Myocardial bridge: clinical case (Coronary stenting for symptomatic myocardial bridging)

Reporters: 6th course students-Awotunde Akinwumi G, Afolabi Omotolani B
Supervisors: MD Shmidt E.Y., MD Medved E.P, PhD Abdueva F.M., Head of the Department of Internal Medicine Professor Yabluchansky N.I.
Definition

- Myocardial bridge is defined as a segment of the major coronary artery running intramurally through the myocardium, deviating from its usual epicardial course.
- Synonyms: (intramural coronary artery, mural coronary artery, coronary overbridging, myocardial loop)
- First described anatomically by Reyman in 1737
- anterior descending artery (LAD), with the mid LAD considered the most common location
Structure of human heart
Etiology

- congenital anomaly of the coronary arteries
- rates much higher than the general population in the following conditions:
  - hypertrophic cardiomyopathy patients
  - heart transplant patients: related to the increased stiffness and hypertrophy of the myocardium post transplant, resulting in increased rates of systolic vessel compression
Morphological variation in tunneling (length and depth of tunneled segment)

Various Depths
- Epicardial
- Partially Tunneled
- Thin Myocardial Bridge
- Thick Myocardial Bridge
- Very Thick Myocardial Bridge

LAD

Various Lengths

Asymptomatic
Potentially Symptomatic

Myocardial BRIDGE
Pathogenesis

- The myocardial bridge causes coronary artery narrowing during systole therefore myocardial bridges should not compromise blood supply to the musculature during diastole.
- Systolic narrowing at the myocardial bridging segment may result in endothelial damage, which may provoke platelet aggregation, coronary vasospasm and eventually acute coronary syndrome.
Pathogenesis

• the vessel segment proximal to the bridge appears to develop atherosclerosis at increased rates, approaching 90% rather than the myocardial bridging segment itself. Research has shown vasoactive agents to be present in higher concentrations in the proximal portion of the myocardial bridging artery compared to the myocardial bridging segment. It can also be an alternative cause of ischemia in patients with myocardial bridging.
Clinical Manifestations (Features)

- Symptomatic patients with myocardial bridging may present with myocardial ischemia,
- acute coronary syndromes,
- coronary spasm,
- exercise-induced dysrhythmias (such as supraventricular tachycardia, ventricular tachycardia, or atrioventricular block),
- myocardial stunning,
- transient ventricular dysfunction,
- syncope,
- sudden death.

When myocardial bridging is associated with heart valve disorder or cardiomyopathies, the patients' symptoms can be different.
Diagnosis

- Coronary angiography: The typical angiographic feature of a myocardial bridge is systolic narrowing of an epicardial artery, which is often completely resolved during the diastolic phase of the cardiac cycle.
- Intracoronary Doppler
- Echocardiography
- Electrocardiography
- Stress test with ECG
- Intravascular ultrasound
- Fractional flow reserve
- Cardiac computed tomography (CT) angiography
Prognosis

• Myocardial bridging is generally considered to be a benign condition, it has been proposed as a cause of angina-like chest pain, coronary spasm, myocardial ischemia, acute coronary syndromes, left ventricular dysfunction/stunning, arrhythmias (including supraventricular tachycardia and ventricular tachycardia), and even sudden cardiac death. Serious events are uncommon, and it is still controversial and unclear whether myocardial bridging can be directly attributed as the cause of the events.
Management

- Pharmacologic therapy:
  - First-line therapy: beta-blockers and non-dihydropyridine calcium-channel blockers (decreased chronotropy and inotropy i.e. prolongation of diastole with reductions in heart rate)
  - Nitrates are contraindicated in patients
- Surgical treatments: surgical myotomy or coronary stenting
Clinical Case

Patient Identifying data

- Age: 67 years old
- Sex: Female
Complaints

Main complaints:
• retrosternal pressing pain that occurs either after emotional stress or without clear connection with any provoking factors, relieves in rest
• Unstable blood pressure

System review:
• review of other organs and systems reveals no complaint
History of the present disease

• The patient has been suffering from hypertension since the last 20 years. Maximum BP level (210/100 mmHg) was noticed 4 years ago when patient lost consciousness, the ambulance was called. No significant changes on ECG were revealed. After those incident she periodically hospitalized in CCH and received antihypertensive treatment (ampril 2.5 mg, bisoprolol 5 mg, physiotens 0.4 mg) «Working» BP 140/80 mmHg,

