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**ECG CASE STUDY**

# Asymptomatic ST Segment Elevation

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Asymptomatic ST segment elevation in a life insurance applicant's ECG raises several prognostically important possibilities such as myocardial infarction, pericarditis, Brugada syndrome and early repolarization. This ECG case study discusses the ECG features involved in the differential diagnosis.

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## CASE SCENARIO

A 34-year-old male dentist is applying for $1,000,000 in whole life insurance. His past health is unremarkable. He works out 4 times a week utilizing a program of both aerobic and resistance exercise.

In the fall of 2002, he had a brief flu-like illness. This was associated with vague chest pain, which occurred intermittently over a 2-day period. His symptoms were not severe enough to interfere with his regular exercise program, and he did not seek medical attention. A year later, following an electrocardiogram (ECG) obtained as part of his annual medical check-up, his attending physician speculated that acute pericarditis might have been responsible for his chest pain in the fall of 2002.

At the time of his current application, he has no symptoms and his physical examination is normal. He is a nonsmoker. Both parents and 2 siblings are alive and well. Routine lab work is normal.

Figure 1 is the ECG obtained during his recent annual check up and was enclosed with the attending physician's statement. Is the ECG normal, or is there a specific ECG feature of concern? What is the differential diagnosis of this feature, and how should we decide the specific diagnosis in this applicant? Finally, does this specific diagnosis have any mortality implications of concern to the medical director who is assessing the long-term risk of a life insurance applicant?

## ECG INTERPRETATION

The prevailing rhythm is a sinus bradycardia with sinus arrhythmia and an average ventricular rate of 52 per minute. The PR interval is 0.25 second indicating mild 1° AV block. The QRS and QT intervals are normal. The QRS electrical axis in the frontal plane is normal (approximately +90°). Small Q waves are present in II, III and AVF. These are normal septal Q waves representing the vector of septal depolarization. In individuals with

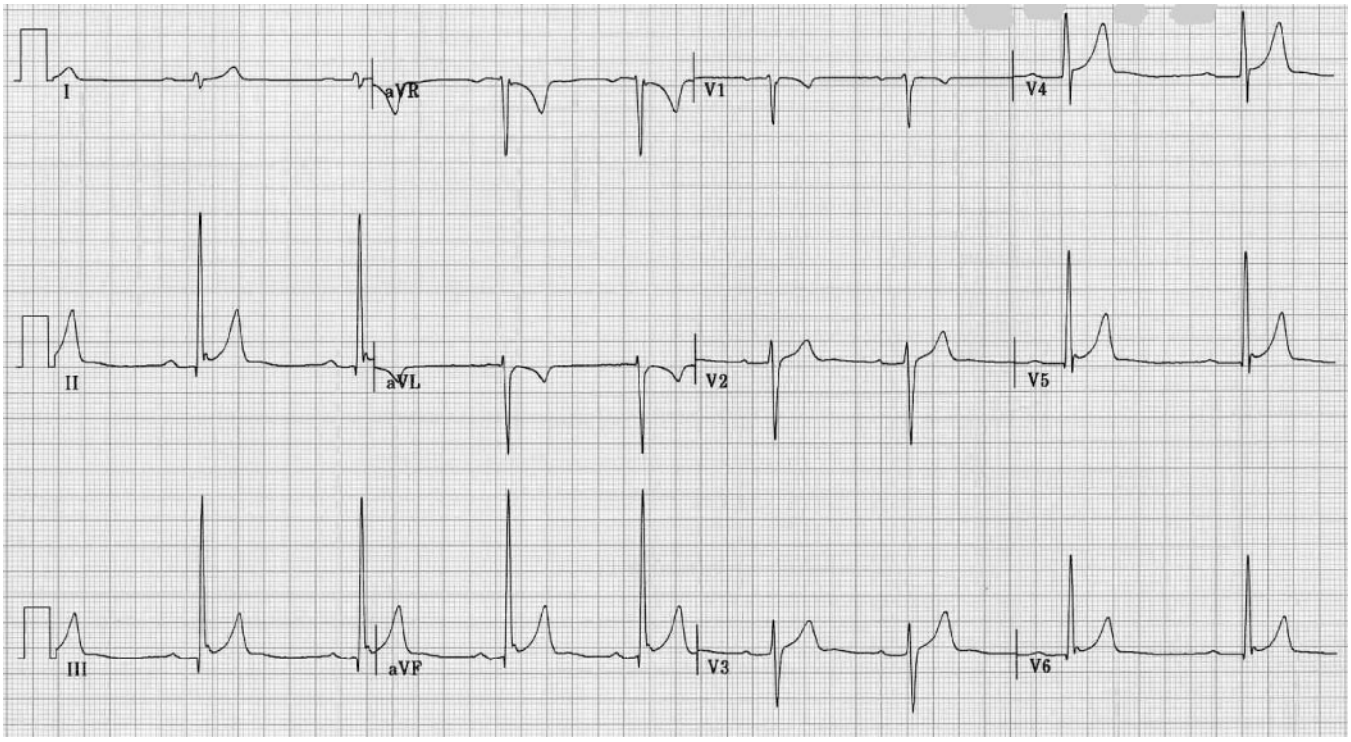


**Figure 1.** *Applicant's Electrocardiogram*

a vertical heart (such as our applicant), the normal initial septal forces are directed right and superiorly away from the inferior leads II, III and AVF. The QRS voltage is increased in II, III and AVF but is probably acceptable in an athletic and relatively young man in the absence of clinical context and other ECG features of left ventricular hypertrophy.

