JACC: CASE REPORTS VOL. 29, 2024

VASCULAR DISEASE

CASE REPORT: CLINICAL CASE: DAVINCI CORNER

Spontaneous Coronary Artery Dissection Resulting in Acute Myocardial Infarction With Cardiac Rupture

Takamasa Tanaka, MD,[a] Ling Li, MD,[b,c] Stephanie A. Dean, MD,[b] Kenji Kawai, MD,[a] Rika Kawakami, MD,[a] Robert Kutys, MS,[a] Thomas Blanchard, PHD,[c] Renu Virmani, MD,[a] Aloke V. Finn, MD[a,c]

ABSTRACT

Spontaneous coronary artery dissection occurs predominantly in women and is associated with fibromuscular dysplasia. We illustrate a rare case of sudden coronary death as a result of cardiac rupture from spontaneous coronary artery dissection in a 54-year-old man without fibromuscular dysplasia. Cardiac rupture has been previously reported in 6 cases, mostly in women. (J Am Coll Cardiol Case Rep 2024;29:102196) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Spontaneous coronary artery dissection (SCAD) is a well-known important cause of acute myocardial infarction and is considered a rare cause of sudden coronary death especially due to mechanical complications such as left ventricular (LV) wall rupture. SCAD has been described largely in women <50 years of age and is associated with pregnancy.[1] The presence of arteriopathy such as fibromuscular dysplasia (FMD) has been reported to occur in 28% to 56% of patients with SCAD.[2] Here, we present a rare case of sudden cardiac death while exercising from LV wall rupture secondary to SCAD in a man without FMD.

LEARNING OBJECTIVES

- To recognize that SCAD can occur in males and is not always associated with FMD.
- To understand that cardiac rupture due to acute transmural myocardial infarction in SCAD is rare.

CASE PRESENTATION

A 54-year-old man without significant cardiac history collapsed while exercising in a gymnasium and became unresponsive. The emergency medical services responded to the scene; however, the subject could not be resuscitated. The subject had a history of small aneurysmal dilatation of the proximal celiac artery, the superior mesenteric artery, and renal arteries. He had median arcuate ligament syndrome with past abdominal surgical ligament release and reported no history of any trauma or drug abuse. The patient's wife described a recent history of chest pain of no specific duration. At autopsy, approximately 250 mL of blood and blood clots were present in the pericardial sac. Gross examination showed cardiac rupture of the posterior apical LV wall, 3 cm in length with ecchymosis (**Figures 1A and 1B**). The histologic

From the [a]CVPath Institute, Gaithersburg, Maryland, USA; [b]Office of the Chief Medical Examiner, Baltimore, Maryland, USA; and the [c]University of Maryland School of Medicine, Baltimore, Maryland, USA.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received October 19, 2023; revised manuscript received November 15, 2023, accepted November 22, 2023.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2023.102196

ABBREVIATIONS AND ACRONYMS

FMD = fibromuscular dysplasia

LV = left ventricle/ventricular

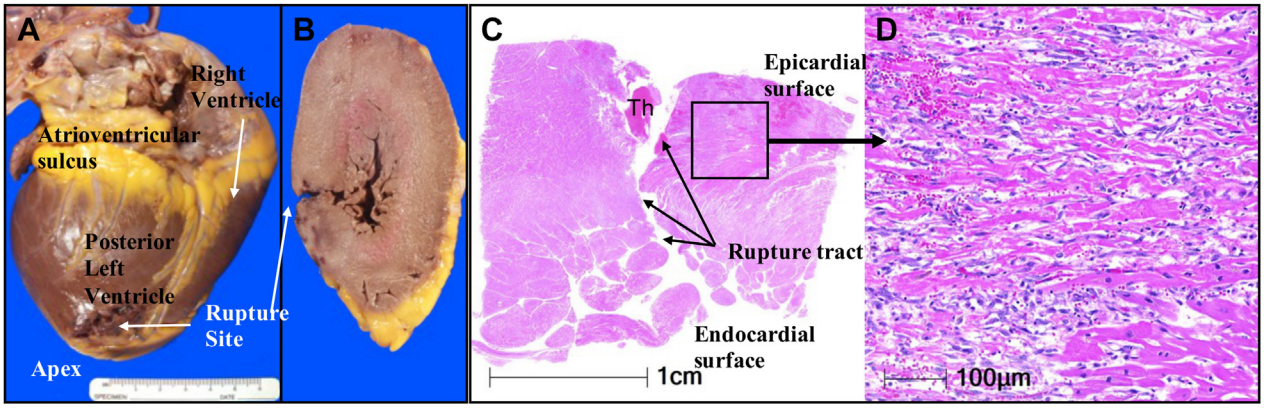
SCAD = spontaneous coronary artery dissection

