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The presentation of spontaneous coronary artery dissection in the emergency department: Signs and symptoms in an unsuspecting population

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Abstract

Objectives: Spontaneous coronary artery dissection (SCAD) has emerged as a common cause of acute coronary syndrome (ACS) in young women, although it is rarely discussed in the differential diagnosis for chest pain in the emergency department (ED). In a population otherwise considered low risk for myocardial infarction, there is a danger of incomplete workup and missed diagnosis. In this study, we aim to describe the clinical presentation of those who present to the ED with SCAD to increase awareness of this potentially fatal diagnosis among emergency practitioners.

Methods: Data were queried from the Mayo Clinic "Virtual" Multicenter SCAD Registry, a large multisite international disease registry. The registry includes demographic information as well as data from both medical records and surveys administered following the SCAD event. Symptom presentation was abstracted from survey narrative responses. Data analysis was performed using descriptive statistics.

Results: Of 1196 subjects included, chest pain was reported during initial SCAD event in 95.7%. Most common chest symptoms descriptors were pain, pressure/weight, and tightness, with radiation most often in one or both arms/shoulders. Other common symptoms included nausea, shortness of breath, and diaphoresis. Most common electrocardiogram (ECG) findings reported were ST elevation, T-wave abnormality, and normal ECG. Initial troponin values were within normal range in 20.1% of patients.

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CONFLICT OF INTEREST

The authors have no potential conflicts to disclose.

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Conclusion: With young healthy women often considered “low risk” for ACS, it is important to understand that SCAD is a cause of ACS, and familiarity with presentation can improve awareness among emergency physicians. Our data can provide insight in helping to identify young women who present with chest pain due to SCAD so they can be appropriately evaluated.

Keywords

acute coronary syndrome; coronary artery dissection; myocardial infarction; women's health

INTRODUCTION

Chest pain is commonly encountered in the emergency department (ED) and in 2017 was the second most common chief complaint, accounting for over 7 million visits to the ED.[1] Furthermore, missed myocardial infarction (MI) results in the highest dollar losses in ED malpractice cases.[2] The differential diagnosis for chest pain is broad and contains several life-threatening causes, including acute coronary syndrome (ACS). Advances such as high-sensitivity troponin testing and risk stratification tools have improved diagnostic efficiency and help to identify those at risk for atherosclerotic heart disease. Despite these advances in identifying patients with unstable plaques/acute plaque rupture as the etiology of ACS, traditional risk stratification tools are often inadequate in identifying patients with spontaneous coronary artery dissection (SCAD), a cause of ACS that predominantly affects otherwise healthy women.[3,4]

SCAD is defined as a noniatrogenic, nontraumatic, nonatherosclerotic dissection of the coronary arteries that, with rare exception, presents as ACS. Intramural hematoma formation with or without a tear of the coronary artery wall leads to coronary artery occlusion and potentially life-threatening myocardial ischemia or infarction. Unlike other causes of ACS, SCAD occurs more frequently in younger, otherwise healthy women with an average age in the range of 42 and 50 years (although cases have been reported as young as the second decade of life and as old as the ninth).[3,4] SCAD is also associated with conditions such as fibromuscular dysplasia, peripartum status, and connective tissue disorders.[3,4] Initially thought to be rare, recent literature has identified SCAD as the cause of at least 4% of all ACS, 35% of ACS among women under the age of 50 years, and as the most common cause of pregnancy-associated MI.[5,6] Unfortunately, patients with SCAD may present with initial normal troponin and electrocardiogram (ECG)[7] and risk stratification scores such as the HEART (History, EKG, Age, Risk Factors, Troponin) Score and the Thrombolysis in Myocardial Infarction (TIMI) score include only atherosclerotic risk factors for ACS and thus may not accurately identify patients with SCAD. As the risk of cardiovascular disease in general in young women is only recently becoming more recognized, this group continues to be at a particularly high risk of being underdiagnosed upon presentation to the ED.[8-10]

This study was performed to better characterize the symptomatic presentation of SCAD in an effort to bring awareness to this life-threatening condition.[7,11] In this study, we aim to characterize the patient population at risk for SCAD, investigating associated symptoms, triggers, and initial troponin and ECG findings in those who were found to have SCAD, as well as discuss the experience of patients with SCAD with the ED setting.

