Revascularization Strategy for STEMI with Multivessel Coronary Artery Disease

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Interventional Cardiology Fellow
Minneapolis Heart Institute
Disclosure

• None

Objective

• Background for MV CAD and STEMI.

• Strategies for treatment of STEMI with MV CAD.

• Overview of the available data and the guidelines.
**Background**

- 40-50% of patients with STEMI have significant lesions in non-IRA.

- MV CAD associated with higher mortality and rate of reinfarction.

- This could be related to multiple vulnerable plaques in non-IRA.

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**Multivessel plaque associated with worse prognosis**

253 Patients with STEMI, 40% had MV CAD

Treatment Strategies for STEMI with MV CAD

• Culprit vessel Intervention only (CVI).
  - Only if the patient has ischemic symptoms or high-risk non-invasive testing.

• CVI with staged PCI of non-culprit vessel.
  - During the index hospitalization or after few weeks from discharge.

• Complete revascularization during index procedure.

Supportive evidence

• Conflicting data:
  - Depend only on observational studies, and meta-analysis
  - Different inclusion criteria
  - End points
  - Small sample size
  - Time of intervention
  - Selection bias
2013 STEMI Guideline

- Long procedure time
- More contrast
- Stent thrombosis
- Procedure complications

O’Gara PT et al. J Am Coll Cardiol 2013

PRAMI Trial

**Preventive:** PCI to non-culprit artery (N 234)

**Non-Preventive:** PCI to culprit artery only (N 231)

**Primary end point:** CV death, MI, & refractory angina

**Result:** 9% in preventive group, 23% in non-preventive. Relative RR 65% & absolute RR 14%

CvLPRIT Trial

Complete revascularization group at index hospitalization (N 150)
Infarct related artery only revascularization group (N 146)

**Primary endpoint**: All cause mortality, recurrent MI, HF, and ischemia driven revascularization

**Results**: 53% reduction in composite endpoint (10 vs 21.2%). No safety differences seen between treatment groups

Which non-IRA we should treat?

- Angiographic severity.
- Vulnerability of the lesion.
- Complexity of the lesion.
- Safety and feasibility of the intervention.
- Coronary physiology.
DANAMI-3-PRIMULTI Trial

- Complete FFR guided PCI to non-culprit artery (N 314)
- PCI to culprit artery only (N 313)

- **Primary endpoint:** All cause mortality, recurrent MI, ischemia driven revascularization
- **Results:** 44% reduction in primary composite endpoints (13% vs 22%)
- MV PCI guided by FFR significantly reduced number of revascularization


Updated STEMI Guideline 2015

- III → IIb
- Ok to treat non-IRA
- Hemodynamic stable
- Either at the index procedure or staged.
FFR in acute MI

- FFR may be inaccurate after acute MI
  - Microvascular spasm.
  - Microvascular flow limitation secondary of edema.

COMPARE-ACUTE

- Complete FFR guided PCI to non-culprit artery (N 295)
- PCI to culprit artery only and FFR to non-culprit artery (N 590)
  - Primary endpoint: MACE (Death, MI, CVS, revascularization)
  - Results: Reduction in primary composite endpoints (7.8 % vs 20.5 %)
  - Safe to defer FFR negative lesion.
  - FLOWER-MI trial showed opposite result?

Smith P, et al. COMPARE-ACUTE Trial, NEJM, March 2017
COMPLET Trial

- NNT to prevent cardiovascular death or myocardial infarction is 37 patients.
- NNT to prevent cardiovascular death, myocardial infarction, or ischemia-driven revascularization is 13 patients.

Shamir M, et al. COMLET, NEJM, October 2019

Complete Versus Culprit-Only Revascularization in Patients Presenting With ST-Segment Elevation Myocardial Infarction

- 10 RCT
- 7114 patients (3426 complete revascularization, and 3688 culprit only revascularization).
- Complete revascularization significantly reduced the risk of MACE compared with culprit only (10.7% vs 20.1%), reinfarction (5.0% vs 6.9%), and revascularization (4.2% vs 12.7%).

