ST Segment Elevation Besides Acute Myocardial Infarction

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Abstract:

ST segment elevation (STE) in electrocardiography (ECG) commonly denotes acute myocardial infarction (AMI)[1,2]. As AMI needs prompt diagnosis and specific treatment like thrombolysis, anticoagulation or primary percutaneous coronary intervention (PCI) which significantly improve outcome[1], recognition of other conditions where STE also occurs are equally important to avoid potential hazards. This review points out to such conditions of ECG tracing.

Key words: ST, elevation, myocardial infraction

Normal variant of ST segment elevation

STE is commonly observed in the electrocardiogram of normal persons (figure 1). A study done on 45 normal volunteers suggest that standard precordial leads do not accurately predict maximal torso potentials during the ST segment, thus STE may commonly be seen without acute myocardial infarction[3]. Another study conducted on 6014 healthy men in the U.S. Air Force who were 16 to 58 years old, 91% had STE of 1-3 mm in one or more precordial leads[4]. Other study by Surawicz and Parikh on 529 males and 544 females, age 5 to 96 years, showed variation of STE between normal male and female that is, prevalence of STE of at least 1 mm in one or more of leads V 1 through V 4 was 93% in the men who were 17 to 24 years old[5]. According to these studies, it is now established that the main feature of the male pattern in electrocardiograms (ECG) is ST elevation at the J-point of =0.1 mV in at least one of the leads V1-V4 with concave upward ST-segments and the prevalence of the male pattern in ECG of healthy male (mostly Caucasians or Blacks) who were 16-58 years old was high (85%)[4,5]. Much higher prevalence was observed in a recent study in Hong Kong (on 202 apparently healthy Chinese adult males, aged 20- 54 years) which was 95.5%[6].
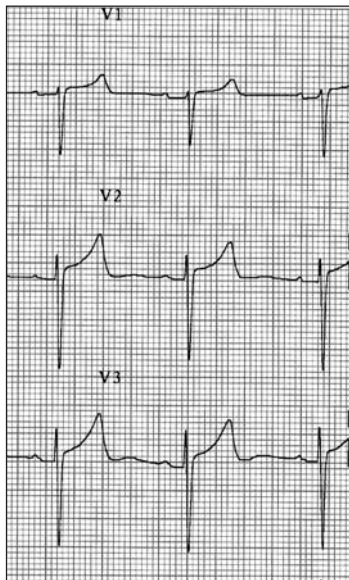
Early repolarization syndrome

The early repolarization syndrome (ERS) is a common electrocardiographic variant characterized by J-point elevation manifested either as QRS slurring (at the transition from the QRS segment to the ST segment) or notching (a positive defection inscribed on terminal S wave), STE with upper concavity and prominent T waves in at least 2 contiguous leads[7-10]. In most of the studies elevation of the J point and/or ST- segment from the baseline by at least 0.1 mV was considered definitive of ER[9,11] (figure 2).

ERS is found in approximately 1% to 2% of the young adult population and in up to 13% to 48% of patients presenting with chest pain in the emergency rooms and coronary care units. It generally occurs in the absence of myocardial disease[12,13]. Its predominance is in young black males and shows disproportionate infrequency in white females[14]. This syndrome is common in people under the age of 30 years old and shows decreasing incidence with
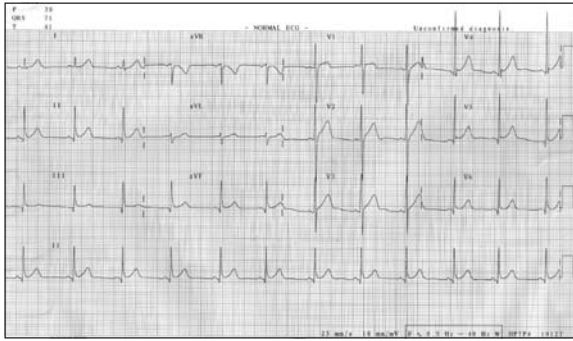

Fig.-1: *ST segment elevation (in V2 & V3) in a normal healthy person*

Fig.-2: *Early repolarization syndrome*

advancing age[15]. It is predominant in more athletically active people and more infrequent in sedentary[16]. There is a higher prevalence of ERS in individuals with spinal cord injury at levels of injury that can disrupt central sympathetic command of the heart (at the C5 - C6 levels) with high vagal tone and loss of sympathetic tone[17].

