JACC: CASE REPORTS VOL. 20, 2023

CASE REPORT

ADVANCED

CLINICAL CASE

Challenge in Diagnosis and Management of a Patient With Myocardial Bridge and Coronary Artery Spasm

Edward T. Ha, MD,[a] Yongxia S. Qu, MD,[a,b] Tatsunori Takahashi, MD,[c] Manish A. Parikh, MD,[a,b] Yuhei Kobayashi, MD[a,b]

ABSTRACT

Positive ischemia by noninvasive imaging studies often results in nonobstructive disease in cardiac catheterization. In this case, we observed ischemia by nuclear stress test in only the anteroseptal area, and the apex is free of ischemia. Coronary angiogram findings were unremarkable, but intravascular ultrasound confirmed the long length of the myocardial bridge. Further testing with spasm provocation and microvascular testing showed diffuse epicardial spasm in this area of myocardial bridge without microvascular dysfunction. We observed the myocardial bridge but no microvascular dysfunction. This case illustrates the coexistence of spasm in the area of a myocardial bridge and the challenges in the medical management of these patients. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2023;20:101950) © 2023 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/).

HISTORY OF PRESENTATION

A 69-year-old man presented with a 4-month history of chest pain mainly on resting. The patient's vital signs on presentation included blood pressure of 142/78 mm Hg and a pulse of 77 beats/min. An electrocardiogram showed normal sinus rhythm and poor R-wave progression through the precordial leads. Physical examination findings were unremarkable. Routine laboratory study results were significant for an elevated hemoglobin A_{1c} of 7.0%. A transthoracic echocardiogram demonstrated a normal ejection fraction. He underwent an exercise nuclear stress test, which demonstrated a reversible defect localized in the mid to basal anterior wall as well as the midseptal wall without apical involvement (**Figure 1**).

LEARNING OBJECTIVES

- To be able to make and confirm the diagnosis of myocardial bridge with nuclear stress imaging and intravascular ultrasound, particularly with normal coronary angiogram findings.
- To understand the importance of testing for epicardial coronary artery spasm in the presence of a myocardial bridge.
- To understand the importance of tailored medical management in patients who have a myocardial bridge and coronary artery spasm at the same time.

PAST MEDICAL HISTORY

The patient had a history of hypertension, hyperlipidemia, and diabetes mellitus. He was also a former cigarette smoker more than 30 years prior.

From the [a]Division of Cardiology, NewYork-Presbyterian Brooklyn Methodist Hospital, Brooklyn, New York, USA; [b]Weill Cornell Medical College, New York, New York, USA; and the [c]Jacobi Medical Center, Bronx, New York, 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 June 13, 2023; accepted June 22, 2023.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2023.101950

