ECG in MI and Pseudoinfarction

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September 5, 2004 21:33



Woman less than 50 yo.

September 6, 2004 05:36



Consensus ECG Criteria for Infarction

Table 3. ECG Changes Indicative of Myocardial Ischemia That May Progress to MI

1. Patients with ST segment elevation:

New or presumed new ST segment elevation at the J point in two or more contiguous leads with the cut-off points $\geq 0.2 \text{ mV}$ in leads V_1 , V_2 , or V_3 and $\geq 0.1 \text{ mV}$ in other leads (contiguity in the frontal plane is defined by the lead sequence aVL, I, inverted aVR, II, aVF, III).

- 2. Patients without ST segment elevation:
 - a. ST segment depression
 - b. T wave abnormalities only

New or presumed new ST segment depression or T wave abnormalities, or both, should be observed in two or more contiguous leads. Also, new or presumed new symmetric inversion of T waves ≥ 1 mm should be present in at least two contiguous leads.

Alpert JS et al. JACC. 2000;36:959.

Consensus ECG Criteria for Infarction

Table 4. Electrocardiographic Changes in Established MI

Any Q wave in leads V₁ through V₃, Q wave > or = to 30 ms (0.03 s) in leads I, II, aVL, aVF, V₄, V₅, or V₆. (The Q wave changes must be present in any two contiguous leads, and be > or = to 1 mm in depth.)

Alpert JS et al. <u>JACC</u>. 2000;<u>36</u>:959.

Ischemia, Injury, Infarction

- Context = resting ECG
- <u>Ischemia</u>: T wave inversion in distribution of affected leads (does localize)
- <u>Injury</u>:
 - Subendocardial injury: ST segment depression (does not localize)
 - Subepicardial/transmural injury: ST segment elevation in affected leads (does localize)
- Infarction: Q wave formation in affected leads (does localize)



ECG-Ischemia





Ischemia, Injury, Infarction - 3

- Infarction irreversible loss of myocardium, replaced eventually by electrically silent collagen
 - ECG initial forces directed away from area of infarction, with abnormal Q wave in affected leads
 - Abnormal Q (Pathologic Q) 0.04 sec wide and depth of 25% of height of R wave in that lead



Ischemia, Injury, Infarction - 3

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Infarction Location

Posterior!!!



Lateral – diagonal branch of LAD Inferior – RCA or LCX Septal – LAD or septal perforator Anterior – Mid LAD Anterolateral – LAD with diagonal Mid Precordial, Anterior, Localized Anterior

Inferior Infarction



Anterior MI





ECG - Pericarditis

 Diffuse ST elevation
 PR segment depression
 No reciprocal changes except aVR



Causes of ST Segment Elevation

- Acute transmyocardial injury
- Hyperkalemia
- Pericarditis
- Normal variant, early repolarization
- Hypothermia (Osborn wave)
- Brugada Syndrome

Less common

- Pulmonary embolism and acute cor pulmonale
- Cardiac tumor
- Aortic dissection
- Post mitral valvuloplasty
- Pancreatitis and GB disease
- Myocarditis
- Septic shock
- Anaphylactic reaction
- Drug overdose (tricyclic, etc)

Time Course of Infarction

- Time course is highly affected by acute intervention ("interruption of the infarction")
- <u>Hyperacute T waves</u> minutes, generally less than 10 minutes, usually missed
- <u>ST elevation</u> onset in seconds to minutes, decreases markedly during the first 7-12 hours, usually gone in few days, represents aneurysm if present more than 8 weeks
- <u>Q wave</u> appears within 6-12 hours (9) and is usually permanent
- <u>T wave inversion</u> onset is after ST elevation and may persist several days, weeks, months, or years

Causes of Prominent Anterior Forces (R>S in V1)

- Normal variant in young adult
- RVH
- True posterior infarction
- Pulmonary disease with displacement of the heart
- WPW pattern ("type A")
- Duchenne's muscular dystrophy
- Lead misplacement
- Dextrocardia

Arrhythmia in Inferior Wall MI

- Usually bradycardia, sinus bradycardia (SA node may be affected, also vagal effect)
- Possible ventricular tachyarrhythmias
- First degree AV block or second degree Mobitz I (Wenckebach) AV block or third degree AV block with narrow QRS escape rhythm (AV Node branch is from the RCA or the LCX); AV block is usually transient and resolves spontaneously