Mohsin S, et al. CRM, December 2020
**BIOVASC Trial**

Immediate complete revascularization (N 764)  
Staged complete revascularization (N 761)

**Primary endpoint:** Death, MI, ischemia-driven revascularization, CVA

**Results:** Immediate complete revascularization was non-inferior to staged complete revascularization.

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**Primary Outcome**

Composite of all-cause mortality, myocardial infarction, any unplanned ischemia-driven revascularization and cerebrovascular events


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**Recommendations for Revascularization of the Non-Infarct Artery in Patients With STEMI**

Referenced studies that support the recommendations are summarized in online data supplement A.

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>1. In selected hemodynamically stable patients with STEMI and multivessel disease, after successful primary PCI, staged PCI of a significant non-infarct artery stenosis is recommended to reduce the risk of death or MI.</td>
</tr>
<tr>
<td>2a</td>
<td>C-EO</td>
<td>2. In selected patients with STEMI with complex multivessel non-infarct artery disease, after successful primary PCI, elective CABG is reasonable to reduce the risk of cardiac events.</td>
</tr>
<tr>
<td>2b</td>
<td>B-R</td>
<td>3. In selected hemodynamically stable patients with STEMI and low-complexity multivessel disease, PCI of a non-infarct artery stenosis may be considered at the time of primary PCI to reduce cardiac event rates.</td>
</tr>
<tr>
<td>3: Harm</td>
<td>B-R</td>
<td>4. In patients with STEMI complicated by cardiogenic shock, routine PCI of a non-infarct artery at the time of primary PCI should not be performed because of the higher risk of death or renal failure.</td>
</tr>
</tbody>
</table>
Conclusion

• MV CAD elevates mortality risk in STEMI

• Evolving in the supportive data, leading to change in the non-IRA strategy (Previous III and now Ia in staging & IIb in index procedure)

• We need more data (Imaging/Physiology, what lesion, and when)

Thank you
Revascularization in ST-Elevation MI and Cardiogenic Shock

Khalid Changal, MD
PGY7
Interventional Cardiology Fellow

Cardiogenic shock

• Clinical syndrome manifested by either a sudden or acute-on-chronic reduction in cardiac output, leading to systemic hypotension and end-organ hypoperfusion.
  • (1) frank or relative hypotension, defined by a systolic BP below 80 or 90 mm Hg or a reduction in mean arterial pressure (MAP) of 30 mm Hg;
  • (2) inadequate cardiac index, defined as less than 1.8 liters/min/m² without mechanical or pharmacologic support, or less than 2.2 liters/min/m² with support;
  • (3) elevated end-diastolic pressures on the right (>10 to 15 mm Hg) and/or left (>18 mm Hg) side of the heart;
  • and (4) evidence of end-organ hypoperfusion.
Early mortality rates have declined in major randomized trials of STEMI patients from 1986 to 2018 with the introduction and improvement in pharmacologic and/or mechanical reperfusion therapy

STEWI and Cardiogenic Shock Mortality

- Mortality remains high, i.e., more than 50%
- CS remains one of the most common causes of hospital mortality after AMI


Mortality by etiology of cardiogenic shock following acute myocardial infarction (AMI).

The high rate of sudden death or cardiac arrest occurs within the first month after MI


Pathophysiology of Cardiogenic Shock in STEMI

- Myocardial infarction
- Myocardial dysfunction
- Systolic dysfunction
- Diastolic dysfunction
- Pulmonary congestion
- Hypoxemia
- Ischemia
- Relief of ischemia
- Decompensation
- Progressive myocardial dysfunction
- Death
- Survival with good quality of life
- Revascularization

Systolic inflammatory response (IL-6, TNF-α, NO)
- Systemic inflammation
- Hypotension
- Hypoxemia
- Ischemia
- Death
- Systolic dysfunction
- Diastolic dysfunction
- Pulmonary congestion
- Hypoxemia
- Ischemia
- Relief of ischemia
- Decompensation
- Progressive myocardial dysfunction
- Death
- Survival with good quality of life
- Revascularization
## Hemodynamic Patterns for Common Clinical Conditions

