

Role of Ultrasound in Evaluating chest pain and dyspnea on exertion

Ultrasound Scholarly Concentration
Case Report
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I. Case

II. Clinical Question

III. Diagnosis

IV. Key Points



63 year-old female with past medical history of hypertension presents to the Emergency Department, complaining of chest pain and dyspnea on exertion.

- Her chest pain has been persistent for the last week; her primary care provider found she has elevated pro-BNP to 2,260 and sinus bradycardia to 54 beats per minute.
 - She was started on Lasix 40mg due to the pro-BNP and had been taking it for 5 days at the time of presentation.
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ROS: simultaneous nausea, vomiting and diarrhea for the last 3 years that have worsened over the last few months, and more acutely over the last 2 weeks. Denies headache, fevers, chills, cough, extremity swelling.

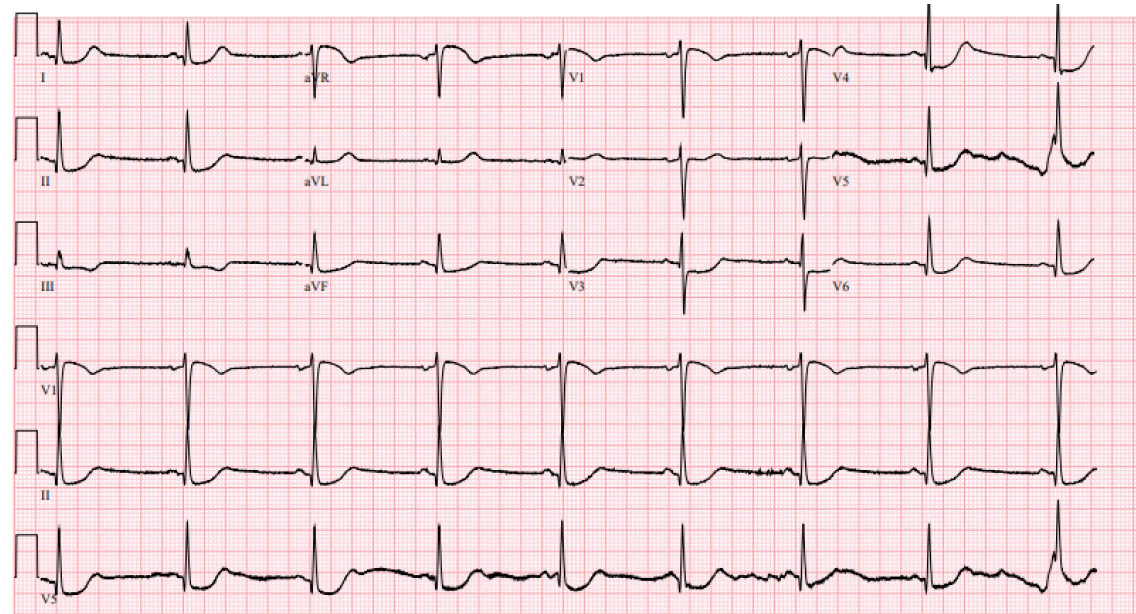
Family history: two maternal aunts passed away suddenly in their sleep – suspected to be due to heart attacks but never confirmed.