Acute pericarditis
Acute pericarditis is a diffuse inflammation of the pericardial sac and superficial epicardium from a multitude of infectious and inflammatory processes. This inflammation results in a current of myocardial injury resulting from the epicardial irritation manifested by a number of electrocardiographic findings. Classically, the electrocardiographic changes have been described as an evolution through several distinct stages involving STE with PR segment depression, normalization of the ST segment abnormality with T wave inversion, and eventual normalization of the electrocardiogram over a period of days to several weeks[18].

The changes in initial stage may be confused with findings of MI or early repolarization; old ECGs help differentiate among these conditions. It is important to remember that no PR-segment depression occurs in MI and no reciprocal changes or Q waves are found in the 12-lead ECG during acute pericarditis (figure 3), which is an important feature in distinguishing acute pericarditis from acute MI[19-22]. In addition, the STE in acute infarction is upwardly convex in concordant leads, and Q waves often appear. The most reliable differential finding in the ECG is the ratio of the magnitude of the STE to the T-wave amplitude in the V6 lead; acute pericarditis is more likely when the ratio is greater than 0.25[23,24].

Left ventricular aneurysm
The persistence of the typical pattern of the fully evolved phase of myocardial infarction – Q wave, STE and inverted T wave – for 3 months or longer after the acute attack
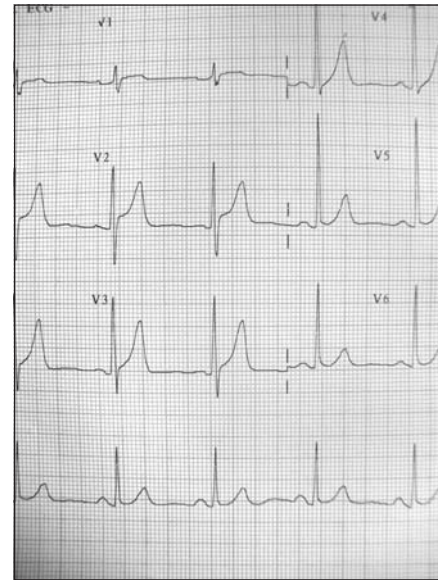

Fig.-3: *ECG from a patient with acute pericarditis. Note the PR depression with mild ST elevation*

suggests the development of a ventricular aneurysm[25]. Left ventricular aneurysm (LVA), also described as dyskinetic left ventricular segment, is defined as a localized area of infarcted myocardium that bulges outward during both systole and diastole. LVAs most often are noted after large anterior wall events but may also be encountered status after inferior and posterior wall injuries. In most cases, LVA is manifested in ECG by varying degrees of STE, which may be difficult to distinguish from ST segment changes caused by acute myocardial infarction. The STE is generally associated with well-developed, completed Q waves in the anterior precordial leads, and there will not be reciprocal ST depression in the contralateral leads[26].

Vasilomanolakis et al reviewed ECG of 77 patients underwent left ventricular aneurysmectomy and observed, post-operatively, significant decreases in ST segment elevation ($p < 0.001$), with anterior ($p < 0.03$) and inferior R wave heights ($p < 0.002$). In addition, abnormal Q waves disappeared in 50% of patients. This indicates that LVA affects ventricular repolarization as evidenced by a significant decrease in ST elevation and left ventricular aneurysmectomy affects ventricular depolarization as evidenced by a significant loss of R wave height and changes in infarct pattern[27].

Left bundle branch block
Left bundle branch block (LBBB) is the result of the delay or interruption of conduction within the left bundle branch. The ST segment and T wave are opposite in direction to the terminal QRS deflection. These are secondary

phenomena, ie they occur secondarily to the abnormal intraventricular conduction and do not indicate primary ST segment and T wave abnormality. In leads oriented to the right ventricle (V1 and V2) the ST segment is elevated and often minimally concave-upward and the T wave is asymmetrical. In leads oriented to the left ventricle (V5 and V6) the ST segment is depressed and often minimally convex-upward and the T wave is inverted with a blunt nadir[28] (figure 4).
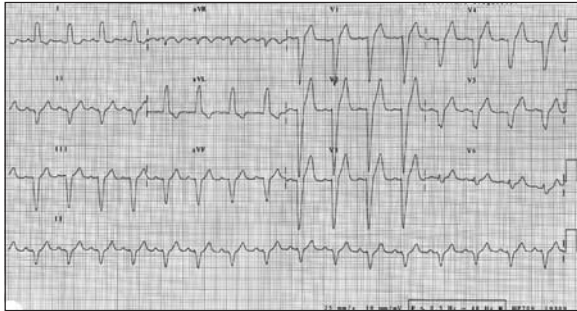

