Clinical Significance of Conditions Presenting with ECG Changes Mimicking Acute Myocardial Infarction

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Abstract

The electrocardiogram (ECG) is the primary tool in the diagnosis of acute myocardial infarction (AMI). However, other clinical conditions, both cardiac and noncardiac originated pathologies, may result in ECG tracing of AMI. This may lead to an incorrect diagnosis, exposing the patients to unnecessary tests and potentially harmful therapeutic procedures. The aim of this report is to increase the still insufficient awareness of clinicians from multiple disciplines, regarding the different clinical syndromes, both cardiac and noncardiac, associated with ECG abnormalities mimicking AMI, to avoid unjustified thrombolytic therapy or intervention procedures. During a 9-year period, the data from six patients (five females, one male; mean age, 50 years [range, 18 to 78 years]) who were admitted to cardiac care unit (CCU) with transient ECG changes resembling AMI were recorded retrospectively. During this 9-year period, 5,400 patients were hospitalized in CCU: 1,350 patients were diagnosed as ST-elevation myocardial infarction (STEMI) and 4,050 patients were diagnosed as non-ST-elevation myocardial infarction (NSTEMI). Only two out of six patients had chest pain with ECG changes criteria suspicious of AMI. STEMI was suspected in four out of six patients. All patients, but one, had normal left ventricular (LV) function. One patient had transient LV dysfunction. All patients, but one, with perimyocarditis, had normal serum cardiac markers. In four out of six patients, who underwent coronary arteries imaging during hospitalization (by angiography or by CT scan), normal coronary arteries were documented. Two patients who underwent ambulatory cardiac CT scan imaging after being discharged from hospital documented patent coronary arteries (case no. 3), or some insignificant irregularities (case no. 4). The discharge diagnoses from CCU were as follows: postictal syndrome, pericarditis, hypothermia, stress-induced ("tako-tsubo") cardiomyopathy, anaphylactic reaction, and status of postchemotherapy. All patients experienced full recovery with normal ECG tracing. During the 5-year follow-up, all patients were alive, and cardiac morbidity was not reported. We conclude that both cardiac and noncardiac clinical syndromes may mimic AMI. Comprehensive clinical examination and profound medical history are crucial for making the correct diagnosis in conditions with ECG changes mimicking AMI.

Keywords

- ECG changes
- mimicking acute MI
- clinical conditions
- cardiac and noncardiac
- normal coronary arteries

Fig. 1 (A) Electrocardiogram in the emergency room demonstrating sinus rhythm and ST elevation in precordial and lateral leads. (B) Electrocardiogram 1 day after admission, demonstrating deep inverted T waves in inferior and lateral leads. (C) Electrocardiogram 8 days after admission with less deep negative T waves in inferior and lateral leads.

Fig. 2 ECG tracing of an 18-year-old girl with perimyocarditis and ST-segment elevation in inferior and lateral leads.

The surface electrocardiogram (ECG), in addition to the clinical presentation, physical examination, and laboratory data, is crucial for the early diagnosis and the correct treatment of acute myocardial infarction (AMI), either by percutaneous intervention (PCI) or by reperfusion thrombolysis. ECG changes mimicking an AMI, such as ST-segment elevation, T-wave inversion, and QRS morphology changes, have been reported in clinical conditions (cardiac and noncardiac) other than acute MI, where a different approach and therapy were needed.^{1,2}

Noncardiac conditions mimicking AMI are normal variants,² cerebrovascular disease,³ subarachnoid hemorrhage,⁴ postictal,⁵ esophageal rupture,⁶ acute pancreatitis,⁷ penetrating duodenal ulcer,⁸ catastrophic abdominal illness,⁹ left pneumothorax,¹⁰ pneumomediastinum,¹¹ hyperkalemia,¹ hypocalcemia¹² and hypercalcemia,¹³ pheochromocytoma,¹⁴ and pulmonary embolism.¹⁵

Transient cardiac contractile impairments (global and segmental with ECG changes) were documented after brain death,¹⁶ and in hypothermia, ST-segment elevations with a prominent J point ("Osborn wave") were described.^{17,18}

