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Simultaneous Occurrence of Spontaneous Coronary Artery Dissection and Embolic Stroke

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Authors' contributions

This work was carried out in collaboration between all authors. Author GA led the conceptual design and wrote the first draft of the manuscript. Authors KB, KA and NJ assisted in obtaining the clinical data and case summary. Authors RP, HA and NA, managed the references, literature review and discussion. Authors RP and MN reviewed the final manuscript and refined its content. All authors read and approved the final manuscript.

Case Study

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ABSTRACT

Acute coronary syndrome (ACS) due to spontaneous coronary artery dissection (SCAD) is rare. Further, concurrent ACS with a cerebrovascular accident is improbable, but possible. We report a case of a young man, with a history of Hodgkin's lymphoma treated with a combination of chemotherapy and radiotherapy ten years ago, presented with acute coronary syndrome caused by an extensive dissection of the right coronary artery, together with acute ischemic stroke. Survivors of Hodgkin's lymphoma are at increased risk for cardiovascular complications due to radiation, which can expedite atherosclerosis and can, eventually, give rise to dissection and cerebrovascular disease, as exemplified in our case. This case report and review outlines the incidence, epidemiology, causes, pathophysiology, diagnosis and treatment of spontaneous coronary artery dissection. Our case report is a remainder to clinicians to be mindful of concomitant occurrence of these two conditions and highlights the significant impact the treatment of each has on the other, especially when the literature does not have clear recommendations about simultaneous management. Spontaneous coronary artery dissection with concomitant ischemic cerebrovascular stroke poses a therapeutic dilemma and requires a multi-disciplinary team to appropriately manage the patient.

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Keywords: *Spontaneous coronary artery dissection; acute coronary syndrome; accelerated atherosclerosis; embolic stroke.*

1. CASE PRESENTATION

A 39 year-old male with a past medical history of hypertension for 5 years, dyslipidemia for 5 years, morbid obesity, and history of Hodgkin's lymphoma 10 years ago treated with chemotherapy and radiotherapy, presented with sudden, severe, typical chest pain at rest, constant for about 30 minutes before presentation associated with palpitations, diaphoresis, vomiting, dyspnea and syncope. He had similar chest pain the day before presentation, which lasted about 2-3 hours and resolved spontaneously. He denied intense physical activity, smoking, alcohol intake or illicit drug use. There was no family history of coronary artery disease.

On arrival BP his blood pressure was 151/93 mm Hg, with a borderline tachycardia of 99 beats per minute, and a normal respiratory rate of 14 breaths per minute, afebrile with temperature 98.2°F, and body mass index (BMI) 51 kg/m^2. Cardiovascular examination revealed normal first and second heart sounds, regular heart sounds, with no splitting, no murmurs, no third or 4th heart sounds and no elevation of jugular venous pressure, Respiratory examination was within normal limits. Extremity exam reveal palpable symmetric peripheral pulses with no pedal edema. EKG (Fig 1) showed sinus rhythm, Q waves and 1 mm ST elevation in inferior leads and tall R waves in leads V1-V2 consistent with posterior wall involvement. CBC and BMP were within normal limits. CK: 302IU/L (40-300IU/L), CK-MB: 17.42ng/ml (1-5ng/ml), Troponin I: 3.71ng/ml (<0.3ng/ml. All blood results with normal reference ranges with are listed in table A. Given the development of Q wave on admission EKG, emergent percutaneous coronary intervention was not carried out. The patient was initially placed on IV unfractionated heparin, dual antiplatelet therapy wit Aspirin and clopidogrel, statin, β-blocker, ACEI and morphine. One-hour later, the patient reported numbness and weakness in his left arm associated with left-sided facial numbness. Neurological examination revealed 1/5 muscular strength in the left upper and lower extremities, decreased pain sensation in the left side of the body, and normal deep tendon reflexes with a Babinski response.

