AORTIC STENOSIS IN 93 Y.O. FEMALE
(case report)
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Abstract: The increase in life expectancy of patients contributes to development of new technology for treatments of patients with comorbidities of advanced age. Transcatheter aortic-valve implantation has been suggested as a less invasive treatment for high-risk patients with aortic stenosis.

Keywords: aortic stenosis, transcatheter aortic-valve implantation

INTRODUCTION

Aortic stenosis (AS) has become the most frequent type of ventricular heart disease (VHD) in Europe and North America. It primarily presents as calcific AS in adults of advanced age (2-7% of the population over 65 years) [1, 2]. The primary treatment for severe aortic stenosis is aortic valve replacement surgery. The 3-year survival in patients with symptomatic aortic stenosis who undergo surgery is 87%; in those who do not have surgery, it is 21% (P <0.001) [3].

AS with a valve area 1.0 cm² is considered severe, critical AS is most likely with a valve area 0.8 cm² [4].

CASE STUDY

A 93-year old women with a stress reaction on death of the husband. In anamnesis are the iron deficiency anemia, sigmodivertikulosis, colonic angiodysplasia (2014), cholecystectomy, appendectomy, chronic renal failure, presented with, paroxysmal atrial fibrillation (under Rivaroxaban therapy), symptomatic aortic (valve) stenosis, arterial hypertension, osteoporosis.

Physical examination:

Anthropometry - 156 cm, 65 kg. BP - 150/60 mm Hg. Loud systolic murmurs were heard in point 2 with right carotid radiating. Weak vesicular breathing was heard in the basal area; basal pleural effusions were noticed. The abdomen was soft, not resistant; the patient denied any tenderness. Discreet lower leg edema was present. She also denied chest pain, shortness of breath. She reported exercise dyspnea.

Chest X-ray demonstrated chronic signs of congestion and low angle effusions on both sides, sclerosis of the aorta without the evidence of infiltration. Laboratory data are presented in Table 1.