Objective Data

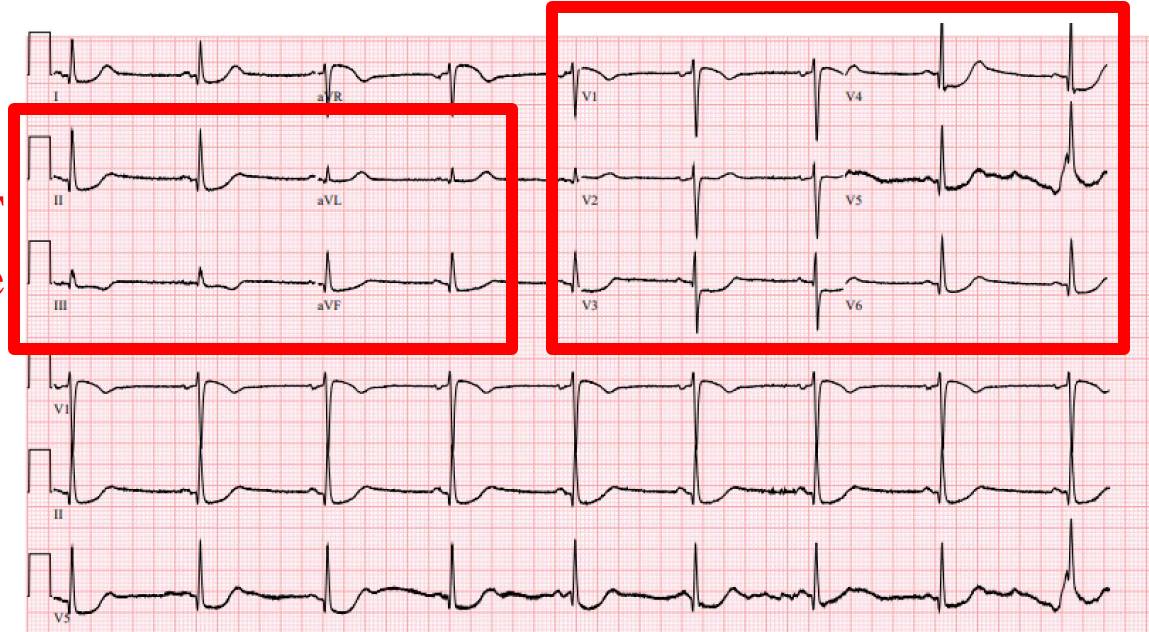


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- VS: Temp = 36.3 °C, **HR 51BPM**, **BP 102/73**, RR=24 breaths/min, SpO₂=94% on room air
- Labs: **D-dimer = 857**, **Cr = 1.90** (baseline 1.1), **Troponin = 85**, **Hb = 16.5**, **pro-BNP = 13,256**. Remaining labs WNL.
- Initial EKG showed no signs of ischemia. Then, 5 hours later, this appears on EKG →



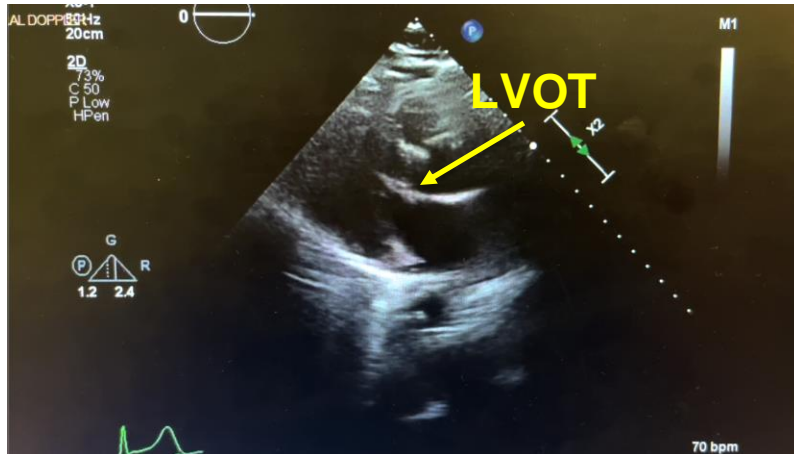
Sinus bradycardia with marked ST abnormality, indicative of possible anterolateral injury





- While awaiting further lab results, she has an episode of **sudden onset shortness of breath, diaphoresis and chest pain** that are accompanied by **hypotension (systolic blood pressure 40 mmHg)** and **bradycardia to the 40s**.
- She is started on a norepinephrine drip and IV fluids.
- What's on your differential?

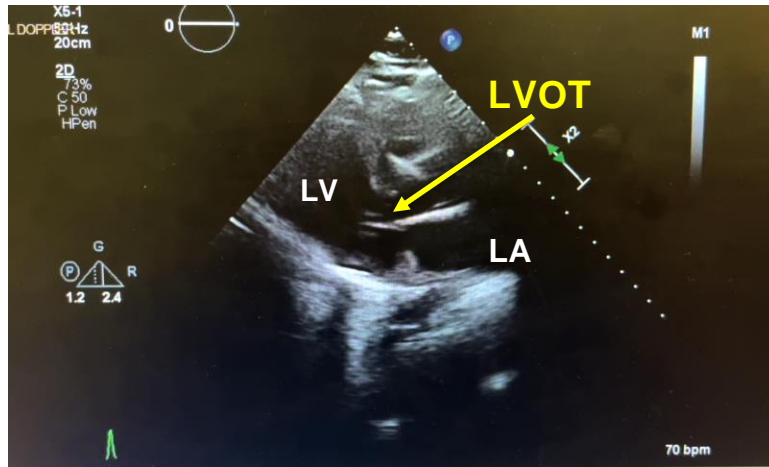
Think broadly: pulmonary embolism, pneumothorax, myocardial infarction, pericardial effusion, cardiac tamponade, just to name a few



