Session Number 304

“MI”MICS: IT MAY LOOK LIKE AN MI, AND ACT LIKE AN MI, BUT IS IT?

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Content Description

Early recognition of acute myocardial infarction (AMI) is vital to optimal treatment. The ability to quickly recognize the changes associated with infarction should be in the repertoire of all critical care nurses. However, early recognition of ECG changes associated with infarction is only part of the puzzle. It is just as important for nurses to be able to recognize when a patient is NOT having an acute myocardial infarction. It is those conditions that can mimic the ECG changes typical of infarction that can cause unnecessary invasive procedures, administration of unneeded medications and excess cost and length of stay in the hospital as well as the critical care units.

Presence of ST-segment elevation or depression, q-waves, and T-wave changes, while frequently indicative of ischemia, injury or infarction, are also present in disorders such as Wolff-Parkinson-White syndrome, pericarditis, Tako-Tsubo syndrome, and left ventricular hypertrophy as well as others. Being able to differentiate these processes from acute coronary syndromes is vital.

Please join us as we explore the classic changes in true acute myocardial infarction using actual case studies and real 12-lead ECGs. We will compare and contrast the classic ECG changes in acute MIs to those found in the various MI mimics, and discuss assessment findings that can help us better interpret the 12-lead ECG for MIs or their Mimic cohorts.

Learning Objectives
At the end of this session, the participant will be able to:

1. List factors that are usually present with acute MI
2. Identify conditions that can mimic acute MI on the 12-lead ECG
3. Describe how to identify an acute MI in specific mimic conditions

Summary of Key Points
1. Classic MI changes review
   A. ST segment changes
      1. Elevation
         i. Convex often merging with the T-wave
         ii. Significant if it is >1mm
         iii. Two or more contiguous precordial leads
         iv. Two or more adjacent limb leads

   Figure 1 -ST elevation  Figure 2 - ST depression

Henry Geiter, Jr, RN, CCRN-CMC  MI Mimos
2. Depression
   i. Subendocardial ischemia
3. A reliable indicator when correlated with clinical findings

B. T-wave changes
1. Hyperacute
   i. Increased amplitude and width
2. Inversions
   i. Follow ST-segment elevation in AMI
3. Non-STEMI

C. Q-waves
1. Pathological
   i. \( \geq 25\% \) of the height of accompanying R-wave
   ii. \( > 1 \) mm wide and \( > 1 \) mm in depth
   iii. Septal depolarization
   iv. Transmural death
2. Positional
   i. Lead III

II. Benign Early Repolarization (BER)
A. Causes
1. Normal finding
2. Young (especially black) males

B. ECG findings
1. ST-segment changes
   i. Concave
   ii. Elevation at the J-point
   iii. No reciprocal changes
   iv. May have no limb lead changes
2. T-wave changes
   i. Tall upright
   ii. Inversions
      a. Normal variant (black males)
3. Limb leads (if present)
   i. ST-segment - lead II > lead III
   ii. Reciprocal changes in aVR
4. Precordial leads
   i. Leads V3-V6

III. Pericarditis
A. Acute pericarditis
1. 5\% of ED patients admitted for C/P

B. Causes
1. Neoplastic
2. Autoimmune
3. Infections
4. Idiopathic

C. Clinical presentation
1. Chest pain
   i. Sudden onset
   ii. Pleuritic in nature
   iii. Worse on inspiration
   iv. Positional
   v. Can be difficult to differentiate from ischemic chest pain
2. Pericardial friction rub
3. Pericardial effusions
4. Cardiac enzymes
   i. Increase in MB fraction
   ii. Troponin
      a) Related to severity of inflammation

D. ECG findings
1. Diffuse ST-segment elevations
   i. Not found in aVR
   ii. Concave
   iii. Most common with TNI > 1.5mcg/L
2. Inflammation
   i. Epicardium
   ii. Pericardial sac
3. T-waves
   i. Low amplitude
   ii. Unlike early repolarization
4. Often increased heart rate

