Οξέα Στεφανιαία Σύνδρομα

Αυτόματος Διαχωρισμός Στεφανιαίων

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Καρδιολογικό Τμήμα Γ.Ν. «Ασκληπιείο Βούλας»
Disclosures

• None for this topic
Mechanisms of SCAD

A

Arterial lumen
Vasa vasorum

B

Intima
Adventitia
Media

C

Intimal rupture
False lumen with intramural hematoma
True lumen

Incidence

• In a Canadian series of women less than 50 years with myocardial infarction, SCAD accounted for 24% of cases.

• Likewise a Japanese registry reported SCAD in 35% of females patients under 50 years presenting with acute myocardial infarction (AMI)

• French series reported SCAD in 36% of women under 60 years with ACS and one or fewer conventional cardiovascular risk factors

• A smaller Australian series describes a SCAD prevalence of 23% in women under 60 years presenting with ACS.
### Patients Characteristics

**Saw J et al. J Am Coll Cardiol 2017;70:1148–58**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>52.5 ± 9.6</td>
</tr>
<tr>
<td>Female</td>
<td>297 (90.8)</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>24.4 (21.5-28.3)</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>268 (82.0)</td>
</tr>
<tr>
<td>East Asian</td>
<td>35 (10.7)</td>
</tr>
<tr>
<td>South Asian</td>
<td>17 (5.2)</td>
</tr>
<tr>
<td>African Canadian</td>
<td>3 (0.9)</td>
</tr>
<tr>
<td>First nation</td>
<td>2 (0.6)</td>
</tr>
<tr>
<td><strong>Diabetes mellitus</strong></td>
<td>15 (4.6)</td>
</tr>
<tr>
<td><strong>Dyslipidemia</strong></td>
<td>84 (25.7)</td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td>119 (36.4)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>32 (9.8)</td>
</tr>
<tr>
<td>Family history of coronary artery disease</td>
<td>109 (33.3)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>3 (0.9)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>13 (4.0)</td>
</tr>
<tr>
<td><strong>Hypothyroidism</strong></td>
<td>43 (13.1)</td>
</tr>
<tr>
<td>Postmenopausal</td>
<td>169* (56.9)</td>
</tr>
<tr>
<td>Migraines</td>
<td>119 (36.4)</td>
</tr>
<tr>
<td>Depression</td>
<td>74 (22.6)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>44 (13.5)</td>
</tr>
</tbody>
</table>

*Note: The value marked with an asterisk (*) is not explicitly provided in the table but can be calculated from the total number of patients (N = 327)."
Predisposing causes

- **Fibromuscular dysplasia**
- **Pregnancy-related**: antepartum, early post-partum, late postpartum, very late post-partum
- **Recurrent pregnancies**: multiparity or multigravida
- **Connective tissue disorder**: Marfan syndrome, Loeys-Dietz syndrome, Ehler-Danlos syndrome type 4, cystic medial necrosis, alpha-1 antitrypsin deficiency, polycystic kidney disease
- **Systemic inflammatory disease**: systemic lupus erythematosus, Crohn’s disease, ulcerative colitis, polyarteritis nodosa, sarcoidosis, Churg-Strauss syndrome, Wegener’s granulomatosis, rheumatoid arthritis, Kawasaki, giant cell arteritis, celiac disease
- **Hormonal therapy**: oral contraceptive, estrogen, progesterone, beta-HCG, testosterone, corticosteroids
- **Coronary artery spasm**
- **Idiopathic**

Precipitating stressors

- Intense exercises (isometric or aerobic activities)
- Intense emotional stress
- Labor and delivery
- Intense Valsava-type activities (e.g., retching, vomiting, bowel movement, coughing)
- Recreational drugs (e.g., cocaine, amphetamines, metamphetamines)
- Intense hormonal therapy (e.g., beta-HCG injections, corticosteroids injections)
Type 1

Type 2a

Type 2b

Type 3

Type 4

Mixed

- Patients with SCAD usually present with ACS.
- Delayed diagnosis is common and SCAD should be actively considered in the differential diagnosis of ACS presentations in low risk patients.
Lesion Characteristics

Diagnostic algorithm of myocardial infarction with no obstructive coronary atherosclerosis

Clinical History, ECG, echocardiography and cardiac biomarkers

Coronary angiography

LV angiogram

Normal o regional wall motion abnormalities with "epicardial pattern"

Epicardic causes

Coronary Artery plaque

Coronary dissection

Normal o regional wall motion abnormalities with "microvascular pattern"

Microvascular causes

CAS

CMS

Takotsubo Syndrome

Myocarditis

Coronary embolism

Additional angiographic findings

• Increased coronary tortuosity
• Predilection for more distal coronary segments (in contrast to atherosclerotic disease)
• Predominant involvement of the left anterior descending coronary artery and its branches reported in most but not all series
• False lumen starting and/or ending at a side branch
• Absence or reduced incidence of co-existent atherosclerosis—unaffected coronaries are usually normal or near-normal
• Coronary FMD
• Association of sites of dissection with myocardial bridging
Multimodality Imaging for Spontaneous Coronary Artery Dissection