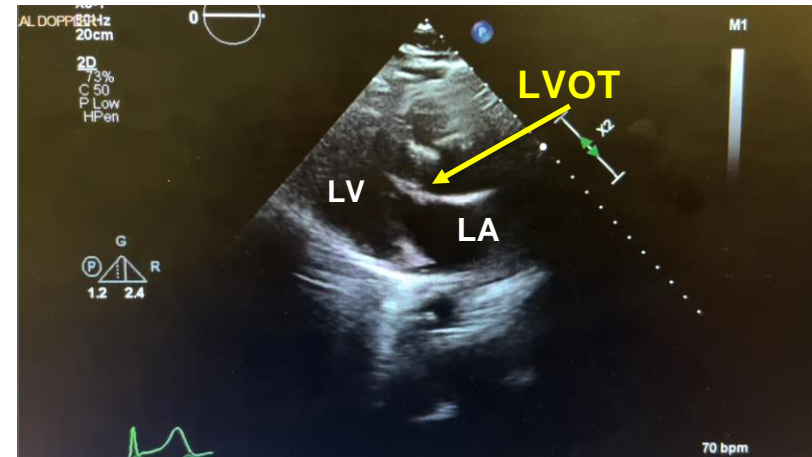
Summary:

1. The left ventricle is normal in size with mildly to moderately increased wall thickness.
2. The left ventricular systolic function is hyperdynamic, LVEF is visually estimated at $>70\%$.
3. Systolic anterior motion of the mitral valve with dynamic LVOT obstruction.
4. There is moderate to severe late peaking mitral valve regurgitation.
5. The right ventricle is normal in size, with normal systolic function.

See next slide for annotated image



Outflow tract **open**



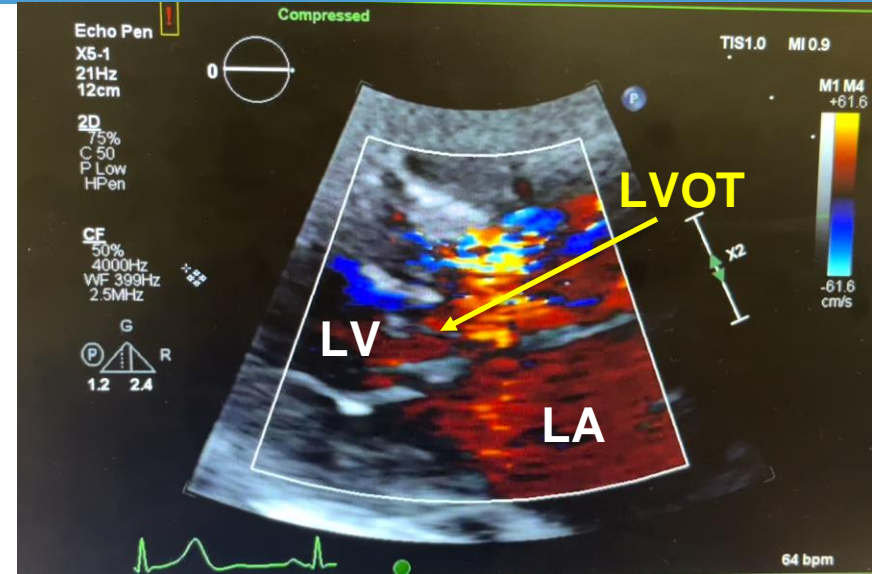
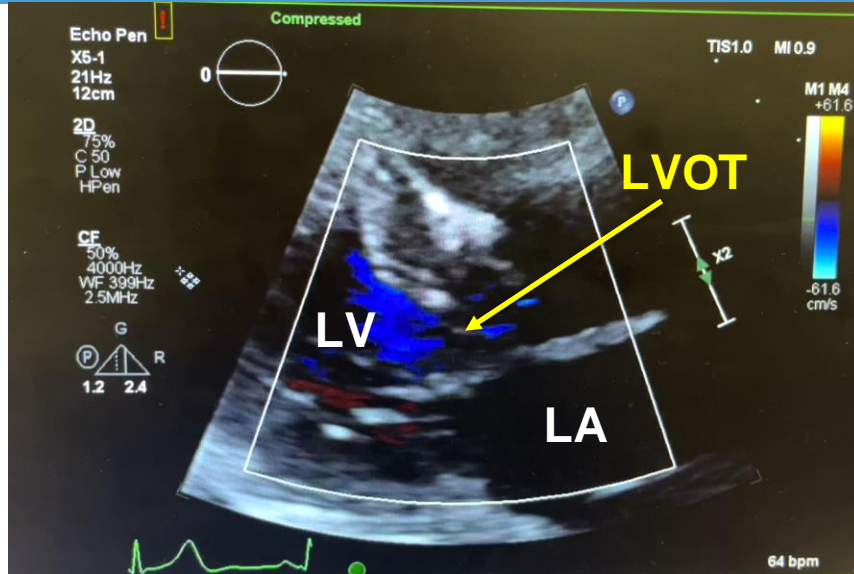
Outflow tract **obstructed**