ABBREVIATIONS AND ACRONYMS

CAS = coronary artery spasm

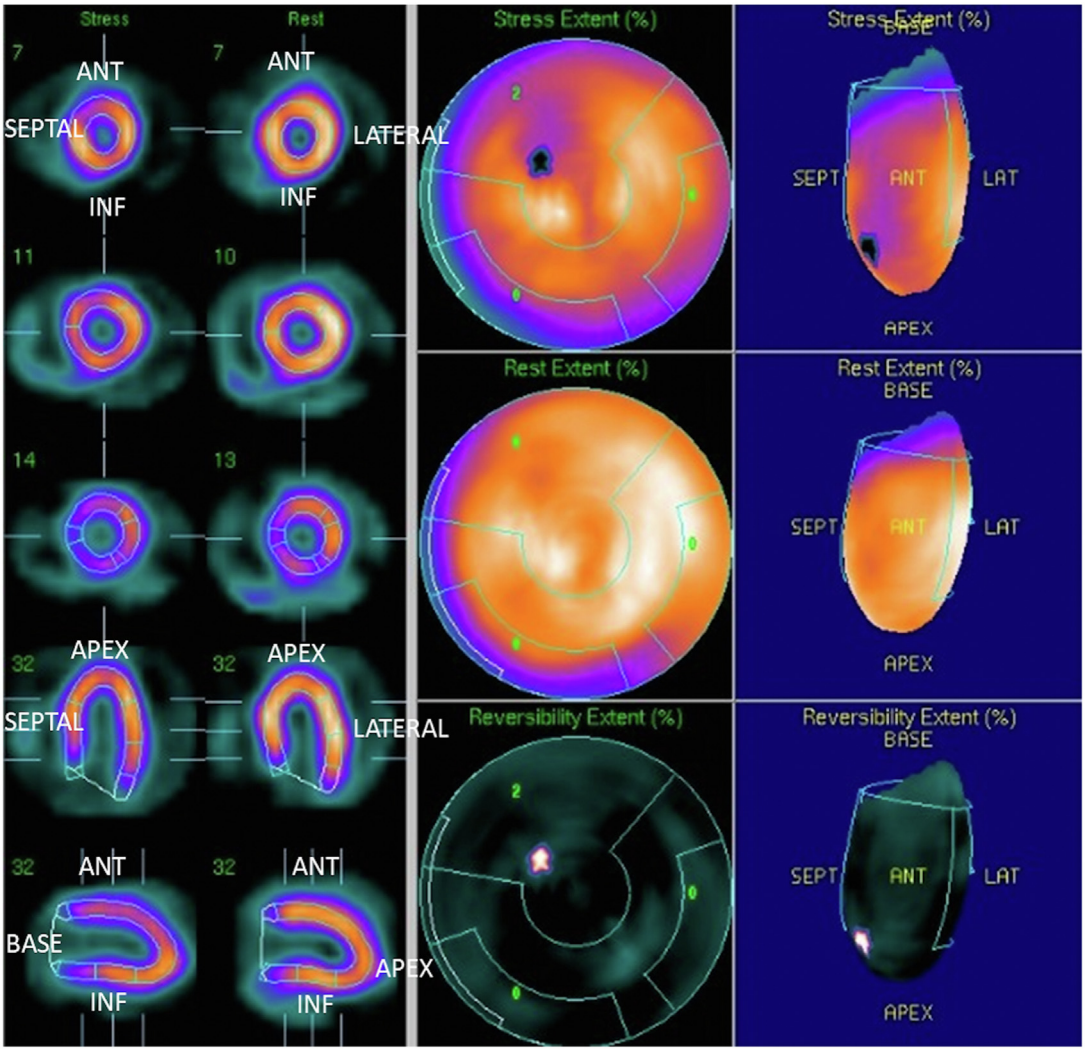
LAD = left anterior descending coronary artery

MB = myocardial bridge

DIFFERENTIAL DIAGNOSIS

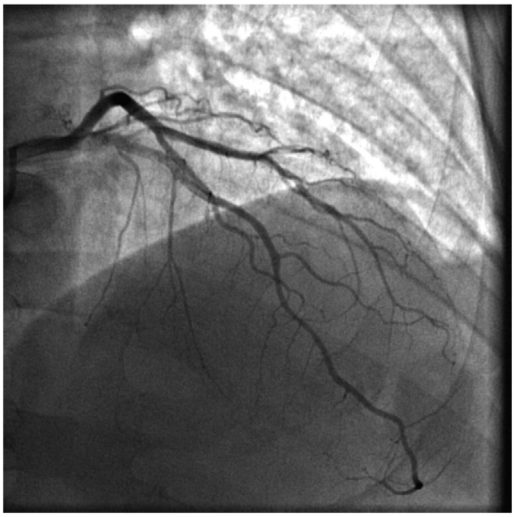
The differential diagnosis at this timepoint was angina pectoris caused by either epicardial or microvascular coronary disease, although the occurrence of symptoms only in the resting state was atypical. However, the pattern of a focal, reversible defect pattern seen on the nuclear stress test was considered to be most consistent with the presence of a myocardial bridge (MB) in the left anterior descending coronary artery (LAD). In patients with an MB, negative pressure created by increased blood flow velocity in the narrow MB segment can pull blood from the adjacent septal

FIGURE 1 Nuclear Stress Test Demonstrating Ischemia in the Left Anterior Descending Coronary Artery Territory But Sparing the Apex



ANT = anterior; INF = inferior; LAT = lateral; SEPT = septal.

FIGURE 2 Coronary Angiogram



and diagonal branches into the LAD during the systolic and early diastolic phases, known as the "Venturi" effect, and can cause localized myocardial ischemia in those side branch territories but typically no ischemia in the apex.[1,2]

