Spontaneous Coronary Artery Dissection (SCAD) in Women

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Facility Presenter Disclosure

Cardiology for the Non-Cardiologist
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- All scientific research related to, reported or used in this CME activity in support or justification of patient care recommendations conforms to the generally accepted standards.

- Clinical medicine is based in evidence that is accepted within the profession.
Overview

- Introduction
- Pathophysiology
- Epidemiology
- Clinical presentation and diagnosis
- Management
- Prognosis
- Pregnancy-related SCAD (P-SCAD)
- Fibromuscular dysplasia (FMD)
- Conclusions
Introduction

- Non-atherosclerotic cause of ACS.

- Important to recognize - *patient characteristics* and *management* differ substantially from typical ACS.
  - Vast majority are women (~90%)
  - Often younger
  - Without or few cardiovascular risk factors
  - Results of revascularization are suboptimal
  - Conservative management is preferred option
Pathophysiology

• Sudden disruption of the coronary artery wall.
  • Separation of intima from vessel wall.
  • Trigger either intimal tear ("inside-out") or bleeding from vasa vasorum ("outside-in") resulting in an intramural hematoma.

• Expansion of hematoma causes propagation of dissection.

• Fragile arterial walls with no atheroma or calcification to limit propagation of dissection.
  • More extensive dissections.
  • Non-affected coronary artery segments appear normal on angiography.
Pathophysiology: sudden disruption of the coronary artery wall
Epidemiology

- True incidence unknown – under diagnosed.
- 2 - 4% of all ACS.
- Important cause of ACS in *young* women.
- Many cases unexplained.

Common identified predisposing factors:
- Pregnancy/Postpartum
- Fibromuscular Dysplasia (FMD)
- Connective tissue disease
- Hormone therapy
Epidemiology

- Potential triggers:
  - Isometric or extreme physical exertion
  - Intense emotional stress
  - Sympathomimetic drugs (e.g. cocaine, amphetamines)
  - Child birth
  - Valsalva (e.g. coughing, retching, vomiting)

- Triggers thought to increase coronary wall shear stress.

- Previously considered primarily a disease of young adults.
  - Described in patients 18–84 years.
  - Mean age in large contemporary series range from 44 to 53 years.

- No ethnic variations reported but there is a strong female predominance.
Key Message

• SCAD has been described across a broad demographic.

• Frequent cause of ACS in young to middle aged women and patients with myocardial infarction in pregnancy or post-partum.
Clinical Presentation and Diagnosis

• Most present with chest pain.
  • Most have elevated hs-TnT.
  • STEMI in 25-55% and NSTEMI in the rest.
  • Minority present with ventricular arrhythmias (3-10%).

Key Message:

• Patients with SCAD usually present with ACS.
• Delayed diagnosis is common - SCAD should be considered in differential of ACS presentations in low risk patients.
Clinical Presentation and Diagnosis

• Diagnosis of SCAD - coronary angiography.
  • LAD most frequently affected.
  • Multivessel dissections common.
  • Dissections more common in mid to distal segments.

• Patients with SCAD have more fragile coronary artery walls.
  • Meticulous technique to avoid catheter-induced dissection and minimize risk of dissection propagation.
Management

- Conservative management preferred in stable patients with SCAD.
  - Most heal spontaneously
- Medical therapy is based upon opinion - no randomized clinical trials.
- Initial treatment similar to standard ACS:
  - Dual antiplatelet therapy
  - Heparin
  - Beta-blockers

Thrombolytics contraindicated - increased risk of bleeding and extension of intramural hematoma.
Management

- **Dual antiplatelet therapy - aspirin and clopidogrel generally accepted but controversial.**
  - Optimal duration of DAPT and subsequent monotherapy with ASA?
  - More potent antiplatelet agents such as ticagrelor and prasugrel not recommended.

- **Heparin** – same concerns about potential adverse impact.

- **Statins** are important for ACS treatment in patients with atheroma, benefit in SCAD unknown and **not recommended**.

- **Beta-blockers recommended.**
  - Potential to reduce arterial shear stress.
  - Facilitate healing?
  - Possibly reduce long-term recurrence?
Management

- **Indications for revascularization:**
  - Complete vessel occlusion
  - Left main involvement
  - *Ongoing ischemia*
  - Hemodynamic instability
  - Sustained ventricular arrhythmias

- **PCI preferred revascularization strategy** but associated with significant challenges.

- **Technical difficulties include:**
  - *Wiring* true lumen
  - Dissection/hematoma extension.
  - Stent placement can result in hematoma propagation and loss of flow.
Management

- Conservative approach to stent implantation preferred.
  - Distal dissections left untreated if good flow.
  - Drug-eluting stents typically used.
  - Increased risk of stent malposition following reabsorption of intramural hematoma.
    - May predispose to late stent thrombosis.
Management

- **CABG in SCAD is generally used as a bail-out strategy.**
  - Left main dissection.
  - PCI unsuccessful or not technically feasible.
  - Rate of emergency CABG for PCI failure is significant.

- In-hospital mortality <2% following CABG for SCAD.