Fig.-4: *Left bundle branch block*

Making the diagnosis of AMI in the presence of LBBB can be problematic. However, the secondary ST–T changes in LBBB described above are shifted to the opposite direction from the major component of the QRS complex (i.e., discordant). When these changes are concordant, they are specific for acute myocardial infarction[29,30]. Another criterion that has been proposed for recognizing an associated anteroseptal infarct is ST-segment elevation of 5 mm or more[29].

Prinzmetal angina
Prinzmetal angina is used to describe recurrent episodes of ischaemic cardiac pain at rest accompanied by reversible STE without cardiac enzyme elevation. This ECG change reflects transmural ischaemia and indicates total or subtotal obstruction to blood flow through a major coronary artery. Although STE is a common finding during spasm, ST-segment depression and T-wave changes may also be observed during different ischaemic episodes in the same patient. These latter changes reflect lesser degrees of ischaemia suggesting varying severity of spasm at the same site or varying extent of involvement of the coronary artery tree by spasm[31,32]. The spasms are most commonly focal and can occur simultaneously in more than one site. Even coronary segments that are apparently normal at the coronary angiography, often show evidence of mural atherosclerosis at the intravascular ultrasound. This can result in localized endothelial dysfunction and coronary spasm[33,34].

The key to the diagnosis of prinzmetal angina is the documentation of the STE in a patient during transient chest discomfort (which usually occurs at rest, typically in the early morning hours, and is non-reproducible during exercise) and that resolves when the chest discomfort abates[32,35].

Left ventricular hypertrophy
Left ventricular hypertrophy (LVH) sometimes show STE in the right oriented leads with a slight upward concavity[36]. Brady et al conducted a study on 202 patients who presented with chest pain and STE showed 31 (15%) patients had STE acute myocardial infarction (AMI) as the final hospital diagnosis which caused the STE; 171 (85%) patients with STE had non-AMI diagnosis responsible for the STE, including LVH 51 (25%), LBBB 31 (15%), benign early repolarization (BER) 25 (12%), right bundle branch block 10 (5%), nonspecific bundle branch block 10 (5%), left ventricular aneurysm 5 (3%), acute pericarditis 2 (1%), ventricular paced rhythm 2 (1%), and undefined ST segment elevation 35 (17%)[37].

Brugada syndrome
Brugada syndrome (figure 5) is characterized by a specific electrocardiographic pattern in the right precordial leads and susceptibility to ventricular arrhythmias and sudden death[38]. Three different ECG patterns were described: a) type I, characterized by a coved-type ST-segment elevation eŠ 2 mm in more than one right precordial lead (V1-V3),
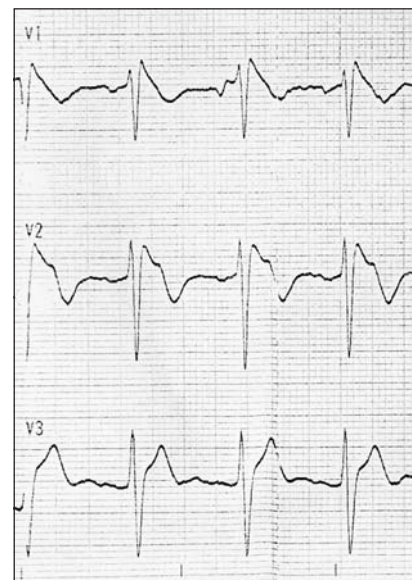

Fig.-5: *Brugada syndrome (type I). Note the characteristic coved-type ST-segment elevation in lead V1-V followed by negative T waves*

followed by negative T waves; b) type II, characterized by ST-segment elevation eŠ 2 mm in right precordial leads followed by positive or biphasic T waves, resulting in a saddleback configuration; and c) type III, defined as any of the 2 previous types if ST-segment elevation is dŠ 1 mm[39,40]. Although the 3 patterns can be observed in Brugada syndrome, and even in the same patient at different times, type I is the only one that is considered diagnostic of the disease[39].