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METHODS

This observational cohort study was approved by the Mayo Foundation Institutional Review Board, and subjects were those who consented to the Mayo Clinic "Virtual" Multicenter SCAD Registry. Recruitment for this registry began after institutional review board approval on August 3, 2011; data for this study extend through March 18, 2020. Initial subject contact for the Mayo Clinic SCAD Registry came from participants in an online community hosted by WomenHeart: the National Coalition for Women with Heart Disease. With initial pilot success of this patient driven recruitment process, subjects have since been recruited from Mayo Clinic and numerous other health care systems with the assistance of disease-specific social media groups, physician referrals, and self-referrals.[12] The Mayo Clinic SCAD Registry now contains patient data from over 1400 individuals. Each subjects' SCAD diagnosis was confirmed angiographically by Mayo Clinic SCAD team cardiologists' and review of medical records and images both from both within, and outside of, the Mayo Clinic system. Objective data were abstracted from medical records into the Mayo Clinic SCAD Registry database by trained nurses and updated prospectively. Both subjective and objective, self-reported follow-up data were also collected prospectively from multiple follow-up surveys, which include questions related to initial SCAD presentation, symptoms, mental health, history of pregnancy, and other pertinent past medical history. Subjects whose data had not yet been abstracted or did not have answers to the questionnaires were excluded.

The question, "Please describe the onset of your symptoms" in the primary questionnaire sent upon initial subject recruitment allowed extraction of chest pain/sensation descriptors as well as radiation or symptoms described in extremities/outside of the chest and other related symptoms. Exact words were extracted when possible; however, synonyms were also used to help classify into common themes (for example, "an elephant on my chest" was included as "pressure/weight on chest"). Radiation to arms, back, or elsewhere outside of the chest or sensations (for example, numbness, tingling, weakness, tightness) in these areas were also similarly quantified, as were associated symptoms including shortness of breath, nausea, vomiting/retching/dry-heaving, and diaphoresis. Patients were also queried on follow-up survey using yes/no questions as to whether or not they experienced any extreme physical or emotional stress prior to their SCAD event, whether or not their SCAD event was associated with postpartum/peripartum status, and whether they required cardiopulmonary resuscitation (CPR) upon initial presentation. Additionally, the follow-up survey provided information about SCAD recurrence and post-SCAD chest pain.

The Mayo Clinic SCAD Registry includes objective data points abstracted from the medical record including vital signs at presentation, presentation type (including unstable angina, ST-elevation MI [STEMI], non-ST-elevation MI [NSTEMI], cardiac arrest, ventricular fibrillation, and ventricular tachycardia), ECG, and troponin results. ECG data were reported as the written ECG report in the medical record, because access to ECG tracings was variable. These data were quantified in our study by categorizing into normal ECG, ST-segment elevation, ST-segment depression, ST-segment abnormality, and T- wave abnormality. Since the numeric value for an abnormal troponin differs substantially between centers and has evolved over time, troponin results are reported as normal or abnormal.

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Statistical analysis consists of descriptive statistics, with demographics, comorbidities, frequency of symptoms, and other data points described reported as percentages. Median and interquartile range (IQR) were used to report age, body mass index (BMI), and vital signs at the time of the SCAD event.

RESULTS

Of the 1276 consented patients in the registry as of March 18, 2020, there were 1196 subjects included in the study. There were 80 subjects excluded including those found not to be SCAD, if there was no abstractor signature or data abstracted for the subject, if there were a significant amount of data missing, if there were no ED records, and if the records were in a language other than English. Demographics and pertinent past medical history are shown in Table 1. Of the 1196 subjects, 95.7% responded “yes” to experiencing chest symptoms (such as pain or pressure) during their SCAD event (3.8% report no chest symptoms and 0.5% did not respond).

Of the 1196 subjects in the registry, 997 had a recorded response to the open-ended question, “Please describe the onset of your symptoms.” The subjective data points of chest pain/sensation quality descriptors, radiation or areas of the body mentioned outside of the chest, and related symptoms extracted from these responses are described in Table 2. The most common descriptors of chest pain/sensation were pain (as the only descriptor), pressure/weight on chest, and tightness with radiation or non–chest pain sensation most often mentioned in one or both arms or shoulders (85.1% left-sided).

Among the 1196 subjects, 199 (16.6%) subjectively reported extreme exercise and 237 (19.8%) extreme stress/emotion prior to their SCAD event. Peripartum status at time of SCAD was self-reported in 10.6% of subjects. Notably, 7.9% of patients reported receiving CPR or defibrillation during their initial SCAD presentation (70.1% did not, 1.4% were not sure if they received CPR or defibrillation, and 20.6% did not have a response). Objective data regarding initial SCAD event presentation, including percentage presenting in cardiac arrest, vital signs, ECG, and troponin, are shown in Table 3. On follow-up survey, 40.2% of subjects reported experiencing chest pain since their SCAD event (55.7% reported no chest pain and 4.1% were unsure), and 197 (16.5%) subjects reported experiencing at least one recurrence of SCAD.