- Follow-up angiographic studies show high rates of graft occlusion.
  - Competitive flow or technical difficulties with distal graft anastomosis?
2 single-centre registries have provided important data on natural history and prognosis of SCAD.

1. Canadian SCAD registry of 164 patients (mean age 52, 92% women)
   - 80% treated conservatively initially.
   - Elective coronary angiography (>26 d) - all spontaneous healing.
   - 33 underwent PCI
     - Complete success in 36.4%.
     - > 50% had procedural extension of dissection.
   - 6 underwent CABG - 3 for failed PCI.
   - No in-hospital mortalities.
   - 2-year MACE rate 10-17%.
2. Registry of 189 patients (mean age 44, 92% women)
   • 94 treated conservatively initially
   • 10% required intervention at a mean of 4 days after initial admission.
   • PCI failure rate 53% - underscoring suboptimal results of PCI.

• **Long-term survival was excellent in both series.**

• **Significant morbidity in both series.**
  • Major adverse cardiac events (MACE) significant.
  • Primarily driven by recurrent SCAD events; average rate 5% per year.
Prognosis

Key points from contemporary series:

• Excellent in-hospital and long-term survival.
• Prolonged inpatient monitoring (up to 1 week) due to risk of recurrent events.
  • In contrast to atherosclerotic ACS - guidelines emphasize early intervention and discharge approach.
• Suboptimal results of PCI.
• Significant risk of future SCAD events.
• Recurrent chest pain after SCAD is common; often cyclical (usually premenstrual), requires careful assessment.
• No effective preventive treatment to reduce long-term risk.
Pregnancy-related SCAD (P-SCAD)

- AMI during pregnancy is uncommon.
  - ~25% of pregnancy and 50% of post-partum coronary events reportedly due to SCAD.

- P-SCAD accounts for ~10% of all SCAD.
  - SCAD should no longer be considered primarily a peripartum condition.

- Canadian Cohort Study; incidence of 1.8 SCAD per 100,000 pregnancies.
  - P-SCAD presentation more severe than SCAD outside of pregnancy; STEMI (64%), cardiogenic shock (24%), cardiac arrest (14%) and maternal death (4.5%).
  - P-SCAD more likely to involve proximal coronaries.
  - P-SCAD associated with worse post infarct left ventricular dysfunction compared to non-P-SCAD.
Pregnancy-related SCAD (P-SCAD)

- Cases tend to occur within 6 weeks of delivery – early postpartum peak.
  - Reported during early pregnancy but most are during third trimester.
  - Also late (6 weeks to 12 months) and very late (12 to 24 months) postpartum, especially in patients breastfeeding.
- Factors that may be associated with increase risk:
  - Multi-parity - structural changes cumulative.
  - Fertility hormones
  - Pre-eclampsia
- Physiology of P-SCAD:
  - Hormonal changes - may influence vascular connective tissue and/or vessel microvasculature.
  - Increased cardiac output and circulatory volume.
  - Acute hemodynamic stress of childbirth.
Pregnancy-related SCAD (P-SCAD)

- Patients should be screened for connective tissue disease and chronic inflammatory conditions.
- Risk of recurrence is significant.

**Key Message:**
Women of reproductive age with a history of SCAD should be carefully counselled regarding risk of recurrent events and more severe P-SCAD phenotype.
Fibromuscular Dysplasia (FMD)

- FMD is a non-atherosclerotic, non-inflammatory disease of arterial walls.
  - Abnormal thickening of vessel wall causing stenosis and aneurysm formation.
  - Can involve all arterial beds.
  - Most are women.
  - Cause unknown.
  - Diagnosis made angiography.

- Classic finding is multifocal disease - "string of beads" appearance.

- Fibromuscular ridges causing arterial stenosis alternating with arterial dilatation.
- Focal disease is less common and results in localized tubular narrowing.
Fibromuscular Dysplasia (FMD)

- First reported association with SCAD - 2005 by Vancouver group – case series of 7 women.
- Same group published results for 50 patients.
  - All screened for FMD
  - FMD in 86%
  - High pick-up rate of FMD may have been due to the frequent use of invasive angiography.
    - CT and MRA are less sensitive than invasive angiography.
- High prevalence FMD in SCAD in these series.
  - Prevalence of SCAD in US FMD registry is very low; <3%.
  - Difficult to see how will alter current management unless there is specific treatment for FMD or difference in prognosis.
Conclusions

• SCAD is an important cause of ACS.

• Pathophysiology and treatment are different compared to ACS caused by atherosclerosis.

• Early angiography should be considered in patients presenting with ACS but at low risk of atherosclerosis; in particular young to middle aged women.

• Clinically stable patients with good coronary flow, a conservative management strategy is recommended.

• Left main involvement, complete vessel occlusion, ongoing ischemia or hemodynamic instability require coronary revascularization.

• PCI results are suboptimal.
Conclusions

• Women of reproductive age with a history of SCAD should be carefully counselled regarding risk of recurrent events and more severe P-SCAD phenotype.

• Patients should be screened for FMD.

• Long-term prognosis is excellent.

• Risk of recurrent SCAD events is significant.
Thank you
SCAD – CTA and parallel angiography at presentation and 3 months (healing)
SCAD – stenting leads to proximal hematoma migration – further stents required.