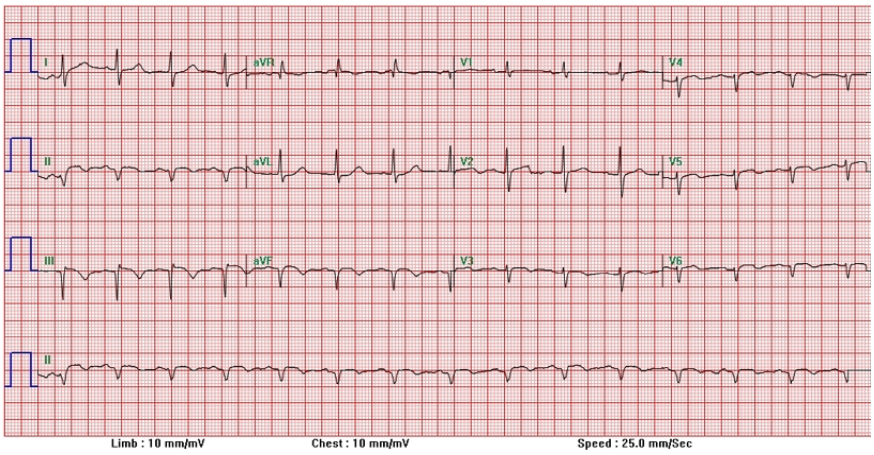

Fig. 1. ECG done at the time of presentation: Normal sinus rhythm with HR 99, Q waves and 1 mm ST elevation in inferior leads and tall R waves in leads V1-V2 consistent with inferior-posterior MI (EKG report as per cardiologist)

Patient's chest pain has not resolved so coronary angiogram was performed. Left cardiac catheterization revealed 70–80% occlusion in mid LAD, 30% in left main and spontaneous dissection of proximal to distal RCA; left ventricular systolic function was mildly impaired with ejection fraction of 40 % (Fig. 2). No interventional therapy was pursued at that time. Transthoracic echocardiogram 48 hours after catheterization did not reveal structural heart disease, neither systolic nor diastolic dysfunction with the former apparently improved secondary to resolved stunned myocardium.

HGB(11.6-16.8) g/dL	16.8	
HCT(35.1-50.0) %	48.9	
WBC(3.6-11.0) K/uL	9.3	
PLT(150-372) K/uL	249.0	
NEUT(43-76) %	75	
LYM(17-45) %	17	
MONO(5-12) %	6	
EOS(0-8) %	2	
BASO(0-1) %	0	
ABSO NEUT(2.0-7.5) K/uL	7.0	
ABSO LYMPHS(0.9-2.9) K/uL	1.6	
RBC(3.80-5.60) M/uL	5.43	
MCV(73.5-96.5) fL	89.9	
MCH(23.9-33.6) pg	31.0	
MCHC(32-35) g/dL	34.4	
RDW(12.1-16.5) %	13.5	
MPV(7.8-11.6) fL	**7.6 L**	
DTYPE	Automated Differential	
INR(0.9-1.1) RATIO	1.0	
PTT(22-35) SEC	25 Heparin Therapeutic Range effective 12/21/2012 is 70 to 107 sec.	
GLUCOSE(70-115) mg/dL	91	
BUN(6-20) mg/dL	11	
CREA(0.7-1.2) mg/dL	0.8	
CALCIUM LEVEL(8.6-10.2) mg/dL	9.3	
ALBUMIN(3.5-5.2) g/dL	4.6	
TOTAL PROTEIN(6.6-8.7) g/dL	7.5	
SODIUM(136-145) mmol/L	136	
POTASSIUM(3.6-5.1) mmol/L	**3.4 L**	
CL(90-110) mmol/L	98	
CO2(22-28) mmol/L	25	
AGP(2.6-10.6) mmol/L	**12.00 H**	
EST GFR NON AFRI(>60) mL/min/1.73 m2	>60	
EST GFR AFRI AM(>60) mL/min/1.73 m2		
AG(1.1-1.5) Ratio	**1.60 H**	
ALK PHOS(40-130) U/L	103	
TOTAL BILIRUBIN(0.1-1.0) mg/dL	0.7	
AST(15-40) U/L	**49 H**	
ALT(5-40) U/L	**44 H**	
Enzyme	**1st Set**	**8 Hours later**
CK(40-300) IU/L	**302.0 H**	**699.0 H**
CKMB(1.0-5.0) ng/mL	**17.42 H**	**71.37 H**
TROPONIN I - ROCHE(<0.30) ng/mL	**3.71 H**	**13.63 H**

