



Unit 8, Case 29—ST Elevation in aVR

What is your interpretation of the EKG?
History/Clinical Picture—middle aged woman with ROSC from a PEA arrest.
Rate—~90
Rhythm—Sinus Rhythm
Axis—Normal
P Waves—normal
Q, R, S Waves—no pathologic Q waves, normal R wave progression
T Waves—Flattening III and aVL
U Waves—no pathologic U waves noted
PR Interval—normal
QRS Width—narrow
ST Segment—2mm of ST elevation in aVR, 1mm of ST elevation in V1. ST depression in V3-6, I, II, II, and aVF
QT Interval—not prolonged
Diagnosis: STE in aVR with diffuse STD consistent with diffuse sub-endocardial ischemia

Discussion: ST-elevation in aVR with diffuse ST-depression suggests diffuse sub-endocardial ischemia. Such a pattern has been associated with acute coronary syndrome in the setting of left main or proximal LAD insufficiency, severe triple vessel atherosclerotic disease, or any process that causes global cardiac ischemia (massive PE, severe anemia, shock, etc). Hypokalemia, defibrillation, and epinephrine administration are also associated with this pattern. Post cardiac arrest, one must evaluate whether the EKG findings are a result of the cardiac arrest itself (either global ischemia from poor perfusion or the resuscitative effort) or if the EKG signals ACS as the cause of the arrest. Cardiac arrest from myocardial infarction is often (though not always) the result of a ventricular dysrhythmia. Correct management of this patient includes hemodynamic stabilization, targeted temperature management, and investigation for the cause of the arrest. Serial troponins, serial EKGs, and early involvement of interventional cardiology are appropriate. If no alternate etiology for the arrest is found, the EKG has evolving ischemic changes, or the troponin rises abruptly, emergent cardiac catheterization may be indicated.

Resource Links: Life in the Fast Lane — great overview

Dr. Steve Smith's Blog – good case





Unit 8, Case 30—High Lateral MI

What is your interpretation of the EKG?
History/Clinical Picture— elderly woman with cardiac risk factors presenting with anginal chest pain
Rate—~100
Rhythm— sinus rhythm
Axis— left axis deviation
P Waves— normal
Q, R, S Waves— no pathologic q waves, normal to slightly early R-wave progression, S-waves III and aVF
T Waves— inferior TWI
U Waves— not seen
PR Interval— normal
QRS Width— narrow
ST Segment— < 0.5mm STE in I, 1.5mm in aVL, 1mm in V1, 1.5mm in V2; ST depression II, II, aVF, and V6</p>
QT Interval— normal

Diagnosis: High lateral ST Elevation

Discussion: This EKG, while not meeting classical STEMI criteria, is highly suggestive of an occluded coronary artery feeding the high lateral wall. In the context of a clinical presentation consistent with acute coronary syndrome (i.e. a patient with a "good story") this EKG is a highly concerning for an evolving STEMI and merits immediate discussion with interventional cardiology and likely cath lab activation. If the interventional cardiologist is reticent to take the patient directly to the cath lab, the ED team in consultation with cardiology should provide maximal anti-anginal therapy (dual antiplatelet load, heparin bolus and drip, nitroglycerin drip) and perform serial EKGs every 20 minutes. If the EKG evolves into a frank STEMI, or the patient has persistent chest pain after maximal medical therapy, the patient should go to the cath lab for emergent PCI.

Resource Links: Life in the Fast Lane – great overview



Unit 8, Case 30—High Lateral MI

An example of High Lateral STEMI



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Courtesy of Edward Burns of Life in the Fast Lane

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Unit 8, Case 31—De Winter's Complex (Anterior STEMI)

What is your interpretation of the EKG?

History/Clinical Picture — Middle aged man with a single known cardiac risk factor presenting with chest pain that started during exertion Rate—between 60 and 70 Rhythm— sinus rhythm Axis— normal P Waves— normal Q, R, S Waves— no pathologic q waves, large S wave in III, relative normal R wave progression T Waves— TWI in III, large peaked TW in V2-V5, large relative to the QRS complex U Waves— not seen PR Interval— normal QRS Width— normal ST Segment — ST segment depression in V2-V6 QT Interval – normal Diagnosis: De Winter's complex suggestive of acute LAD occlusion. A de Winter's complex is characterized by tall, peaked T waves following upsloping ST depression in the precordial leads.

What is the eponym for the morphology of the ST-T complex seen in this EKG? De Winter's T Waves

What does this ST-T morphology signify? Acute LAD occlusion versus subtotal occlusion

How should this patient be managed?

This EKG and clinical history are highly suggestive of acute LAD occlusion and should be considered a STEMI equivalent. Cath lab activation is the appropriate management. De Winter's T-waves are thought to be present in about 2% of acute LAD occlusions and are often under-recognized by clinicians. Some experts believe that a de Winter's pattern suggests severe subtotal occlusion (much like Wellen's waves) as opposed to total occlusion. Nevertheless, de Winter's waves in this clinical setting suggest unstable plaque in a proximal artery with a large infarct territory and proper management includes emergent cardiac catheterization with PCI.

Resource Links: Life in the Fast Lane — great overview

Dr. Steve Smith's Blog – good case

















Unit 8, Case 32—RV Infarct

What is your interpretation of the EKG?

History/Clinical Picture — patient with coronary disease risk factors and classical anginal pain

Rate— ~75

Rhythm— sinus rhythm

Axis- normal axis

P Waves— normal

- Q, R, S Waves— no pathologic q-waves
- T Waves— flattening and inversion anterolaterally

U Waves- not present

PR Interval – normal

QRS Width- normal

ST Segment— inferior STE with lead III>II, and ST depression in the anterior and lateral leads. STE is also seen in V4R, V5R, and V6R.

QT Interval—mildly prolonged ~471

Diagnosis: Inferior STEMI with right ventricular infarct

Management: Like any other STEMI, immediate cath lab activation is paramount. Infarction of the right ventricle leads to a highly preload dependent state, and nitrates can precipitate hypotension and should be avoided. Unlike most variants of post-MI cardiogenic shock, fluids are the appropriate initial therapy for hypotension in the setting of RV infarct. Be suspicious of RV infarct when an inferior STEMI has ST-elevation in lead III > lead II, or when there is ST-elevation in lead V1.

Resource Links: <u>Life in the Fast Lane</u> — great overview

Dr. Steve Smith's Blog – good case



Unit 8, Case 32—RV Infarct

<u>Right sided EKG</u>—remember only 0.5mm STE in a single lead rules in STEMI (except men < 30 =1mm)



