Right ventricular infarction: Prevalence and outcome

N. Koutsogiannis.
Department of Cardiology
University Hospital Of Patras
Clinically significant RV infarction when proximal or mid occlusion of RCA and impaired flow in RV branches occurs.

**Angiographic Hallmarks of RV infarction**

- Proximal lesion in RCA
- Severely impaired flow in RV branches

Bowers et al. Circulation 2002
Same **cardiac output** as the left ventricle

**One sixth** of muscle mass of LV

**One fourth** of the stroke work of LV

**Pulmonary vascular resistance** is one tenth of systemic resistance

- **Biphasic nature** of blood flow during both systole and diastole
- **Extensive collateral flow** from rich left-to-right collateral system
- **Favorable oxygen supply-demand ratio and resistance to ischemia**

- In many proximal occlusions of RCA there is no evidence of right ventricular involvement
- Recovery of right ventricular function in the majority of patients after hospital discharge even without reperfusion

**Smaller mass and stroke work**
Pathophysiology of RV infarction

Acute RV dysfunction and dilatation

- Decreased RV cardiac output
- Increased RV diastolic pressure and septal shift towards LV
- Decreased LV preload
- Decreased LV cardiac output

Hypotension

Decreased coronary perfusion

Cardiogenic Shock

Adverse effects on hemodynamics when:

- The **interventricular septum** is involved (piston like effect on right heart systole)
- Loss of **atrioventricular synchrony** (AV nodal artery from RCA)
- **Volume depletion** (diuretics, nitrates)

Decreased RV cardiac output
Pathognomonic triad for RV infarction in a patient with inferior myocardial infarction but low sensitivity 25%
Diagnosis - ECG

ST elevation in V4R
sensitivity 88%
specificity 78%
accuracy 83%
Echocardiography in clinically significant RV Infarction

- Right ventricular dilatation (RV≥LV)
- Right ventricular free wall motion asynergy and systolic dysfunction
- Diastolic septal shift
- Interatrial septal bowing towards left atrium
- Tricuspid regurgitation
- IVC plethora
200 patients with inferior MI
54% had ST elevation in V4R

Thrombolysis in 36%

64% VS 28% in hospital complications

In – hospital mortality

Zehender et al NEJM 1993
Meta analysis of 22 studies (13 prospective and 9 retrospective) involving a total of 7,136 patients with AMI of whom 1,963 had Right Ventricular Infarction, mostly in the thrombolytic era.

Prevalence: 27.5%
Left ventricular infarct size and function were similar between patients with and those without RV infarction.
Relative risk of Complication Increase in AMI patients with RV infarction

Cardiogenic shock
Relative Risk = 2.9

Ventricular arrhythmias
Relative Risk = 2.29

Advanced AV block
Relative Risk = 3.27

Mechanical complications
Relative Risk = 3.01

Hamon et al Critical Care Med 2008
Right Ventricular free wall motion index after PCI of RCA

Unsuccessful reperfusion with PCI

Successful reperfusion with PCI

In hospital mortality 2%

In hospital mortality 53%

RV recovery even without reperfusion

Bowers et al NEJM 1998
In hospital mortality for RV shock = 53.1%
In Hospital mortality for LV shock = 60.8%
P = 0.296

Similar mortality of patients with RV and LV shock in SOCK registry

Jacobs a et al. JACC 2001

RV shock patients:
- Younger age
- Higher prevalence of single vessel disease
- Similar benefit from revascularization

Logrank P-value = 0.685
Primary reperfusion in acute right ventricular infarction: An observational study


- 2679 patients with first acute inferior infarction from 1996 to 2009
- 25.3% (679) of them had Right Ventricular Infarction by ECG
Clinical Classification of RV Infarction

No Right Heart Failure
- SBP > 100mmHG
- No Right Heart Failure by clinical/echo or hemodynamics

Right Heart Failure
- Clinical or
- Echo or
- Hemodynamics

Cardiogenic shock with all the following
- SBP < 90mmHG
- CO < 2.1l/min/m²
- ↓End-organ perfusion

64%  24%  12%

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### Echocardiographic data at baseline

<table>
<thead>
<tr>
<th>RV dilatation</th>
<th>No RVF</th>
<th>RVF</th>
<th>Cardiogenic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV/LV = 1</td>
<td>0%</td>
<td>65%</td>
<td>17%</td>
</tr>
<tr>
<td>RV/LV &gt; 1</td>
<td>0%</td>
<td>25%</td>
<td>83%</td>
</tr>
<tr>
<td>WMA only inferior wall of RV</td>
<td>84%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>WMA inferior + other wall of RV</td>
<td>16%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>TR</td>
<td>17%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>LVEF &lt; 50%</td>
<td>11%</td>
<td>22%</td>
<td>34%</td>
</tr>
</tbody>
</table>
### Hemodynamic data at baseline