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### Table 1.
Results of laboratory investigation of the female patient with aortic stenosis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>14.06 - 2016</th>
<th>17.06 - 2016</th>
<th>Unit</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood cells</td>
<td>10.93</td>
<td>10.63</td>
<td>G./L</td>
<td>3.60-10.50</td>
</tr>
<tr>
<td>Platelets</td>
<td>565</td>
<td>503</td>
<td>G./L</td>
<td>160-370</td>
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<td>Red Blood Cells</td>
<td>3.49</td>
<td>4.19</td>
<td>T/L</td>
<td>3.85-5.20</td>
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<tr>
<td>HGB (Hb, hemoglobin)</td>
<td>7.1</td>
<td>8.6</td>
<td>g/dl</td>
<td>11.8-15.8</td>
</tr>
<tr>
<td>HCT (hematocrit)</td>
<td>23.7</td>
<td>28.8</td>
<td>%</td>
<td>35-45%</td>
</tr>
<tr>
<td>NEUT%</td>
<td>77.8</td>
<td>72.3</td>
<td>%</td>
<td>50.0-70.0</td>
</tr>
<tr>
<td>LYM%</td>
<td>11.5</td>
<td>153.</td>
<td>%</td>
<td>20.0-44.0</td>
</tr>
<tr>
<td>Hypochromic Ery.</td>
<td>53.1</td>
<td>41.5</td>
<td>%</td>
<td>0-2.5%</td>
</tr>
<tr>
<td>Kreatinin</td>
<td>1.56</td>
<td>1.37</td>
<td>mg/dl</td>
<td>0.51-0.95</td>
</tr>
<tr>
<td>GFR</td>
<td>28.38</td>
<td>33.21</td>
<td>mL/min/1.7 m²</td>
<td>&gt;90</td>
</tr>
<tr>
<td>BUN</td>
<td>32</td>
<td>22</td>
<td>mg/dl</td>
<td>8-23</td>
</tr>
<tr>
<td>Uric Acid</td>
<td>8.3</td>
<td>8.8</td>
<td>mg/dl</td>
<td>2.4-5.7</td>
</tr>
<tr>
<td>Na</td>
<td>140</td>
<td>138</td>
<td>mmol/L</td>
<td>136-145</td>
</tr>
<tr>
<td>K</td>
<td>4.8</td>
<td>4.4</td>
<td>mmol/L</td>
<td>3.5-4.6</td>
</tr>
<tr>
<td>Chlorid</td>
<td>101</td>
<td>102</td>
<td>mmol/L</td>
<td>3.5-4.6</td>
</tr>
<tr>
<td>Iron</td>
<td>16</td>
<td>102</td>
<td>µg/dL</td>
<td>33-193</td>
</tr>
<tr>
<td>Transferrin</td>
<td>2.45</td>
<td></td>
<td>g/l</td>
<td>2.0-3.6</td>
</tr>
<tr>
<td>Transferrin saturation</td>
<td>4.63</td>
<td></td>
<td>%</td>
<td>16.0-45.00</td>
</tr>
<tr>
<td>Ferritin</td>
<td>14</td>
<td></td>
<td>µg/L</td>
<td>15-150</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>341.7</td>
<td></td>
<td>Pg/mL</td>
<td>197-771.0</td>
</tr>
<tr>
<td>CRP</td>
<td>9.4</td>
<td></td>
<td>mg/l</td>
<td>0.0-5.0</td>
</tr>
<tr>
<td>ESR 1h</td>
<td>57</td>
<td></td>
<td>mm</td>
<td>0-45</td>
</tr>
<tr>
<td>Calcium</td>
<td>2.29</td>
<td>2.37</td>
<td>mmol/L</td>
<td>2.05-2.40</td>
</tr>
<tr>
<td>CK</td>
<td>38</td>
<td></td>
<td>U/L</td>
<td>20-180</td>
</tr>
<tr>
<td>Troponin T-hs</td>
<td>32.69</td>
<td></td>
<td>ng/L</td>
<td>0.0-14.0</td>
</tr>
<tr>
<td>ALAT (GPT)</td>
<td>27</td>
<td></td>
<td>U/L</td>
<td>0-35.0</td>
</tr>
<tr>
<td>Gamma-GT</td>
<td>39</td>
<td></td>
<td>U/L</td>
<td>0-40</td>
</tr>
<tr>
<td>Alk. Phosphatase (AP)</td>
<td>71</td>
<td></td>
<td>U/L</td>
<td>35-105</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>0.27</td>
<td></td>
<td>mg/dL</td>
<td>0.00-1.20</td>
</tr>
<tr>
<td>LDH</td>
<td>317</td>
<td></td>
<td>U/L</td>
<td>0-250</td>
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<tr>
<td>Glucose</td>
<td>148</td>
<td></td>
<td>mg/dL</td>
<td>50-100</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>126</td>
<td></td>
<td>mg/dL</td>
<td>150-200</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>22</td>
<td></td>
<td>mg/dL</td>
<td>&gt;65</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td></td>
<td></td>
<td>mg/dL</td>
<td>60-130</td>
</tr>
<tr>
<td>Chol./HDL-Ch.-ratio</td>
<td>5.7</td>
<td></td>
<td></td>
<td>0.3-6.</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>198</td>
<td></td>
<td>mg/dL</td>
<td>50-150</td>
</tr>
<tr>
<td>Total proteins</td>
<td>65</td>
<td></td>
<td>g/L</td>
<td>66-87</td>
</tr>
<tr>
<td>TSH (hormone)</td>
<td>2.50</td>
<td></td>
<td>µU/ml</td>
<td>0.27-4.20</td>
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<tr>
<td>Hemostasis</td>
<td>aPTT</td>
<td>48.5</td>
<td>sek</td>
<td>26.0-38.0</td>
</tr>
<tr>
<td></td>
<td>INR</td>
<td>1.84</td>
<td></td>
<td>1.07</td>
</tr>
</tbody>
</table>

ECG revealed sinus rhythm, HR was 74 bpm. left axis, left ventricular hypertrophy, AV-Block I, T- neg.: I, II, V5-6 (Repolarization problem) (fig.1).

![Fig. 1. ECG](image1)

Echocardiography demonstrated a normal borderline, normal Left Ventricular Ejection Fraction LVEF( EF 54%); Mitral Insufficiency grade II; Severe Aortic Stenosis, max. gradient 105 mm Hg, mean AV Pg 62 mm Hg., AVA V max/VTI 0.8 cm 2, pulmonary hypertension 65 mm. Hg. (fig.2). Conclusively aortic stenosis together with mitral insufficiency and pulmonary hypertension were diagnosed.

