Supplement

Common Errors in ECG Diagnosis of Coronary Artery Disease

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Abstract

A variety of clinical conditions not associated with ischemic heart disease can have ECG features mimicking those of ischemic etiology. Some of these entities are: ventricular hypertrophy, intraventricular conduction defects, pre-excitation syndrome, pericarditis, electrolyte disturbances, etc. This can lead to erroneous diagnosis and uncalled for hazardous treatment. A careful comprehensive evaluation with in depth analysis of the ECG can overcome such errors.

We have reviewed some of these conditions and elaborated upon the differentiating features. ©

Introduction

Conditions other than coronary artery disease (CAD) may lead to ST – T changes mimicking those found in CAD, often leading to erroneous diagnosis and unnecessary treatment. Of patients with chest pain and ST segment elevation, 171 of 202 patients (85%) in one study[1] and 63 of 123 (51%) in another study[2] had diagnosis other than infarction after detailed investigations.

Similarly Sharkey *et al*[3] and Khoury *et al*[4] reported that 11% and 5.7% patients treated with thrombolytic therapy on the basis of ECG changes infact did not have infarction. Thus it is imperative that the distinctive differentiating factors of several of these conditions should be kept in mind in order to avoid such errors.

In this brief review we discuss the various conditions, which often lead to erroneous diagnosis of ischemic heart disease.

A. Pulmonary Diseases

1. Chronic Obstructive Pulmonary Disease (COPD) and Corpulmonale: In patients with COPD and corpulmonale, poor 'r' wave progression, loss of R waves in right sided and mid precordial leads may mimic anterior wall myocardial infarction.[5] Abnormal Q waves may appear in inferior leads to suggest inferior wall myocardial infarction (IWMI). These are usually secondary to vertical displacement of the heart secondary to low lying flattened diaphragms and the intervention of hyper inflated lungs.[6] If recordings are taken one intercostal space lower the morphology of the QRS forces is partially normalized. Other clues to non ischemic etiology are associated P pulmonale, rightward displacement of QRS and T wave axes, low voltage of QRS complexes in limb leads, $S_1 S_2 S_3$ pattern and large S waves in $V_5 – V_6$.

2. Spontaneous Pneumothorax: Due to combined effects of cardiac displacement, rotation and interposition of air between the heart and chest wall symmetrical T wave inversion, loss of R wave in precordial leads along with reduced QRS voltage may be seen. Careful clinical and radiological examination may point towards the correct diagnosis. The lost R wave amplitude may be regained after lung expansion.

3. Pulmonary Embolism (PE): Following acute PE abnormal Q waves may appear in leads III and aVF suggesting IWMI. These are secondary to acute right ventricular dilatation with clockwise rotation of the heart along its longitudinal axis. The ventricular septum (IVS) becomes so oriented such that right septal surface is directed superiorly. Hence Q waves are recorded in III and aVF. However sharply in contrast to classical inferior wall myocardial infarction Q waves are not seen in lead II. Also acute right ventricular ischemia may lead to ECG changes similar to anterior wall injury patterns.

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B. Myocardial Diseases

1. Primary myocardial disease: Hypertrophic cardiomyopathy may lead to pseudoinfarction patterns with Q waves in leads $V_2 – V_6$ and leads II, III, aVF (in 20 – 50% of patients)[7]. These are secondary to intense hypertrophy, because of which the initial left to right septal vector is exaggerated and recorded as prominent Q in left sided chest leads. Even dilated CMP many present with poor R wave progression, anterior Q waves and ST – T wave changes when there is extensive myocardial fibrosis.

Apical HCM may present with deep symmetrical T wave inversion (Fig. 1) in anterior leads and may be mistaken for ischemia, even when the condition itself is of a benign nature.

2. Secondary myocardial diseases: Several forms of myocarditis, neuromuscular and neurological disorders such as progressive muscular dystrophy, Freidriechs ataxia, scleroderma, amyloidosis and primary and metastatic tumors of the heart may present with pseudoinfarction patterns. Myocardial fibrosis or electrically inert myocardial tissue may produce such patterns.