<table>
<thead>
<tr>
<th></th>
<th>No RVF</th>
<th>RVF</th>
<th>Cardiogenic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>mRAP (mmHg)</strong></td>
<td>4.6 ± 2.1</td>
<td>12.9 ± 3.6</td>
<td>21.4 ± 5.15</td>
</tr>
<tr>
<td><strong>REVDP (mmHg)</strong></td>
<td>3.4 ± 1.7</td>
<td>11.2 ± 4</td>
<td>16 ± 5.4</td>
</tr>
<tr>
<td><strong>sPAP (mmHg)</strong></td>
<td>16.2 ± 4.4</td>
<td>16.4 ± 3.9</td>
<td>36.8 ± 9.3</td>
</tr>
<tr>
<td><strong>dPAP (mmHg)</strong></td>
<td>9.7 ± 3.7</td>
<td>13.4 ± 2.2</td>
<td>22.2 ± 3.9</td>
</tr>
<tr>
<td><strong>mPWP (mmHg)</strong></td>
<td>8.6 ± 3.1</td>
<td>12.1 ± 1.8</td>
<td>19.9 ± 6.2</td>
</tr>
<tr>
<td><strong>CI (L.min/m²)</strong></td>
<td>3.4 ± 0.71</td>
<td>2.4 ± 0.21</td>
<td>1.67 ± 0.5</td>
</tr>
<tr>
<td><strong>mSAP (mmHg)</strong></td>
<td>108.0 ± 7</td>
<td>78.7 ± 12.3</td>
<td>62.7 ± 9.5</td>
</tr>
<tr>
<td><strong>RAP/PWP ≥ 0.8</strong></td>
<td>2%</td>
<td>96%</td>
<td>92%</td>
</tr>
</tbody>
</table>
## Angiographic data at baseline

<table>
<thead>
<tr>
<th>Condition</th>
<th>No RVF</th>
<th>RVF</th>
<th>Cardiogenic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA culprit vessel</td>
<td>91%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>RCA proximal</td>
<td>16%</td>
<td>69%</td>
<td>92%</td>
</tr>
<tr>
<td>Without collateral</td>
<td>40%</td>
<td>53%</td>
<td>89%</td>
</tr>
<tr>
<td>Multi vessel disease</td>
<td>45%</td>
<td>68%</td>
<td>86%</td>
</tr>
<tr>
<td>TIMI flow 3 in all RVB</td>
<td>72%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Impaired flow in 1RVB</td>
<td>21%</td>
<td>8.9%</td>
<td>0%</td>
</tr>
<tr>
<td>Impaired flow in ≥ 2RVB</td>
<td>6%</td>
<td>33%</td>
<td>7%</td>
</tr>
<tr>
<td>No flow in all RVB</td>
<td>0%</td>
<td>57%</td>
<td>92%</td>
</tr>
</tbody>
</table>
### In Hospital Mortality

<table>
<thead>
<tr>
<th></th>
<th>No reperfusion therapy</th>
<th>Thrombolysis</th>
<th>Primary PCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>No RV failure</td>
<td>7.9%</td>
<td>4.4%</td>
<td>3.2%</td>
</tr>
<tr>
<td>RV failure</td>
<td>27%</td>
<td>13%</td>
<td>8.3%</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>80%</td>
<td>100%</td>
<td>58%</td>
</tr>
</tbody>
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★ P <0.01 vs previous

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### Complications in “silent” RV

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<th>Thrombolysis</th>
<th>Primary PCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrio-ventricular block</td>
<td>14%</td>
<td>6%</td>
<td>5%</td>
</tr>
<tr>
<td>SVT /VF</td>
<td>16%</td>
<td>5%</td>
<td>4%</td>
</tr>
<tr>
<td>Reinfarction</td>
<td>5%</td>
<td>7.3%</td>
<td>0.9%</td>
</tr>
<tr>
<td>Urgent target vessel revascularization</td>
<td>5%</td>
<td>3.6%</td>
<td>0.9%</td>
</tr>
<tr>
<td>tamponate/rupture</td>
<td>0%</td>
<td>1.8%</td>
<td>0%</td>
</tr>
<tr>
<td>Sustained hypotension</td>
<td>16%</td>
<td>1%</td>
<td>0%</td>
</tr>
<tr>
<td>evolution to Cardiogenic Shock</td>
<td>7%</td>
<td>5%</td>
<td>0%</td>
</tr>
</tbody>
</table>
Conclusions

- Always do right precordial ECG in inferior infarctions
- Avoid medications who reduce right ventricle performance (diuretics, nitrates, b-blockers, morphine)
- Perform timely reperfusion ideally with primary PCI with special attention to restore flow in right ventricular branches

Should we open an occluded RCA when clinically significant RV infraction occurs even after 48 hours?