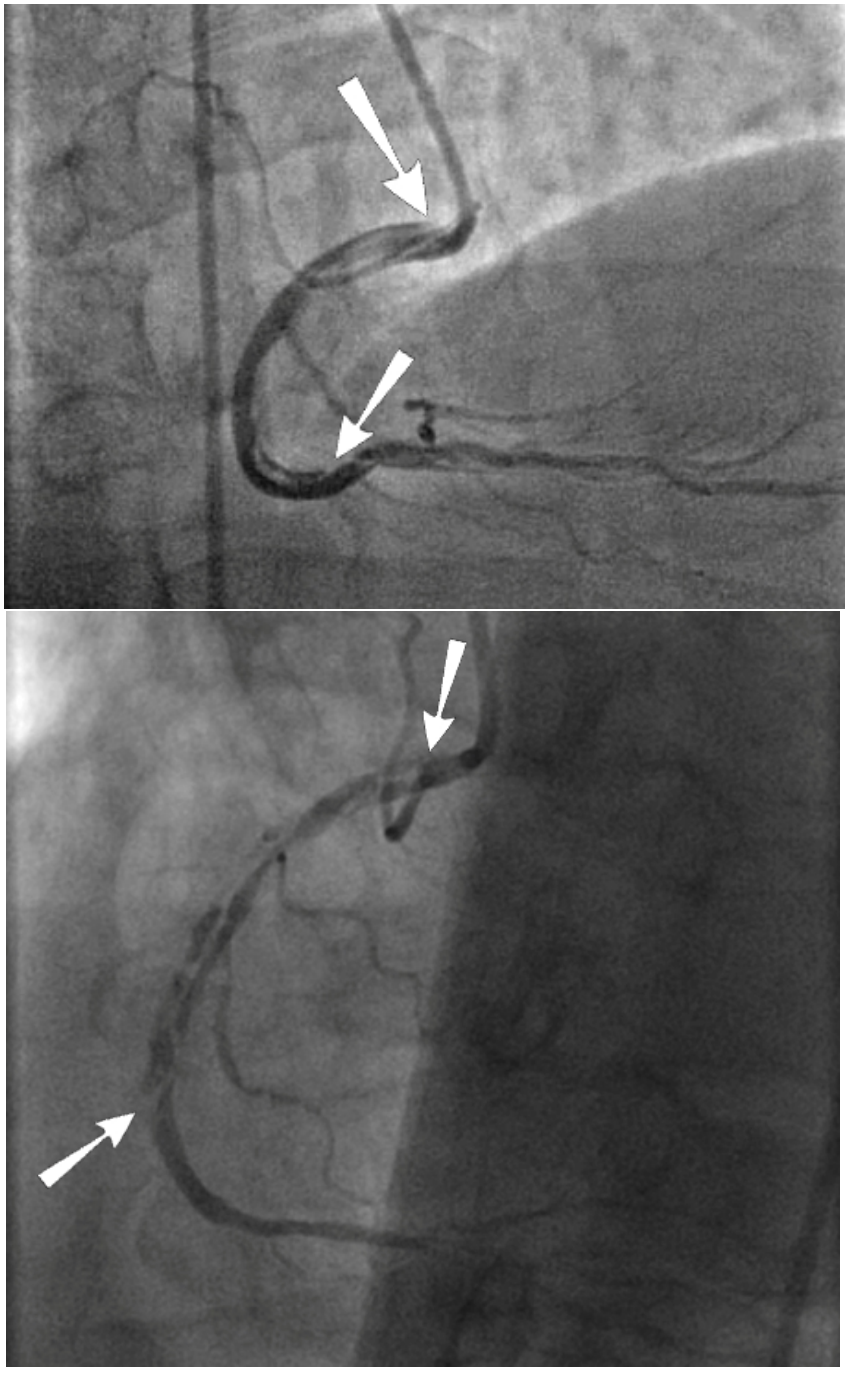

Fig. 2. Left Heart Catheterization: Spontaneous dissection of right coronary artery from proximal to distal with 30% occlusion in main LAD and 70-80% occlusion in mid LAD

Computerized tomography (CT) of the patient's head, without contrast, revealed no evidence of intracranial hemorrhage or infarct. CT angiography of head and neck did not show caroti or aortic dissection findings.

Thrombolytic therapy for acute ischemic stroke was not administered due to recent anticoagulation therapy. Magnetic resonance imaging (MRI) of his brain confirmed two small bilateral non-hemorrhagic embolic infarcts over the right frontal lobe and left anterior parietal cortex (Fig. 3). Were these old infarcts? They are new infarcts and report is mentioned below as per radiology. Please use arrows to indicate abnormalities visible on the cardiac angiogram.

