JACC: CASE REPORTS VOL. 7, 2023

CASE REPORT

BEGINNER

CLINICAL CASE

Dynamic Left Ventricular Outflow Tract Obstruction Exacerbated by Thoracic Kyphosis

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ABSTRACT

We present a 74-year-old woman with kyphosis and symptoms of pre/syncope. Heart catheterization revealed dynamic left ventricular outflow tract obstruction (DLVOTO) with Brockenbrough Braunwald response only when kyphotic posture was assumed. She had a positive response to metoprolol. DLVOTO is a challenging diagnosis in the absence of resting LVOTO. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2023;7:101710) © 2023 The Authors. 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/).

A 74-year-old woman presented with recurrent near-syncope/syncopal episodes over the past year. These symptoms started after she experienced compression fractures resulting in a 12-cm height loss. They happened after she had stood for long periods, and a full syncopal event happened after she had stood for approximately 1 hour 9 months previously. The patient also had intermittent right upper quadrant (RUQ) pain that would improve with lying flat.

LEARNING OBJECTIVES

- To keep DLVOTO on the differential diagnosis list of syncope, especially when the clinical context is suggestive, even when no left ventricular outflow tract obstruction is detected on resting echocardiogram.
- To consider potential triggers for DLVOTO when diagnosing this entity, and to reproduce these conditions during testing to help demonstrate the obstruction if present.

A previous work-up, including ambulatory electrocardiogram monitoring for 14 days, showed brief runs of supraventricular tachycardia and otherwise was unremarkable. She underwent a transthoracic echocardiogram (TTE), which showed mild aortic regurgitation and grade 1/3 diastolic dysfunction. In our clinic, the result of her physical examination was significant for normal body mass index, blood pressure of 162/82 mm Hg, kyphosis, and a faint diastolic murmur.

MEDICAL HISTORY

Her medical history was significant for a history of multiple-level compression fractures (T12, L1, L3) resulting in kyphosis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis list included arrhythmia, dynamic left ventricular outflow tract obstruction (DLVOTO), valvular disorders, and heart failure.

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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 Author Center.

Manuscript received November 4, 2022; accepted November 15, 2022.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2022.101710

ABBREVIATIONS AND ACRONYMS

CBC = complete blood count

DLVOTO = dynamic left ventricular outflow tract obstruction

IVC = inferior vena cava

LVOTO = left ventricular outflow tract obstruction

PVC = premature ventricular contraction

RUQ = right upper quadrant

TTE = transthoracic echocardiogram

INVESTIGATIONS

The results of complete blood count, basic metabolic panel, and liver profile were unremarkable. Chest and spine imaging showed thoracic kyphosis (**Figure 1**). Computed tomography of the abdomen and pelvis showed dilation of the intrahepatic inferior vena cava (IVC), with the main portal vein at the upper limits. The electrocardiogram was significant for normal sinus rhythm and left bundle branch block. Repeated TTE was consistent with her previous one (Videos 1 to 3) in addition to showing sigmoid ventricular septum (basal septal prominence 15 mm) (**Figure 2**). There was no DLVOTO (rest, Valsalva). O_2 saturation was consistent at 97% to 99% (rest, Valsalva, standing). Ejection fraction was 57%. The IVC was enlarged but with normal inspiratory collapse.

The patient underwent heart catheterization. At baseline, the right and left atrial filling pressures and the right ventricular and pulmonary artery pressures were normal. The left ventricular filling pressures were mildly elevated (left ventricular end-diastolic pressure 16 mm Hg) in the setting of moderate systemic hypertension (aortic pressure 162/81/110 mm Hg). The cardiac index was normal at 4.5 L/min/m^2 with normal pulmonary vascular resistance of 1.1 WU. No LVOTO was present at rest or after premature ventricular contractions (PVCs). With arm exercise there was further elevation in the aortic pressure to 196/105/141 mm Hg, with severe elevation in the left ventricular and left atrial pressures (left ventricular end-diastolic pressure 26 mm Hg, mean left atrial pressure 26 mm Hg). With isoproterenol up to 4 μg/min, LVOTO developed with a resting gradient of 30 mm Hg and post-PVC gradient up to 50 mm Hg. When a wedge was placed under the back to simulate her kyphosis, severe LVOTO developed, with peak-to-peak gradient >100 mm Hg and a Brockenbrough Braunwald response (**Figure 3**). A coronary angiogram revealed normal coronaries. The findings were consistent with systemic hypertension associated with diastolic dysfunction and heart failure with preserved ejection fraction. There was evidence of provocable severe DLVOTO (isoproterenol + stooped/kyphotic position).

