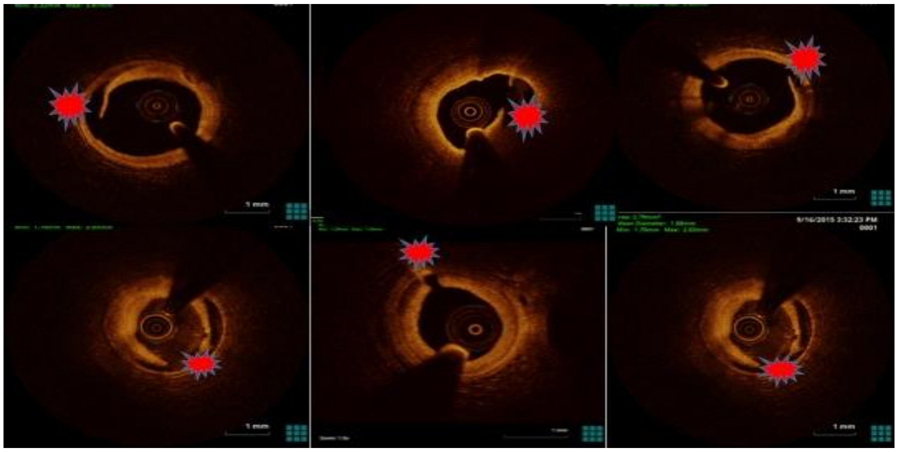
In the cases involving the LMS dissection, IVUS can help to locate the external elastic lamina (EEL) due to high penetration in comparison to other imaging modalities including OCT. **[**1**]**

Large- scale registry data have shown that there is an advantage of OCT in detailed coronary artery imaging and it can be utilized for assessment for the plaque. **[**37, 38**]**

OCT however uses a contrast medium to create a bloodless field and hence can cause hydraulic extension of a dissection. This restricts its potential usage when a large dissection is apparent angiographically despite having excellent imaging resolution.

There is not much information about the role of physiological assessment to help in the decision-making process.

It may be deduced from the literature to have a low threshold of suspicion regarding the presence of dissection and appropriate timing of treatment needs to be decided accordingly. Besides, an appropriate imaging modality, if available, may help in decision-making **[**34**].** An example of use of OCT is mentioned below in Figure 10.



(Figure 10): Various images of coronary dissection as seen on OCT. The high resolution allows for clear identification of the dissection flap.

MANAGEMENT OF SCAD:

The management of SCAD is dependent on the early and prompt diagnosis.

Medical therapy:

The medical management is considered on scenarios where the patient is clinically stable or in cases where revascularization is not feasible.