Cardiac conditions with ECG changes mimicking acute MI were described in early repolarization,² pericarditis,^{19,20} myocarditis²¹⁻²³ hypertrophic cardiomyopathy,²⁴ Brugada syndrome,²⁵ and stress-induced (apical ballooning) cardiomyopathy,²⁶⁻²⁹ and also in patients with left ventricular hypertrophy,² left bundle-branch block,² after direct current countershock to the precordium,² and in Prinzmetal angina.²

Purpose

The purpose of this report is to present six patients with different clinical conditions, cardiac and noncardiac, with ECG changes mimicking AMI.

Following the fact that many disciplines are involved in the interpretation of ECG, all of them should be aware of the possible false-positive diagnosis of STEMI. Thus, the purpose

is to increase the still insufficient awareness to this documented entity of noncoronary syndromes, with ECG abnormalities mimicking AMI, and consequently avoid unjustified intervention procedures, or therapy.

Methods and Patients

We present six patients with different clinical conditions and with ECG changes mimicking AMI.

Case 1: A 62-year-old woman who was admitted to cardiac care unit (CCU) after epileptic seizures, syncope, normal brain CT scan, and pathological electroencephalogram (EEG) pattern. The ECG was suggestive of evolving AMI (ST elevation in precordial leads [V₁₋₅] and QT interval prolongation [QT = 400 msec, QTc = 310 msec], converted the next day to T-wave inversion; ▶**Fig. 1**). Cardiac serum markers (such as protein: Troponin I) and coronary angiography were normal.

Case 2: An 18-year-old girl who suffered acute rheumatic perimyocarditis, repetitive ventricular fibrillation, and ECG changes mimicking AMI (▶**Fig. 2**), while coronary arteries were patent.

Case 3: A 35-year-old woman with schizophrenia, who was admitted to CCU with severe hypothermia and shock, bradycardia, and ST-T changes mimicking AMI (▶**Fig. 3**). Cardiac CT demonstrated normal coronary arteries.

Case 4: A 78-year-old woman, with known colon cancer, was admitted 6 days following 5FU chemotherapy, with ECG changes mimicking AMI, but without clinical or biochemical evidence of AMI (▶**Fig. 4**). Cardiac CT demonstrated patent coronary arteries with some irregularities.

Case 5: A 57-year-old man was admitted to CCU after anaphylactic shock, following a bee-sting that was treated with adrenaline and corticosteroids. The ECG demonstrated a transient ST elevation in the anterior wall (▶**Fig. 5A**) with no

Fig. 3 ECG tracing of a female patient with hypothermia in the emergency coronary care unit: (A) frontal leads and (B) precordial leads. J wave (Osborn wave) in diaphragmatic and anterolateral wall.

Fig. 4 ECG tracing after chemotherapy, demonstrating T-wave inversion in anterior and lateral leads.

clinical or biochemical evidence of AMI. Cardiac CT demonstrated normal coronary arteries (►Fig. 5B).

Case 6: A 48-year-old woman was admitted to CCU, following a few days of chest pain, exacerbated on the day of admission, as a result of the death of a first-circle family member. An ECG recorded inverted T waves in precordial and inferior leads (►Fig. 6A).

Coronary arteriography was normal. Echocardiogram and left ventriculogram documented apical ballooning, typical of stress-induced (“tako-tsubo”) cardiomyopathy (►Fig. 6B).

In summary, during a 9-year period, the data from six patients (five females and one male; mean age, 50 years [range, 18 to 78 years]) who were admitted to CCU with transient ECG changes resembling AMI were recorded retrospectively. During this 9-year period, 5,400 patients were hospitalized in CCU; 1,350 were diagnosed as ST-elevation myocardial infarction (STEMI); and 4,050 patients were diagnosed as non-ST-elevation myocardial infarction (NSTEMI). Only two out of six patients had chest pain with ECG changes criteria suspicious of AMI. STEMI was suspected in four out of six patients.