section of the posterior wall of the LV surrounding the rupture site showed an acute transmural myocardial infarction with macrophage infiltration and early fibroblast proliferation, consistent with an infarct of 5 to 7 days' duration (Figures 1C and 1D). Epicardial coronary artery examination revealed a right-dominant coronary system with the presence of spontaneous dissection involving the mid and distal left circumflex artery with completed occlusion of the lumen from a hematoma in the false lumen. The rest of the coronary tree showed focal areas of luminal irregularity without any significant coronary atherosclerosis. Histologic sections showed the presence of dissection at the medial adventitial border with adventitial chronic inflammation consisting of eosinophils, lymphocytes, and macrophages (Figure 2). The left obtuse marginal branch of the circumflex artery showed replacement fibrosis of the media from a focal healed coronary dissection (Figure 3). The rest of the coronary tree showed absence of FMD (Figure 4), and the renal, superior, and inferior mesenteric arteries did not show any abnormality.

DISCUSSION

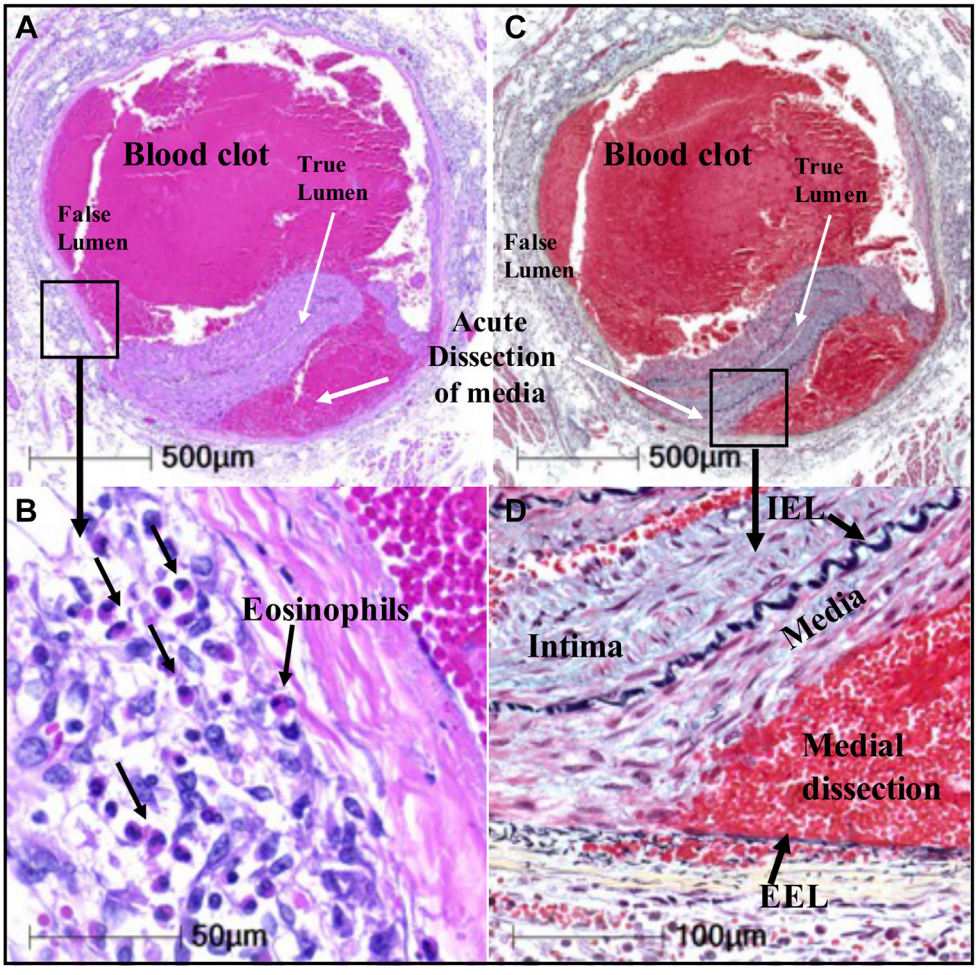
Our male patient had acute SCAD with LV wall rupture and had healed coronary artery dissection of the media without FMD. Recent studies report that SCAD is responsible for 1% to 4% of all acute coronary syndromes. Of SCAD patients presenting with clinical symptoms, approximately 28% present with ST-segment elevation myocardial infarction.[2] Overall, the incidence of mechanical complications is reported as 0.27% of all cases presenting with ST-segment elevation myocardial infarction.[3] Sudden cardiac death is rare in patients with acute SCAD. A recent paper reported 6 cases of mechanical complications related to SCAD (5 female and 1 male) with 2 cases from papillary muscle rupture, 1 with ventricular septal perforation, and 3 with anterior wall rupture.[4] The 1 male patient survived the anterior wall rupture. In our case, SCAD caused a myocardial infarction leading to LV wall rupture that led to the patient's demise. Although the pathophysiology of SCAD is unclear, various reports show that SCAD is caused by a combination of factors that relate to the sex of the individual, occurring mostly in females, especially during the peripartum period, perhaps due to hormonal imbalance, and coexistent arteriopathy. It is likely that factors that predispose to SCAD in men are different from those in women. One-third of male SCAD cases are associated with FMD, while 40% are linked to prior isometric exercise.[2] It has been reported that SCAD may be missed even at autopsy; therefore, careful assessment of coronary arteries with histopathologic examination is important for accurate diagnosis.[5] Saw et al[6] reported a 3-year follow-up of 750 cases of SCAD. Mortality was