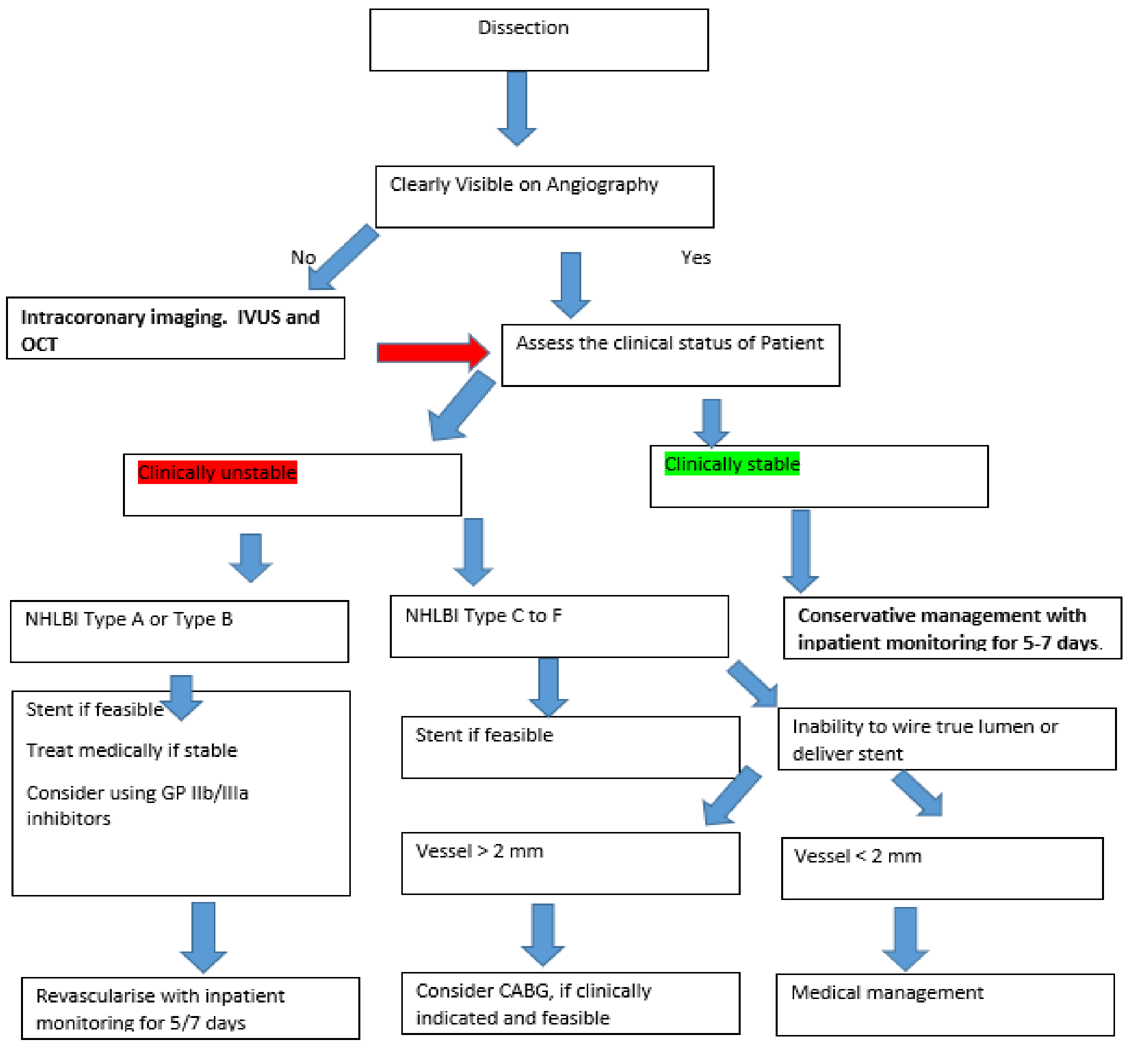
There is no clear evidence that traditional medical therapies used in the treatment of ACS have any beneficial impact. Aspirin and clopidogrel have been used for the treatment of SCAD. [34] There is no clear evidence for use of potent P2Y12 antagonists (prasugrel and ticagrelor) for the treatment of SCAD. Anticoagulant agents, like heparin are usually discontinued after confirmed diagnosis of SCAD to avoid the extension of IMH. Beta-blockers have shown evidence to reduce arterial shear stress and can potentially be used in cases of SCAD.[33] The role of ACE inhibitors and statins is not clear.

Revascularization Therapy:

The decision to revascularize in cases of SCAD can be very challenging and it depends on the patient's clinical status, affected coronary anatomy, and area of myocardium at risk.[23]

In those cases where the patient is stable without ongoing pain, it is reasonable to continue with conservative management and in-patient monitoring.

The decision of revasculariztion is based on patient's overall condition of ischemic chest pain and hemodynamic instability in which case revascularization strategy is recommended provided major arteries are affected and the technical feasibility is available **[**23**][**34**]**. This concept is explained in the Algorithm below.



ALGORITHM

PCI in these cases may be challenging and the long-term outcomes are often not good. **[**23**]** The wiring in these dissected arteries can be a challenge in order to navigate into distal true lumen. The IMH of the dissected segment can also propagate antegradely or retrogradely during the procedure and making the situation worse.

Thus, it is recommended that PCI to be considered for patients with ongoing chest pain and ischemia when the lesion is amenable to stenting, and to consider CABG for extensive dissections involving the left main.[23]

Conclusion:

In this review article, we have discussed coronary artery dissection, its etiology, and various types. Several images outlining the various types of dissection are also mentioned. The algorithm mentioned, provides a step-by-step approach for the reader to understand the pathophysiology and treatment of the underlying disorder. A lack of case series without a follow up may pose a limitation to our manuscript but it is beyond the scope of this article.

