Unusual finding

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History

- Male patient 55 years, farmer, non smoker, not hypertensive nor diabetic.
- Presented with recurrent typical anginal pain.
Examination

- Average built
- BP: 140/70
- Pulse: 90.
- Cardiac ex.: NAD
- Chest ex.: NAD

ECG
Cardiac biomarkers

- CPK, CK-MB and Troponin were normal.
• Huge vascular structure anterior and lateral to the left ventricle, with spontaneous echo contrast inside with no color flow.

Lab

• Creatinine: 2.9 mg/dl.
• GFR: 23 mL/min/ 1.73 m²
• Normocytic normochromic anemia.
• Albumin: 4.1 gm
• SGOT, SGPT: normal
• Electrolytes & ABG: average.
What is the next step?

1. Coronary Angiography.
2. MSCT
3. Cardiac MRI

• Before decision making:
  ✓ Renal US was done showing:
    • Right kidney with poor Corticomedullary differentiation.
    • Left kidney shows grade I nephropathy.
  ✓ Nephrological consultation didn’t recommend any contrast agents unless if life saving condition.
Decision

• Non contrast CMR was preferred to diagnose this vascular structure.
What is next?

- Whole heart axial CMR sequence without contrast was done to evaluate the aorta and coronaries.
- Post-processing reconstruction was done.
• The diagnosis is huge aneurysm originating from the Left main taking the course of LAD with a large thrombus inside.
  • Multiple RCA aneurysms.

Coronary artery aneurysm (CAA)

• Focal or diffuse dilatation of a coronary artery >1.5 times of its original diameter.
## Causes

<table>
<thead>
<tr>
<th>Cause</th>
<th>Age</th>
<th>Description</th>
<th>Pathogenetic Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerosis</td>
<td>Adults</td>
<td>Most common cause of CAA, clinical importance depends on association with significant coronary artery stenosis</td>
<td>Local mechanical stress from stenosis, atherosclerotic pathologic findings extending into tunica media</td>
</tr>
<tr>
<td>Vasculitis</td>
<td>Childhood</td>
<td>Most common cause of CAA in childhood in Japan, spontaneous resolution occurs in 50%</td>
<td>Autoimmune, vasculitis</td>
</tr>
<tr>
<td>Takayasu</td>
<td>Young adults</td>
<td>Common cause of CAA in young Asian females in Japan</td>
<td>Cellular immunity associated with chronic infection</td>
</tr>
<tr>
<td>Polycystic Kidney Disease</td>
<td>Young adults</td>
<td>Necrotizing inflammatory lesions in small- and medium-sized arteries</td>
<td>Characterized by fibronectin necrosis and infiltration by predominantly polymorphonuclear leukocytes</td>
</tr>
<tr>
<td>Connective tissue disorders</td>
<td>Young adults</td>
<td>Ehlers-Danlos syndrome, Marfan syndrome, cystic medial necrosis</td>
<td>IL-6, TNF-α, C-reactive protein, MMP-2, MMP-9</td>
</tr>
<tr>
<td>Mycotic</td>
<td>Any age</td>
<td>Infection with Staphylococcus aureus or Pseudomonas aeruginosa, syphilis, Lyme disease</td>
<td>Microembolization to vasa vasorum, direct pathogen invasion of arterial wall, immune complex deposition</td>
</tr>
<tr>
<td>Trauma/iatrogenic</td>
<td>Adults</td>
<td>Clinical history helps establish diagnosis because of antiproliferative treatment with corticosteroids, calcineurin, and anti-inflammatory drugs</td>
<td>Trauma from overinflated balloon or high inflation pressures, coronary dissection, interventions in the setting of acute myocardial infarction, inadequate</td>
</tr>
</tbody>
</table>

## Classification of CAA

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Categories</th>
<th>Luminal Diameter of the Aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shape</strong></td>
<td>Secular, Fusiform</td>
<td>Maximum Transverse Diameter &gt; Longitudinal Diameter</td>
</tr>
<tr>
<td><strong>Vascular Wall Integrity</strong></td>
<td>True Aneurysm, Pseudoaneurysm</td>
<td>Longitudinal Dimension &gt; Maximum Transverse Diameter</td>
</tr>
<tr>
<td><strong>Topographical Extent</strong></td>
<td>Type I, Type II, Type III, Type IV</td>
<td>All Vascular Layers Present, Loss of the Vascular Wall Integrity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diffuse Dilatation of Two or Three Vessels</td>
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<tr>
<td></td>
<td></td>
<td>Diffuse Dilatation in One Vessel and Localized in Another</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diffuse Dilatation of One Vessel Only</td>
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<tr>
<td></td>
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<td>Localized or Segmental Dilatation</td>
</tr>
</tbody>
</table>

Modified from Antoniades et al., 2008, Diaz-Zamudio et al., 2009.
Presentation

• Asymptomatic and diagnosed incidentally in coronary angiograms.
• They are mostly located in RCA followed by left main, LAD, and LCX.
• Atherosclerosis is the most common cause of CAAs.

Prognosis

• The prognosis of CAAs differs according to the severity of obstructive CAD.
Management

1. In asymptomatic patients without severe CAD, **conservative** approach is recommended.
2. **Covered stents** may be concerned in eligible symptomatic patients.
3. Surgery