DISCUSSION

Our analysis of the largest cohort of patients with SCAD provides insight into their presenting symptoms, vital signs, troponin results, and ECG findings. The overwhelming majority of those who had confirmed SCAD experienced chest pain or other chest symptoms that radiated (specifically to the arms, back, jaw, and neck) and was associated with other symptoms such as diaphoresis, nausea, vomiting, and shortness of breath, consistent with the typical atherosclerotic ACS symptom presentation. In addition, 40.2% of individuals experienced recurrence of their chest pain after initial hospitalization for SCAD, and 16.5% had been diagnosed with recurrent SCAD at the time of survey.

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Our study of patients presenting with SCAD found relatively lower rates of typical atherosclerotic risk factors compared to those with atherosclerotic disease—specifically hypertension and diabetes (as an example, one study showed rates of 52.3% and 22.4% in atherosclerotic disease, respectively). Of note, our study showed hyperlipidemia was comparable to what has been shown in non-SCAD MI.[13,14] Rather, our data shows high rates of comorbidities such as migraine and FMD, which are known to be more prevalent among patients with SCAD than the general population.[15] Furthermore, patients with SCAD have a higher percentage of normal ECGs and initial troponin values when compared to patients with ACS due to atherosclerotic disease.[13,14] Thus, risk stratification tools for atherosclerotic causes of ACS (for example, the HEART score) do not identify patients with SCAD. As practitioners in the ED are more familiar with atherosclerotic risk factors and causes of ACS, these risk stratification tools may offer false reassurance when evaluating a patient with chest pain.

As with atherosclerotic ACS, the diagnosis of SCAD is usually made with invasive coronary angiography, with intracoronary imaging such as optical coherence tomography as an adjunctive tool for confirmation.[5] The treatment of SCAD, however, varies considerably from that of atherosclerotic ACS and making the correct diagnosis will prevent the poor outcomes that result from the standard treatment of ACS.[16] For example, while timely percutaneous coronary intervention (PCI) is the preferred treatment for atherosclerotic ACS, PCI is associated with high rates of procedural complications or unsuccessful results among patients with SCAD.[3,5,17] Furthermore, most coronary arteries with SCAD heal over time.[17,18] Therefore, many patients with SCAD should be managed conservatively without PCI while reserving revascularization for the subset of patients with SCAD who have poor coronary blood flow or clinical instability.[3,17,18] As cardiologists ultimately make the diagnosis and provide appropriate treatment for patients with SCAD, it is crucial that the emergency practitioner have a high enough clinical suspicion to obtain a cardiology consult upon initial presentation.

Failure or a delay in diagnosing SCAD can have devastating consequences including mismanagement and/or unmonitored progression of dissection.[16] Unfortunately, missed or delayed diagnosis is common.[3,4,9] As an example, there are numerous cases reported where patients with SCAD did not receive a full cardiac evaluation on presentation despite reporting potential ACS symptoms or did not trigger the standard response to elevated biomarkers, leading to significant comorbidity or death.[19,20] With the underdiagnosis and misdiagnosis of SCAD, in addition to poor outcomes reported in women who present with a cardiac events in general, it is important to address the implicit bias likely underlying this patient population to ensure the appropriate diagnostic testing of those “atypical” patients who present with chest pain.[8,21,22,23,24,25,26,27,28]

LIMITATIONS

As with any descriptive study there are important limitations to recognize. Without an appropriate comparison group, we are unable to statistically validate any differences between this group and other causes of ACS. Notably, extreme emotion and physical activity prior to SCAD was lower in this population than has been previously reported in smaller

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studies and can be due to the sample size or due to differences in how patients were surveyed.[29] This study is limited by participant recall bias and selection bias. Inclusion criteria into the registry requires a diagnosis of SCAD so this study does not include persons with SCAD who died, who did not present for care, or who did not undergo an appropriate diagnostic evaluation for their symptoms, an important limitation in a population who is already at high risk for being missed. Despite SCAD affecting all race/ethnicity groups, the population in our study is also predominately white, and this may limit applicability of these findings to non-White patients with SCAD.[30] With these limitations in mind, however, this remains the largest SCAD cohort to date and provides valuable, large-scale descriptive data as to how patients with SCAD present. This information can aid in the more appropriate and expedient diagnosis of SCAD in the ED, reducing the consequences of a missed diagnosis for both physicians and, most importantly, patients.