<table>
<thead>
<tr>
<th>Cardiac Condition</th>
<th>Hemodynamic Parameter</th>
<th>RA</th>
<th>RV</th>
<th>PA</th>
<th>PAPI</th>
<th>PCW</th>
<th>CI</th>
<th>CPO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>0–6</td>
<td>25/0–6</td>
<td>25/0–12</td>
<td>&gt;1</td>
<td>6–12</td>
<td>≥2.5</td>
<td>&gt;0.6</td>
</tr>
<tr>
<td>AMI without LVF</td>
<td></td>
<td>0–6</td>
<td>25/0–6</td>
<td>30/12–18</td>
<td>&gt;1</td>
<td>≤18</td>
<td>≥2.5</td>
<td>&gt;0.6</td>
</tr>
<tr>
<td>AMI with LVF</td>
<td></td>
<td>0–6</td>
<td>30–40/0–6</td>
<td>30–40/18–25</td>
<td>&gt;1</td>
<td>&gt;18</td>
<td>May be &lt;2.0</td>
<td>&lt;0.6</td>
</tr>
<tr>
<td>Biventricular failure</td>
<td>&gt;6</td>
<td>50–60/6–12</td>
<td>50–60/25</td>
<td>May be &lt;1</td>
<td>18–25</td>
<td>May be &lt;2.0</td>
<td>&lt;0.6</td>
<td></td>
</tr>
<tr>
<td>RVMI</td>
<td></td>
<td>12–20</td>
<td>30/12–20</td>
<td>30/12</td>
<td>Often &lt;1</td>
<td>≤12</td>
<td>May be &lt;2.0</td>
<td>&lt;0.6</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
<td>12–16</td>
<td>25/12–16</td>
<td>25/12–16</td>
<td>Often &lt;1</td>
<td>12–16</td>
<td>&lt;2.0</td>
<td>&lt;0.6</td>
<td></td>
</tr>
<tr>
<td>Acute Pulmonary embolism</td>
<td>12–20</td>
<td>30–50/12–20</td>
<td>30–50/12</td>
<td>Often &lt;1</td>
<td>&lt;12</td>
<td>&lt;2.0</td>
<td>&lt;0.6</td>
<td></td>
</tr>
</tbody>
</table>

### Exclude Mechanical Complications

- Their treatment usually requires prompt invasive management with intervening mechanical support of the circulation.
- May Require Urgent Surgical Treatment
Survival

- Revascularization improves survival.
- Inotropes/vasopressors, MCS: useful temporizing maneuvers.

Major categories of nonsurgical mechanical circulatory support.

- IABP
- Impella
- VA-ECMO
- Tandem Heart
Mechanical Circulatory Support

- (1) maintain end-organ perfusion and prevent progressive shock,
- (2) reduce intracardiac filling pressures and congestion,
- (3) reduce LV volumes, wall stress, and myocardial oxygen consumption,
- (4) augment coronary perfusion,
- (5) allow time for recovery of stunned or hibernating myocardium, and
- (6) limit infarct size

SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock?)

- shock caused by LV failure complicating STEMI were randomly assigned to emergency revascularization ($n = 152$), accomplished by either CABG or angioplasty, or to initial medical stabilization ($n = 150$).
How Urgently Should Revascularization be Pursued in Cardiogenic Shock and STEMI?

FITT-STEMI trial

- \( n = 12,675 \) STEMI patients who used emergency medical service transportation and were treated with primary percutaneous coronary intervention (PCI)
- In CS patients with no OHCA, every 10-min treatment delay resulted in 3.31 additional deaths in 100 PCI-treated patients.
PCI after 12 hours

- There are no RCTs examining the benefit of PCI in patients with STEMI presenting >12 hours after symptom onset who have clinical evidence of ongoing ischemia, acute severe heart failure, or life-threatening arrhythmias.
- **Expert Consensus**: a strategy of delayed reperfusion in these unstable patient subsets expected to improve symptoms and outcomes, and for this reason PCI should be considered.

Fibrinolysis

*Immediate transfer to a PCI-capable hospital is recommended in patients with shock or acute severe HF regardless of the time delay.*
Non-Infarct artery in Cardiogenic Shock

- Multivessel disease affects 70% to 90% of patients with cardiogenic shock and acute MI
- The optimal extent of initial revascularization has undergone intense clinical investigation

CULPRIT SHOCK

- 706 patients with cardiogenic shock onset within 12 hours in the setting of acute MI
CULPRIT
SHOCK

DEATH FROM ANY CAUSE

<table>
<thead>
<tr>
<th>Patients Who Died From Any Cause (%)</th>
<th>Multivessel PCI</th>
<th>Culprit-lesion-only PCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>10</td>
<td>10</td>
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<tr>
<td>5</td>
<td>20</td>
<td>20</td>
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<td>10</td>
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<td>40</td>
<td>90</td>
<td>90</td>
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<tr>
<td>45</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

