American Journal of Medical Case Reports, 2015, Vol. 3, No. 6, 181-183
Available online at http://pubs.sciepub.com/ajmcr/3/6/9

DOI:10.12691/ajmcr-3-6-9

A Bridge to Sudden Cardiac Death

Umayr Azimi*, Hamza Hashmi

Internal Medicine, Grand Rapids Medical Education Partners/Michigan State University, Grand Rapids, Michigan, USA
*Corresponding author: umayr.azimi@grmep.com

Received April 28, 2015; Revised May 24, 2015; Accepted May 29, 2015

Abstract Coronary arteries normally traverse the epicardium of myocardial tissue. Myocardial bridging occurs when a portion of coronary artery passes intramurally through myocardial tissue. We present a case of pulseless arrest during a bike race in an otherwise healthy individual. Coronary angiography showed extensive myocardial bridging of the mid-LAD and mild anteroapical hypokinesis. The case demonstrates situations when myocardial bridging may be of clinical significance.

Keywords: *MI, myocardial infarct, myocardial bridging, bridging, bridge, SCD, sudden cardiac death, arrhythmia, AICD, ICD*

Cite This Article: Umayr Azimi, and Hamza Hashmi, "A Bridge to Sudden Cardiac Death." *American Journal of Medical Case Reports*, vol. 3, no. 6 (2015): 181-183. doi: 10.12691/ajmcr-3-6-9.

1. Introduction

Coronary arteries normally traverse the epicardium of myocardial tissue. Myocardial bridging occurs when a portion of coronary artery passes intramurally through myocardial tissue. During systole, the myocardial tissue compresses the intramural vessel. Because the heart receives blood during diastole, this only becomes clinically significant with persistence of vessel narrowing in early diastole, resulting from severe compression during systole or with increased contractility, which decreases diastole.

2. Patient Description

The patient was a previously healthy 38-year old Caucasian male, with a BMI of 25, who was an avid bike rider. He exercised five times weekly, had no previous cardiac risk factors, no history of substance abuse and no cardiac family history. He presented after pulseless arrest during a bike race. CPR was performed immediately for 10 minutes until defibrillation for pulseless ventricular tachycardia could be delivered.

After presentation to the hospital, the patient was found to have elevated troponins initially 0.020 with a maximum value reaching 0.360. EKG exhibited large T waves, but no ST segment or T wave changes as seen in Figure 1 below. Electrolytes were seen to be within normal limits. CT of the thorax with contrast failed to show any irregularities aside from a fractured rib and signs of aspiration pneumonitis. Drug screen was seen to be negative. Transthoracic echocardiogram showed a left ventricular ejection fraction of 50% but no other abnormalities. There were no recurrent arrhythmias on telemetry during his hospitalization. The patient underwent coronary angiography showing extensive myocardial bridging of his mid-LAD measuring 60mm in length with mild antero-apical hypokinesis as seen in Figure 2 below. There was no evidence of coronary spasm, stenosis or coronary artery disease. After discussion of treatment options, the patient was started on a beta-blocker and asingle chamber AICD was placed, which he then tolerated well. About 1 month post-discharge, the patient underwent exercise stress testing, which showed an ejection fraction of 46% with a small fixed perfusion abnormality in the inferior wall, with the patient reaching exercise to 12.9 METS. Follow up echocardiogram, showed an EF of 50% to 55% with very subtle apical hypokinesis and no further changes were made.

3. Case Discussion

Myocardial bridging is the most common coronary anatomic variant, reported to be mostly limited to the mid to distal LAD. It was first discovered as an autopsy finding in 1737 but it was not until 1976, that the bridge was found to undergo transient narrowing during systole angiographically [2]. It is estimated to be found in 0.5-16% of angiograms incidentally [1]. It has been reported to be seen in an average of 25% of individuals on autopsy with a range of 5- 86% [1,2,5].

Typical symptoms associated with symptomatic myocardial bridging include chest pain, palpitations, fatigue or other non-specific symptoms. In extreme cases MI or sudden cardiac death can occur. In most cases these symptoms are usually not attributed to the bridging, however it is important to seriously consider myocardial bridging in the differential diagnosis as the cause in otherwise young healthy individuals without substantial risk factors for CAD. No unified treatment or classification methods are currently in place. The best current practice per case reports and small-scale studies employ negative inotropic agents such as beta-blockers and calcium channel blockers as first line medical therapy.

These are seen to decrease time in systole and increase diastole thereby increasing blood flow through the coronary vessels. AICDs are increasingly being used to prevent recurrent malignant arrhythmias that can lead to sudden cardiac death and was placement was employed in this case. Stent placement has also been attempted, but with high risk of failure, mostly due to occlusion within 1-2 years. If these fail to adequately control symptoms, surgical intervention may ultimately be necessary with myotomy or CABG. In small non-randomized studies, it has been seen that both can be effective but myotomy is seen to be associated with higher risk of ventricular rupture, bleeding, or aneurysm formation [3]. This patient underwent cardiac catheterization, however no evidence for stenosis/ischemia was seen and after discussion of the several treatment options previously mentioned, he felt placement of AICD and medical management was the better option for him. He was given instructions to not exercise, until cleared by his cardiologist on outpatient follow up visits.

