Dynamic LVOT obstruction

Sherif M Helmy, MD, FASE
Sr Consultant of Cardiology
Heart Hospital, HMC, Qatar
“Speaker disclosure - I do not have an affiliation (financial or otherwise) with a pharmaceutical, medical device, or communication and event planning company.”
Dynamic LV Outflow Tract Obstruction

Outline

• Introduction
• DLVOTO in HCM
• Causes of DLVOTO other than HCM
• Case studies
• Conclusions
Dynamic LV Outflow Tract Obstruction

Definition

Obstruction to the LVOT by structural abnormalities which can be provoked or altered by a change in the physiological conditions.
• Dynamic LVOT obstruction has been associated with HCM.
• Recently it was noted that it also occurs in numerous diseases and may be found absence of a noticeable cardiac disease (<1% cases).
• It is usually transient and accompany certain clinical situations.
• It is one of the more common causes of unexplained hypotension.
• The diagnosis of LVOTO is important to eliminate the factors that can potentially intensify the obstruction.
Hallmarks of Dynamic LV Outflow Tract Obstruction

• Obstruction occurs in mid to late systole > Dagger shaped Doppler signal
• Associated with SAM
• Posteriorly directed MR jet
• Mid systolic closure of the aortic cusps
• Provoked by or altered by loading conditions
Hallmarks of dynamic LVOT obstruction

Fixed obstruction

Dynamic obstruction
Hallmarks of Dynamic LV Outflow Tract Obstruction

SAM

Mid systolic notch of the aortic cusps
Cardiomyopathy: Definition

- “A myocardial disorder in which the heart muscle is structurally and functionally abnormal, in the absence of coronary artery disease, hypertension, valvular disease and congenital heart disease sufficient to cause the observed myocardial abnormality.”

ESC Working Group on Myocardial Pericardial Diseases (Elliott P et al. EHJ 2007)
Increased left ventricular wall thickness not solely explained by abnormal loading conditions

**ADULTS:**
- LV wall thickness ≥15 mm in one or more LV myocardial segments measured by any imaging technique

**CHILDREN:**
- LV wall thickness more than two standard deviations above the predicted mean (z-score >2)

Case 1

HOCM

Sherif M Helmy, MD, FASE, ICU, Qatar 2019
Differential diagnosis of unexplained LV hypertrophy

Familial LVH
- Sarcomeric HCM
- Lysosomal Storage Disease
  - Anderson-Fabry disease
  - Gaucher disease
  - Hurler disease
- Mitochondrial Myopathies

Syndromic HCM
- Noonan syndrome
- LEOPARD syndrome
- Friedreich ataxia

Acquired LVH
- Athletes’ Heart
- Arterial Hypertension
- Infiltrative Disorders
  - Amyloidosis
  - Sarcoidosis
- Aortic Valve Disease

ASE’s comprehensive Echocardiography, 2015
Multimodality approach for the diagnosis of unexplained LVH

ASE’s comprehensive Echocardiography, 2015
# Clinical History: Differential Diagnosis of LVH

## Clinical History

<table>
<thead>
<tr>
<th>Hypertension</th>
<th>Hypertensive heart disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple myeloma or chronic inflammatory disease</td>
<td>Cardiac amyloidosis</td>
</tr>
<tr>
<td>Neurological problems related to ataxia</td>
<td>Freidreich’s cardiomyopathy</td>
</tr>
<tr>
<td>Multiple organ involvements (kidneys or nervous system)</td>
<td>Fabry’s disease</td>
</tr>
</tbody>
</table>

Weidman et al, JASE, 2010;23, 791-801
Interpret images in context of clinical features and other tests:
A: HCM
B: Normal
C: Amyloidosis
D: Fabry’s
E: Non compaction
Physiological variants of HCM

- Latent obstruction (40%)
- Non-obstructive (30%)
- Resting obstructive (30%)

70% of the HCM population demonstrate obstructive physiology
Case 2
Mild septal hypertrophy with SAM......419689
Dynamic LVOTO in HCM develops due to

- **structural defects:**
  Narrowing by septal hypertrophy;

  **THAT IS NOT THE WHOLE STORY**

- Hydrodynamic forces causing SAM
Dynamic LVOTO in HCM develops due to

• Multiple structural defects:
  a) Narrowing by septal hypertrophy;
  b) Mitral leaflets abnormalities;
  c) anterior displacement of the MV apparatus;
  d) anterior malposition of the papillary muscles.

• Hydrodynamic forces causing SAM
Diverse geometric changes related to DLVOTO

• In hypertrophic cardiomyopathy patients without significant LV hypertrophy, in addition to basal septal thickness, anterior MV length, abnormal chordal attachment, and bifid PM mobility are associated with LVOT obstruction. In such patients, additional procedures on MV and PM (±myectomy) could be considered.