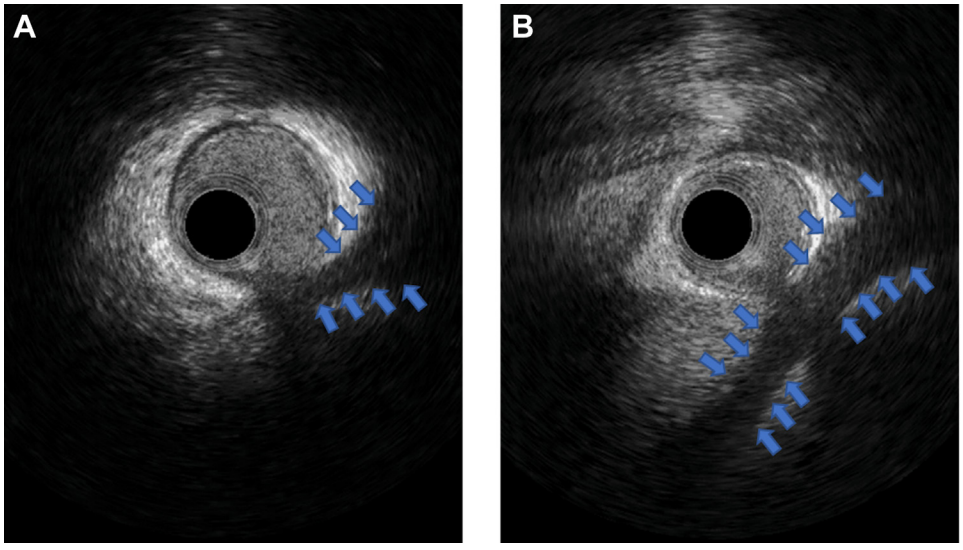
INVESTIGATIONS

Subsequent coronary angiogram demonstrated only mild atherosclerotic disease at the proximal LAD without evidence of an angiographically detectable MB (**Figure 2**, Video 1). Invasive coronary physiologic assessment with resting full-cycle ratio and fractional flow reserve was performed in the distal LAD, the results of which were both negative for ischemia and confirmed no significant epicardial disease; the microvascular function was also borderline as assessed by coronary flow reserve and the index of microcirculatory resistance (**Figure 3**).[3] A decision was made to assess the LAD with intravascular ultrasound and perform provocative spasm testing to identify the cause of his symptoms. Intravascular ultrasound revealed a typical "halo" finding from the mid to distal LAD, confirming the presence of a long segment of an MB (**Figure 4**, Video 2).[3,4] During a spasm provocation test with intracoronary injection of incremental acetylcholine doses up to 100 μg, significant epicardial coronary artery spasm (CAS) was observed in the segment of the MB, with reproduction of his usual chest pain and ischemic electrocardiogram changes (**Figure 5**, Video 3).

FIGURE 3 Physiology Tracing: FFR/RFR and CFR/IMR

CFR = coronary flow reserve; FFR = fractional flow reserve; IMR = index of microcirculatory resistance; RFR = resting full-cycle ratio.

FIGURE 4 Intravascular Ultrasound Showing Diastole vs Systole of Myocardial Bridge

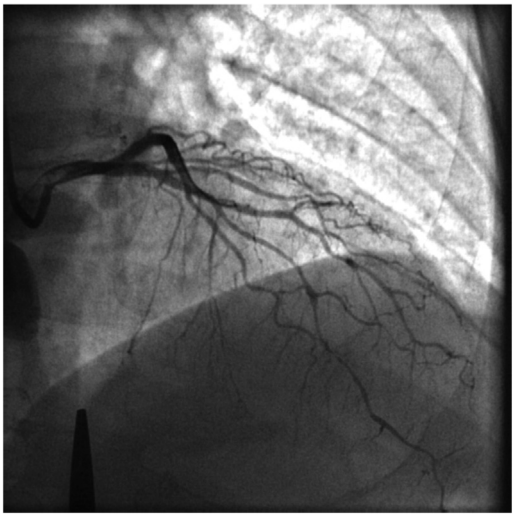


(A) Diastole. **(B)** Systole. Blue arrows demonstrate the typical band of echolucent area commonly referred to as the "half moon" or "halo" sign.

MANAGEMENT

Accordingly, the patient was diagnosed with severe coronary vasospastic angina associated with a long segment of MB. Based on the result of this comprehensive invasive assessment and persistent symptoms while on a beta-blocker, the decision was made to discontinue the beta-blocker and initiate verapamil with nitroglycerin as needed. The patient's symptoms improved with this tailored medical therapy.

FIGURE 5 Angiogram After 100 μg of Intracoronary Acetylcholine Injection



DISCUSSION

An MB is presumed to be a benign entity, although ischemic symptoms have been described and are usually treated with a beta-blocker because it typically presents with proximal atherosclerotic disease[5] and lowering heart rate will also increase diastolic time and thereby lessen the degree of ischemia from MB.[6,7]

CAS is increasingly recognized as a cause of angina with no obstructive coronary arteries and is usually diagnosed by invasive provocative spasm testing because there is currently no established, noninvasive method to accurately identify CAS.[8] The provocation of CAS is typically done with intracoronary acetylcholine administration, and recent meta-analysis demonstrated its safety profile in patients with angina with no obstructive coronary arteries.[9]