• Since last year she has been suffering from retrosternal pain that occurs either after emotional stress or without connection with any provoking factors, relieves in rest without taking any medicines. The complaints on retrosternal pain brought patient to the hospital for examination and treatment
Life history

• No previous surgery
• No history of tuberculosis, diabetes mellitus, myocardial infarction, gastroesophageal reflux disease, pericarditis, valvular heart diseases.
Physical examination

• No physical abnormalities were detected by clinical examination and blood pressure was 140/80 mmHg (on the background of antihypertensive medication), HR 61 bpm
• Respiratory rate: no significant changes
• Auscultation: clear vesicular sound
• Accentuated second sound over the aorta
• Abdomen without any changes
Preliminary diagnosis

- Arterial hypertension stage II 3 degree.
  Hypertensive heart HF 0-1
- IHD. Stable angina
Plan of investigation

• Minimum investigation:
  – Complete blood count
  – Urine analysis
  – Blood analysis (glucose, creatinine, lipid profile, total bilirubin, AST, ALT)
  – ECG
  – EchoCG
  – Coronary arteriography
  – Exercise treadmill test
ECG

- Conclusion: Sinus rhythm. Signs of left ventricular hypertrophy
Lipid profile/Blood test

- triglycerides 3.3 mmol/L (≤ 2.3)
- high density lipoprotein cholesterol 1.43 mmol/L (≥ 0.9), total cholesterol 7.93 mmol/L (<5.2),
- low density lipoprotein cholesterol 0.77 mmol/L (<3.5),
- blood glucose 5.5 mmol/L (3.5-5.5)
- Atherogenic coefficient 54 (≤ 3)

Conclusion: mild hyperlipidemia (triglyceride level was slightly increased)
All other blood tests also were normal
EchoCG

Diameter of aorta 32mm(20-37mm)

- Mitral valve opening: 29mm(26-35mm)
- Left atrium: 32mm
- Left ventricle, end diastolic diameter: 40mm(35-55), end systolic diameter: 25mm(23-38mm), ejection fraction: 65%(55-78%), systolic fraction 34%(28-44%)
- Interventricular septum: 11.8mm(6-11mm)
- Right atrium: 28mm, Right ventricle: 18mm(9-26)
- Thickness of LV posterior wall- 12.7 mm(6-11mm)
- Conclusion: there is atherosclerotic changes of the aorta, hypertrophy of the left ventricle
Exercise treadmill test

• While doing exercise treadmill test (protocol Bruce): blood pressure, heart rate and 12-leads ECG were recorded during several steps with increased physical exertions (from 4,6 METs). The ECG and ST-segment were continuously displayed and measured automatically by a computer-assisted system in all 12 leads.

• Max reached BP was 180/100 mmHg, max HR 127 bpm. At heart rate of 127 beat/minute (7,0METs), the ST segment showed progressive depression more then 1,0 mm in leads II, III, avf, V4, V5, V6 that necessitated termination of the test. The patient felt only mild dyspnea and tiredness. During 4 minutes of restitution period there was complete recovery of ST-segment.

• Conclusion: test is positive.
Exercise treadmill test: Early phase

- No significant changes was observed
Exercise treadmill test: Maximum Exertion

- ST depression on V4-V6 and on lead II, III, and aVF
Coronary angiography

• The right type of coronary blood supply. Significant coronary tortuosity. Left coronary artery - prolonged myocardium bridging in the middle segment of the left anterior descending coronary artery with systolic compression 90%.

• The circumflex artery branches of the left coronary artery and right coronary artery - with signs of atherosclerotic lesions without hemodynamic significance
Compression of coronary artery in systole