Arrhythmia in Anterior Wall MI

- Sinus tachycardia (MI is large resulting in poor stroke volume so increased HR to maintain cardiac output)
- Ventricular tachyarrhythmias
- Conduction abnormalities are usually below the AV node in the bundle branches
- Second degree AV block may be Mobitz II
- AV block usually has a wide escape rhythm and is usually permanent

RV Myocardial Infarction

- Cause: proximal RCA lesion
- Hemodynamics: low BP, elevated JVP, clear lungs (RV can't get blood across to the LV), may have severe hypotension with NTG, RA pressure equal or greater than PAW
- ECG indicators: ST elevation in V1 greater than V2, or RV4 ST elevation of at least 1 mm

Inferior MI, Naming the Artery

- Simple inferior MI RCA
- Inferior MI with RV involvement RCA
- Inferior MI with posterior involvement LCX
- Inferior MI with lateral involvement LCX

Clinical Presentation of MI in Different Vessel Involvement

- <u>LAD</u>: large MIs, anterior lateral septal inferoapical and proximal bundle branches
- <u>RCA</u>: sinus node in 55%, RV, AVN, posteromedial pap muscle, inferior LV and variably posterior and lateral; vagal activation, sinus bradycardia, RV involvement, papillary muscle dysfunction and MI
- <u>LCX</u>: posterior and variably inferior and lateral ... abnormalities in second half of the QRS complex and frequently cause underestimation of the area at risk and undertreatment of the patient

Localization of Anterior MI Lesion by ECG

- 100 pts, first anterior MI, tracing with most pronounced ST change before reperfusion therapy; ST elev >2mm in V2 and V3
- Excluded LBBB, prior CABG, LVH, prior cardiac surgery
- ST elev in aVR in 43% prox to S1 and 5% dist to S1, 0.4 (0.2-1.8mm)

Engelen DJ et al (Wellens). J Am Coll Cardiol. 1999;34:389

ECG in Anterior MI

- ST elevation in V2-4
- 40% are proximal to 1SP and D1
 - ST elevation in aVR and ST elev >2.5 mm in V1, ST depression in 2, 3, and F and maybe V5-6, and Q in aVL
- 40% are <u>distal to both</u>
 - Dominance of inferoapical area, absent ST depression and maybe elevation in 2, 3, and F, sometimes Qs in V4-6,
- 10% are proximal to D1 but not 1SP
 - Q in left lateral leads, ST depression in III, and no ST depression in II
- 10% are proximal to 1SP but not D1
 - ST elevation in aVR and >2.5 mm elevation in V1, and ST depression in V5, and maybe ST elevation in V3R, and ST depression in aVL, a very specific finding, also ST elevation in 2, 3, and F

Site of Occlusion of LAD

Criterion	Occlusion Site	Sens	Spec	PPA	NPA
CRBBB	Prox to S1	12	100	100	62
ST ↑ V1>2.5	Prox S1	12	100	100	61
ST ↑ aVR	Prox S1	43	95	86	70
ST↓V5	Prox S1	17	98	88	62
Q in L	Prox D1	44	85	67	69
ST ↓ II>1.0 mm	Prox S1/D1	34	98	93	68
Q V5	Dist S1	24	93	71	53
ST ↓ in L	Dist D1	22	95	87	46
No ST ↓ III	Dist S1/D1	41	95	92	53

Localizing LAD Lesion

Table 2. Electrocardiographic Predictors of Left Anterior Descending Coronary Artery (LAD) Occlusion Proximal to the First Septal Perforator (S1) and/or the First Diagonal Branch (D1)

	Predictors of LAD Occlusion Proximal to S1								
	Sens	Spec	PPV	NPV	LR	p-Value			
$ST \uparrow_{aVR}$	43	95	86	70	8.6	0.000			
$ST \downarrow_{II} \ge 1.0 \text{ mm}$	36	100	100	68		0.000			
$ST \downarrow_{III} \ge 1.0 \text{ mm}$	60	71	60	71	2.1	0.002			
$ST \downarrow_{III} \ge 2.5 \text{ mm}$	33	97	88	67	11.0	0.000			
$ST \downarrow_{aVF} \ge 1.0 \text{ mm}$	52	84	71	71	3.3	0.000			
$ST \downarrow_{aVF} \ge 2.0 \text{ mm}$	26	97	85	64	8.7	0.002			
cRBBB	14	100	100	62		0.004			
$ST \downarrow V_{V5}$	17	98	88	62	8.5	0.009			
$ST \uparrow_{V1} > 2.5 mm$	12	100	100	61		0.011			
	Predictors of LAD Occlusion Proximal to D1								
ST↓ _{II} ≥1.0 mm	34	98	93	68	17.0	0.000			
$ST \downarrow_{III} \ge 1.0 \text{ mm}$	66	75	64	76	2.6	0.000			
$ST \downarrow_{III} \ge 2.5 \text{ mm}$	32	95	81	67	6.4	0.001			
$ST \downarrow_{aVF} \ge 1.0 \text{ mm}$	54	85	71	72	3.6	0.000			
$ST \downarrow_{aVF} \ge 2.0 \text{ mm}$	27	97	85	66	9.0	0.001			
Q _{aVL}	44	85	67	69	2.9	0.002			