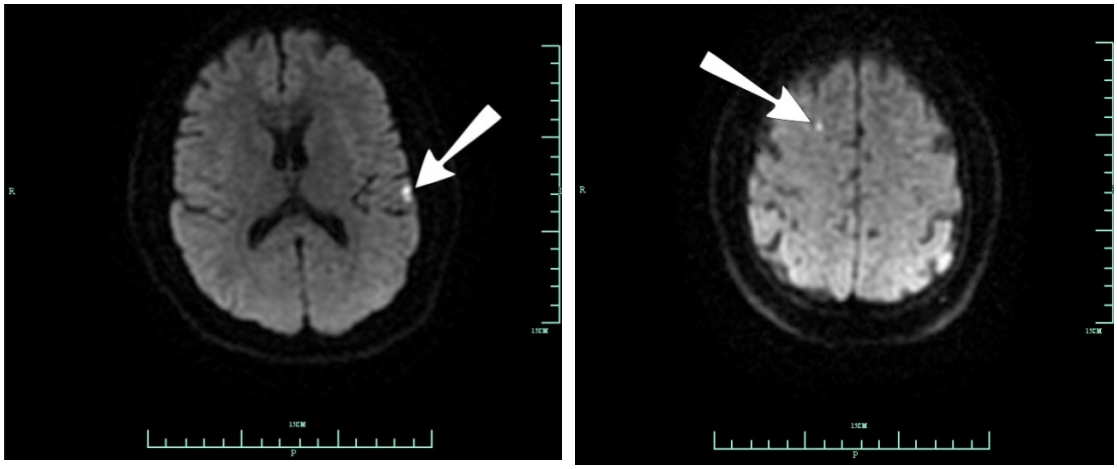

Fig. 3. Brain MRI with and without gadolinium contrast: There are punctuate foci of restricted diffusion in the right frontal lobe white matter and left anterior parietal cortex, consistent with small nonhemorrhagic acute infarct

After cardiovascular surgery, interventional cardiology and neurology evaluation, patient was advised to follow conservative medical therapy. He was given aspirin 325 mg and clopidogrel 300mg time one dose at presentation and then aspirin 81mg/day and clopidogrel 75mg/day as maintenance therapy Two months later, the patient denied recurrent symptoms but persistence of left sided hemiplegic was noted.

2. REVIEW OF THE LITERATURE AND DISCUSSION

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS) of unclear etiology commonly affecting young women. Before the era of advanced cardiac imaging, most cases were found on postmortem examination. The first case of SCAD was documented in 1931 in an autopsy of a 42-year-old woman who had sudden cardiac death [1]. Forker et al. reported the first angiographically confirmed case in 1973 [2]. Now several cases are being recognized due to increased use of coronary angiography and other cardiac imaging. SCAD could recur in as many as 50% of the cases within two months of presentation [3,4].

Recently, literature has found a strong link between fibromuscular dysplasia (FMD) and SCAD. Tweet et al. recently reported the largest series (87 patients from 1979-2009) of non-atherosclerotic SCAD. They found the disease was extremely common in females with 82%

prevalence and an average age of 42.6 years with approximate 30% recurrence rate over the following ten years. Interestingly, mortality was lower in SCAD group compare to acute coronary syndrome controls [5]. Incidentally, FMD of iliac arteries was observed in 50% of patients and it was speculated that FMD might potentially be linked to the occurrence of SCAD [5]. Saw et al. in their recent review of 50 patients (98% women) identified concurrent occurrences of FMD and non-atherosclerotic SCAD. They examined three non-coronary vascular territories: renal, iliac, or cerebrovascular with either angiography, magnetic RA or CTA without histological confirmation. It was found that 86% of the patients had FMD of at least one non-coronary artery, and in fact almost 42% had more than one non-coronary vessel FMD. The rest of the 14% either (10%) did not complete screening for FMD or (4%) were not found to have FMD. Authors described that underlying FMD of coronary artery potentially predisposed them to SCAD, and future studies will be needed to confirm this hypothesis with histological analysis [6].

The spectrum of clinical presentation can range from asymptomatic to ACS, ventricular arrhythmias, heart failure and sudden death [4,5,6]. It can involve either single or multiple coronary arteries [7,8]. LAD is more commonly affected followed by RCA [9]. Exact incidence of SCAD is not known, but various studies have reported that SCAD is detected in 0.07% to 1.1% of all coronary angiograms performed [9] Females are more commonly affected than males [10,11].

