

Epicardial Ultrasound In A Case of Myocardial Bridge and Apical Hypertrophic Cardiomyopathy

Lindsey Whalen¹, Stephen Davies¹, Karen Singh¹, and Gorav Ailawadi¹

¹University of Virginia Health System

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Abstract

A 59-year-old male with a history of unstable angina was diagnosed with a myocardial bridge of the left anterior descending artery (LAD) and apical variant hypertrophic cardiomyopathy (AHCM). He underwent unroofing of the myocardial bridge and a left ventricular apical myectomy. Intraoperatively, epicardial ultrasound was used to identify the myocardial bridge with systolic compression of the LAD and confirm resolution of this compression postoperatively. Furthermore, epicardial ultrasound was used for guiding the degree of apical resection of the decompressed heart. This novel use of intraoperative epicardial ultrasound can help guide surgeons preoperatively and confirm results immediately after an operation.

Title : Epicardial Ultrasound In A Case of Myocardial Bridge and Apical Hypertrophic Cardiomyopathy

Running Head : Epicardial US for Myocardial Bridge and Apical HCM

Authors : L. Brett Whalen BS¹, Stephen W. Davies MD¹, Karen Singh MD², Gorav Ailawadi MD, MBA¹

Institutions and affiliations : University of Virginia Health System Department of Surgery, Division of Cardiac Surgery Charlottesville, VA, USA¹,

University of Virginia Health System Department of Anesthesiology Charlottesville, VA, USA²

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Corresponding author :

Gorav Ailawadi, MD, MBA

University of Virginia

1215 Lee Street

Charlottesville VA, 22908

Email: GA3F@hscmail.mcc.virginia.edu

Phone: (434) 924-5052

Abstract :

A 59-year-old male with a history of unstable angina was diagnosed with a myocardial bridge of the left anterior descending artery (LAD) and apical variant hypertrophic cardiomyopathy (AHCM). He underwent unroofing of the myocardial bridge and a left ventricular apical myectomy. Intraoperatively, epicardial ultrasound was used to identify the myocardial bridge with systolic compression of the LAD and confirm resolution of this compression postoperatively. Furthermore, epicardial ultrasound was used for guiding the

degree of apical resection of the decompressed heart. This novel use of intraoperative epicardial ultrasound can help guide surgeons preoperatively and confirm results immediately after an operation.

Introduction :

Apical variant hypertrophic cardiomyopathy (AHCM,) characterized by non-obstructive hypertrophy of the myocardium in the left ventricular apex,¹ accounts for just 3% of hypertrophic cardiomyopathy (HCM) cases in the United States.² Patients with AHCM, although often asymptomatic, can present with chest pain, heart failure, dyspnea, or syncope.² A myocardial bridge, with resultant compression of a coronary artery during systole, is often asymptomatic, but can similarly lead to morbid events.³ Patients with HCM who require myectomy are rare and only 15% of patients with HCM have a myocardial bridge.⁴ Since just 3% of HCM cases are of the apical variant, the combination of these pathologies as reported here is exceedingly uncommon.

Herein, we present a patient who underwent surgical repair of a myocardial bridge of the LAD and a myectomy for AHCM . We report the novel use of a sterile, epicardial, high frequency ultrasound (HFUS) with a 15 MHz imaging probe and MiraQTM system (Medistim ASA, Oslo, Norway) to document the myocardial bridge throughout systolic LAD compression as well as guide the amount of apical resection required on a decompressed heart.

Case Report :

A 59 year-old male presented to us in 2018 with a 10-year history of intermittent chest tightness, shortness of breath and fatigue now reporting worsening, unstable angina over the last two years. A previous cardiac MRI in 2016 demonstrated modest asymmetric thickening of the apical septum, left ventricular free wall and apical lateral wall. His history was significant for hyperlipidemia, hypertension, hypothyroidism and nephrolithiasis.

A cardiac catheterization was done due to worsening exertional angina and showed a left anterior descending artery (LAD) myocardial bridge with 60% mid LAD compression (Figure 1A). A subsequent CT coronary angiography demonstrated a 1cm segment myocardial bridge with a short segment of compression in the proximal LAD. Given his significant NYHA Classification III symptoms and low surgical risk, he was referred to us for surgical repair.

