JACC: CASE REPORTS VOL. 2, NO. 11, 2020

MINI-FOCUS ISSUE: CORONARIES

BEGINNER

CASE REPORT: CLINICAL CASE

A Novel Case of Spontaneous Coronary Artery Dissection During Cabergoline Therapy for Prolactinoma

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ABSTRACT

We present a case of spontaneous coronary artery dissection associated with cabergoline treatment for prolactinoma. A 31-year-old woman with history of hypertension and prolactinoma, treated with cabergoline, presented with chest pain. She had non-ST-segment elevation myocardial infarction with double vessel coronary artery dissection and was treated with coronary artery bypass grafting. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:1684–7)
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HISTORY OF PRESENTATION

A 31-year-old woman presented with complaint of chest pain. She denied tobacco, drug, or alcohol use, and she reported there was no known family history of genetic disease, cardiac disease, or sudden death. On initial evaluation, vital signs demonstrated a heart rate of 62 beats/min and a blood pressure of 164/110 mm Hg. Physical examination was significant for an anxious female with physical distress caused by intermittent chest pain but no focal abnormal findings were identified. An electrocardiogram showed diffuse T-wave inversions in II, III, aVf, and V_1 to V_6. Laboratory tests showed an initial troponin I of 0.06 ng/ml (normal range, <0.40 ng/ml) but reflected a normal complete blood count and basic metabolic panel. Patient was given aspirin (325 mg) and clopidogrel (600 mg) at the outside hospital she initially presented to. On presentation to our facility, her troponin I level increased to 2.8 ng/ml.

LEARNING OBJECTIVES

- SCAD commonly occurs in young females without risk factors of atherosclerotic CAD.
- The use of cabergoline to treat hyperprolactinemia could be related to its incidence.
- CABG was effective in the case of a symptomatic patient with ongoing ischemia despite medical therapy. PCI could be associated with an increased risk of iatrogenic dissections or extensions of dissection.

PAST MEDICAL HISTORY

The patient had a previous medical history of hypertension treated with amlodipine (10 mg/day). She delivered 1 child 11 years ago. The patient had reported symptoms characteristic of amenorrhea, galactorrhea, and infertility 2 years ago. Her prolactin level was elevated to 160 ng/ml (normal range, 2.8 to

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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 *JACC: Case Reports* author instructions page.

Manuscript received June 11, 2020; accepted July 1, 2020.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2020.07.024

29.2 ng/ml) but she was not treated for prolactinoma for 1 year because of noncompliance with medical recommendations. A magnetic resonance imaging detected a 2.3-cm pituitary mass and a prolactin level of 236 ng/ml 7 months ago. She was started on cabergoline (0.25 mg twice weekly) for prolactinoma. After completing 3 months of therapy, she stopped refilling her prescription. Three weeks later (3 months before this presentation), she visited the emergency department with complaint of chest pain. She was discharged home because electrocardiogram did not show significant ST-segment changes and echocardiogram revealed normal ejection fraction. Her endocrinologist restarted her back on cabergoline at her previous dose.

DIFFERENTIAL DIAGNOSIS

Based on the initial presentation, electrocardiogram findings, and elevated troponin, differential diagnosis included non-ST-segment elevation myocardial infarction, myocarditis, and stress cardiomyopathy.

INVESTIGATION

A coronary angiogram revealed normal left main, diffuse 99% stenosis of the proximal to mid left anterior descending artery, 90% stenosis at the first diagonal branch, normal left circumflex, and total occlusion of the mid right coronary artery (RCA) without visible collaterals (**Figure 1**, Video 1). RCA was likely an acute flow-limiting dissection and the culprit lesion. An echocardiography showed 60% of ejection fraction with hypokinesis of inferior and septal wall. A lipid panel test was not significant (cholesterol, 164 mg/dl; low-density lipoprotein, 123 mg/dl; high-density lipoprotein, 52 mg/dl; and triglyceride, 53 mg/dl).

MANAGEMENT

Considering the patient's young age and staccato chest pain, which was related to high blood pressure, coronary artery dissection was suspected. Percutaneous coronary intervention (PCI) was not performed. Blood pressure control resolved chest pain. An intra-aortic balloon pump was placed and the patient was treated with intravenous heparin and nitroglycerine infusions in anticipation of coronary artery bypass grafting (CABG) for definitive treatment. Ten hours later, the patient had a recurrence of chest pain with increasing troponin I of 6.5 ng/ml and was taken for on-pump CABG. The left internal thoracic artery was anastomosed to left anterior descending artery and the right internal thoracic artery to RCA. Radial artery was anastomosed to the diagonal artery. Tissue along the diagonal artery and left anterior descending artery was inflamed and swollen (**Figure 2**). Once arteries were opened, they were found to have evidence of dissection with false lumen, chronic thrombosis, and a dissection flap. The RCA was similarly dissected. The chest was left open because of profound hypotension during an attempt to close.

DISCUSSION

Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome or sudden cardiac death (1). It is identified in 0.2% to