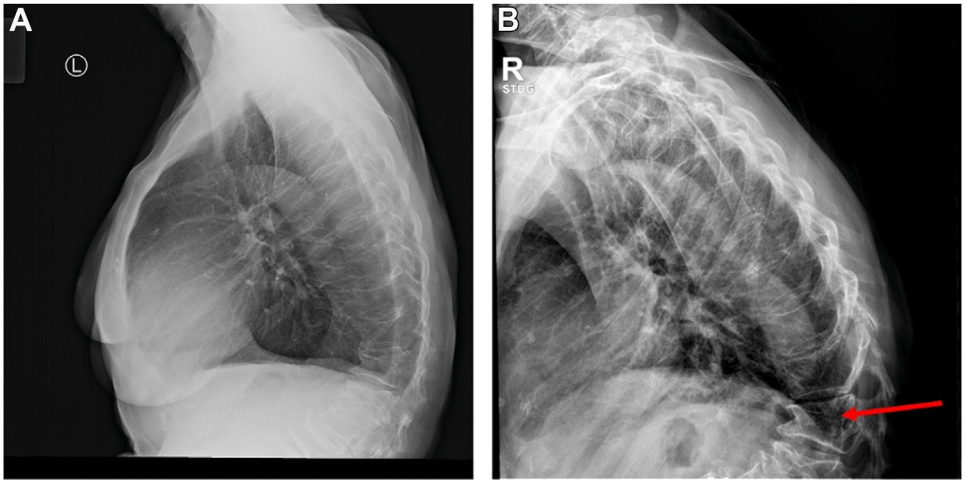
MANAGEMENT

The patient was given an initial prescription for metoprolol succinate 25 mg daily, with recommendation to up-titrate for a goal of <130/80 mm Hg. Given the propensity for DLVOTO, it was recommended that she avoid diuretics and vasodilators. She was advised to meet with physical medicine and rehabilitation specialists to discuss management strategies for kyphosis.

DISCUSSION

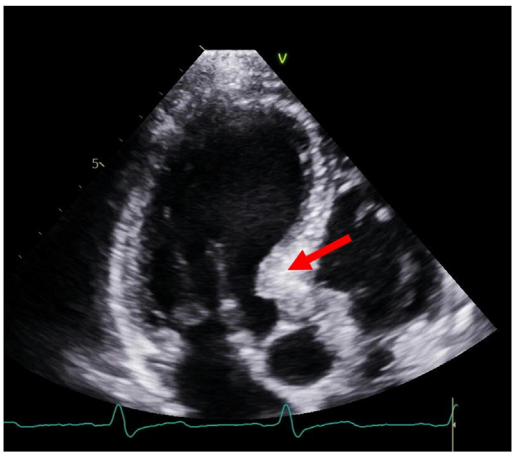
DLVOTO may occur in a wide variety of pathologic conditions, most commonly hypertrophic cardiomyopathy.[1] LVOTO results in decreased stroke volume and increased left atrial pressure, which may result in

FIGURE 1 Chest and Spine Radiography Showing Kyphosis



Lateral chest **(A)** and spine **(B)** radiography showing kyphosis in a patient with compression fracture at T12 **(red arrow)**.

FIGURE 2 **Transthoracic Echocardiogram Apical Long-Axis View, Sigmoid Septum**

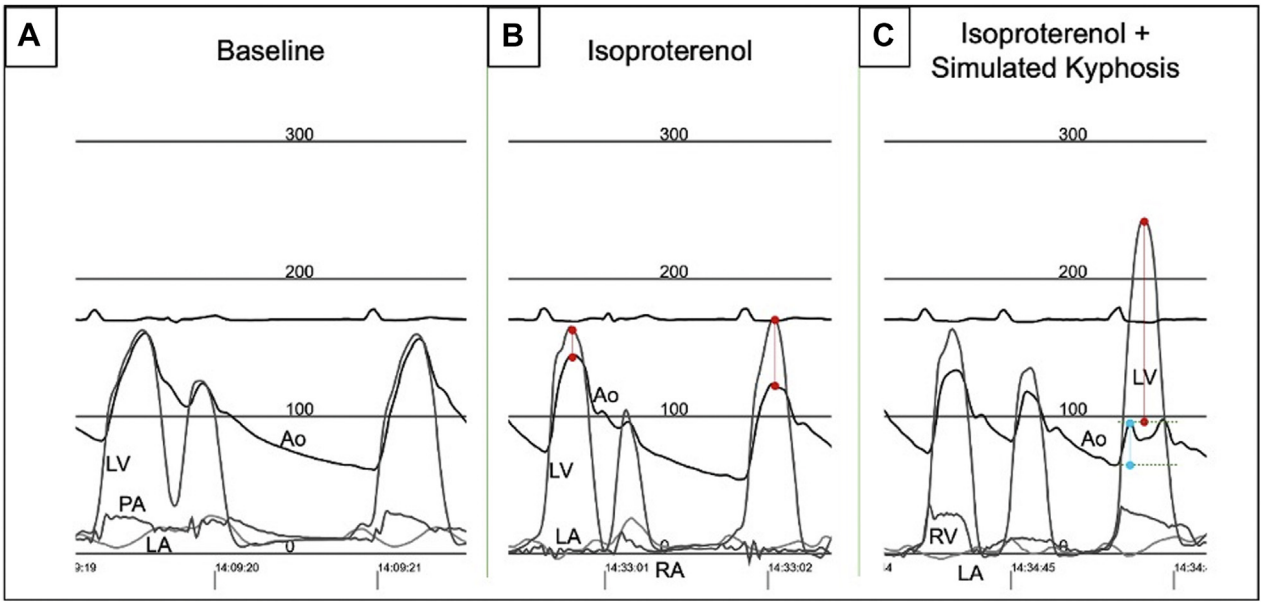


Resting transthoracic echocardiogram showing sigmoid septal hypertrophy **(red arrow)**.

