A case of post myocardial infarction ventricular septal rupture

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Female, 81yo

Hx: diabetes mellitus, hypertension, chronic anaemia

presented to our hospital with acute - precordial chest pain starting 5h ago. She reported a 7-10 days history of (on and off) same pain

clinical examination revealed tachycardia at 103bpm, BP 110/55mmHg, respiratory rate of 22 breaths/min, S1, S2 heart sounds and a systolic murmur of 2/6
ECG showed ST elevation with Q waves in V2-V5 leads, T wave inversion in I and aVL leads and left axis deviation

**hs-Troponin T** = 1366 → 3793
Coronary angio
Primary PCI to LAD
Echo
Echo
3 days post discharge she appeared again with severe discomfort, weakness, tachycardia and hypotension. She also reported diarrhea.

- Lab: multiple-organ failure
- Chest auscultation: tachycardia, systolic-diastolic murmur
- Emergency echo revealed **ventricular septal rupture**
- ICU admission - IABP - intubation – infusion of inotropes
introduction

Ventricular rupture (VR): rare but fatal mechanical complication of AMI
2\textsuperscript{nd} leading cause of in-hospital death during AMI
Responsible for as many as 15\% of total early deaths in AMI patients
Occurring within the first two weeks after acute phase

**Types of rupture:**
LV free wall rupture, ventricular septal rupture and papillary muscle rupture
Ventricular double rupture (VDR): co-existence of 2 of the above

Becker RC et al, JACC 1996;27:1321-6
## Incidence

### Table 1  Ventricular septal rupture incidence, time to identification, and resultant mortality in four contemporary clinical series compared with historical data

<table>
<thead>
<tr>
<th>Source</th>
<th>MI treatment (overall cohort)</th>
<th>Incidence of VSR, % (n)</th>
<th>Time to VSR identification</th>
<th>Mortality, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Historical data</td>
<td>Pre-thrombolysis</td>
<td>1–2</td>
<td>3–5 days</td>
<td>With surgery: 45 With: 90</td>
</tr>
<tr>
<td>MIDAS(^2)</td>
<td>PCI (19%)</td>
<td>0.25–0.31 (408)</td>
<td>Not reported</td>
<td>In-hospital: 1990–92: 41 2005–07: 44 1-year: 1990–92: 60 2005–07: 56</td>
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<td>1990–2007</td>
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<tr>
<td></td>
<td>(n = 148,881) patients with STEMI</td>
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<tr>
<td>GUSTO-1(^3)</td>
<td>Thrombolysis (100%)</td>
<td>0.20 (84)</td>
<td>1 day</td>
<td>30-day: with VSR: 73.8 without VSR: 6.8</td>
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<tr>
<td>1990–1993</td>
<td></td>
<td></td>
<td>(range 0–47)</td>
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<tr>
<td></td>
<td>(n = 41,021) patients with STEMI</td>
<td></td>
<td>(94% ≤ 1 week)</td>
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<tr>
<td>GRACE(^4)</td>
<td>Primary PCI (15% overall; 38% for STEMI)</td>
<td>0.25 (155)</td>
<td>Not reported</td>
<td>In-hospital: 41</td>
</tr>
<tr>
<td>2000–2007</td>
<td>Lysis (12% overall; 28% for STEMI)</td>
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<tr>
<td></td>
<td>(n = 60,198) patients with ‘ACS’</td>
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<tr>
<td>APEX-AMI(^5)</td>
<td>Primary PCI (94%)</td>
<td>0.17 (10)</td>
<td>7.7 h (range 5.5–23.5)</td>
<td>30-day: 80</td>
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<td>2004–2006</td>
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<tr>
<td></td>
<td>(n = 57,45) patients with STEMI</td>
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</tbody>
</table>
Incidence

60198 pts enrolled from 2000 to 2007

273 pts (0.45%)

118 pts (0.2%)

155 pts (0.26%)

Free wall

Ventricular septal

STEMI 0.9%

NSTEMI 0.17%

UA 0.25%
mortality

Hospital mortality rate 58% in pts with VR vs. 4.5% in those without

80% mortality (free wall ventricular rupture)
41% mortality (ventricular septal rupture)

Eur Heart J 2010;31:1449-56
pathophysiology

- After full-thickness (transmural) infarction at any anatomic location. VSR: similar frequency in anterior and inferior/lateral infarctions.

- Newly formed communication → left to right shunting (from high-pressure LV to lower pressure RV).

- Clinical presentation: depending on the size: from hemodynamic stability to frank circulatory collapse.

- Subacute: coagulation necrosis of ischemic tissue with neutrophilic infiltration, causing thinning and weakening of the septal myocardium (3-5d).

- Acute: dissection of an intramural haematoma or haemorrhage into ischemic myocardium (physical shear stressor at the border of an infarct zone, combined with a hypercontractile remote myocardial segment (24h).
The impact of acute ischemia on tissue damage

The higher values the higher the damage and the extend of ischemia and so the risk of rupture

Absence of pre-existing collateral vessels contribute to extended tissue damage
Factors related to ventricular rupture

- Age (>70yo → up to 10%)
- Female sex
- Previous stroke
- ST segment elevation
- HR and BP at presentation
- Positive initial cardiac biomarkers
- Higher GRACE score
- Delay to reperfusion therapy

Eur Heart J 2010;31:1449-56
Initial management post VSR

1\textsuperscript{st} line: Haemodynamic stabilization and afterload reduction

(50\textendash70\% of pts have multivessel disease, \rightarrow hypotension, reduced coronary perfusion)

→ intra-aortic balloon counterpulsation (IABP)

→ PA catheter

→ mechanical circulatory support (Impella, TandemHeart, ECMO) as a bridge to definite repair
management

Eur Heart J 2014;35:2060-68
Transcatheter closure

A demanding procedure

- Fragile myocardial tissue
- Acute vs subacute closure (>2-3 weeks post-infarct)
  (acutely: some pts are in severe cardiogenic shock)
- The procedure itself has several limitations: delivery sheath might tear the borders of VSD, available device sizes are frequently not sufficient
- Healing of infarcted tissue may increase the size of VSD thus resulting to malposition of the device
Unfortunately our patient had numerous risk factors for post AMI VR

- advanced age
- Female sex
- delayed admission
- no ST segment resolution
- anterior infarction
- History of hypertension
- apical location of septal perforation

She died the next day
Thank you for your attention