References:

1. Parviz, Y., et al., “Utility of intracoronary imaging in the cardiac catheterization laboratory: comprehensive evaluation with intravascular ultrasound and optical coherence tomography”. Br Med Bull, 2018. 125(1): p. 79-90.

2. Saw, J., “Spontaneous coronary artery dissection”. Can J Cardiol, 2013. 29(9): p. 1027-33.

3. Leape, L.L., et al., “Effect of variability in the interpretation of coronary angiograms on the appropriateness of use of coronary revascularization procedures”. Am Heart J, 2000. 139(1 Pt 1): p. 106-13.

4. Girasis, C., et al., “Validity and variability in visual assessment of stenosis severity in phantom bifurcation lesions: a survey in experts during the fifth meeting of the European Bifurcation Club”. Catheter Cardiovasc Interv, 2012. 79(3): p. 361-8.

5. Fleming, R.M., D.M. Fleming, and R. Gaede, “Training physicians and health care providers to accurately read coronary arteriograms”. A training program. Angiology, 1996. 47(4): p. 349- 59.

6. Gertz, S.D. and W.C. Roberts, “Hemodynamic shear force in rupture of coronary arterial atherosclerotic plaques”. Am J Cardiol, 1990. 66(19): p. 1368-72.

7. Loree, H.M., et al., “Turbulent pressure fluctuations on surface of model vascular stenoses”. Am J Physiol, 1991. 261(3 Pt 2): p. H644-50.

8. Binns, R.L. and D.N. Ku, “Effect of stenosis on wall motion. A possible mechanism of stroke and transient ischemic attack”. Arteriosclerosis, 1989. 9(6): p. 842-7.

9. Barger, A.C., et al., “Hypothesis: vasa vasorum and neovascularization of human coronary arteries”. A possible role in the pathophysiology of atherosclerosis. N Engl J Med, 1984. 310(3): p. 175-7.

10. Richardson, P.D., M.J. Davies, and G.V. Born, “Influence of plaque configuration and stress distribution on fissuring of coronary atherosclerotic plaques”. Lancet, 1989. 2(8669): p. 941-4.

11. van der Wal, A.C., et al., “Site of intimal rupture or erosion of thrombosed coronary atherosclerotic plaques is characterized by an inflammatory process irrespective of the dominant plaque morphology”. Circulation, 1994. 89(1): p. 36-44.

12. Boyle, A.J., et al., “Catheter-induced coronary artery dissection: risk factors, prevention and management”. J Invasive Cardiol, 2006. 18(10): p. 500-3.

13. Brinker, J.A., Geeez! Oh my God! Oops! #&*&! Cathet Cardiovasc Diagn, 1998. 43(3): p. 280- 1.

14. Al-Lamee, R., et al., “Percutaneous coronary intervention in stable angina (ORBITA): a double-blind, randomised controlled trial”. Lancet, 2018. 391(10115): p. 31-40.

15. Guo, J., et al., “Optical coherence tomography assessment of edge dissections after drug- eluting stent implantation in coronary artery”. Chin Med J (Engl), 2012. 125(6): p. 1047-50.

16. Chamie, D., et al., “Incidence, predictors, morphological characteristics, and clinical outcomes of stent edge dissections detected by optical coherence tomography”. JACC Cardiovasc Interv, 2013. 6(8): p. 800-13.

17. Saw, J., et al., “Spontaneous coronary artery dissection: prevalence of predisposing conditions including fibromuscular dysplasia in a tertiary center cohort”. JACC Cardiovasc Interv, 2013. 6(1): p. 44-52.

18. Tweet, M.S., et al., “Clinical features, management, and prognosis of spontaneous coronary artery dissection”. Circulation, 2012. 126(5): p. 579-88.

19. Alfonso, F., et al., “Spontaneous coronary artery dissection: long-term follow-up of a large series of patients prospectively managed with a "conservative" therapeutic strategy”. JACC Cardiovasc Interv, 2012. 5(10): p. 1062-70.