Intraoperatively, the transesophageal echo (TEE) exam revealed (Figure 2A) severe apical hypertrophy with an apical thickness measurement of 2.5 cm. The biplane method of disks calculated a low normal end-diastolic volume, an ejection fraction of 50%, and a low stroke volume index of 19 ml/m². Given his severity of symptoms unlikely to be explained by the myocardial bridge, the decision was made to address the AHCM as well. Once the sternum had been opened and the pericardial well created, HFUS was utilized to evaluate the submyocardial LAD trajectory and left ventricle apical myocardial thickness. Using this technology, the LAD was first identified lying deep to a thick layer of epicardial adipose tissue. We then traced the LAD more proximally to localize the overlying myocardial bridge. During systole, the LAD was almost completely occluded, yet widely patent during diastole (Video 1, Figure 1B). After crossclamp, the myocardial bridge was released along the length of the compressed LAD with cautery. Our attention was then turned to the left ventricular (LV) apex.

The hypertrophic apex was assessed and measured with the probe (Video 3, Figure 2C) to help guide the extent of resection necessary, which was roughly 1.5 cm depth circumferentially. The LV apex was opened using a #10 scalpel blade 2-3 cm parallel to the LAD. The apical septum and walls were aggressively cored out with a #10 blade and metzenbaum scissors, taking care not to injure the papillary muscles. The LV apex was widely opened leaving roughly 1 cm of residual LV wall thickness, confirmed by HFUS. After thoroughly irrigating and clearing of residual debris, the LV apex was closed using felt and a double layered, 2-0 prolene horizontal mattress, followed by running closure. The suture line was further secured with Bioglue (CryoLife, Inc., Kennasaw, GA). The patient was rewarmed and the aortic cross clamp was removed. TEE demonstrated improved end-diastolic volume despite an underfilled left ventricle immediately after cardiopulmonary bypass;

however, a normal contour of the LV apex was achieved (Figure 2B) and repeat Medistim evaluation of the LAD now showed a widely patent vessel throughout its course (Video 2, Figure 1C).

The patient's postoperative course was unremarkable. Postoperatively, he noted dramatically improved symptoms with resolution of his angina and was able to resume all activities including returning to full-time work by 2 months after surgery. Now at 1.5 years follow-up, he remains in NYHA Class I functional class with no residual angina or shortness of breath.

Comment :

Our patient underwent a rare combination of a myotomy of a myocardial bridge and an apical myectomy. Although a previous case report documented a patient with this combination,⁶ the present report shows the utility of epicardial ultrasound to define the extent and guide the resection of both pathologies. HFUS helped rapidly identify the myocardial bridge, clearly demonstrating the pathophysiology of the disease, as well as confirmed uninterrupted blood flow after myotomy. HFUS was also highly useful in guiding the depth of resection of the AHCM, allowing the surgeon to determine how much to resect on an arrested, empty heart, unlike most preoperative imaging modalities (MRI, TEE), which are in the setting of a full, beating heart.

Preoperative diagnosis of both AHCM and myocardial bridging is commonly performed with cardiac MRI.^{1,2} AHCM is also seen on ventriculography and echocardiography as a "spade-shaped" end-diastolic volume^{1,2}. Myocardial bridging is typically diagnosed via coronary angiography.^{3,5} However, none of these modalities provide real-time guidance to the surgeon intraoperatively, particularly on a flaccid heart. In fact, HFUS may be further useful in more conventional cases of hypertrophic cardiomyopathy to guide the resection depth and avoid ventricular septal defects.

In summary, this case describes a rare combination of two distinct pathologies. Similar to the real time feedback cardiologists get when performing coronary or valvular procedures on the beating heart, epicardial ultrasound gives the surgeon useful real-time guidance intraoperatively that may provide a safer, more effective result.

Keywords: cardiovascular pathology; cardiovascular research; perfusion

Author contributions:

L. Brett Whalen: drafting article

Gorav Ailawadi: concept/design, critical revision

Stephen W. Davies: critical revision

Karen Singh: data collection, analysis, interpretation

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Figure legend:

1. A. A cardiac catheterization image indicates compression (arrow) of the left anterior descending artery (LAD) due to a myocardial bridge. B. A frame of the Medistim video during systole shows the lumen of the LAD (arrow) is compressed by myocardial bridge above (arrowhead). C. A frame of the Medistim video post-myectomy shows the LAD (arrow) again in systole, now with a patent lumen with no residual muscle above the vessel.