SCAD is often classified into three types: atherosclerotic, non-atherosclerotic type and idiopathic forms. Most of the literature has shown that non-atherosclerotic SCAD occurs three times higher in women than in men [6,10,11]. Conditions associated with non-atherosclerotic group include the post-partum period, intense exercise, emotional stress, systemic lupus erythematosus, antiphospholipid antibody, Ehlers-Danlos syndrome, Osler-Weber-Rendu disease, polyarteritis nodosa, sarcoidosis, hypertension, smoking, cyclosporine therapy and cocaine abuse. Pregnancy is a well-documented cause of SCAD with the highest risk in the peripartum period. Proposed mechanisms include significant hemodynamic changes predisposing to shear stress, intimal rupture and hormonal changes leading to medial dissection through degeneration of vessel wall collagen [12-17].
SCAD is caused by a hemorrhage separating the arterial walls. The pathophysiology of SCAD remains poorly understood. Two mechanisms have been advocated behind SCAD:

1. Intimal rupture propagating into dissection: presenting with double lumen, intimal flap, or slow clearing of dye on angiograms.
2. Dissecting hematoma caused by rupture of vasa vasorum: presenting as narrowed lumen vessel, thus mimicking atherosclerotic disease [18].

Vascular wall fragility defect caused by an extracellular matrix cross linking defect such as lysyl oxydase deficiency was described in a case report of SCAD. Lysyl oxydase is involved in the cross linking of arterial wall collagen and elastin [19].

Histopathological analysis has revealed increasing amount of eosinophilic infiltrate in adventitia particularly in non-atherosclerotic dissecting vessels [20,21] Eosinophils contain many granules including collagenase, which can presumably degrade vessel wall collagen and predispose to SCAD. Cases of SCAD have been described in hyper-eosinophilic states like Churg-Strauss syndrome [22]. This assumption gets little support from an anecdotal report where immunosuppressant therapy such as cytoxan and prednisone have produced some favorable results by neutralizing the effect of eosinophilic-induced inflammation [23]; however, this is not sufficient to link eosinophilic infiltrate as an actual pathogenetic factor of

SCAD. Therefore, it remains unclear whether this eosinophilic infiltrate is a primary causative or secondary reactive phenomenon. Further studies are needed to clarify the association.

Atherosclerosis associated SCAD predominantly occurs in up to 80% of male patients [24]. Prognosis of atherosclerotic SCAD appears to be either similar or better than non-atherosclerotic isolated SCAD [24]. Radiation-induced SCAD is a well-known entity especially after radiotherapy to the chest area in patients with Hodgkin's disease or breast cancer; long-term follow up is recommended. It can occur as early as three years after radiotherapy [25]. Our patient received radiotherapy to the chest ten years ago.

Diagnosis of SCAD should be considered in young populations, particularly in females who present with an acute coronary event. Diagnosis can be made largely by coronary angiography by finding a dissection plane and double lumen. Newer advanced cardiac imaging techniques are very promising in diagnosis and management of SCAD. [26-29]

Noninvasive techniques for assessment of SCAD include Multi-detector CT (MDCT) and Dual-source computed tomography. MDCT is used to tell the details about the extent and thickness of hematoma as well as to document healing and follow up [30,31]. The cost and expertise required for these advanced technologies in cardiac catheterization may limit widespread availability and use.

3. MANAGEMENT OF SPONTANEOUS CORONARY ARTERY DISSECTION (SCAD)

Consensus guidelines are lacking for the optimal management of SCAD due to scarcity of data and limited clinical experience. Available therapies include either conservative medical management or aggressive interventional therapy in the form of percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery (CABG). Choice of therapy depends upon clinical presentation, timely diagnosis, site and extent of dissection, and availability of therapy.

Tweet et al. [5]. argued against the more liberal use of early interventional therapy. In his review of 87 patients of non-atherosclerotic SCAD, patients managed with initial conservative therapy were found to have a better outcome, and resolution was observed on follow up angiography. PCI was associated with high rates of complications and technical procedure failure. Two major problems encountered during PCI were either progression of dissection leading to poor hypoperfusion or procedure failure owing to cannulation of wire into false lumen. This highlights the different mechanism involved in SCAD as compared with atherosclerotic narrowing. Despite these potential problems, the role of emergent PCI in critical occlusion to restore coronary perfusion remains crucial and cannot be ignored. Due to the above complications and relatively good prognosis, the authors suggested that conservative therapy should be first line especially if angiography showed good perfusion in coronaries, and PCI could possibly be limited to conditions of ongoing ischemia or infarction. Judicial use of newer cardiac imaging modalities like IVUS and OCT may assist in deciding either conservative or interventional therapy.[5] Possible options to avoid the propagation of dissection due to PCI include the placement of multiple stents covering the dissection or to seal the intimal tear by putting a stent at the entry point of dissection and allowing the dissection to resolve with time.