LA = left atrium, LV = left ventricle, LVOT = left ventricular outflow tract

Echo, with Doppler



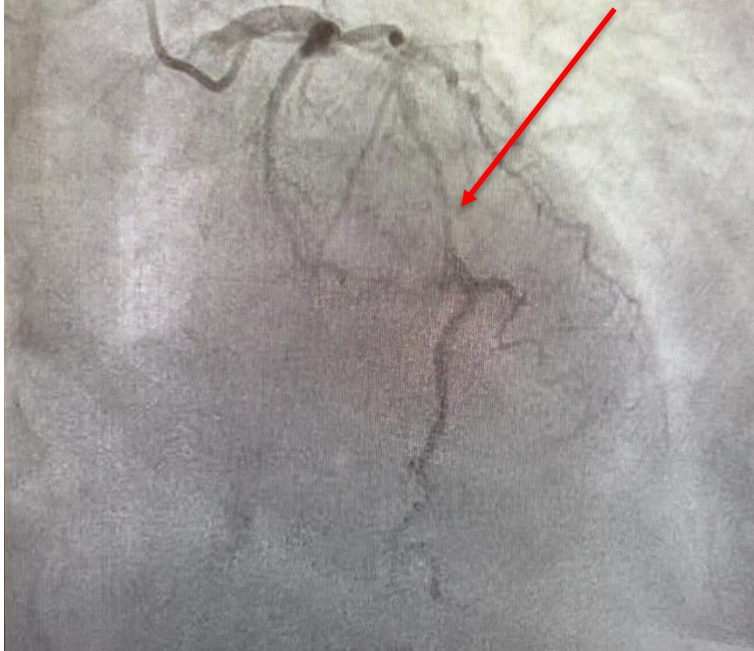
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Color represents mean velocity of flow across the opening.
Blue is moving away from the probe, yellow is towards the probe.

The figure on the right shows how turbulent the flow is as well as regurgitation into the LA.

Note compression of vessel



Left Heart Cath:

- Mean LVOT 30mmHg at rest
- Peak LVOT gradient 50mmHg at rest

The important finding here is the Peak LVOT gradient $>30\text{mmHg}$ at rest. If it is $<50\text{mmHg}$ at rest, you can do provocative maneuvers like Valsalva or exercise during a treadmill stress Echo to see if the patient develops a gradient. If the gradient is $>50\text{mmHg}$ either at rest or on exertion and symptomatic, they are clinically at the threshold for septal reduction therapy.