FIGURE 1 The Gross and Histological Section of the Cardiac Rupture Site at the Posterior Left Ventricle



(A) Gross image of the heart showing cardiac rupture on the posterior surface of the left ventricle, near the apex. (B) Apical slice of the left ventricle showing rupture site with surrounding mottled appearance of the myocardium. (C) Histologic section (hematoxylin and eosin stain) of the site of rupture showing acute myocardial infarction in the surrounding posterior left ventricular wall. (D) A high-power image of the boxed area in panel C showing myocyte necrosis with fibroblast infiltration and focal macrophage infiltration changes consistent with a 5- to 7-day-old myocardial infarction. Th = thrombus.

FIGURE 2 Mid Left Circumflex Artery



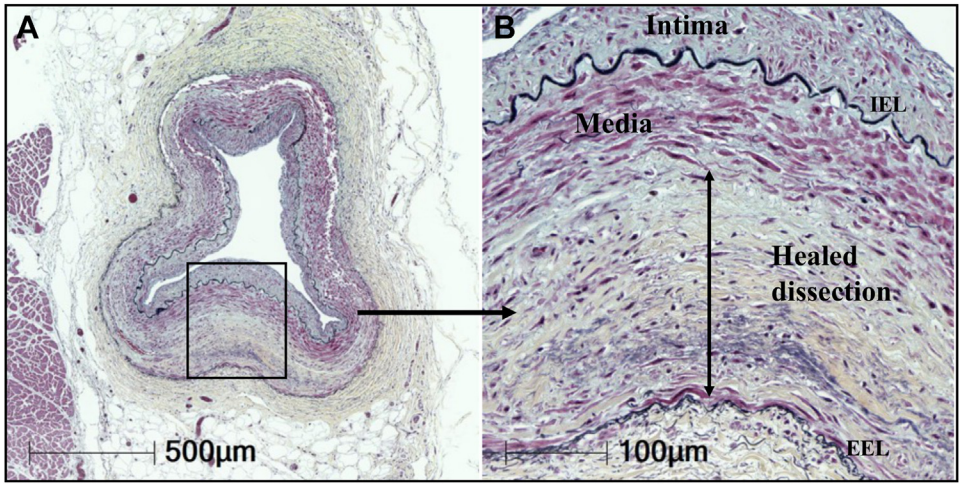
(A) Mid left circumflex artery with acute spontaneous coronary artery dissection (hematoxylin and eosin). (B) High-power images of the boxed area in panel A showing adventitial inflammatory infiltrate consisting of eosinophils (arrows), macrophages, and a few lymphocytes and scattered fibroblasts. (C) The adjoining section as in panel A stained with Movat pentachrome stain of the left circumflex artery with complete occlusion of the lumen from the hematoma present in the false lumen created by the medial adventitial dissection. (D) High-power image from panel C; note that the dissection is at the medial adventitial border. EEL= eternal elastic lamina; IEL = internal elastic lamina.