The beneficial effect of antiplatelet agents in SCAD treated with PCI is very clear, and anecdotal reports have also shown favorable results in patients with SCAD treated with conservative medical therapy. Beta blocker therapy produces favorable results by reducing the shear stress and should be used in all cases if no contraindication [8].

Shamloo et al. [9]. have recently analyzed the management of 440 published cases of both atherosclerotic and non-atherosclerotic SCAD type from 1931 to 2008. They found that aggressive interventional therapy has provided better results as compared to medical management alone. The majority of patients in the interventional group had a superior outcome; and only 2.5% required repeat intervention in contrast with conservative management, where 21.2% patients ultimately necessitated interventional therapy. Another significant finding was the deleterious effect of thrombolytic therapy in SCAD. Almost 60% of patients who received thrombolytics (before diagnosis of SCAD) eventually required emergent surgical or catheter-based intervention because of worsening clinical conditions. This clearly implies the importance of early diagnosis of SCAD and avoidance of thrombolytic agents in the treatment of this entity. It is notable that this is a retrospective study and associated bias cannot be ignored.Even though patients of SCAD with associated atherosclerosis are more frequently older males with multiple coronary risk factors, their prognosis was surprisingly not different than non-atherosclerotic SCAD. CABG should be considered when PCI is either unsuccessful or practically not possible or suitable such as in a multivessel dissection.

More recently, a prospective study by Alfanso et al. has also found that a conservative management strategy (defined as selecting interventional therapy only for patients with ongoing or recurrent Ischemia) has superior outcome, and its long-term prognosis was favorable in these patients [32].

Conservative medical therapy appears to be a favorable strategy especially in the case of uncomplicated ACS. The question remains to be solved about the utility of thrombolytics, heparin and glycoprotein IIb-IIIa inhibitors during an acute coronary event due to SCAD. Further studies are needed to better define their role [33,34,35,36,37]. There is a concern that the clinical condition can be worsened by dissection propagation because of these agents [38]. Prognosis of SCAD is reasonably excellent in both atherosclerotic and non-atherosclerotic SCAD.

Concurrent occurrence of SCAD and stroke is a very rare scenario, and to the best of our knowledge, only three case reports have been documented until today [39,40,41]. The exact relationship between concomitant occurrence of SCAD and embolic stroke is uncertain and data is restricted only to case reports. The cause of embolic stroke in our case was unclear but it is mentioned in the literature that hematologic disorders (Hodgkin in this case) can lead to ischemic stroke of unusual etiology [42]. In-hospital mortality rate on cardioembolic stroke remained around 20% and cardioembolic stroke is the subtype of ischemic infarct with the highest in-hospital mortality. The short-term prognosis of patients with cardioembolic stroke is poor in comparison with other ischemic stroke subtypes [43].

Our case represents a challenge in reference to the therapeutic decision-making since the use of thrombolytic therapy in stroke within the first three hours of presentation has shown to improve neurologic deficit, whereas the use of fibrinolytic agents could be detrimental in the case of SCAD. After a multidisciplinary assessment, the patient was advised to follow conservative medical therapy and a positive outcome was achieved since there was no evidence of recurrent symptoms of SCAD despite the persistent neurologic deficit.

4. CONCLUSION

In conclusion, we believe the treatment of SCAD should be individualized depending upon clinical presentation and concomitant conditions such as stroke as presented in our clinical case. It is our intention to familiarize the medical community with this difficult clinical scenario and emphasize its evidence-based management.