DAYS SINCE RANDOMIZATION

No. at Risk
Multivessel PCI  341  199  172  162  156  153  152
Culprit-lesion-only PCI  344  219  207  198  192  189  184

CULPRIT
SHOCK

RENAL-REPLACEMENT THERAPY

<table>
<thead>
<tr>
<th>Patients Who Underwent Renal-Replacement Therapy (%)</th>
<th>Multivessel PCI</th>
<th>Culprit-lesion-only PCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>5</td>
<td>5</td>
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<tr>
<td>5</td>
<td>10</td>
<td>10</td>
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<tr>
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<td>95</td>
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<td>95</td>
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Recommendations for Revascularization of the Non-Infarct Artery in Patients With STEMI

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</table>

FUTURE
National Cardiogenic Shock Initiative

- Single arm, 80 Centers recruiting
- Historical Cohort for comparison
- Non-Randomized Data

Upfront Use of Impella Prior to PCI
RESULTS

- A survival rate of 71% to hospital discharge in 406 patients who presented with acute myocardial infarction (AMI) complicated by cardiogenic shock

Door-To-Unload in STEMI Pilot Trial
No difference in Delayed vs. Immediate Reperfusion

- Major adverse cardiovascular and cerebrovascular event rates were not statistically different between the U-IR versus U-DR groups (8% versus 12%, respectively, \( P=0.99 \)).
- In comparison with the U-IR group, delaying reperfusion in the U-DR group did not affect 30-day mean infarct size measured as a percentage of LV mass (15±12% versus 13±11%, U-IR versus U-DR, \( P=0.53 \)).
THANK YOU!

Cerebral Embolic Protection during Transcatheter Aortic Valve Replacement

Konstantinos V. Voudris MD, PhD
Advanced Adult Structural and Congenital Heart Disease Interventions Fellow
Abbott Northwestern – Minneapolis Heart Institute
Stroke and TAVR

- TAVR use has significantly increased over the past 10 years
- Clinical stroke occurs in about 2.5% of cases and is usually peri-procedural

**Increased Mortality**

<table>
<thead>
<tr>
<th>In-Hospital Mortality</th>
<th>0%</th>
<th>5%</th>
<th>10%</th>
<th>15%</th>
<th>20%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any stroke</td>
<td>2.8%</td>
<td>2.8%</td>
<td>2.8%</td>
<td>2.8%</td>
<td>2.8%</td>
</tr>
<tr>
<td>No stroke</td>
<td>12.7%</td>
<td>12.7%</td>
<td>12.7%</td>
<td>12.7%</td>
<td>12.7%</td>
</tr>
</tbody>
</table>

P<0.001

<table>
<thead>
<tr>
<th>30-Day Mortality</th>
<th>0%</th>
<th>5%</th>
<th>10%</th>
<th>15%</th>
<th>20%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any stroke</td>
<td>3.7%</td>
<td>3.7%</td>
<td>3.7%</td>
<td>3.7%</td>
<td>3.7%</td>
</tr>
<tr>
<td>No stroke</td>
<td>16.7%</td>
<td>16.7%</td>
<td>16.7%</td>
<td>16.7%</td>
<td>16.7%</td>
</tr>
</tbody>
</table>

P<0.001

**Decreased Home Discharge**

<table>
<thead>
<tr>
<th>Location</th>
<th>0%</th>
<th>10%</th>
<th>20%</th>
<th>30%</th>
<th>40%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Home</td>
<td>54%</td>
<td>54%</td>
<td>54%</td>
<td>54%</td>
<td>54%</td>
</tr>
<tr>
<td>Extended care/rehab</td>
<td>36%</td>
<td>36%</td>
<td>36%</td>
<td>36%</td>
<td>36%</td>
</tr>
<tr>
<td>Nursing home</td>
<td>4%</td>
<td>4%</td>
<td>4%</td>
<td>4%</td>
<td>4%</td>
</tr>
</tbody>
</table>

Huded et al. JAMA. 2019;321(23):2306-2315

Mori et al. J Am Coll Cardiol. 2021 Nov;78(22):2161-72
Stroke and TAVR: Incidence