E. Four “typical” phases
1. ST-segment and PR-segment deviations
   i. Occurs in the first hours to days
   ii. Reciprocal ST-segment changes
      a) Depression in aVR and V₁
   iii. PR-segment deviation
      a) Atrial inflammation
      b) Elevation in aVR
      c) Depression in limb leads and left precordial leads
2. ST-segments and PR-segments normalize
3. T-wave inversions
4. ECG returns to normal

IV. Left bundle Branch Block

A. Causes
1. Previous MI
2. Hypertension
3. Cardiac Issues
4. Digitalis intoxication
5. Congenital defects
6. Hemochromatosis

B. ECG findings
1. QRS complex
   i. >0.12 seconds in complete LBBB
   ii. >0.10 seconds in incomplete LBBB
   iii. QS or rS in V₁ to V₄
   iv. Poor R-wave progression
   v. Large terminal R wave in I, aVL and V₆
   vi. Terminal S-wave in lead V₁
   vii. Left-axis deviation
2. ST-segment changes
   i. Elevation
   ii. Depression
3. T-wave changes
   i. Discordant with QRS in all leads

Figure 8 - PR elevation and ST depression

Figure 9 - Typical changes in LBBB. ST elevation in V₁, and ST depression in V₆
C. Ventricular pacing – single, right ventricular lead
   1. Associated with a LBBB pattern

V. Left ventricular hypertrophy

A. Causes
   1. Cardiomyopathy
   2. Hypertension
   3. Aortic valve abnormalities

B. Clinical presentation
   1. CHF
   2. Arrhythmias
   3. Displaced PMI
   4. Possible S₄ heart sound

C. ECG findings
   1. Increased QRS voltage
   2. Increased QRS duration
   3. Left axis deviation
   4. Horizontal or downsloping ST-segment
   5. T-wave inversions
      i. “Strain pattern”
   6. Left atrial abnormality
      i. M-shaped P waves
   7. Sokolow-Lyon indices
      i. (S-wave in V₁ + R-wave in V₅ or V₆) ≥ 3.5mV (35mm) AND
      ii. R-wave in aVL ≥ 1.1mV (11mm)

VI. Wolff-Parkinson-White

A. Causes
   1. Conduction abnormality
   2. Small genetic influence

B. Clinical presentation
   1. Low incidence of arrhythmias
      i. AVRT
      ii. Atrial fibrillation
      iii. Atrial flutter

C. ECG changes
   1. Delta waves
      i. Fusion of two signals
      ii. Accessory pathway
      iii. Normal AV nodal pathway

Figure 10 - Typical changes in LVH. ST depression and T-wave inversion in lateral leads

Figure 11 - Typical changes in WPW showing inferior pseudoinfarction. Positive delta wave in V-leads.
2. Short PR-interval
3. QRS duration is >0.12 sec.
4. Two types of QRS patterns
   i. Type A (Left Kent bundle – positive delta)
   ii. Type B (Right Kent bundle – negative delta)
5. Excacerbation with AV-nodal blockers

VII. Tako-Tsubo Cardiomyopathy
   A. Also known as: Stress-induced CMO, Broken Heart syndrome, Left ventricular apical ballooning
   B. Causes
      1. Extreme psychological stressor
      2. Extreme catecholamine release
      3. Microvascular coronary spasm (ischemia)
   C. Clinical presentation
      1. Clean coronaries
      2. Lethal arrhythmias
      3. Post-menopausal women
      4. LV apical ballooning on echo or in cath lab
      5. Diffuse wall motion abnormalities
      6. Rise in cardiac enzymes
   D. ECG changes
      1. ST-segment elevation (most common)
         i. Anterior leads
      2. ST-segment depression
      3. T-wave inversions
      4. Prolonged QT-interval