CONSENT

All authors declare that 'written informed consent was obtained from the patient for publication of this case report and accompanying images.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Pretty H. Dissecting aneurysms of coronary artery in woman aged 42: BMJ. 1931;1:667.
2. Forker AD, Rosenlof RC, Weaver WF, Carveth SW, Reese HE. Primary dissecting aneurysm of the right coronary artery with survival. Chest. 1973;64:656–8.
3. Kamineni R, Sadhu A, Alpert J. Spontaneous coronary artery dissection: report of two cases and a 50-year review of the literature. Cardiol Rev. 2002;10:279–284.
4. Mulvany N, Ranson D, Pilbeam M. Isolated dissection of the coronary artery: a post-mortem study of seven cases. Pathology. 2001;33:307–311.
5. Marysia S. Tweet, Sharonne N. Hayes, Sridevi R. Pitta, Robert D. Simari, Amir Lerman, Ryan J. Lennon, Bernard J. Gersh, Sherezade Khambatta, Patricia J.M. Best, Charanjit S. Rihal and Rajiv Gulati. Clinical Features, Management, and Prognosis of Spontaneous Coronary Artery Dissection. Circulation. 2012;126:579-588; Originally Published Online July 16, 2012.
6. Jacqueline Saw, MD, Donald Ricci, MD, Andrew Starovoytov, MD, Rebecca Fox, MSC, Christopher E. Buller MD. Spontaneous Coronary Artery Dissection Prevalence of Predisposing Conditions Including Fibromuscular Dysplasia in a Tertiary Center Cohort. JACC: Cardiovascular Interventions. 2013;6(1):44–52.
7. Rogers IS, Rinaldi MJ, Humphrey CB, Boden WE, Dougherty JE. Postpartum dissection of the left main coronary artery. Clin Cardiol. 2006;29:175–178.
8. 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.
9. Behrooz K. Shamloo, Rajesh S. Chintala, Ali Nasur, Mohammad Ghazvini, Parastoo Shariat, James A. Diggs, Steven N. Singh. Spontaneous coronary artery dissection: aggressive vs conservative therapy. J Invasive Cardiol. 2010;22(5):222-8.

10. Vanzetto G, Berger-Coz E, Barone-Rochette G, Chavanon O, Bouvaist H, Hacini R, Blin D, Machecourt J. 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.
11. 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.
12. Soutkas V, Dragovic L. Sudden deaths from eosinophilic coronary monoarteritis: a subset of spontaneous coronary artery dissection. Am Forensic Med Pathol. 2009;30:268–269.
13. Vrints CJ. Spontaneous coronary artery dissection. Heart. 2010;96:801–8.
14. Motreff P, Souteyrand G, Dauphin C, Eschalier R, Cassagnes J, Lusson JR. Management of spontaneous coronary artery dissection: review of the literature and discussion based on a series of 12 young women with acute coronary syndrome. Cardiology. 2010;115:10–8.
15. Tehrani S, Khawaja M, Redwood S. Successful treatment of a late presentation exercise-induced spontaneous left anterior descending artery dissection. J Invasive Cardiol. 2011;23:E260–E262.
16. Hampole C, Philip F, Shafii A, Pettersson G, Anesi G, Patel J, Menon V. Spontaneous coronary artery dissection in Ehlers-Danlos syndrome. Ann Thorac Surg. 2011;92:1883–1884.
17. Oliveira SM, Goncalves A, Dias P, et al. Spontaneous coronary artery dissection: a diagnosis to consider in acute coronary syndromes. Rev Port Cardiol. 2009;28:707–13.
18. Maehara A, Mintz GS, Castagna MT, et al. Intravascular ultrasound assessment of spontaneous coronary artery dissection. Am J Cardiol. 2002;89:466–8.
19. Sibon I, Summer P, Lamaziere JM Daniel, and Bonnet J. Lysyl oxidase deficiency: a new cause of human arterial dissection. Heart. 2005;91(5):e33.
20. Borczuk AC, van Hoeven KH, Factor SM. Review and hypothesis: The eosinophil and peripartum heart disease (myocarditis and coronary artery dissection)-coincidence or pathogenetic significance? Cardiovasc Res. 1997;33:527–32.
21. Basso C, Morgagni GL, Thiene G. Spontaneous coronary artery dissection: a neglected cause of acute myocardial ischemia and sudden death. Heart. 1996;75(5):451.
22. Hunsaker JC, O'Connor WN, Lie JT. Spontaneous coronary dissection and isolated eosinophilic coronary arteritis: Sudden cardiac death in a patient with limited variant of Churg-Strauss syndrome. Mayo Clin Proc. 1992;67:761–6.
23. Koller PT, Cliffe CM, Ridley DJ. Immunosuppressive therapy for peripartum type spontaneous coronary artery dissection: Case report and review. Clin Cardiol. 1998;21:40–6.
24. 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;20:573–6.
25. Miltenyi Zsofia; Keresztes Katalin; Garai Ildiko; Edes Istvan; Galajda Zoltan; Toth Laszlo; Illes Arpad. Radiation-induced coronary artery disease in Hodgkin's disease. Cardiovascular Radiation Medicine. 2004;5(1):38-43.
26. Deftereos S, Giannopoulos G, Mavrogianni A, Sykiotis A, Pyrgakis V, Bobotis G. Role of grey-scale intravascular ultrasound and Chroma-Flo in deciding on treatment approach for spontaneous coronary dissection in a young woman. Hellenic J Cardiol. 2011;52:364–6.