0.8%, recurrent myocardial infarction was 9.9% (extension of previous SCAD 3.5%, de novo recurrent SCAD 2.4%, iatrogenic dissection 1.9%), and the overall major adverse cardiac events rate was 14.0%.[6] Tweet et al,[7] from the Mayo Clinic, reported a 10-year survival rate of 92% in patients with SCAD. The prevalence of recurrent SCAD is variably reported as 29%.[8] Our subject demonstrated healed SCAD in the left obtuse marginal branch in the absence of symptoms. In summary, our male subject had SCAD that led to acute myocardial infarction of 5 to 7 days' duration that resulted in cardiac rupture due to mechanical stress during exercise, with evidence of an old healed coronary dissection of unknown duration but without any healed infarction.

CONCLUSIONS

Our rare case involved a male subject who died suddenly during exercise from LV wall rupture that was triggered by SCAD in left circumflex coronary artery

FIGURE 3 Left Obtuse Marginal Coronary Artery



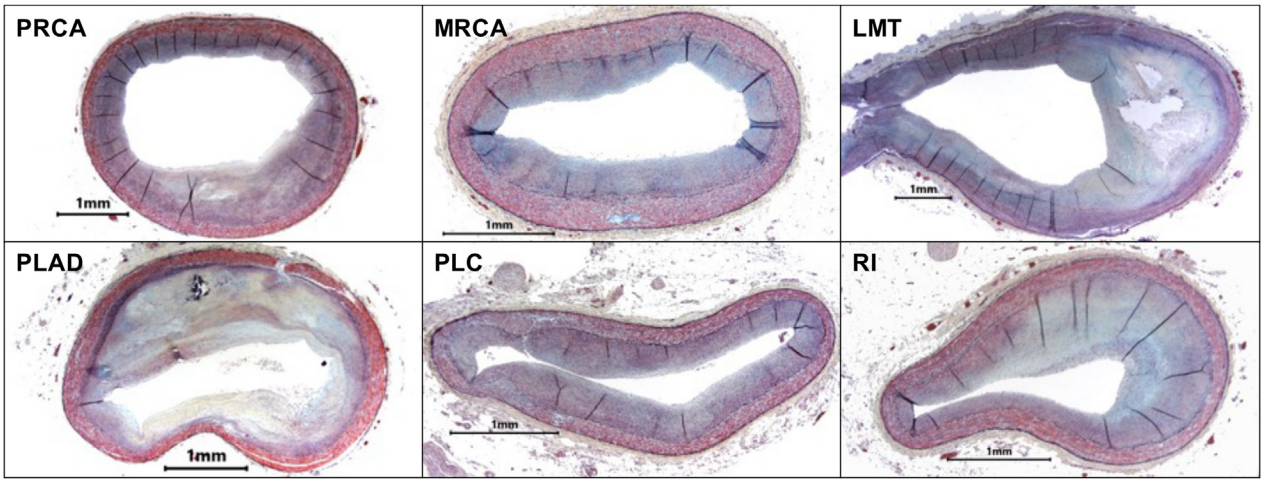
(A) Histological section showing a healed coronary artery medial dissection in the left obtuse marginal branch of the circumflex artery stained by Movat pentachrome. (B) High-power image of the boxed area in panel A showing the full thickness of the media; note that the double arrow shows the healed dissection at the medial adventitial border. Abbreviations as in Figure 3.

(with a right-dominant coronary artery) without FMD, in the presence of healed SCAD.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Drs Virmani and Finn have received institutional research support from the National Institutes of Health (HL141425), the Leducq Foundation, 4C Medical, 4Tech, Abbott Vascular, Ablative Solutions, Absorption Systems, Advanced NanoTherapies, Aerwave Medical, Alivas, Amgen, Asahi Medical, Aurios Medical, Avantec Vascular, BD, Biosensors, Biotronik, Biotyx Medical, Bolt Medical, Boston, Scientific, Canon, Cardiac Implants, Cardiawave, CardioMech, Cardionomic, Celonova, Cerus, EndoVascular, Chansu Vascular Technologies, Childrens National, Concept Medical, Cook Medical, Cooper Health, Cormaze, CRL, Croivalve, CSI, Dexcom, Edwards, Elucid Bioimaging,

FIGURE 4 Representative Sections of Mild-to-Moderate Coronary Atherosclerosis



Representative sections of mild-to-moderate coronary atherosclerosis, with greatest luminal narrowing seen in the proximal left anterior descending artery (PLAD) 50% cross-sectional area luminal narrowing. LMT = left main trunk; MRCA = mid right coronary artery; PLC = proximal left circumflex artery; PRCA = proximal right coronary artery; RI = ramus intermedius.