 $cRBBB = complete right bundle branch block; LR = likelihood-ratio; NPV = negative predictive value; PPV = positive predictive value; sens = sensitivity; spec = specificity; ST \downarrow = ST-depression; ST \uparrow = ST-elevation; Q = abnormal Q-wave.$

Engelen DJ et al (Wellens). J Am Coll Cardiol. 1999;34:389

RCA vs LCX

- <u>RCA</u> STel in III>II (STdep aVL>I)
- <u>LCX</u> STel in II>III (I with ST 0 or elev)
- <u>STEL in V5-V6</u> of little value in differentiating, just larger area at risk
- <u>V4R</u>: Proximal RCA ST elev in V4R and upright T wave, distal RCA no ST elev and upright T wave, LCX no ST elev and inverted T wave (reliability requires significant ST elevation in inferior leads)
- <u>Anterior ST depression</u>: may extend V1-V6, if maximal in V4-6 likely 3-V dz and lower EF, absence indicates RCA and presence unhelpful to distinguish; maximal ST depression in V2-3 predicts LCX
- <u>Isolated RVMI</u>, minor changes inferiorly, ST elevation in V1-2 and V3-4R (I've also heard that if V1>V2, indicates RVMI)

CIRCUMFLEX CORONARY ARTERY MI RIGHT CORONARY ARTERY MI 111 ш

ST SEGMENT VECTOR IN RIGHT CORONARY ARTERY VS CIRCUMFLEX MYOCARDIAL INFARCTION

FIGURE 53-9 Schematic presentation of the ST-segment vector with inferoposterior MI caused by a right coronary artery (RCA) or circumflex coronary artery (CX). As shown, RCA occlusion leads to predominant ischemia in the inferoseptal area with an ST-segment vector pointing toward lead III. In CX occlusion, the ischemic area is located posterolaterally, resulting in an ST-segment vector directed toward lead II.



FIGURE 53-11 Diagram showing the coronary arteries and the possible sites of coronary artery occlusion leading to inferoposterior MI. In the right coronary artery (RCA), the occlusion may be before (proximal) the right ventricular (RV) branch or after it (distal). As shown in proximal RCA occlusion, the RV is involved in the MI.

VALUE OF ST-T SEGMENT CHANGES IN LEAD V4R IN ACUTE INFERO-POSTERIOR MYOCARDIAL INFARCTION



FIGURE 53-12 Characteristic ST-T-segment changes in lead V_{4R} in cases of proximal RCA, a distal RCA occlusion, or a CX occlusion (see text).

LCX Occlusion

- 84 patients: 2 normal, 35 with Q waves, 43 true posterior MI, 2 ST-T abnormalities, 2 LBBB
- Inferior Q: peripheral stenoses
- Lateral Q: central stenoses
- True posterior, both central and peripheral

Surawicz, B. Ch. 8, 2001

Conduction Problems

- AVN delay in inferior MI and proximal RCA, frequently with RV involvement
- RBBB +/- AFB indicates proximal LAD
- AFB in Inferior MI indicates LAD disease

LMCA disease

- Severe hemodynamic deterioration
- Subtotal with collateral from RCA is more common as UA with marked ST depression in I, II, and V4-V6 and ST elevation in aVR
- Total LMCA obstruction aVR with ST elevation >0.05 mV in 88% as opposed to LAD 43% or RCA 8%, and higher amplitude 0.16mV

Atrial infarction

- Atrial repolarization: elevation in I, II, III, V5 or V6, or depression in precordial leads
- May be seen in 10% inferoposterior MI
- Indicates proximal occlusion of RCA or LCX
- Complication atrial fibrillation or flutter, MAR, sinus arrest, atrial rupture (rare)