2. A. The pre-apical myectomy TEE mid-esophageal 2 chamber view shows hypertrophied apical myocardium with a classic spade-shaped LV cavity in diastole. B. A Medistim probe image reveals the thickened apical myocardium (arrow) overriding the narrowed LV chamber (thin line). C. The post-CPB TEE mid-esophageal 2 chamber view shows an underfilled LV with a slightly higher estimated end-diastolic volume, as compared to pre-CPB imaging. The shape of the LV apical cavity appears normal after surgical reduction of the apical myocardium.

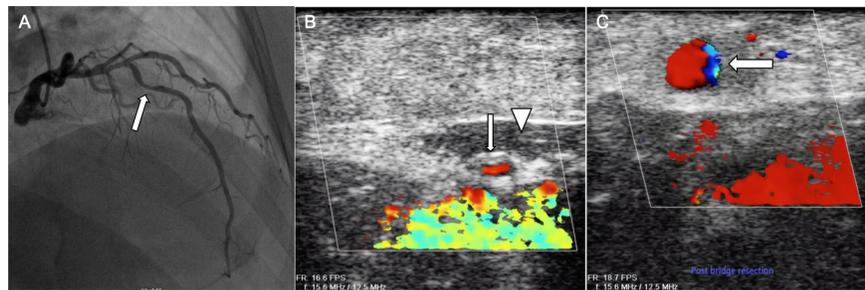
TEE: transesophageal echocardiogram

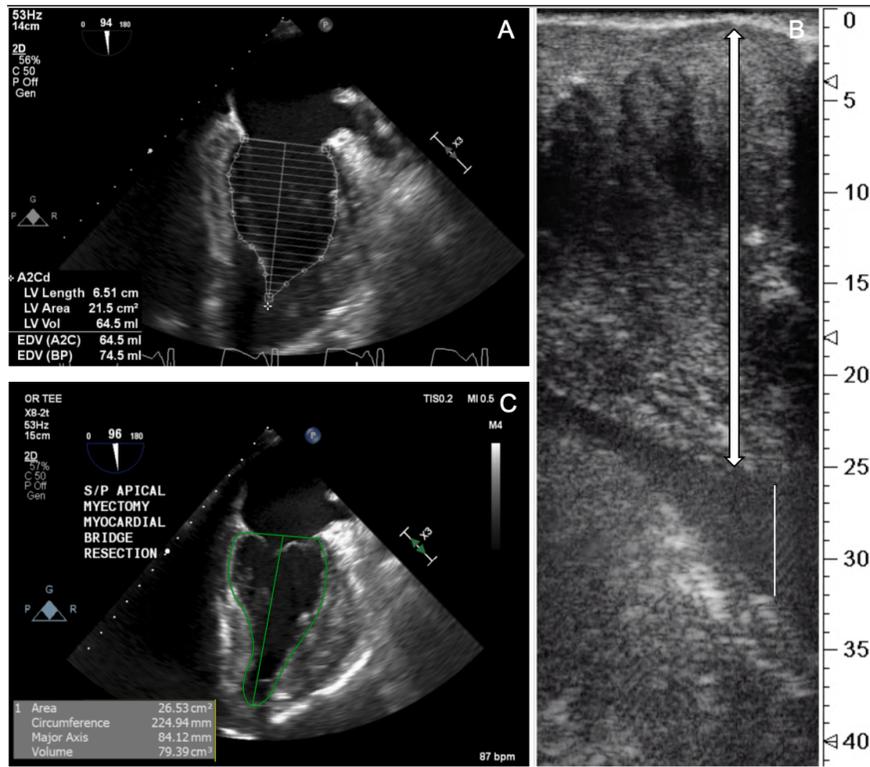
CPB: cardiopulmonary bypass

Video 1. Medistim pre-myectomy LAD.

Video 2. Medistim post-myectomy LAD.

Video 3. Medistim pre-myectomy apical myocardium.





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