Patal et al., Circ Cardiovasc Imaging. 2015;8:e003132. DOI: 10.1161/CIRCIMAGING.115.003132.)
Abnormal Papillary Muscles and Dynamic LVOT Obstruction

Desai MY et al. Heart August 2007
Case 3:
Accessory mitral valve tissue (AMVT)
Differential diagnosis of SAM

- Hypercontractile states (post operative inotropes)
- Post MV repair
- Anomalous papillary muscle insertion
- Anteroapical MI
- Elderly with sigmoid septum and hyperdynamic LV function
- Takotsubo cardiomyopathy
Other than HOCM causes of DLVOTO

• Complicating acute MI
• Catecholamine excess
• Exercise overload
• Anorexia
• Dehydration
• Concentric LV hypertrophy
• After AVR of AS
• After MV repair
• Tako – tsubo (approx. 20%)
• Amyloidosis
• Anaemia
• After anaesthesia for non-cardiac surgery
CASE 4a

Acute MI
Dynamic LVOTO as a complication of acute MI

- Numerous reports have highlighted the occurrence of transient dynamic LVOTO as a complication of AMI.
- Incidence is unclear (probably under diagnosed).
- 20 cases have been reported to 2015 (7 LAD, 7 no LAD, 6 no CAG).
- Mimic cardiogenic shock in an acute care setting.
- Potentially reversible complication.
- Cases experiencing myocardial rupture and death were reported.
Pathogenesis of dynamic LVOTO as a complication of acute MI

Chockalingam et al LV Outflow Obstruction in MI, Circulation 2007
Management algorithm for LVOTO in acute MI

Chockalingam et al LV Outflow Obstruction in MI, Circulation 2007
<table>
<thead>
<tr>
<th>LVOTO Precipitating Factors</th>
<th>Suggested Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal Hyper contracatility</td>
<td>- B blockers;</td>
</tr>
<tr>
<td></td>
<td>- nondihydropyridine calcium blockers</td>
</tr>
<tr>
<td>Apical dysfunction</td>
<td>Urgent revascularization: PCI, thrombolysis</td>
</tr>
<tr>
<td>Hypotension</td>
<td>IV fluids phenyepherine</td>
</tr>
<tr>
<td>SAM</td>
<td><strong>Avoid/discontinue</strong></td>
</tr>
<tr>
<td></td>
<td>Inotropes</td>
</tr>
<tr>
<td></td>
<td>IABP</td>
</tr>
<tr>
<td></td>
<td>Nitrates</td>
</tr>
<tr>
<td></td>
<td>Diuretics</td>
</tr>
</tbody>
</table>

Chockalingam et al LV Outflow Obstruction in MI, Circulation 2007
Dynamic LVOTO as a complication of acute MI

• Clinical suspicion, early recognition, and appropriate management of LVOTO, along with the independent addressing of STEMI, would significantly improve the outcome in this critically ill patient subset.

• Withdrawing inotropes and initiating intravenous b-blockers usually improves hypotension and reduced the LVOTO gradient.
Case 5

**Takotsubo**

- Takotsubo cardiomyopathy is an increasingly recognized clinical syndrome.
- Dynamic LVOT obstruction occurs in ~ 20% of those cases.
- The typical patient is an elderly, hypertensive female with sigmoid deformity of the intraventricular septum.
Case 5

**Takotsubo**

- Dynamic LVOT obstruction should be considered in patients presenting with persistent hypotension or shock.
- Diagnosis of dynamic LVOT obstruction is essential to prevent institution of potentially detrimental therapies.
- Treatment consists of fluid resuscitation and beta blockers.
- Prognosis is good and does not seem to differ from takotsubo patients without LVOT obstruction.
Our patient had a complex presentation with echocardiogram revealing extensive anteroapical akinesis, SAM and MR. The sequence of our patient's presentation suggests that the apical ballooning caused geometric alterations in her LV that in turn led to acute and severe MR, SAM and LVOT obstruction. Despite maximal medical therapy including IABP placement, the SAM persisted and probably prevented the usual recovery seen in this syndrome.
Case 6

DSE
# Dynamic LVOTO during stress echocardiography

<table>
<thead>
<tr>
<th>Definition</th>
<th>Systolic velocity &gt; 2.5 m/sec (25 mm Hg) across the LVOT with stress</th>
</tr>
</thead>
</table>
| Incidence  | Dobutamine: up to 21%  
Exercise : up to 13% |
| Significance | May explain exertional dyspnea and angina in absence of ischemia and with normal coronary angiography.  
Not related to hypotension or CAD. |
| Patient characteristics | Elderly, females, hypertensive, diastolic dysfunction, small LV, RWT, LVOT<19mm/m2 |
| References  | Pellika et al., 1992  
Cabrera Bueno et al., 2004  
Park SM et al., 2015 |
Mechanisms of left ventricular outflow tract obstruction during hypovolemia

Case 7

**MV repair 13082**

- 63 y lady, DM type2, Hypertension, mild CAD, ESRD on regular hemodialysis.
- Shortness of breath and fatigue
Summary

• LVOT obstruction is a dynamic phenomenon
• Its occurrence requires the coexistence of predisposing anatomic factors and a physiological condition that induces it.
• Diagnosis of LVOT obstruction should entail immediate implementation of the therapy to eliminate the factors that can potentially intensify the obstruction.
• Echocardiography is the basic modality in its diagnosis and treatment.
Thank You