All patients, but one, had normal left ventricular (LV) function. One patient had transient LV dysfunction. All patients, but one, with perimyocarditis had normal serum cardiac markers. All six patients underwent coronary arteries imaging (through coronary angiography—cases 1, 2, and 6 [50%]—or through CT coronary angiography—cases 3, 4, and 5 [50%]). Patent coronary arteries were documented, with some insignificant irregularities (case no. 4).

The discharge diagnoses from CCU were postictal syndrome, pericarditis, hypothermia, stress-induced (“tako-

tsubo”) cardiomyopathy, anaphylactic reaction, and status of postchemotherapy. All patients experienced full recovery with normal ECG tracing. During the 5-year follow-up, all patients were alive, and cardiac morbidity was not reported.

Discussion

Despite the availability of newer diagnostic tests such as radionuclide imaging and echocardiography, the ECG remains of prime importance in the evaluation of patients with suspected ischemic heart disease. Recognition of the uses and limitations of this test is therefore essential. Different etiologies, mechanisms, including normal variants and pathologic abnormalities, which may simulate the ECG pattern of acute ischemia and AMI, are responsible for this issue. It concerns the appearance of Q wave or markedly diminished R wave³⁰ and repolarization changes in the ECG: ST-segment deviation (elevation or depression); increased T wave positivity (hyperacute T waves), seen also in hyperkalemia; prominent T-wave inversion seen also in electrolyte disturbances; and in long QT syndrome.^{1,2,30–33}

Multiple studies using animal models have documented the link between ventricular repolarization and depolarization of body surface ECG tracing with transmembrane electrical recordings, which have given rise to a pathophysiological basis and explanation of ECG changes, under different clinical conditions.³² Although most of the ECG abnormalities detected in patients in the emergency department or CCU are caused by primary cardiac diseases, ECG changes do not invariably imply, as described by us in our six patients, cardiac abnormality, or an acute MI.

Fig. 5 (A) ECG tracing after bee-sting and treatment with adrenaline and corticosteroids, demonstrating transient ST-segment elevation in precordial leads. (B) Cardiac CT demonstrating normal coronary arteries.

One of the mechanisms proposed for the clinical syndrome (and the ECG changes accompanying it), of stress-induced (tako-tsubo like) cardiomyopathy, and also in subarachnoid hemorrhage, is the activation of cardiac adrenoreceptors. By using an animal model, the researchers documented myocardial contraction band necrosis, as evidence of sympathetic discharge and its consequences.^{34–36} Others documented impaired endothelial function and vascular response to neural stress,^{33,37} which caused changes in repolarization patterns of the ECG, simulating acute ischemia or AMI. Others attributed this phenomenon to the transient QRS changes simulating AMI (such as Q wave, or markedly diminished R wave) even though no etiology could be determined. Transient conduction block of the septal fascicle of the left bundle,

in clinical settings with no myocardial infarction and with normal profiles of cardiac markers, was also described as a possible mechanism for this observation.³² Different mechanisms have been proposed for the ECG changes (reduced R voltage and T-wave inversion), in other clinical settings such as iatrogenic left pneumothorax and even in emphysema. Rotation of the heart and interference with conductance of the electrical impulse, by retrosternal air, was also cited as a possible mechanism.^{10,32}

Summarizing our observations and the available reported clinical data, it is apparent that ECG changes mimicking AMI may exist in various clinical situations, cardiac and noncardiac, and may not always originate from an acute ischemia or AMI.

Fig. 6 (A) Electrocardiogram of a patient with apical ballooning ("tako-tsubo") cardiomyopathy. (B) The ventriculogram of a patient with an apical ballooning ("tako-tsubo") cardiomyopathy: (1) in systole; (2) in diastole.

Conclusions

We conclude that prompt and correct identification, based on clinical data and serial ECG, is crucial for making the correct diagnosis and treatment in patients with conditions that may still be confused with an AMI.

Although knowledge of ECG manifestation of noncardiac and cardiac conditions (other than AMI) commonly seen in critical care settings may result in rapid diagnosis and correct treatment of potentially life-threatening disorders, misinterpretation of the ECG and the clinical setting may expose the patient to wrong therapeutic options, with unwarranted risks.³⁸

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