![Fig. 2. Aortic valve velocity and pressure gradient.](image2)

Coronary angiography revealed coronary l-vessel stenosis with focal (about 70 %) proximal left arteria descending (LAD) stenosis.

The patient was diagnosed:

I35.0 - Aortic (valve) stenosis, valve area 0.8 cm²
I25.1 - Atherosclerotic heart disease. Focal stenosis proximal LAD.
I34.0 - Mitral (valve) insufficiency.
I13.1 - Hypertensive heart and renal disease with renal
failure

I48. - Paroxysmal atrial fibrillation
I50.0 - Congestive heart failure
E14 - Diabetes mellitus
N18.9 - Chronic renal failure
D50 - Iron deficiency anemia.
K57 - Sigmodivertikulosis.
K55 - Angiodysplasia of the colon.

Treatment:
1. Oral rehydration therapy according standard protocol under the control of kidney function.
2. The earlier treatment was continued:
   • Actonel 35 mg vis Calcimagnon 1 weekly
   • Pantoloc 40 mg in the morning (1/0/0)
   • Lexotanil 3 mg in the evening (0/0/1)
   • Rasilez (aliskeran) / HCT 300 /12.5 mg in the morning (1/0/0)
     • Concor 5 mg (2.5 mg twice per day) (1/2/0/1/2)
     • Furon 40 mg (20 mg twice per day) (1/2/1/2/ 0)
     • Digimerc 0.07 mg (0.035 in the morning) (1/2/0/0)
     • Zanidic (lercanidipine) 10mg per day (0/1/0),
     • Xarelto 15 mg per day (1/0/0) (under current International Normalized Ratio (INR) and glomerular filtration rate (GFR)
       • Metformin 850 mg twice per day (1/0/1)
3. The erythrocyte concentrate infusion was successfully prescribed for treatment hypochromic microcytic anemia (no side effects with appropriate rise of Hb level).
4. Oral iron therapy for approximately 1 month, after a reassessment of the iron status. GIT examination was recommended one month after the discharge from hospital (iron therapy should be stopped for 10 days before it).
5. Transcatheter aortic valve implantation (TAVI) was considered.

Discussion
Aortic valve replacement (AVR) is the definitive therapy for severe AS. In contemporary series, operative mortality of isolated AVR for AS is 1-3% in patients younger than 70 years and 4-8% in selected older adults [1,4 - 13].

Surgery has been shown to prolong and improve quality of life, even in selected patients over 80 years of age [10 - 13].

Contraindications for transcatheter aortic valve implantation can be absolute, clinical and relative.

Absolute contraindications include:
• Absence of a ‘heart team’ and no cardiac surgery on the site
• Appropriateness of TAVI, as an alternative to AVR, not confirmed by a ‘heart team’

Clinical contraindications comprise:
• Estimated life expectancy <1 year
• Thrombus in the left ventricle
• Active endocarditis
• Elevated risk of coronary ostium obstruction (asymmetric valve calcification, short distance between annulus and coronary ostium, small aortic sinuses)
• Plaques with mobile thrombi in the ascending aorta, or arch
• For transfemoral/subclavian approach: inadequate vascular access (vessel size, calcification, tortuosity)

Relative contraindications are:
• Bicuspid or non-calcified valves
• Untreated coronary artery disease requiring revascularization
  • Haemodynamic instability
  • LVEF<20%
• For transapical approach: severe pulmonary disease, LV apex not accessible.

In patients with high surgical risk, TAVI has been shown to be feasible (procedural success rates 90%) using transfemoral, transapical or, less commonly, subclavian or direct trans-aortic access [13,14 - 22].

4 CONCLUSIONS
The Considering the patient’s clinical condition: Severe aortic valve stenosis, the absence of decompensation of the cardiovascular and respiratory systems- patient is need of replacement surgery Trans catheter implantation of the aortic valve.
REFERENCES


Received: 28-Jul. - 2016
Accepted: 20-Sep. - 2016