Caveats

- Many exceptions
- Most reliable with first MI
- Impaired with multivessel disease, prior MI, collateral circulation, LBBB, preexcitation and paced rhythms

Pseudo-Infarction - 1

- Loss of Viable Myocardium: Duchenne-type muscular dystrophy, inferoposterior MI pattern but less wide Q waves, also Friedrich's ataxia
- <u>Altered distribution of myocardial mass</u>: HCM, LVH, RVH
- Pulmonary embolism and acute cor pulmonale
- Hyperkalemia
- Q waves in PVCs

Surawicz, B et al. ECG in clinical practice

Pseudo-Infarction - 2

- <u>Altered sequence of depolarization</u> or abnormal cardiac position:
 - stocky people inferior MI
 - poor R progression in
 - R to L septal depolarization: incomplete LBBB, LVH, dextrocardia, "corrected" TGA
 - Inferior deviation of initial QRS force (LAFB, thin persons)
 - Downward displacement of origin of initial QRS (emphysema)
 - Position of electrodes (posterior pericardial effusion, high electrodes)
 - Pseudo-Q wave with perpendicular orientation of initial QRS deflection to the lead axis
 - Pneumothorax, scoliosis

Surawicz, B et al. ECG in clinical practice



Rhythm sinus with irregularities

Dramatic ST elevation in inferior and lateral precordial leads Significant ST depression in right precordial leads Early (marked ST change and no T evolution, not much Q wave)

Acute inferoposterolateral MI



Sinus rhythm, normal PR interval Insignificant inferior Q waves Diffuse T wave abnormality Significant anterior T wave inversion In the setting of unstable angina, this finding is anterior ischemia These "Wellens T waves" indicate LAD disease



Sinus rhythm, rate near 100

Anterior MI, old, with R wave decrement from V2 to V3 Possible anterolateral involvement, because R waves in V4-6 are small

Q in III is inconsequential if in isolation, but here has a Q in aVF Association of localized anterior and inferior may indicate "apical"



Sinus rhythm rate about 80

Significant Qs in II, III, and aVF, with a little ST elevation and With no T wave inversion, but also With prolonged QT interval I guess this inferior MI is probably acute or recent but

I'd want good clinical correlation



Sinus tachycardia rate 150...(be sure it is not atrial flutter) Significant diffuse ST depression in 9 leads Subendocardial ischemia No infarction Q waves Does not look like hypertrophy



Sinus rhythm, one PAC, very acute inferoposterolateral MI

Practice 6B



RBBB pattern has developed – is this rate-related or VT? I find no P waves, rate about 130

Practice 6C



Back to sinus, still very early, but ST elevation is less

Practice 6D



Now later with inferior Q waves and T wave inversion The Peaked T in V2 is an analogy of posterior T inversion



Acute anterolateral MI, old enough for Q waves But T waves still upright and ST segments still up somewhat

Practice 7B



Slightly later, with now some evolutionary terminal T inversion Notice ST elevation looks worse, due to double-standard



Sinus rhythm with inferior MI and RBBB An unusual combination, usually pre-existent RBBB



Another acute anterolateral MI, notice ST elevation in I and aVL

Practice 9B



Slightly later, notice the evolutionary T inversion Especially in V4 and V5



Acute anterolateral MI and RBBB and leftward axis Also sinus rhythm with frequent supraventricular ectopy T inversion in V1 is from the RBBB and not the MI

175

Practice 10B



Later, loss of RBBB and with IVCD – QRS looks completely different Is there 2:1 AV block? Anterior MI and RBBB is risk for AV block! Complete heart block with ventricular escape?

V5



Sometimes sick patients make for hard tracings Inferior ST elevation and V1 ST elevation too

Practice 11B



In inferior MI, when ST elevation in V1 is greater than V2, There is RV MI and not anterior injury



Acute inferior MI

Notice the chest leads have reverse progression, they are right chest leads ST elevation in V4R indicates RV myocardial infarction

Practice 12B



Inferior MI one hour later, now left chest leads Complete heart block with narrow junctional escape





Sinus tachycardia, diffuse ST segment elevation Pericarditis

But Q in V2-3 already happening and ST elevation also

Practice 15B

3 years later, there are Qs inferiorly as well as anteriorly Persistent ST and T abnormality

Wellens T wave

Practice 17B

Same patient with Wellens T wave

Close to hyperacute inferior T waves, but some ST elevation Also reciprocal change in aVL and some elevation in V6