systole

diastole
Day-night BP monitoring

<table>
<thead>
<tr>
<th>No</th>
<th>Beginning</th>
<th>HR</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11:28:00</td>
<td>68</td>
<td>146</td>
<td>78</td>
</tr>
<tr>
<td>2</td>
<td>11:59:34</td>
<td>77</td>
<td>163</td>
<td>89</td>
</tr>
<tr>
<td>4</td>
<td>12:00:12</td>
<td>66</td>
<td>149</td>
<td>70</td>
</tr>
<tr>
<td>12</td>
<td>15:51:09</td>
<td>64</td>
<td>137</td>
<td>78</td>
</tr>
<tr>
<td>16</td>
<td>17:00:41</td>
<td>60</td>
<td>149</td>
<td>81</td>
</tr>
<tr>
<td>22</td>
<td>19:43:39</td>
<td>70</td>
<td>136</td>
<td>59</td>
</tr>
<tr>
<td>26</td>
<td>21:22:16</td>
<td>76</td>
<td>123</td>
<td>52</td>
</tr>
<tr>
<td>27</td>
<td>21:53:52</td>
<td>62</td>
<td>122</td>
<td>59</td>
</tr>
<tr>
<td>28</td>
<td>22:25:32</td>
<td>51</td>
<td>123</td>
<td>57</td>
</tr>
<tr>
<td>32</td>
<td>23:33:07</td>
<td>70</td>
<td>133</td>
<td>69</td>
</tr>
<tr>
<td>33</td>
<td>00:33:48</td>
<td>57</td>
<td>114</td>
<td>51</td>
</tr>
<tr>
<td>34</td>
<td>01:35:25</td>
<td>49</td>
<td>119</td>
<td>58</td>
</tr>
<tr>
<td>37</td>
<td>03:38:52</td>
<td>45</td>
<td>91</td>
<td>44</td>
</tr>
<tr>
<td>38</td>
<td>04:40:30</td>
<td>47</td>
<td>115</td>
<td>60</td>
</tr>
<tr>
<td>39</td>
<td>05:41:52</td>
<td>51</td>
<td>148</td>
<td>69</td>
</tr>
<tr>
<td>40</td>
<td>06:43:20</td>
<td>61</td>
<td>162</td>
<td>76</td>
</tr>
<tr>
<td>42</td>
<td>08:17:09</td>
<td>96</td>
<td>141</td>
<td>70</td>
</tr>
</tbody>
</table>

- BP monitoring was done on the background of antihypertensive drugs
- Changes for BP is typical for mild hypertension
Clinical Diagnosis

• Main disease: Myocardial bridge of LAD with systolic compression 90%. Coronary stenting of LAD. HF 0-1. High risk.

• Concomitant diseases: Arterial hypertension II st 3degree. Hypertensive heart.
Treadmill after stenting (early phase)
Treadmill after stenting (Maximum exertion)

Treadmill test was negative. No complaint of angina
Treatment during hospitalisation

Antihypertensive drugs:
- ecvator (amlodipine besylate/lisinopril dihydrate) 20/10mg in the morning
- physiotens (moxonidine) 0.4mg in the evening
- Amlodipine 5mg in the evening

Antihyperlipidemic drug:
- Crestor (rosuvastatin calcium) 40mg in the evening

Stent protecting drug:
- Plavix (clopidogrel bisulfate) 75mg
Drug recommendation after discharge

Antihypertensive drugs:
✓ Bisoprolol 5mg daily
✓ Ampril (ramipril) 5mg daily
✓ Physiotens (moxonidine) 0.4mg in the evening

Antihyperlipidemic drug:
✓ Crestor (rosuvastatin calcium) 40mg in the evening

Stent protecting drugs:
✓ Plavix (clopidogrel bisulfate) 75mg
✓ Aspirin 100mg
Conclusion

• Significant myocardial bridge in a symptomatic patient can simulate angina. Coronary angiography might be useful to better characterize the length, location and significance of the MB. In asymptomatic patients a myocardial bridge is not treated. In patients with symptoms, medicines such as beta-blockers and calcium channel blockers are usually the first line of treatment. But in refractory to medication cases or in cases with significant systolic compression of coronary artery stent implantation can be one of the method for surgery management of myocardial bridge.
References

• Stefan Möhlenkamp, Waldemar Hort, Junbo Ge, Raimund Erbel. AHA Journals Update on Myocardial Bridging(http://circ.ahajournals.org/content/106/20/2616)
• Michael S. Lee, MD and Cheng-Han Chen, MD Myocardial Bridging: An Up-to-Date Review(https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4818117/)
• Mohamed Abdou Myocardial bridging causing ischemia and recurrent chest pain: a case report (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3224392)
• Dr Umamaheswara Reddy V et al. Myocardial bridging of the coronary arteries(https://radiopaedia.org/articles/myocardial-bridging-of-the-coronary-arteries)
• Netter Atlas of human anatomy
Thank you