**IMAGING OF CORONARY ARTERY ANATOMY**

- **Type 1:** Contrast staining in false lumen

**IMAGING OF MYOCARDIAL FUNCTION ± PERFUSION**

- **TTE**
- **CTA**
- **CMR**
- **MPI**

Tweet MS et al. J Am Coll Cardiol Img 2016;9:436–50
Advantages and Disadvantages of Intracoronary Imaging for SCAD

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definitive diagnosis of SCAD</td>
<td>Invasive, requires anticoagulation</td>
</tr>
<tr>
<td>Confirm true lumen entry by coronary wire</td>
<td>Costly</td>
</tr>
<tr>
<td>Facilitate stent sizing</td>
<td>Not available in all laboratories</td>
</tr>
<tr>
<td>Confirm adequate stent apposition</td>
<td>Possible risks of extending dissection by:</td>
</tr>
<tr>
<td>Confirm full coverage of dissected segment</td>
<td>- Guide catheter, coronary wire</td>
</tr>
<tr>
<td>Facilitate diagnosis of potential arteriopathy</td>
<td>- Imaging catheter</td>
</tr>
<tr>
<td></td>
<td>- Hydraulic extension (with OCT)</td>
</tr>
<tr>
<td></td>
<td>- Vessel occlusion (by catheter, embolization)</td>
</tr>
</tbody>
</table>
Intravascular imaging

Early Coronary Angiography

Type 1 SCAD appearance (arterial wall stain, multiple lumen)?

No

Type 2 SCAD appearance (diffuse, smooth stenosis)?

No

Type 3 SCAD appearance (mimics atherosclerosis)?

Perform OCT or IVUS

Yes

Give IC nitroglycerin, and consider:
- Perform OCT/IVUS, or
- Repeat angiogram in 4-6 weeks

SCAD
Multimodality Imaging for Spontaneous Coronary Artery Dissection

- Acute SCAD on angiography
  - No
  - Yes
    - OCT/IVUS: False lumen or intramural hematoma?
      - Yes
        - TIMI flow assessment
          - TIMI 0-1 OR clinically unstable
            - Revascularize with inpatient monitoring for 5-7 days, consider CABG in high volume surgical centers
          - TIMI 2-3 AND clinically stable
            - Conservative management with inpatient monitoring for 5-7 days
Revascularization Versus Conservative Therapy

Interventional Challenges

• Increased risk of secondary iatrogenic dissection (2% risk during coronary angiography vs. 0.2% during non-SCAD angiography, 14.3% during PCI)

• Guidewire passage into the false lumen

• Proximal and/or distal false lumen propagation during stent deployment

• Persistent distal dissection

• Major side branch restriction or occlusion by propagation of hematoma

• Often extensive dissected segments require long stents, increasing stent restenosis

• Risk of stent malapposition after resorption of IMH, with risk of late stent thrombosis
Suggestions if PCI is pursued for SCAD

• Meticulous guide catheter manipulation, preferably through femoral access approach
• OCT/IVUS guidance to ensure wire in true lumen (or over-the-wire catheter injections) and optimize stent apposition
• Long stents covering 5 – 10 mm of proximal and distal edges of IMH
• Placing short stents at proximal and distal edges first, before placing long stent in the middle
• Targeting an intimal tear or ‘flap’ for focal stenting or stenting just the proximal extent of the dissection to prevent proximal propagation
• Consider bioabsorbable stents (temporary scaffold to avoid long-term malapposition)
• Minimal plain old balloon angioplasty (POBA) to restore flow followed by a conservative strategy
• Possible and careful use of cutting balloon (to fenestrate IMH)
• Consider follow-up OCT to assess for malapposed/uncovered struts before stopping DAPT

Pharmacology

• β – blockers
• Antiplatelets
• ACE – i
• Lipid lowering therapy
Prognosis according to HTN status and β-blocker use

Prognosis: mortality, recurrence risk, and major adverse cardiac events

<table>
<thead>
<tr>
<th>Study lead author</th>
<th>Death (in hospital)</th>
<th>Death post discharge (median follow-up in months)</th>
<th>Recurrent AMI post discharge</th>
<th>Recurrent de Novo SCAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tweet³</td>
<td>1/189</td>
<td>3/189 (27)</td>
<td>37/189 (19%)</td>
<td>29/189 (15.3%)</td>
</tr>
<tr>
<td>Lettieri⁵</td>
<td>3/134</td>
<td>4/127 (22)</td>
<td>2/127 (1.5%)</td>
<td>6/127 (4.7%)</td>
</tr>
<tr>
<td>Rogowski⁷</td>
<td>1/64</td>
<td>0/64 (54)</td>
<td>3/64 (4.6%)</td>
<td>3/64 (4.7%)</td>
</tr>
<tr>
<td>Nakashima⁸</td>
<td>0/63</td>
<td>1/63 (34)</td>
<td>18/63 (28.5%)</td>
<td>7/63* (11.1%)</td>
</tr>
<tr>
<td>Saw¹⁹</td>
<td>0/327</td>
<td>4/327 (37)</td>
<td>55/327 (16.8%)</td>
<td>34/327 (10.4%)</td>
</tr>
</tbody>
</table>
FMD and SCAD
Long term issues

- Post spontaneous coronary artery dissection chest pain and its management (invasive angiography should be reserved for patients with hard evidence of ischaemia or myocardial necrosis)
- Cardiac rehabilitation and exercise
- Post traumatic stress disorder and the emotional and psychological consequences of SCAD