CONCLUSION

Our evaluation of over 1000 patients with spontaneous coronary artery dissection reveals that patients diagnosed with spontaneous coronary artery dissection lack common atherosclerotic risk factors such as diabetes mellitus, hypertension, and tobacco use. Although this condition causes chest pain due to cardiac ischemia, these patients do not have the usual risk factors for atherosclerotic acute coronary syndrome and so risk stratification protocols do not apply. Diagnosis is dependent on a high level of suspicion for young to middle-aged patients, particularly women, with concerning presentations. The “low-risk” profile of these patients puts them at risk for adverse outcomes, despite commonly presenting with radiating chest symptoms. Our study emphasizes the need for spontaneous coronary artery dissection awareness and inclusion of spontaneous coronary artery dissection on the chest pain differential and advocates for an appropriate diagnostic workup in young women without classic risk factors who present with chest pain.

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Table 1

Demographics

Age (years), median (IQR)	54 (47–61)
Sex, *n* (%)	
Female	1143 (95.6)
Male	47 (3.9)
BMI, median (IQR)	25.0 (21.8–29.2)
Race, n (%)	
White	1104 (92.3)
Black	27 (2.3)
Hispanic/Hispanic-White	27 (2.2)
Asian/Asian-White	16 (1.3)
Asian Indian	2 (0.2)
Native American/Native American-White	5 (0.4)
Polynesian/Puerto Rican	1 (0.1)
Other/unknown	2 (0.2)
Comorbidities, *n* (%)	
Classic ACS risk factors	
Hypertension	385 (32.2)
Hyperlipidemia	397 (33.2)
Diabetes Mellitus	35 (2.9)
Previous tobacco use, *n* (%)	316 (26.4)
SCAD risk factors	
Migraine	420 (35.1)
Hypothyroid	179 (15.0)
Hyperthyroid	26 (2.2)
FMD	
No	242 (20.2)
Yes	461 (38.5)
Possible	53 (4.4)
Non-FMD endovascular aneurysm	59 (4.9)
Not Screened	373 (31.2%)

Abbreviations: BMI, body mass index; FMD, fibromuscular dysplasia.

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TABLE 2

Sensation, radiation, and other related symptoms

	n **(%)**
Chest pain/sensation quality	
No chest pain	177 (17.8)
Chest pain	820 (82.2)
Pain as the only descriptor	273 (27.4)
Pressure/weight on chest	200 (20.1)
Tightness	79 (7.9)
Burning	59 (5.9)
Heaviness	49 (4.9)
Crushing	35 (3.5)
Sharp	29 (2.9)
Discomfort	26 (2.6)
Other[a]	70 (5.9)
Radiation/sensation outside of the chest	
Arm/shoulder	694 (69.6)
Shoulder blades/back	154 (15.4)
Jaw	149 (14.9)
Neck	100 (10.0)
Related symptoms	
Nausea/vomiting/retching/dry heaving	249 (25.0)
Shortness of breath	177 (17.8)
Diaphoresis	171 (17.2)

Note: Extracted from the responses to the inquiry "Please describe the onset of your symptoms." There were 997 responses, *n* = is the number of subjects that mentioned each term in their response.

[a]"Other" includes squeezing, cramping, tearing, and dull.

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TABLE 3

Initial SCAD presentation

Vital signs	**Mean (IQR)**
HR (beats/min)	76 (65–84)
Blood pressure	
Systolic (mm Hg)	133 (116–147)
Diastolic (mm Hg)	79 (68–90)
	***n* (%)**
Troponin	
Troponin data available	1023 (85.5)
Initial troponin negative	206 (20.1)
Presentation[a]	
Unstable Angina	20 (1.7)
NSTEMI	688 (57.5)
STEMI	460 (38.5)
Cardiac Arrest	105 (8.8)
Ventricular Fibrillation	94 (7.9)
Ventricular Tachycardia	106 (8.9)
ECG Findings	
Missing ECG data	117 (9.8)
ECG[b]	1079 (90.2)
Normal ECG	170 (15.8)
ST elevation	494 (45.8)
ST depression	62 (5.7)
ST abnormality	93 (8.6)
T-wave abnormality	235 (21.8)

Note: Data in this table were abstracted from medical records. Abbreviations: HR, heart rate; NSTEMI, non–ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction.

[a]Note that presentation sum is greater than 100% as subjects may have presented with more than one presentation type.

[b]ECG data taken from physician interpretation reported in medical record. $n = 1196$.