Huded et al. JAMA. 2019;321(23):2306-2315

30-day severe stroke (major and disabling stroke rate %)

High volume does not reduce stroke rate

Embolic CNS Injury – MRI

- Extremely frequent (70-93%)
- More frequent than SAVR
- Size of lesion significantly smaller
- Majority silent

Cerebral Embolic Protection - Sentinel

- 6-Fr radial access, deflectable catheter
- 2 independent filter baskets (140um pores)
- Protects 3 of 4 great vessels (90% circulation)
- Debris capture in >99% of TAVR cases
Sentinel IDE Trial

A. 30-day MACCE Rates

B. New Lesion Volume on MRI

Debris Capture

Type of Debris Captured

Finn. CRT 2021
Cerebral Embolic Protection - Use

PROTECTED TAVR trial – Study Design

- 3,000 patients across North America, Europe, and Australia undergoing TF-TAVR
- Randomized 1:1
- 1,501 were assigned to the cerebral embolic protection device group and 1,499 to the control group
- Primary end point - stroke within 72 hours after TAVR or before discharge (whichever comes first)
- Secondary end points - Disabling stroke, death, transient ischemic attack, delirium, major or minor vascular complications at the CEP access site, and acute kidney injury
- Neurologist examined all patients at baseline and after TAVR
PROTECTED TAVR trial – Baseline Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Control (N=1499)</th>
<th>CEP (N=1501)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>78.9±7.8</td>
<td>78.9±8.0</td>
</tr>
<tr>
<td>Female Sex</td>
<td>37.8%</td>
<td>42.0%</td>
</tr>
<tr>
<td>Society of Thoracic Surgeons score, %</td>
<td>3.4±2.8</td>
<td>3.3±2.7</td>
</tr>
<tr>
<td>STS score &lt;3%</td>
<td>58.2%</td>
<td>56.6%</td>
</tr>
<tr>
<td>Surgical Risk (per Heart Team)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extreme/High Risk</td>
<td>30.4%</td>
<td>30.4%</td>
</tr>
<tr>
<td>Intermediate Risk</td>
<td>34.2%</td>
<td>33.2%</td>
</tr>
<tr>
<td>Low risk</td>
<td>35.4%</td>
<td>36.3%</td>
</tr>
<tr>
<td>Native Valve Calcification Severity (site-reported)</td>
<td></td>
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</tr>
<tr>
<td>None/Mild</td>
<td>15.2%</td>
<td>16.2%</td>
</tr>
<tr>
<td>Moderate</td>
<td>29.5%</td>
<td>29.4%</td>
</tr>
<tr>
<td>Severe/Extreme</td>
<td>55.3%</td>
<td>54.4%</td>
</tr>
<tr>
<td>CHA₂DS²-VASC score</td>
<td>4.2±1.3</td>
<td>4.2±1.3</td>
</tr>
</tbody>
</table>

Operative risk was well-balanced

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PROTECTED TAVR trial – Results

Negative trial

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Control</th>
<th>CEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Stroke</td>
<td>2.9%</td>
<td>2.3%</td>
</tr>
<tr>
<td>Non-Disabling Stroke</td>
<td>1.3%</td>
<td>1.7%</td>
</tr>
<tr>
<td>Disabling Stroke</td>
<td>1.3%</td>
<td>0.5%</td>
</tr>
</tbody>
</table>

Primary Endpoint

Secondary Endpoint

Operative risk was well-balanced
PROTECTED TAVR trial – Safety

<table>
<thead>
<tr>
<th>Event at ≤72h / Discharge ITT population</th>
<th>Control (N=1499)</th>
<th>CEP (N=1501)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause Mortality</td>
<td>0.3% (4)</td>
<td>0.5% (8)</td>
</tr>
<tr>
<td>Cardiovascular Mortality</td>
<td>0.3% (4)</td>
<td>0.5% (8)</td>
</tr>
<tr>
<td>Safety composite</td>
<td>3.0% (45)</td>
<td>2.7% (41)</td>
</tr>
<tr>
<td>(all-cause mortality and stroke)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CEP Access Site-related Vascular Complication (Major or Minor)</td>
<td>N/A</td>
<td>0.1% (1)</td>
</tr>
<tr>
<td>Acute Kidney Injury (stage 2 or 3)</td>
<td>0.5% (7)</td>
<td>0.5% (8)</td>
</tr>
</tbody>
</table>