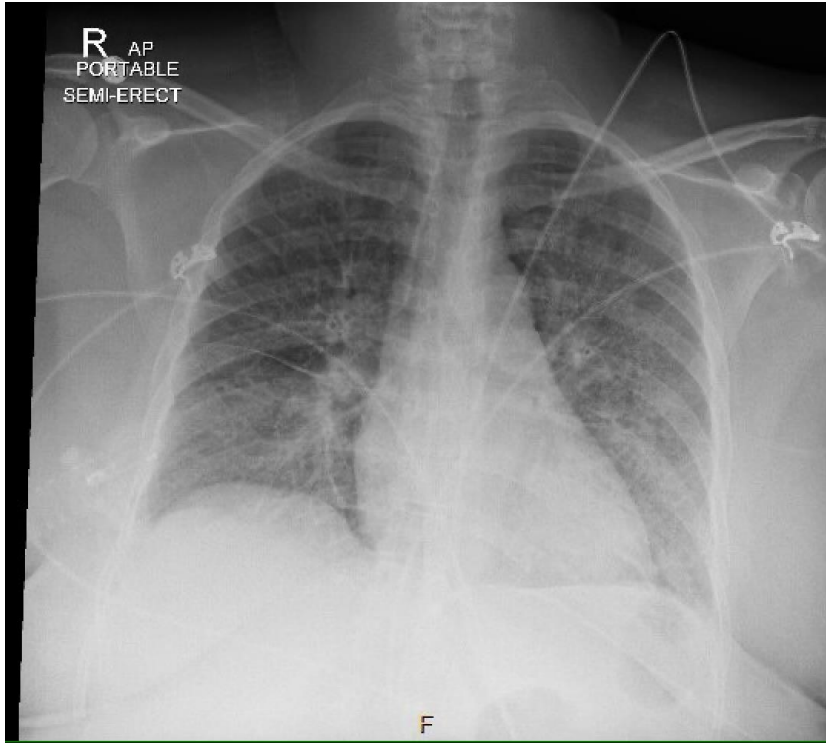
Based on her imaging, labs and symptoms, what was she suffering from?

**Hypertrophic obstructive cardiomyopathy
or HOCM**

Complication #1



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Suddenly, after returning from her left heart catheterization, she said:

"Wait, I'm having a really hard time breathing."

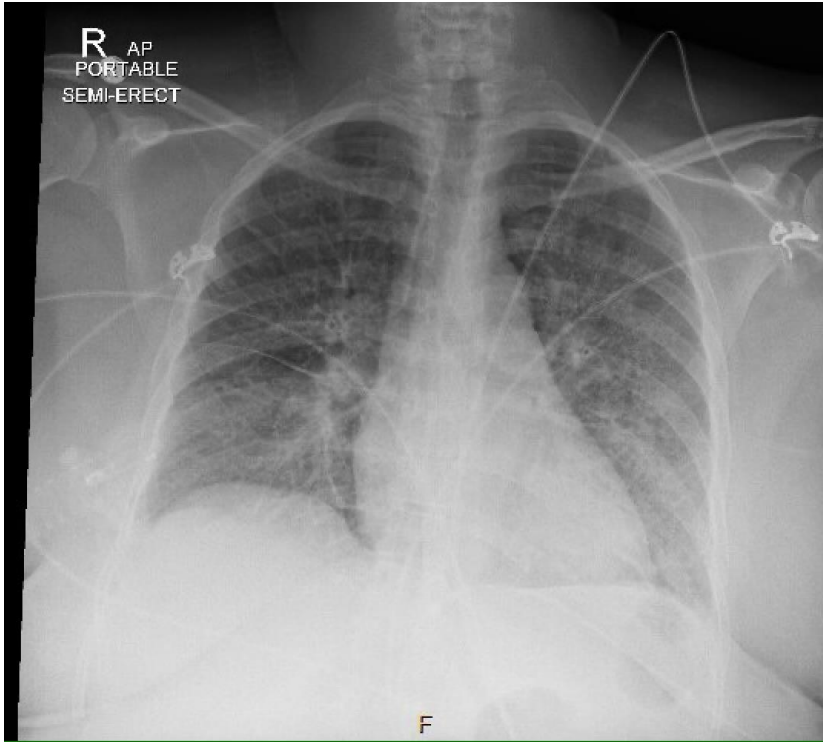
She was acutely dyspneic and hypoxic. To the left is her chest X-ray.