VIII. Brugada syndrome
   A. Causes
      1. Mutations in the sodium channel gene
      2. Gentic transmission
   B. Clinical presentation
      1. Predominantly found in males
      2. Ventricular arrhythmias
      3. Sudden near cardiac death
      4. Arrhythmias appear late in life
      5. Atrial fibrillation
      6. Arrhythmogenic right ventricular dysplasia
      7. Nocturnal arrhythmias and ECG changes
         i. Related to autonomic tone
      8. Can be induced by:
         i. Cocaine use and overdoses of
         ii. Neuroleptics
         iii. Cyclic antidepressants
         iv. EP studies
   C. ECG changes
      1. ST-segment elevation in V1 to V3
      2. Type I ‘coved’ - Convex ST-segment elevation ≥ 2mm followed by a negative T-wave
      3. Type II and III (“saddle back” type) - elevated ST segment with upright or biphasic T wave.
         i. Type 2 – ST segment is elevated ≥ 1 mm

Figure 12 A&B – (A) shows LV diastole, (B) shows LV systole with apical ballooning.

Figure 13 - Typical changes with the three major types of Brugada's syndrome.
ii. Type 3 – ST segment is elevated < 1 mm
   a. Lead placement
4. Long QT interval
5. Transient ECG changes are common

IX. Myocarditis
   A. Causes
      1. Usually viral
      2. Other infectious etiologies
      3. Autoimmune
      4. Cardiotoxins
      5. Hypersensitivity reaction
      6. Systemic disorders
   B. Clinical presentation
      1. Febrile
      2. “Aches and Pains”
   C. ECG findings
      1. T-wave inversions
      2. Small or absent Q-waves in V5 and V6
      3. Low voltage QRS
      4. “Strain pattern”
      5. Prolonged PR and QT segments

X. CNS disease
   A. Causes
      1. Trauma
      2. Infectious
   B. ECG findings
      1. Giant, negative T-waves
      2. Prolonged QT-interval
         i. Subarachnoid hemorrhage

XI. Dextrocardia
   A. Causes
      1. “dextro” = “right”
      2. Embryonic arrest
      3. Situs inversus
   B. Clinical Presentation
      1. Right -sided chest pain
      2. Displaced heart sounds and PMI
      3. Structurally normal
      4. Often incidental finding
   C. ECG Findings
      1. Negative P-wave in lead I and aVL
      2. Reversed R-wave Progression
      3. Net positive QRS in aVR

XII. Others
   A. Hypothermia
   B. Osborne Waves

Figure 14 - Deep symmetrical T-waves in SAH with increased ICP

Figure 15 – 12-lead ECG showing dextrocardia

Figure 16 – Chest X-ray showing dextrocardia
XIII. Recognizing AMI in the presence of mimics

A. Pseudonormalization

1. Causes
   i. AMI
   ii. Preexisting angina
   iii. Ischemia

2. Clinical presentation
   i. Preexisting T-wave inversion
   ii. Preexisting ST-segment elevation/depression
   iii. “Normal” ECG with chest pain

3. ECG changes
   i. When pain free
      a) Patients typical T-wave inversion
      b) Patients typical ST-segment elevation/depression
   ii. When having chest pain
      a) ECG changes of ischemia “normalize”

B. Identifying AMI in the presence of LBBB, pacemaker rhythms and ventricular rhythms

1. T-wave inversions

2. Sgarbossa criteria
   i. Concordant ST segment elevation of 1 mm or in any lead — score 5
   ii. Concordant ST segment depression of 1 mm or more in any lead from V1 to V3 — score 3
   iii. Discordant ST segment elevation of 5 mm or more — score 2

3. Score of 3 or more is 90% specific for AMI

4. QR complexes in leads I, V5, or V6, or in II, III, and aVF suggests underlying infarction

5. S waves in left leads with preexisting LBBB.

6. Cabrera’s sign
   i. Notching of S waves in the mid-precordial leads

7. Chapman’s sign
   i. Notching of a wide R wave in V5 or V6

BIBLIOGRAPHY


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