C. Conduction Abnormalities

1. Left bundle branch block (LBBB): LBBB may be characterized by small or no R waves in right precordial leads. QS deflections may extend from leads $V_1 – V_4$ or reversal of R wave progression may occur. The secondary ST elevation in right chest leads may falsely suggest

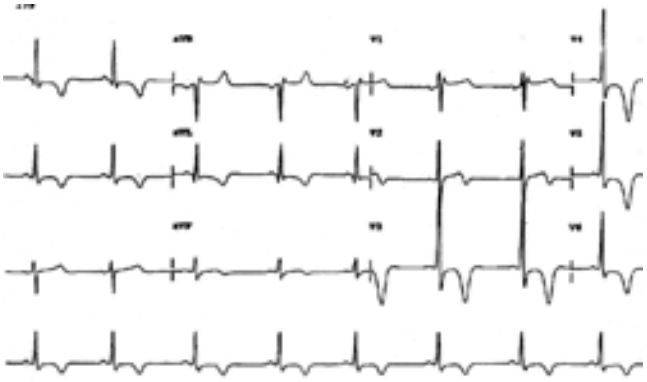

Fig. 1 : Apical HCM deep symmetrical T wave inversions seen in leads I, aVL, $V_4 – V_6$.

an acute infarction. QS defections may be seen in leads II, III and aVF. Hence it is essential that one be aware of the features of myocardial infarction in presence of underlying left bundle branch block. The criterion for the same is shown in Table 1.[8]

Points for each criterion met an added. Total scores ≥ 3 yields 90% specificity and an 88% positive predictive value.

2. Left anterior hemiblock (LAHB): LAHB is characterized by left axis deviation; small Q wave in lead I and rS in leads II and III. Small Q waves may be recorded in right and mid precordial leads. In presence of LAHB the early activation of the septal portion supplied by the left posterior fascicle results in the vector being displaced inferiorly such that they are oriented to the negative side of the lead axes of the right precordial leads and hence Q waves recorded in the same. When placed one intercostal space below QRS deflections in leads V_1 and V_2 are replaced by RS deflections. Also in presence of acute inferior wall myocardial infarction the initial small r waves cause by LAFB in II, III and aVF mask the Q waves of infarction.

3. Ventricular Preexcitation Syndrome (PES): In type A tracts the delta waves may be downward in I, aVL resembling lateral wall myocardial infarction. In type B tracts initial deflection in leads II, III, aVF or leads $V_1 – V_2$ may mislead one to interpret an old inferior (Fig. 2) or anterior wall myocardial infarction. The short PR interval may be the clue to the diagnosis of accessory bypass tract. The differential diagnosis may be difficult when PR interval is normal as is seen in PES variants.

D. Intracranial Causes

Cerebrovascular events, IC bleeds especially SAH may lead to ST elevation, depression, large, wide upright or inverted T waves with QT prolongation (also c/d CVA "T" wave pattern) and prominent Q waves. Connor reported focal myocytolysis in 8% patients who died of SAH and had such T wave changes.[9] This myocytolysis is due to excessive sympathetic stimulation mediated via the hypothalamus.

E. Left ventricular hypertrophy (LVH)

In LVH poor 'R' wave progression, loss of septal R wave in right to mid precordial leads and QS complexes in $V_1 – V_2$ may be seen (rarely extend beyond V_3). ST elevation and tall T waves may suggest anterior infarction. ST depression in the lateral chest leads referred to as LVH with strain pattern may be mistaken for ischemia (Fig. 3). Features in favor of LVH are

Table 1 : Scoring system for diagnosis of AMI in presence of left bundle branch block

	Criterion	Point Score
1.	ST segment elevation ≥ 1mm concordant with QRS	5
2.	ST segment depression ≥ 1 mm in leads $V_1 – V_3$.	3
3.	ST elevation ≥ 5 mm discordant with QRS	2

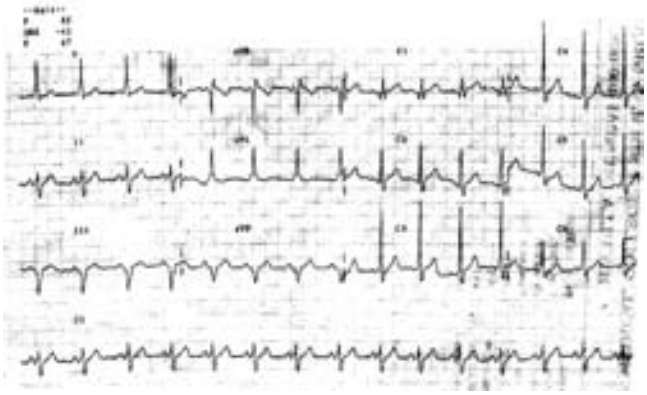

Fig. 2 : Preexcitation syndrome QS pattern in III, aVF resembles IWMI. Short PR interval and delta waves in $V_4 – V_6$ are strong clues to correct diagnosis.

1. The concave upwards initial portion of the ST – T segment compared to the flattened or convex pattern as seen in acute myocardial infarction.
2. Persistence of changes as opposed to the dynamic changes seen in AMI

F. Hyperkalemia

Can cause striking ST elevation. Other ECG features that favor Hyperkalemia are wide QRS, tall, pointed and tented T waves (Fig. 4) and low amplitude or no P waves. In contrast to myocardial infarction where ST segment has a plateau or a shoulder or is up sloping, in Hyperkalemia the elevated ST is down sloping.