eLum Technologies, Emboline, Endotronix, Envision, Filterlex, Imperative Care, Innovalve, Innovative, Cardiovascular Solutions, Intact Vascular, Interface Biolgics, Intershunt Technologies, Invatin, Lahav, Limflow, L&J Bio, Lutonix, Lyra Therapeutics, Mayo Clinic, Maywell, MDS, MedAlliance, Medanex, Medtronic, Mercator, Microport, Microvention, Neovasc, Nephronyx, Nova Vascular, Nyra Medical, Occultech, Olympus, Ohio Health, OrbusNeich, Ossiso, Phenox, Pi-Cardia, Polares Medical, Polyvascular, Profusa, ProKidney, LLC, Protembis, Pulse Biosciences, Qool Therapeutics, Recombinetics, Recor, Regencor, Renata Medical, Restore Medical, Ripple Therapeutics, Rush University, Sanofi, Shockwave, SMT, SoundPipe, Spartan Micro, Spectrawave, Surmodics, Terumo, The Jacobs Institute, Transmural Systems, Transverse Medical, TruLeaf, UCSF, UPMC, Vascudyne, Vesper, Vetex Medical, Whiteswell, W.L. Gore, and Xeltis. Dr Finn has received honoraria from Abbott Vascular (stents), Biosensors (stents), Boston Scientific (stents), Celonova (stents), Cook Medical (stents), CSI (catheters), Lutonix Bard (balloons), Sinomed (stents), and Terumo Corporation (stents); and has served as a consultant for Amgen, Amgen (Farma), Abbott Vascular (stents), Boston Scientific (stents, valves, balloons), Celonova (stents), Cook Medical (stents), Lutonix Bard (stents, balloons), and Sinomed (stents). Dr Virmani has received honoraria from Abbott Vascular, Biosensors, Boston Scientific, Celonova, Cook Medical, Cordis, CSI, Lutonix Bard, Medtronic, OrbusNeich Medical, CeloNova, SINO Medical Technology, ReCor, Terumo Corporation, W.L. Gore, and Spectranetics; and served as a consultant for Abbott Vascular, Boston Scientific, Celonova, Cook Medical, CSI, Edwards Lifesciences, Bard BD, Medtronic, OrbusNeich Medical, ReCor Medical, SinoMedical Technology, Surmodics, Terumo Corporation, W.L. Gore, and Xeltis. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Aloke V. Finn, CVPath, Institute, Inc, 19 Firstfield Road, Gaithersburg, Maryland 20878, USA. E-mail: afinn@CVPath.org. @CVPath_MD, @alokefinn.

REFERENCES

1. Di Fusco SA, Rossini R, Zilio F, et al. Spontaneous coronary artery dissection: overview of pathophysiology. *Trends Cardiovasc Med*. 2022;32:92–100.

2. McAlister C, Alfadhel M, Samuel R, et al. Differences in demographics and outcomes between men and women with spontaneous coronary artery dissection. *J Am Coll Cardiol Intv*. 2022;15:2052–2061.

3. Elbadawi A, Elgendy IY, Mahmoud K, et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. *J Am Coll Cardiol Intv*. 2019;12:1825–1836.

4. Ahmed T, Honaker O, Misumida N, Messerli AW. Spontaneous coronary artery dissection (SCAD) complicated with post-infarction ventricular septal rupture and a comparative review on mechanical complications related with SCAD. *Curr Probl Cardiol*. 2023;48:101229.

5. Margaritis M, Saini F, Baranowska-Clarke AA, et al. Vascular histopathology and connective tissue ultrastructure in spontaneous coronary artery dissection: pathophysiological and clinical implications. *Cardiovasc Res*. 2022;118:1835–1848.

6. Saw J, Starovoytov A, Aymong E, et al. Canadian spontaneous coronary artery dissection cohort study: 3-year outcomes. *J Am Coll Cardiol*. 2022;80:1585–1597.

7. Tweet MS, Hayes SN, Pitta SR, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. *Circulation*. 2012;126:579–588.

8. Krittanawong C, Kumar A, Virk HUH, et al. Recurrent spontaneous coronary artery dissection in the United States. *Int J Cardiol*. 2020;301:34–37.

KEY WORDS acute myocardial infarction, acute spontaneous coronary artery dissection, cardiac rupture, sudden cardiac death