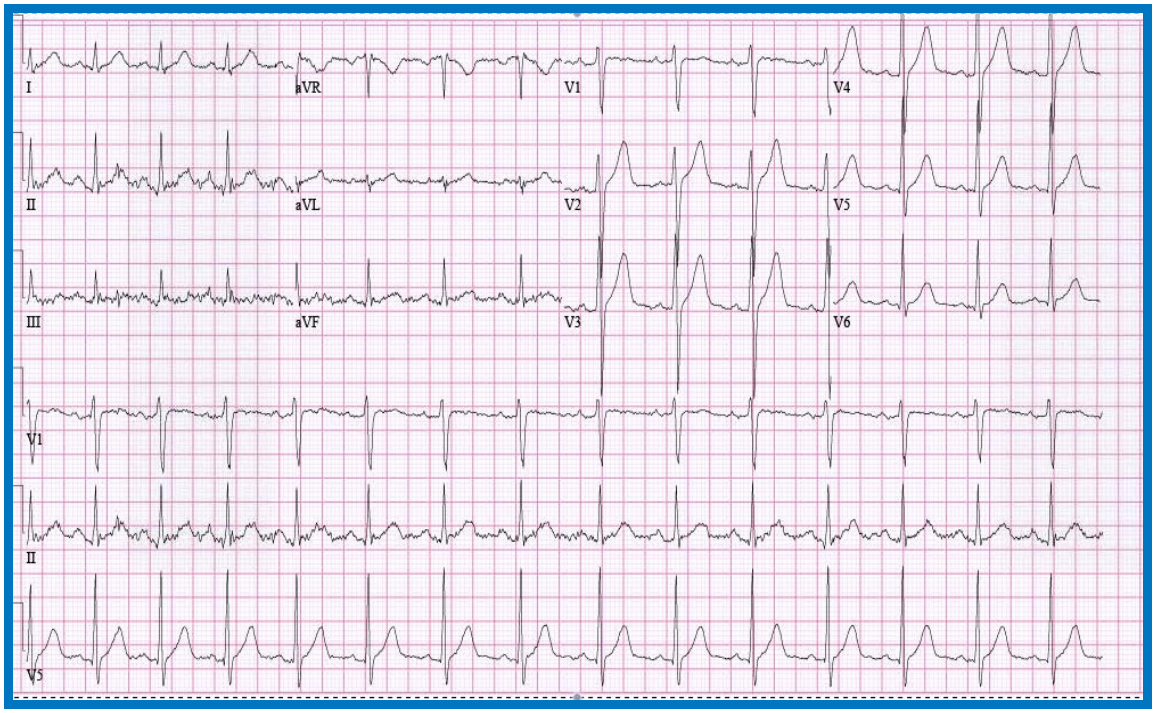

Figure 1. showing large T waves in leads V2- V5.