20. Alfonso, F. and T. Bastante, “Spontaneous coronary artery dissection: novel diagnostic insights from large series of patients”. Circ Cardiovasc Interv, 2014. 7(5): p. 638-41.

21. Saw, J., “Pregnancy-Associated Spontaneous Coronary Artery Dissection Represents an Exceptionally High-Risk Spontaneous Coronary Artery Dissection Cohort”. Circ Cardiovasc Interv, 2017. 10(3).

22. Saw, J., Reply: “Pregnancy-Associated Coronary Artery Dissection: A Therapeutic Dilemma”. J Am Coll Cardiol, 2018. 71(4): p. 470-471.

23. Saw, J., et al., “Spontaneous coronary artery dissection: association with predisposing arteriopathies and precipitating stressors and cardiovascular outcomes. Circ Cardiovasc Interv, 2014. 7(5): p.645-55.

24. Rogowski, S., et al., “Spontaneous Coronary Artery Dissection: Angiographic Follow-Up and Long-Term Clinical Outcome in a Predominantly Medically Treated Population”. Catheter Cardiovasc Interv, 2017. 89(1): p. 59-68.

25. Vijayaraghavan, R., et al., “Pregnancy-related spontaneous coronary artery dissection”.Circulation, 2014. 130(21): p. 1915-20.

26. Saw, J., “Coronary angiogram classification of spontaneous coronary artery dissection”. Catheter Cardiovasc Interv, 2014. 84(7): p. 1115-22.

27. Prasad, M., et al., “Prevalence of extracoronary vascular abnormalities and fibromuscular dysplasia in patients with spontaneous coronary artery dissection”. Am J Cardiol, 2015. 115(12): p. 1672-7.

28. Tweet, M.S., et al., “Spontaneous coronary artery dissection: revascularization versus conservative therapy”. Circ Cardiovasc Interv, 2014. 7(6): p. 777-86.

29. Mortensen, K.H., et al., “Spontaneous coronary artery dissection: a Western Denmark Heart Registry study. Catheter Cardiovasc Interv”, 2009. 74(5): p. 710-7.

30. Kanaroglou, S., V. Nair, and J.R. Fernandes, “Sudden cardiac death due to coronary artery dissection as a complication of cardiac sarcoidosis”. Cardiovasc Pathol, 2015. 24(4): p. 244-6.

31. Saw, J., G.B.J. Mancini, and K.H. Humphries, “Contemporary Review on Spontaneous Coronary Artery Dissection”. J Am Coll Cardiol, 2016. 68(3): p. 297-312.

32. S, Y.H., R. Themudo, and E. Maret, “Spontaneous coronary artery dissection and takotsubo syndrome: The chicken or the egg causality dilemma. Catheter Cardiovasc Interv”, 2017. 89(7): p. 1215-1218.

33. S, Y.H., “Spontaneous coronary artery dissection and takotsubo syndrome: An often overlooked association; review”. Cardiovasc Revasc Med, 2018.

34. Yip, A. and J. Saw, “Spontaneous coronary artery dissection-A review. Cardiovasc Diagn Ther”, 2015. 5(1): p. 37-48.

35. Mintz, G.S., et al., "American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies (IVUS). A report of the American College of Cardiology Task Force on Clinical Expert Consensus Documents. J Am Coll Cardiol, 2001. 37(5): p. 1478-92.

36. Mariani, J., Jr., et al., "Intravascular ultrasound guidance to minimize the use of iodine contrast in percutaneous coronary intervention: the MOZART (Minimizing cOntrast utiliZation With IVUS Guidance in coRonary angioplasTy) randomized controlled trial. JACC Cardiovasc Interv, 2014. 7(11): p. 1287-93.

37. Ali, Z.A., et al., "Optical coherence tomography compared with intravascular ultrasound and with angiography to guide coronary stent implantation (ILUMIEN III: OPTIMIZE PCI): a randomised controlled trial. Lancet", 2016. 388(10060): p. 2618-2628.

38. Ali, Z.A., et al., "Outcomes of Optical Coherence Tomography Compared With Intravascular Ultrasound and With Angiography to Guide Coronary Stent Implantation: One-Year Results from the ILUMIEN III: OPTIMIZE PCI trial". EuroIntervention, 2020.