The presence of an MB may hinder the identification of underlying CAS, but the 2 entities can frequently coexist.[7,10] Correct diagnosis of coronary

vasospasm in patients with an MB is integral because management schemes differ. As discussed, a beta-blocker is the typical first choice for patients with an MB but can worsen CAS. The use of calcium-channel blockers is the first choice for CAS and has the added benefit of possibly improving symptoms of MB through similar mechanisms to beta-blockade of reducing heart rate.[11,12]

In this case, interpreting the specific pattern of noninvasive stress testing was a key to suspecting the presence of an MB and prompted the use of intravascular ultrasound, which confirmed the presence of the MB. Furthermore, the patient, who is taking a beta-blocker, continued to have resting anginal symptoms, which also made us consider the coexistence of CAS. A clinical dilemma exists in patients with an MB, because the MB itself, proximal atherosclerotic disease, and CAS can cause anginal symptoms through the different mechanisms. Therefore, detailed invasive assessments and tailored medical management is a key for the management of patients with an MB.

CONCLUSIONS

This case demonstrates the importance of combining "functional" coronary angiography, which focuses on the functional aspect of coronary artery disease, in addition to traditional "structural" coronary angiography for the evaluation of ischemic symptoms in the setting of a positive stress test result with findings of non-obstructive coronaries with myocardial bridging.[13]

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Parikh serves on the advisory boards of Abbott Vascular, Boston Scientific, and Medtronic. Dr Kobayashi serves as a consultant to Abbott Vascular. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Yuhei Kobayashi, New York-Presbyterian Brooklyn Methodist Hospital/Weill Cornell Medicine, Division of Cardiology, 506 6th Street, Brooklyn, New York 11215, USA. E-mail: dxl9003@med.cornell.edu.

REFERENCES

1. Gould KL, Johnson NP. Myocardial bridges: lessons in clinical coronary pathophysiology. *J Am Coll Cardiol Img*. 2015;8:705–709.

2. Klues HG, Schwarz ER, vom Dahl J, et al. Disturbed intracoronary hemodynamics in myocardial bridging: early normalization by intracoronary stent placement. *Circulation*. 1997;96:2905–2913.

3. Fearon WF, Kobayashi Y. Invasive assessment of the coronary microvasculature: the index of microcirculatory resistance. *Circ Cardiovasc Interv*. 2017;10:e005361.

4. Aoi S, Maehara A, Takahashi T, Latib A, Kobayashi Y. OCT-defined myocardial bridge as a homogenous band: validation with a hybrid IVUS-OCT catheter. *Cardiovasc Revasc Med*. 2023;46:122–123.

5. Yamada R, Tremmel JA, Tanaka S, et al. Functional versus anatomic assessment of myocardial bridging by intravascular ultrasound: impact of arterial compression on proximal atherosclerotic plaque. *J Am Heart Assoc*. 2016;5:e001735.

6. Schwarz ER, Klues HG, vom Dahl J, Klein I, Krebs W, Hanrath P. Functional, angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication. *J Am Coll Cardiol*. 1996;27:1637–1645.

7. Lee BK, Lim HS, Fearon WF, et al. Invasive evaluation of patients with angina in the absence of obstructive coronary artery disease. *Circulation*. 2015;131:1054–1060.

8. Pargaonkar VS, Kobayashi Y, Kimura T, et al. Accuracy of non-invasive stress testing in women and men with angina in the absence of obstructive coronary artery disease. *Int J Cardiol*. 2019;282:7–15.

9. Takahashi T, Samuels BA, Li W, et al. Safety of provocative testing with intracoronary acetylcholine and implications for standard protocols. *J Am Coll Cardiol*. 2022;79:2367–2378.

10. Montone RA, Gurgoglione FL, Del Buono MG, et al. Interplay between myocardial bridging and coronary spasm in patients with myocardial ischemia and non-obstructive coronary arteries: pathogenic and prognostic implications. *J Am Heart Assoc*. 2021;10:e020535.

11. Corban MT, Hung OY, Eshtehardi P, et al. Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. *J Am Coll Cardiol*. 2014;63:2346–2355.

12. Sternheim D, Power DA, Samtani R, Kini A, Fuster V, Sharma S. Myocardial bridging: diagnosis, functional assessment, and management: *JACC* state-of-the-art review. *J Am Coll Cardiol*. 2021;78:2196–2212.

13. Ha ET, Attallah M, Parikh MA, Kobayashi Y. Positive nuclear stress test after STEMI: importance of invasive physiologic assessment and acetylcholine testing. *J Am Coll Cardiol Intv*. 2023;16:1116–1117.

KEY WORDS ANOCA, coronary artery spasm, myocardial bridge

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