Introduction

- Wellen’s syndrome describes a distinct pattern of subtle electrocardiographic abnormalities associated with significant proximal left anterior descending (LAD) coronary artery occlusion 1-3.
- The ischemia manifests as bicuspid or deeply inverted T waves in leads V2 and V3 with limited ST segment deviation 2,3.
- EKG changes are typically only present during pain-free intervals. Patients may have a normal EKG while symptomatic 4.
- Cardiac markers are often negative or only mildly elevated 2,3,6.
- Patients with Wellen’s syndrome require urgent cardiac catheterization as they are at risk for progression to a devastating anterolateral wall myocardial infarction (MI) 1,3.
- Additional diagnostic testing (such as exercise stress test) is not appropriate and will precipitate deterioration 1,4.
- Without rapid definitive treatment, 75% of patients will go on to develop an extensive ST segment elevation MI 7.

History

- 34-year-old male presented to the ED with 2 days of mild constant chest pain described as non-radiating left-sided tightness associated with mild dyspnea, worse when laying flat and unchanged with deep inspiration.
- The patient’s symptoms prevented him from sleeping the previous night.
- He was seen at an urgent care facility that morning and was referred to the ED for further evaluation.
- Initial EKG in triage was unchanged from urgent care with no ST abnormalities. Patient was instructed to remain in waiting room to await room placement and lab results.
- Approximately 2-3 hours later the patient was brought back to a room upon return of critical troponin level at 4.6 ng/mL.
- The patient’s symptoms had completely resolved upon repeat evaluation.
- Denied nausea, vomiting, palpitations, diaphoresis, hemoptyis, syncope, and back pain.

Physical Exam

- Vital signs: 146/82 / 76 bpm / 14 breaths/min / 98.3F / 98% RA
- Well appearing Caucasian male resting comfortably on stretcher in no acute distress, no diaphoresis
- No JVD
- Lungs clear to auscultation. No wheezes, rales, or rhonchi.
- Regular rate and rhythm, no murmurs or rubs. Clear S1, S2 without splitting. Chest wall non-tender.
- Peripheral pulses equal and 2+ bilaterally. No lower extremity edema.
- Abdomen soft, non-tender non-distended. No palpable masses.

Surgical Hx:

- None

Family Hx:

- No family hx of cardiac disease

Social History:

- Intermittent use of intranasal cocaine, last use 2 days ago
- ½ PPD-current smoker
- Denied use of alcohol, marijuana, or other illicit substances

Initial Diagnostic Testing

- Figure 1 shows repeat EKG during pain-free interval. There are biphasic T waves in V2-V5 with 1 mm ST elevation in V2 and 2 mm ST elevation in V2. No reciprocal changes
- Troponin 4.6 ng/mL (<0.04 ng/mL)
- Mild hypokalemia at 3.2
- AST elevated at 52
- CK, CK-MB, LDH, lipase, PT/INR otherwise within normal limits
- Portable chest x-ray unremarkable

Differential Diagnosis:

- Wellen’s Syndrome
- Cocaine-induced vasospasm
- GERD
- Electrolyte disturbance
- Pulmonary embolism
- Aortic dissection
- NSTEMI
- Unstable angina
- Pernicarditis
- Myocarditis

Management

- Consulting cardiologist was concerned the EKG changes were due to cocaine-induced vasospasm as opposed to an obstructing lesion.
- Emergent catheterization revealed 100% occlusion of proximal LAD.
- Patient did not have collateral from large posterior descending artery.
- Thrombectomy with placement of drug eluting stent to proximal LAD with TIMI-3 reperfusion.
- Serial troponins peaked at 11.84 five hours post PCI. No significant change in post PCI EKG.
- Moderately decreased EF 35%-40% with apical akinesis and mild mitral regurgitation on echocardiogram.
- Aspirin, Clopidogrel showed marked symmetric T wave inversions in V2-V5 (Figure 2).
- Patient remained hemodynamically stable without chest pain or SOB during hospitalization.
- Patient was started on aspirin, ticagrelor, atorvastatin, and metoprolol.
- Discharged POD#2 with outpatient cardiology follow up. Details regarding the patient’s care following hospital discharge are not known.

Discussion

- Deeply inverted T waves can be seen following thrombosis, supporting the idea that Wellen T waves may actually represent reperfusion of the LAD following a brief MI.

Conclusion

- Wellen’s syndrome can easily be missed in the emergency department setting, as subtle and transient T wave changes are sometimes the only marker of significant coronary thrombosis.
- Because the lesion is highly unstable and likely to re-occlude, it is important for clinicians to recognize this rare entity quickly and advocate for urgent catheterization.

References


Figure 1. EKG with biphasic T waves in V2-V5

Figure 2. EKG at discharge, two days s/p stent to LAD

Table 1. Initial diagnostic testing showing myocardial infarction (MI) associated with significant coronary artery occlusion.

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
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<tbody>
<tr>
<td>Troponin</td>
<td>4.6 ng/mL</td>
</tr>
<tr>
<td>CK-MB</td>
<td>Elevated</td>
</tr>
<tr>
<td>PT/INR</td>
<td>Normal</td>
</tr>
<tr>
<td>Electrolytes</td>
<td>Normal</td>
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Figure 2. EKG at discharge, two days s/p stent to LAD

Table 2. Differential diagnosis of Wellen’s syndrome.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Description</th>
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<tbody>
<tr>
<td>Wellen’s Syndrome</td>
<td>Acute coronary syndrome with diffuse T wave inversions</td>
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<tr>
<td>Cocaine-induced Vasospasm</td>
<td>Acute coronary syndrome with diffuse T wave inversions</td>
</tr>
<tr>
<td>GERD</td>
<td>Acute coronary syndrome with diffuse T wave inversions</td>
</tr>
<tr>
<td>Electrolyte Disturbance</td>
<td>Acute coronary syndrome with diffuse T wave inversions</td>
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Figure 3. EKG at discharge, two days s/p stent to LAD