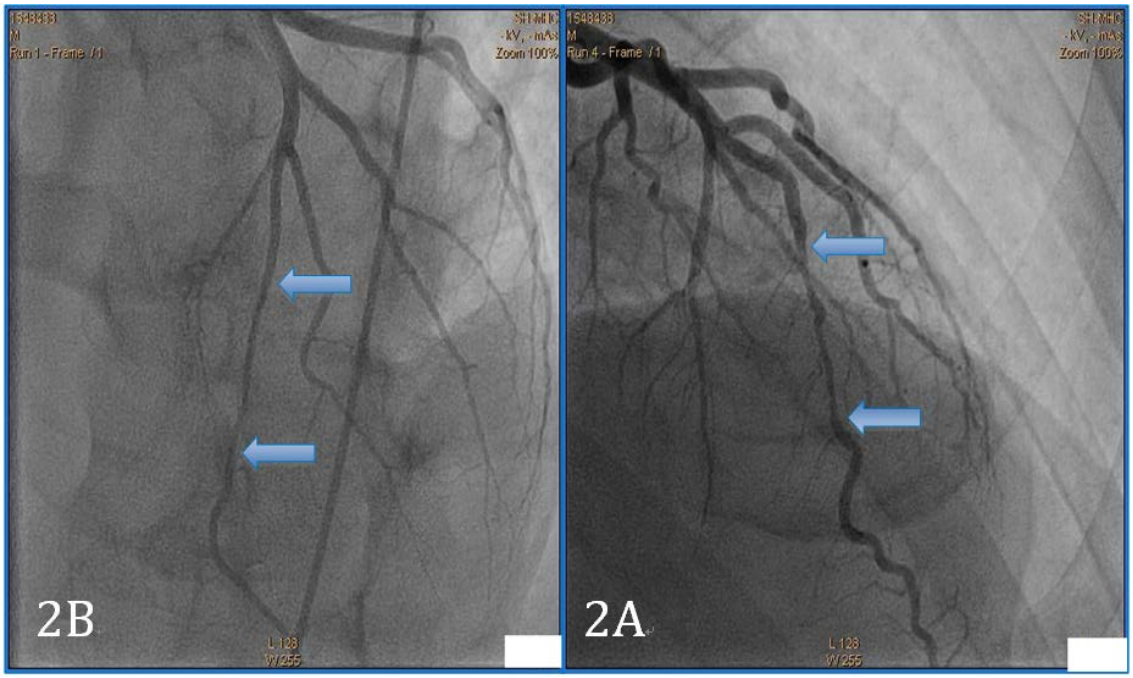

Figure 2. showing extensive myocardial bridging between blue arrows. 2a during diastole and 2b during systole

The case presented demonstrates the possible malignant nature of extensive myocardial bridging in an otherwise healthy individual without risk factors for heart disease. Either symptomatic patients or individuals with extensive bridging usually greater than 25mm with evidence for ischemia or arrhythmias should be treated appropriately. This case in particular was challenging from a treatment perspective. While the patient exhibited low normal

ejection fraction and troponin leak, there was no evidence of stenosis albeit a fixed perfusion deficit was seen on stress testing during follow up. While CABG and Myotomy were discussed as potential treatment options for the future, it was felt these options are promising but still controversial therapies and the patient decided to pursue medical management with Metoprolol and AICD placement for secondary prevention of malignant arrhythmia. It will be important for future similar cases to be reported so more evidence based practice can be supported with larger scale trials for more definitive treatment conclusions.

4. Conclusion

This case demonstrates an uncommon cause for sudden cardiac arrest in the young, which is more commonly caused by arrhythmia or hypertrophic cardiomyopathy. The finding of myocardial bridging as an anatomic variant can be of clinical importance with extensive bridging as was seen in our patient. There is currently no consensus therapeutic intervention. Beta-blockers are one option as they can reduce cardiac contractility while increasing time in diastole, thus decreasing coronary artery compression in those with myocardial bridging. AICD placement is another non-surgical treatment that can help prevent arrhythmia caused by extensive myocardial bridging from leading to sudden cardiac death. If these medical therapies fail, surgical intervention may be necessary with myotomy or CABG. It is important to determine whether myocardial bridging is the cause of cardiac symptomatology and this case helps demonstrate an otherwise “benign” condition as the cause of sudden cardiac death in young, healthy individuals.

Acknowledgements

None.

Statement of Competing Interests

The authors do not have any statement of competing interests.

Abbreviations

AICD = Automatic Implantable Cardioverter-Defibrillator
BMI = Body Mass Index
CABG = Coronary Artery Bypass Grafting
CAD = Coronary Artery Disease
CPR = Cardiopulmonary Resuscitation.

References

[1] Schwarz, Ernst R., Rajiv Gupta, Philipp K. Haager, JuergenVom Dahl, Heinrich G. Klues, JuergenMinartz, and Barry F. Uretsky. "Myocardial Bridging in Absence of Coronary Artery Disease: Proposal of a New Classification Based on Clinical-Angiographic Data and Long-Term Follow-Up." *Cardiology* 112.1 (2009): 13-21.

[2] Sciahbasi, Alessandro, Francesco Summaria, Roberto Patrizi, and Ernesto Lioy. "Cardiac Arrest and Myocardial Bridging." *Italy Heart Journal* 5 (2004): 869-71.

[3] Attaran, S., M. Moscarelli, T. Athanasiou, and J. Anderson. "Is Coronary Artery Bypass Grafting an Acceptable Alternative to Myotomy for the Treatment of Myocardial Bridging?" *Interactive CardioVascular and Thoracic Surgery* 16.3 (2013): 347-49.

[4] Faruqui, AzharM.a., William C. Maloy, Joel M. Felner, Robert C. Schlant, William D. Logan, and PanagiotisSymbas. "Symptomatic Myocardial Bridging of Coronary Artery." *The American Journal of Cardiology* 41.7 (1978): 1305-310.

[5] Hostiuc, S., G. Curca, D. Dermengiu, S. Dermengiu, M. Hostiuc, and M. Rusu. "Morphological Changes Associated with Hemodynamically Significant Myocardial Bridges in Sudden Cardiac Death." *The Thoracic and Cardiovascular Surgeon* 59.07 (2011): 406-10.

[6] Mohlenkamp, S. "Update on Myocardial Bridging." *Circulation* 106.20 (2002): 2616-622.