ABBREVIATIONS AND ACRONYMS

CABG = coronary artery bypass grafting

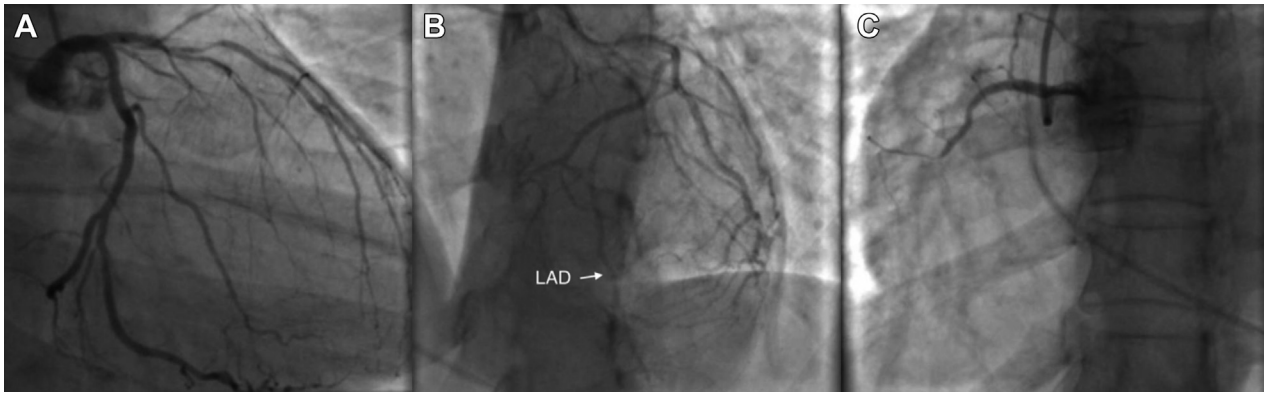
CAD = coronary artery disease

PCI = percutaneous coronary intervention

RCA = right coronary artery

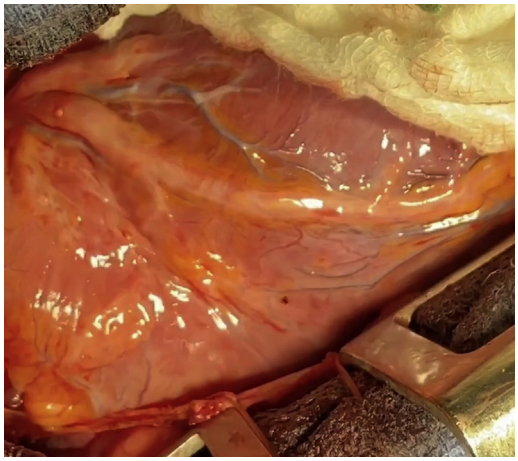
SCAD = spontaneous coronary artery dissection

FIGURE 1 Pre-Operative Coronary Angiogram



(A) In the right anterior oblique caudal view, left main and the left circumflex was normal. **(B)** In the left anterior oblique cranial view, the proximal to mid left anterior descending artery showed diffuse 99% stenosis. There was a 90% stenosis at the bifurcation of the first diagonal branch. **(C)** In the left anterior oblique cranial view, the mid right coronary artery was 100% occluded.

FIGURE 2 Intraoperative Findings of Coronary Artery Dissection



The tissue along the diagonal artery and left anterior descending artery was inflamed and swollen.

1.1% of coronary angiographies performed for acute coronary artery syndrome (2). SCAD predominately affects young females with some experiencing the condition in a life-threatening manner than others. Although it is a rare disease, it can be underdiagnosed because of the perception that coronary artery disease (CAD) does not affect young patients. In our case, the patient presented to the emergency department with chest pain 3 months before undergoing CABG. She may have had a SCAD event at that time, from which she recovered, followed by another event that led to the second presentation. It is clear that CAD may not likely be considered when young patients complain of chest pain. Intramural hematoma within the wall of a coronary artery is characteristic after SCAD. This is caused by intimal tear or a spontaneous hematoma arising from the vasa vasorum within the vessel wall (3). The cause of SCAD could be multifactorial, including underlying arteriopathies, genetic factors, hormonal influences, or systemic inflammatory diseases. Among arteriopathies, the association of SCAD with fibromuscular dysplasia has been reported. Fibromuscular dysplasia affects any arteries and can manifest as arterial stenosis, aneurysm, or dissection (4). There is only 1 previous case report about SCAD during use of cabergoline (5). Cabergoline is an ergot derivative, which can cause vasospasm leading to dissection of arteries (6). There may be a relationship between cabergoline and SCAD. Besides this, the underlying disease of hyperprolactinemia and associated hormonal changes may have a similar effect as pregnancy and cause SCAD.

There is not a well-established guideline for treatment of SCAD. Studies have demonstrated that SCAD can heal spontaneously in most patients, ranging from 70% to 97% (7). Therefore, SCAD can be managed conservatively as long as the patient is clinically stable. However, in cases of ongoing ischemia or hemodynamic instability, PCI or CABG should be considered. PCI for treatment is associated with an increased risk of iatrogenic dissections or extensions of dissection as a result of underlying arteriopathies (8). The indication for CABG includes left main, proximal left descending artery, or multiple vessel diseases; technical failure or complications of PCI; and refractory ischemia despite conservative management. As a result of subsequent healing of native dissected arteries leading to competitive flow, a high rate of graft occlusions has been reported (8).

FOLLOW-UP

The patient was transferred to intensive care unit. Chest closure occurred on post-operative day 2. She was extubated on post-operative day 3 and discharged home on post-operative day 9 without complications. Patient was discharged home on aspirin (81 mg/day), carvedilol (25 mg/day), isosorbide mononitrate (30 mg/day), and rosuvastatin (5 mg/day). Prolactin level during admission was 21 ng/ml (normal range, 2.8 to 29.2 ng/ml). Cabergoline was discontinued and prolactinoma was going to be followed by her endocrinologist.

CONCLUSIONS

SCAD commonly occurs in young females without risk factors of atherosclerotic CAD. Although there are multiple factors that could cause SCAD, the use of cabergoline for the treatment of hyperprolactinemia could be related to its incidence. CABG was effective in this symptomatic patient. Further follow-up is necessary to assess long-term outcomes of CABG in SCAD. More recognition about the disease could lead to earlier diagnosis and treatment as well as prevent sudden death.

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KEY WORDS acute coronary syndrome, coronary angiography, coronary artery bypass

APPENDIX For a supplemental video, please see the online version of this paper.