G. Normal variants

Include three patterns.[10] These are:

1. Normal male pattern: - Seen in 90% of healthy young men. The concave upwards ST elevation usually is 1 –3 mm and most marked in V_2.
2. Early repolarization: - Seen in young black men. The ST elevation is 1-4 mm, most marked in V_4 with notching at J point. Tall upright T waves may be seen (Fig. 5). There may be reciprocal ST depression in aVR, not in aVL, when limb leads are involved.
3. ST elevation of normal variant: - Seen in V_3 through V_5 with inverted T waves, short QT and high QRS voltage. Usually considered a combination of early repolarization and persistent juvenile T wave pattern.

H. Acute pericarditis

ECG changes may be confused with acute ischemia. The various

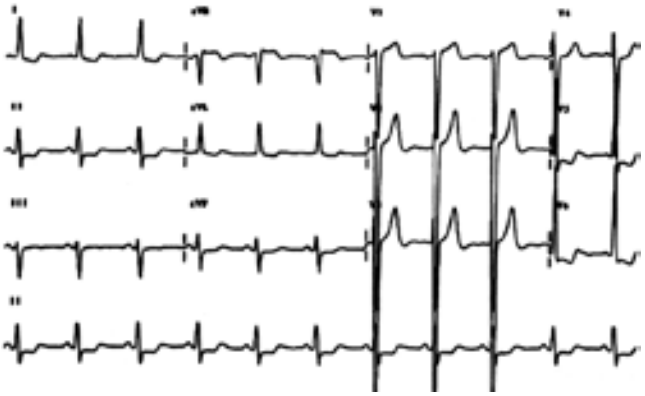

Fig. 3 : LVH with strain pattern. Note the ST depression in I, aVL, $V_5 – V_6$ and tall T in $V_2 – V_3$.

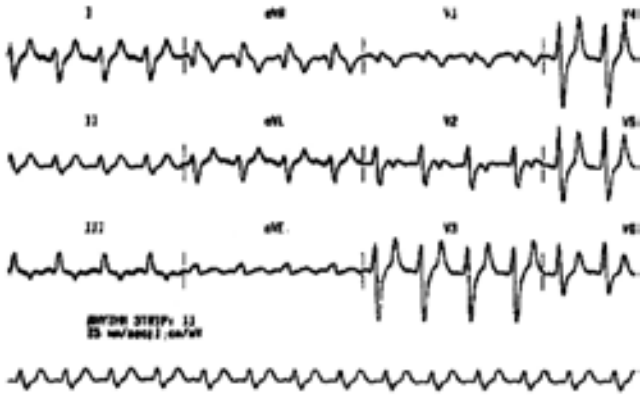

Fig. 4 : Hyperkalemia tall T waves seen in $V_3 – V_5$ may be mistaken for hyperacute anterior wall myocardial infarction.

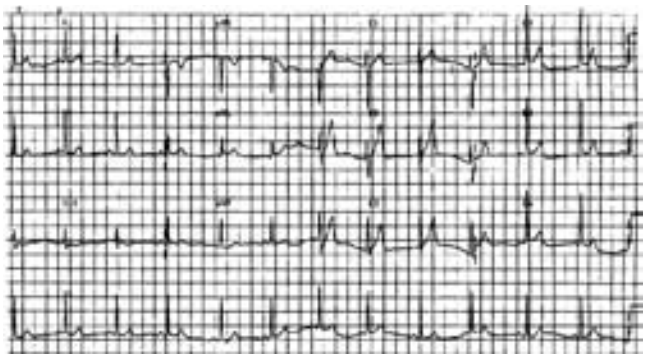

Fig. 5 : Early repolarization flow. Note ST elevation in V_4, J point notching and tall T waves.

differences are as showing Table 2.[11]

I. The Brugada syndrome

Brugada and Brugada in 1992 described a disorder linked to mutations in cardiac sodium channel gene characterized by right bundle branch block and ST elevation in right precordial leads in the absence of QT prolongation and high risk of ventricular fibrillation. The ST elevation limited to leads V1 – V2 and ST segment typically begins from the top of the 'R' wave, is down sloping and ends with an inverted T wave. This is very district from myocardial infarction patterns.

Table 2 : ECG features differentiating between acute pericarditis and acute ischemia

	Acute Pericarditis	Acute Ischemia
J- ST	Diffuse elevation, concave, no reciprocal depressions	Localized deviation usually convex
PR depression	Common	Uncommon
Q waves	None unless infarction	Common
T waves	Inverted after J points return to baseline	Inverted while T is still elevated
Arrhythmia	None	Frequent
Conduction anomalies	None	Frequent

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