What do you think happened?

Chest X-ray findings



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Chest X-ray shows:

Interval development of diffuse bilateral hazy opacities. Perihilar peribronchial cuffing. Small right pleural effusion.

How did we end up with these x-ray findings?



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The patient had been receiving vasopressors to help counteract her hypotensive episodes.

Unfortunately, that meant we increased her afterload and thereby worsened her mitral regurgitation (MR).

This resulted in pulmonary edema that required diuretics to remove the back up of fluid.

Her hospital course was complicated by this delicate fluid balance.

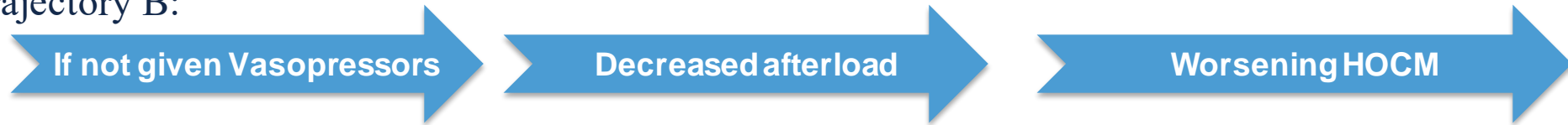


We struggled to find the balance between pressor support and volume status:

Trajectory A:



Trajectory B:

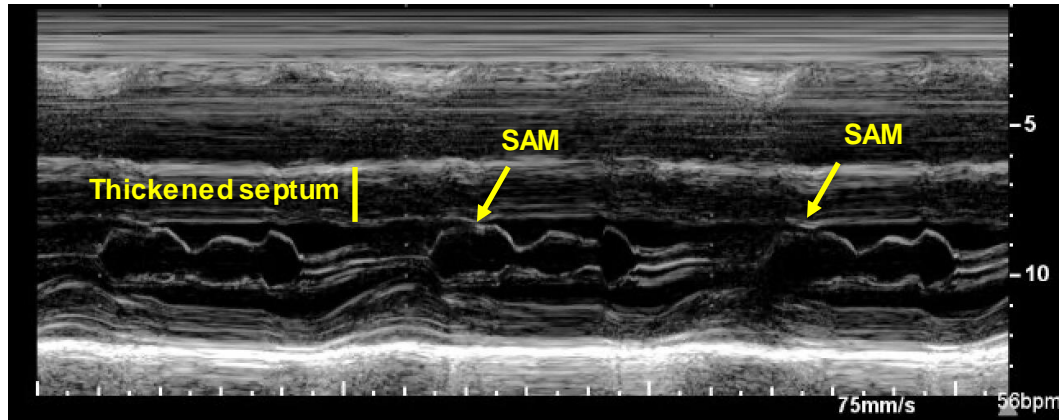


What's so interesting about her presentation?



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- Late diagnosis of persistently symptomatic HOCM with paroxysmal EKG changes concerning for acute coronary syndrome.
 - Transient T-wave inversions seen on EKG were due to her thickened myocardium undergoing periodic ischemia due to physiologic obstruction of blood flow.
 - It was notable that her severe presentation required intensive-level care.
 - Given her long history of gastrointestinal symptoms, she was likely chronically borderline hypovolemic and the recent addition of a diuretic and decreased oral intake compounded their effects and caused her hospitalization.
 - The fine line between hyper- and hypovolemia was difficult to navigate, prolonging her hospitalization.
 - We theorize that if she had not begun taking a diuretic, she likely would have continued to live independently in the community, with transient hypotensive episodes without a clear diagnosis.
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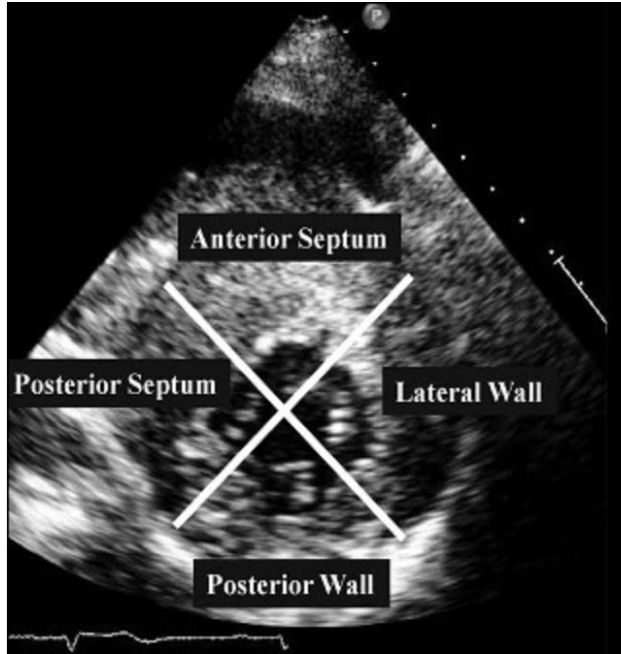