Patients with Brugada syndrome usually remain asymptomatic. Nevertheless, it has been reported that 17% to 42% of them present syncope or SCD as a consequence of a ventricular arrhythmia at some time during their lives[41-44]. An ICD is the only treatment with demonstrated efficacy in Brugada syndrome. ICD implantation is recommended for all patients who have already had symptoms and for asymptomatic patients in whom the EPS induces ventricular arrhythmias, especially if they present a spontaneous type I ECG pattern. In the asymptomatic patients, without a family history of SCD and whose type I ECG pattern is only documented after the administration of sodium channel blockers, periodic follow up is recommended without the need of an EPS for risk stratification[40].

Pulmonary embolism
Pulmonary embolism is a common cardiovascular emergency, but it is still often misdiagnosed due to its unspecific clinical symptoms, so various models have been developed to help its diagnosis[45]. ECG changes are very unspecific and range from most common sinus tachycardia, rightward shift in QRS axis, complete or incomplete right bundle branch block, precordial T wave inversion, S1Q3T3 pattern and more uncommon ST segment elevation[46,47]. A few case reports describe ECG presentation of pulmonary embolism as STE in precordial leads, but exact mechanism is still unclear[47-49].

Electrical cardioversion: Transient elevation of the ST segment after electrical cardioversion has been very well reported in the literature, with an incidence as high as 16%. The mechanism of these ST-segment elevations is not clear[50]. Two main explanations have been suggested: myocardial injury or coronary vasospasm[51].

Hypercalcemia: Severe hypercalcaemia provoking ECG changes mimicking acute myocardial infarction is infrequently reported. STE is usually seen in leads V1–V3[52]. Nishi et al reported a case of hypercalcemia presented with STE which resolved after correction of hypercalcemia[53]. The ECG changes of severe hypercalcemia (>14 mg/dl) can mimic acute myocardial

infarction in two ways: First, calcium shortens phase 2 of the cardiac muscle action potential, leading to shortening of the ST segment. Thus the shortening of QaTc seen in hypercalcemia produces a high takeoff of the ST segment simulating acute myocardial ischemia. As the ST segment is difficult to measure however, QT intervals have been used to evaluate the ECG effects of hypercalcemia. Secondly, flattened or biphasic T waves are prominent in moderate to severe hypercalcemia[54,55].

In a situation in which emergency calcium assays are not available, the ability to diagnose hypercalcemia from the ECG is valuable, and a QTc interval of 0.27 second or less is a reliable indicator in differentiating hypercalcemia from true myocardial ischemia[56].

Hypertrophic cardiomyopathy
Hypertrophic cardiomyopathy (HCM) is a primary disease of myocardium resulting in myocardial hypertrophy without any inciting pressure or volume overload. The electrocardiographic features of hypertrophic cardiomyopathy are numerous, including ST segment elevation that may simulate other STE conditions. Several cases of HCM are reported having STE in their ECGs without AMI[57,58]. Chronic ST segment elevation has been occasionally described in patients with HCM complicated with apical necrosis and aneurysm formation, but not in uncomplicated cases of apical HCM[58].

Sickle cell anaemia
Sickle cell anaemia is associated with significant electrocardiographic abnormalities. A descriptive cross sectional study was done on 60 sickle cell anaemia patients by Oguanobi et al showed that non specific STE of 2 mm and T-wave inversion in the right precordial leads occurred in 10% and 13.33% of the patients respectively. Although it has been suggested that the ST – T wave abnormality might be an index of myocardial ischaemia, no clear evidence of myocardial infarction was demonstrated by this study and isolated cases of ST segment and T-wave changes were observed and there were no significant Q-wave abnormality were noted[59]. Aluko[60] and Sergeant[61] found ST segment and T-wave changes in 9% and 5% respectively of adult steady state patients. Racial differences may explain the slightly higher values in the study of Oguanobi et al when compared with the findings of Sergeant on Jamaican patients[61].

Conclusion:
Though STE most commonly is due to AMI, many other conditions can mimic this important electrocardiographic

feature. In patients who present with chest pain, STE is likely to be cardiac in origin and prompt recognition and treatment improves outcomes. However, unnecessary treatment with thrombolytic therapy or anticoagulation can be much harmful, and in patients who are at low risk of cardiac disease other causes must be ruled out. Pattern of STE in AMI is commonly convexity upward but in non-AMI it is usually somewhat depressed or concave along the ascending limb of the T wave[2]. Still, if confused, it is mandatory to confirm AMI by cardiac markers, echocardiography or coronary angiogram to avoid case fatality.

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