27. Arnold JR, West NE, Van Gaal WJ, Karamitsos TD, Banning AP. The role of intravascular ultrasound in the management of spontaneous coronary artery dissection. Cardiovasc Ultrasound. 2008;6:24.
28. Suter MJ, Nadkarni SK, Weisz G, Tanaka A, Bourma BE, Tearney GJ. Intravascular optical imaging technologies for investigating the coronary artery. J Am Coll Cardiol Img. 2011;4:1022-1039.
29. Lim C, Banning A, Channon K. Optical coherence tomography in the diagnosis and treatment of spontaneous coronary artery dissection. J Invasive Cardiol. 2010;22:559–60.
30. Manghat NE, Morgan-Hughes GJ, Roobottom CA. Spontaneous coronary artery dissection appearance and follow up on multi-detector row CT coronary angiography. Clin Radiol. 2005;60:1120–5.
31. Zheng M, Li J, Xu J, Chen K, Zhao B, Huan Y. Spontaneous dissection of left anterior descending coronary artery: The diagnostic role of dual-source computed tomography. J Thorac Imaging. 2010;25:W79–81.
32. Fernando Alfonso, Manuel Paulo, Vera Lennie, Jaime Dutary, Esther Bernardo, Pilar Jiménez-Quevedo, Nieves Gonzalo, Javier Escaned, Camino Banuelos, Marıa J. Pérez-Vizcayno, Rosana Hernandez, Carlos Macaya. 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):1062-70.
33. Monodeep Biswas, Arjinder Sethi, and Stephen J. Voyce Spontaneous Coronary Artery Dissection: Case Report and Review of Literature. Heart Views. 2012;13(4):149–154.
34. Karaahmet T, Tigen K, Gurel E, Cevik C, Mutlu B, Başaran Y. Spontaneous dissection of the left main coronary artery regressed with thrombolytic therapy: Evaluation with multi-slice computed tomography angiography. Anadolu Kardiyol Derg. 2009;9:E2–3.
35. Behnam R, Tillinghast S. Thrombolytic therapy in spontaneous coronary artery dissection. Clin Cardiol. 1991;14:611–4.
36. 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;52:226–30.
37. Cheung S, Mithani V, Watson RM. Healing of spontaneous coronary dissection in the context of glycoprotein IIB/IIIA inhibitor therapy: A case report.Catheter Cardiovasc Interv. 2000;51:95–100.
38. Sebastian C, Scherlag M, Kugelmass A, Schechter E. Primary stent implantation for acute myocardial infarction during pregnancy: Use of abciximab, ticlopidine, and aspirin. Cathet Cardiovasc Diagn. 1998;45:275–9.
39. Rajendra N, Lim F, Shaukat N. Spontaneous coronary artery dissection presenting as an ischaemic stroke in a middle-aged man with anti-cardiolipin antibodies: A case report. J Med Case Rep. 2010;4:94.
40. Acikel S, Yildirir A, Muderrisoglu H. A rare presentation of primary spontaneous coronary dissection: left ventricular apical thrombus associated with ischemic cerebrovascular event. Int J Cardiol. 2009;134(2):e53-5.
41. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med. 1995;333(24):1581.

42. Arboix A, Bechich S, Oliveres M, García-Eroles L, Massons J, Targa C Ischemic stroke of unusual cause: clinical features, etiology and outcome. Eur J Neurol. 2001;8(2):133-9.
43. Arboix A, Alió J.Cardioembolic stroke: clinical features, specific cardiac disorders and prognosis. Curr Cardiol Rev. 2010;6(3):150-61. doi: 10.2174/157340310791658730.

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