M-mode imaging of case patient

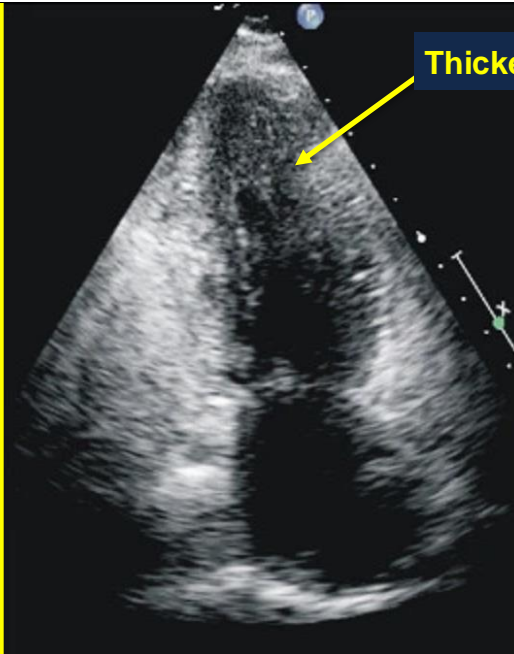
M-mode imaging can show diagnostic criteria important for identifying hypertrophic cardiomyopathy:

- asymmetrical septal hypertrophy
- systolic anterior motion of the mitral valve (SAM)
- a small LV cavity
- septal immobility,
- premature closure of the aortic valve

POCUS views of Hypertrophic Cardiomyopathy



Short axis view



Apical view

LV hypertrophy cut-off
suggestive of HCM in
adult population: **15 mm**

*Note: the patient under
discussion had LV
thickness of 18mm*




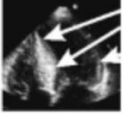
Using different POCUS views, various scores can be calculated to assess severity of hypertrophy.

For example, Wigle's score considers thickness >15mm and the extent of hypertrophy extending to the apex.

In contrast, Spirito's score calculates the magnitude of hypertrophy based on LV segmental involvement.

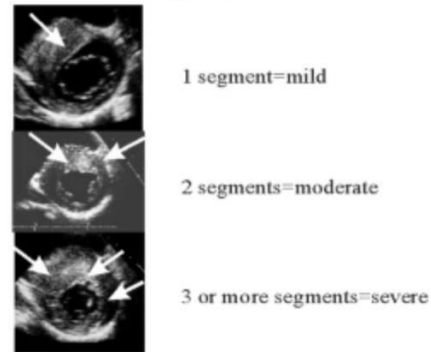
Wigle's score

Thickness of Basal Septum	Points
 15-19	1
20-24	2
25-29	3
≥30	4

LV hypertrophy extension to	
 Mid septum	2
Apical septum	2
Lateral wall	2

Maximal points score 10

Spirito's score (LV hypertrophy extension)



While we did not calculate this patient's Wigle or Spirito's score, these calculators would be helpful to measure the extent of her LV hypertrophy.

So, what happened to our patient?



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- She was titrated on beta-blockers and calcium channel blockers while inpatient
 - In the outpatient setting, she was initiated on Mavacamten (reversible cardiac myosin ATPase inhibitor)
 - She continues to follow with outpatient cardiology
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- She could have been diagnosed sooner if POCUS was considered at her initial presentation, reducing her symptom burden
 - Bedside-POCUS is a key tool in the diagnosis of HOCM, even if you do not have access to a formal echocardiogram
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