57 year old male with chest pain

- No prior history CAD
- No risk factors
- Active, excellent functional capacity

- 8/14/14
  - Presents to ANW ER with 1 hour constant chest pain
  - Started at rest
  - Significant stress recently, otherwise feeling well
- Tn 7.1
- CBC, BMP, LFT's unremarkable
• Diffuse mid to distal stenosis
• No change with 100mcg IC NTG
• “Findings most consistent with arteritis”

• DDx
  • Vasculitis
  • Diffuse vasospasm
  • Diffuse dissection
  • Atherosclerotic CAD

• ASA / Plavix / Statin
• Nifedipine / Isosorbide
• Vasculitis panel
  • Rheumatology / vascular medicine consults

• Further Hx from wife: ‘severe’ stress, not sleeping
  • Utox negative

• No symptoms suggestive of inflammatory disorder
• Negative ANA / ANCA
• CRP 5.1 (minimally elevated), ESR normal
- Vascular surgery
  - “Likely vasculitis, not c/w spasm”

- Rheumatology
  - “Not likely vasculitis”

- CV Surgery
  - “Atherosclerotic CAD”

- Cardiology
  - “Suspect vasospasm”
• Multiple discrete scattered areas of edema
  • Some confined to coronary territories
  • Others to mid myocardial segments sparing endocardium

  “Suggests non-coronary etiology such as myocarditis”

• Normal thoracic / abdominal aorta and branch vessels
• Repeat coronary angiogram 4 days later (on vasodilators)
  • Coronary anatomy unchanged
  • RV biopsied
    • No evidence of myocarditis

• Discharged HD 5

• Cardiology followup 10 days post discharge
  • Clinically doing well, cardiac rehab
  • On nifedipine, d/c’d isosorbide for headache
  • CCTA ordered
**Contemporary Update – Vasospastic Angina**

### Angina Pectoris’

1. A Variant Form of Angina Pectoris

**Preliminary Report**

*Monson Penningt*, M.D., *Renee Kenamer*, M.D., *Becky Miller*, M.D.,
*Talisha Wang*, M.D., and *Nina Fox*, M.D.

**Los Angeles, California**

*Classic Hibernation’s angina pectoris is a distinct syndrome with two major characteristics:* (1) the pain which is preceded by the onset of the pain is triggered by stress and (2) the patient has a prior history of coronary artery disease and angina. The classic form of angina is characterized by anginal pain which is present with physical exertion, usually in the morning or late at night. The patient often experiences exercise-induced chest pain.

### Table 1: Coronary Artery Vasospastic Disorders

Vasospastic angina diagnostic criteria elements:

1. 
   - **Menstrual-vessels:** During spontaneous episode, with at least one of the following:
     - Acute angina—especially between night and week morning
     - Marked diurnal variation in exercise tolerance—reduced in morning
     - Hypertension can precipitate an episode
     - Calcium channel blockers (but not β-blockers) suppress episodes

2. 
   - **Transient ischemic EEG changes:** During spontaneous episode, including one of the following:
     - ST segment elevation ≥0.1 mV
     - ST segment depression ≥-1 mV
     - No negative T waves

3. 
   - **Coronary angiography:** Defined as transient total or subtotal coronary artery occlusion (≥90% concomitant) with angina and ischemic EEG changes either spontaneously or in response to a provocative stimulus (typically exercise, ergon, or hyperventilation)

*Definition of vasospastic angina:* The diagnosis is made if typical responses to provocation tests are observed during spontaneous episodes and either transient ischemic EEG changes during the spontaneous episodes or coronary artery spasm criteria are fulfilled. Supraventricular vasospastic angina is diagnosed if nitroglycerin-responsive angina is elicited during spontaneous episodes for transient ischemic EEG changes are reproducible or unstable and coronary artery spasm criteria are met.