PROTECTED TAVR trial – Subgroup analysis

- Limited ability to predict who will benefit
Upcoming Devices

<table>
<thead>
<tr>
<th>Competitive Landscape (Feb 2022)</th>
<th>Sentinel</th>
<th>TriGuard</th>
<th>CAFTIS</th>
<th>Embolix</th>
<th>Embolinker</th>
<th>PointGuard</th>
<th>Profilimbo</th>
<th>Embrace</th>
<th>ArticLab</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral-embolic protection</td>
<td>Partial</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Mesh pore size</td>
<td>140</td>
<td>115-145</td>
<td>115-145</td>
<td>125</td>
<td>130</td>
<td>105</td>
<td>60</td>
<td>70</td>
<td></td>
</tr>
<tr>
<td>Full-body embolic protection</td>
<td>X</td>
<td>X</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Capture and removal</td>
<td>Partial</td>
<td>Partial</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Protects aortic surface</td>
<td>X</td>
<td>X</td>
<td>✓</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Stable anchoring</td>
<td>X</td>
<td>X</td>
<td>✓</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Access</td>
<td>6F LMA</td>
<td>5F LMA</td>
<td>6F LMA</td>
<td>6F LMA</td>
<td>6F LMA</td>
<td>6F LMA</td>
<td>6F LMA</td>
<td>6F LMA</td>
<td>6F LMA</td>
</tr>
<tr>
<td>Regulatory status</td>
<td>FDA + CE</td>
<td>CE</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>
Conclusion

- Clinical stroke occurs in about 2.5% of TAVR cases and is usually peri-procedural
- Associated with significant morbidity and mortality
- DW-MRI lesions frequent after TAVR (70-93%) – Majority silent
- Cerebral Embolic Protection with Sentinel
  - Safe
  - Does not reduce all strokes
  - ? Disabling strokes – more data needed (BHF PROTECT – TAVI)
- Multiple different devices coming

Thank you!
Penetrating Aortic Ulcers
Sarah Langdon, MD, Christopher Pedersen, MD
4/10/23

- No financial disclosures
Case

- 84M with PAU found during AVR/CABG

Background

- Atherosclerotic lesion with disruption of internal elastic lamina
- Can progress into intramural hematoma (IMH), dissection, pseudoaneurysm, rupture, or degenerate into aneurysms
- More common in the descending thoracic aorta
- Often seen with severe atherosclerotic disease
- Much less common than true IMH or dissection
Presentation and Diagnosis

- Asymptomatic or incidentally found on imaging
- Chest pain, back pain, abdominal pain – similar to aortic dissection
- Hypertension common

- CTA gold standard imaging modality
  - Outpouching of the aortic wall

Management

- Anti-impulse control with goal SBP <120, HR 60-80
  - Beta-blockade first line

- Symptomatic PAU or those associated with IMH should be repaired
- Asymptomatic PAUs with high-risk features should be considered for repair

- Historically treated with open aortic repair, but there is growing evidence for endovascular repair
  - Open treatment remains important for ascending aorta and aortic arch
  - Endovascular therapies for descending thoracic aorta
  - New technologies for disease near the arch
High Risk Imaging Features

- Maximum diameter >13-20mm
- Maximum depth >10mm
- Significant growth
- PAU associated with a saccular aneurysm
- PAU with increasing pleural effusion

Outcomes

- Asymptomatic
  - 6.5% developed symptoms, radiographic progression, or rupture over 10 years
- Open Repair
  - 9-19% perioperative mortality
- Meta-analysis of 310 TEVARs performed for PAU
  - 98.3% success rate, 30d mortality 4.8%, aortic related mortality of 4.1 at 18 months
Endovascular Repair

- Thoracic endovascular aortic repair (TEVAR)
- GORE® TAG® Thoracic Branch Endoprosthesis (TBE)

Back to Case

- Decided to perform TBE
• Intra-operative graft deployment

• Completion angiogram
Follow-up Imaging

Summary

- Uncommon aortic pathology
- Medical management is sufficient for a majority of asymptomatic patients however patients with symptoms or high risks features should be considered for repair
- TEVAR and Branched TEVAR devices provide less invasive options
References

- 2022 ACC/AHA Guidelines for the Diagnosis and Management of Aortic Disease