hypotension, shortness of breath, and syncope/presyncope. Extracardiac contributors to DLVOTO are uncommonly recognized. Previous examples of severe kyphosis associated with DLVOTO[2] and LVOT stenosis[3] have been reported, but both patients had marked kyphosis, with extreme distortion of the thoracic cavity and evidence of LVOTO at rest.

LVOTO can be classified as fixed versus dynamic. Fixed obstruction includes structural narrowing that is present throughout the cardiac cycle, whereas DLVOTO depends on loading conditions. In DLVOTO (ie, hypertrophic cardiomyopathy), there is no fixed obstruction; however, in systole and owing to septal thickening, turbulent blood movement in combination with elongated anterior mitral leaflet can result in systolic anterior motion.[4] Both drag effects on the anterior leaflet and the Venturi effect play important roles in this phenomenon.[1] Many factors may exacerbate this state, including dehydration, catecholamine excess, and basal hyperkinesis.[4] Kyphosis could potentially limit left ventricular filling/preload, which is an important determinant of the severity of systolic anterior motion because they are inversely related. Furthermore, the angulation between the aorta and the left ventricle may be increased by worsening kyphosis, with the associated upward pressure of the abdomen on the thoracic cavity leading to a more horizontal heart axis. Our patient likely had a triad of kyphosis, sigmoid septum, and catecholamine surge. Prolonged standing with

FIGURE 3 **Cardiac Catheterization With Drug and Physiologic Maneuvers to Assess for Dynamic Left Ventricular Outflow Tract Obstruction**



Heart catheterization showing pressures at baseline **(A)**, with isoproterenol **(B)**, and with isoproterenol + thoracic kyphosis **(C)**. **Red line** = peak-to-peak gradient (PTPG). **(B)** PTPG of approximately 30 mm Hg that increased to 50 mm Hg after premature ventricular contractions. **(C)** PTPG of >100 mm Hg with evidence of Brockenbrough Braunwald response. **Blue line** = increased intraventricular gradient with decreased pulse pressure.

kyphotic posture[5] and perhaps vertebral fractures could lead to pain and catecholamine release. Kyphosis decreases intrathoracic space, and it negatively affects the ribs' mobility and lung function,[6] which seems to affect women more than men.[6] This suggests that a reduction in the thoracic cavity in the presence of sigmoid septum and certain triggers (ie, catecholamines) were enough to cause DLVOTO. In our case, the degree of kyphosis and the absence of LVOTO on resting TTE were interesting findings, and different from those by Choi et al.[2] Perhaps the severe kyphosis in their case was enough to cause LVOTO, whereas in our case the kyphosis was not of a similar severity; thus, the induction of kyphosis and catecholamine presence were important to trigger the LVOTO.

Interestingly, the severe kyphosis may have also contributed to her RUQ pain, which improved when she was lying supine. In kyphosis, the reduction in thoracic cavity volume can potentially lead to increased venous congestion and hepatic capsule stretching, resulting in pain or discomfort. This was supported by the evidence of dilated intrahepatic IVC on computed tomography. TTE is performed while patients are in the supine position. In supination, thoracic kyphosis decreases[7] as the spine and the back passively relax[8], and this is expected to allow the thoracic cavity to expand. In view of the temporal relationship between kyphosis and the development of our patient's symptoms, normal liver profile, and evidence of intrahepatic IVC dilation with normal right-sided pressures, this vascular phenomenon could potentially explain her RUQ pain.

Conditions that could trigger DLVOTO include reduction of preload, afterload, or diastolic time. Understanding these factors helps in the symptomatic management of DLVOTO. The improvement our patient experienced with β-blockers supports the theory that her presyncope symptoms were related to LVOTO.

FOLLOW-UP

Over the next 3 weeks, metoprolol was up-titrated to 50 mg, and the patient reported fewer symptoms of lightheadedness.

CONCLUSIONS

This is an interesting case of DLVOTO secondary to severe kyphosis. Kyphosis developed secondary to osteoporotic fractures, and soon thereafter presyncope/syncopal episodes started. Although resting TTE could not explain these symptoms, cardiac catheterization revealed DLVOTO triggered by the presence of kyphotic posture. Identification of DLVOTO can be challenging in the absence of classic findings; thus, it is important to understand what triggers patients may have so as to reproduce them when testing for DLVOTO.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS dynamic, kyphosis, left ventricular outflow tract, obstruction

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