### Table 2: Indications for provocative coronary artery spasm testing

<table>
<thead>
<tr>
<th>Class I (prior indication)</th>
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<tbody>
<tr>
<td>- History suggestive of VSA without documented evidence, especially if</td>
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<tr>
<td>- Nitroglycerin-sensitive angina, and/or</td>
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<tr>
<td>- Marked diurnal variation in symptoms, exercise tolerance,</td>
</tr>
<tr>
<td>- Unexplained exercise with chest pain</td>
</tr>
<tr>
<td>- Recurrent rest angina following angiographically successful PCI</td>
</tr>
</tbody>
</table>

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*MINNEAPOLIS HEART INSTITUTE*
### Sensitivity and Specificity of Intracoronary Injection of Acetylcholine for the Induction of Coronary Artery Spasm

**Ken Okamura, MD, Hirofumi Yasue, MD, Koshi Matsumoto, MD, Kazuo Goto, MD, Hiroshi Miyage, MD, Hisao Ogasawara, MD, Shun-jiro Matsumoto, MD**

**Abstract**

Coronary artery spasm has been cited as a major cause of variant angina. To evaluate the sensitivity and specificity, intracoronary injection of acetylcholine (ACh) was performed in 75 patients with variant angina and angiography was performed in 48 patients with normal coronary arteries. The ACh-induced coronary spasm was defined as at least 75% or abrupt, severe, transient narrowing of the lumen. ACh was used in a concentration of 10-6 M. The mean heart rate was 80 beats/min and the mean systolic blood pressure was 120 mmHg. In patients with variant angina, ACh was injected for 5 minutes (40 microliter per minute) and the coronary lumen was observed by angiography. The lumen was normal in all cases. In patients with normal coronary arteries, ACh was injected for 2 minutes (40 microliter per minute). The coronary lumen was normal in all patients. Coronary artery spasm was induced in total 138 cases (28%) of patients with variant angina. ACh was used in a concentration of 10-6 M. The mean heart rate was 80 beats/min and the mean systolic blood pressure was 120 mmHg. In patients with normal coronary arteries, ACh was injected for 2 minutes (40 microliter per minute). The coronary lumen was normal in all patients.

**Provoking Coronary Vasospasm for Diagnosis of Variant Angina**

**Outdated Trick of the Trade or a Resurgent Diagnostic Modality?**

Raezal M. Ward, MD, Debra Fyler, MD, Ahmed A. Elgyalal, MD

**Table 1: Vasospasm as a ‘culprit’ of ACS?**

<table>
<thead>
<tr>
<th>Population</th>
<th>Vasospasm as a Culprit of ACS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caucasian</td>
<td>16%</td>
</tr>
<tr>
<td>Taiwanese</td>
<td>74%</td>
</tr>
</tbody>
</table>

**Table 2: Europeans Presenting with ACS and No Culprit**

- 488 angiographies
  - 138 (28%) no culprit
  - 86 = no other cause identified (stress CM, PE, etc)
  - Underwent graded IC ACH
  - 42 (49%) > 75% spasm (majority reproducing initial symptoms)
  - Tn elevation 10%

**3 Year Clinical Event Rate**

- 0% death or non-fatal MI
- 4% repeat angiography
- High rate of vasodilator therapy
• 2100 pts with chest pain and non-obstructive CAD
  • ‘Positive’: > 90% stenosis + EKG changes
  • ‘Intermediate’: 50-90%, +/- EKG
  • ‘Negative’: < 50%
    • Positive: 21%
      • 2 years: higher rates of adverse events (4% vs 1%)
        • 94% on CCB or long acting Ntg
      • Worse: multi-vessel spasm, current smoking, multiple episodes

Coronary Heart Disease

Clinical Usefulness, Angiographic Characteristics, and Safety Evaluation of Intracoronary Acetylcholine Provocation Testing Among 921 Consecutive White Patients With Unobstructed Coronary Arteries

(Circulation, 2014;129:1728-1738.)

• 139 patients with exertional angina
  • 54 yo, 77% female
  • 72% abnormal stress test
  • Angiogram: Non-obstructive CAD (<50% stenosis)

Comprehensive invasive assessment
  • Epicardial vessels
    • Functional evaluation w/ IC ACH (endothelial dysfunction = spasm > 20%)
    • Physiologic evaluation with FFR of LAD
    • Anatomic evaluation with IVUS of LAD
  • Microvasculature
    • Coronary flow reserve
• Endothelial dysfunction: 44%
• Microvascular dysfunction: 21%
• FFR < 0.8: 5%
• Myocardial bridging: 58%
  • Multiple findings: 54%

"The 'less than very good' outcomes of these patients must be recognized so that a near-normal angiogram does not drive diagnostic and therapeutic complacency"