The ST segments are elevated within a concave upwards pattern in leads II, III, AVF, $V_2$–$V_6$. In these same leads, the J-point (which marks the end of the QRS complex and the beginning of the ST segment) is elevated and in leads II, III, AVF, $V_5$–$V_6$, there is a distinct notch (J wave). Upright T waves are noted in all the leads except AVR, AVL and $V_1$, where they are inverted. In leads II, III, AVF, $V_4$–$V_5$, the T waves are tall with asymmetric sides, a rounded peak and a wide base (vs the T waves of hyperkalemia, which are tall with a sharp peak, symmetric sides and a narrow base).

A small U wave is visible in most leads especially the precordial leads. It has the same polarity as the T wave and is less than one third the amplitude of the T wave with an ascending limb moving more rapidly than the descending limb. These are features of a normal U wave.

## ECG DIFFERENTIAL DIAGNOSIS

The main reason for concern and the one feature in this ECG, which poses both a diagnostic and prognostic challenge to the medical director, is the elevation of the ST segments. The presence of ST segment elevation raises several possibilities of prognostic importance including: myocardial infarction or transmural ischemia, ventricular aneurysm, acute pericarditis, Brugada syndrome and the early repolarization normal variant pattern.

The ECG in our applicant illustrates what is commonly referred to as an *early repolarization pattern.* This ECG pattern classically consists of a prominent notch or slur on the downsloping portion of the QRS complex, followed by a diffuse upward ST segment concavity concordant with the QRS and a positive T wave in the same lead.[1,2] The degree of ST segment elevation is usually less than 2 mm but may be as great as 5 mm. Another ECG feature that defines this ECG

phenomenon is the localization of the ECG pattern. Mid-to-lateral precordial leads ($V_2$–$V_5$) generally show the greatest ST segment elevation. Similar, but usually less prominent changes, may appear in the other leads (our case is an exception to this feature). It is rare for the ST elevations to be found only in the limb leads. Slower heart rates tend to increase the degree of ST elevation and the amplitude of the T waves. Fast heart rates (greater than 100 times per minute) have the opposite effect with normalization of ST segments and reduction of T wave amplitudes. This normalization of the ST segment elevation does not prevent the usual ECG manifestations of exercised-induced myocardial ischemia during exercise ECG stress testing.[3]

Additional ECG criteria for the early repolarization pattern include reciprocal ST depression in AVR and a waxing and waning of the ST segment elevation over time.[2]

In the differential diagnosis of ST segment elevation, the shape of the ST segment is often a more important factor to consider than the actual height of the ST segment above the baseline.

In acute myocardial infarction or transmural ischemia, the elevated ST segment usually has a coved or convex upward contour with an abrupt ''take off'' following the QRS complex.[4] In addition, because coronary artery disease is a focal disease involving only the myocardium subtended by the occluded culprit coronary artery, ST segment elevation is localized to the leads reflecting the area of infarction, and reciprocal ST depression is present in those leads opposite the area of infarction. Therefore, in addition to the absence of acute chest pain and lack of evidence of cocaine use in our applicant, the wide distribution of ST segment elevations, their upward concave contour and the lack of reciprocal ST depression, are points against the diagnosis of transmural ischemia, injury and infarction.[5,6] The absence of a past history of myocardial infarction and the lack of abnormal Q waves in our applicant makes the diagnosis of ventricular aneurysm untenable.[5]

Concave upward ST segment elevation involving nearly all leads could be compatible with an early stage of acute pericarditis in a clinical context. However, in acute pericarditis, the ST segment elevation is usually equal to or above 25% or more of the T wave amplitude, and the T waves are normal or low in amplitude. In acute pericarditis, the PR segment is depressed in all leads, except AVR and $V_1$, where the PR segment is often elevated due to subepicardial atrial injury.[5]

Like early repolarization, the Brugada syndrome has been identified predominantly in young, otherwise healthy, men. There is a predisposition to familial occurrence and transient normalization of the ECG manifestations of the Brugada syndrome may occur.[7] Two ECG features permit differentiation of the ECG signatures of the early repolarization pattern and the Brugada syndrome: the ST segment pattern itself and the lead localization.[2]

In the precordial leads, the elevated ST segment in individuals with early repolarization is usually localized to leads $V_2$–$V_5$ and is accompanied by a notched J point, an upward concavity of the elevated ST segment and a positive T wave. In contrast, the electrocardiogram of Brugada patients generally displays a prominent J point elevation, followed by a downsloping ST segment and negative T wave localized only to the right precordial leads ($V_1$–$V_3$).[2,6]

## DISCUSSION

Although not considered a marker of cardiovascular disease, the early repolarization pattern's traditional clinical significance relates to its potential to mimic the electrocardiographic patterns of acute myocardial infarction, pericarditis, ventricular aneurysm, hyperkalemia and hypothermia. Clinical interest in this pattern has been rekindled recently by 2 developments: 1) the important role of ST segment elevation in determining the therapeutic approach to acute myocardial infarction[11] and 2) the overlapping similarities of the early repolarization pattern with

the electrocardiographic manifestations of the highly arrhythmogenic Brugada syndrome.[2,12]

This distinctive pattern of ST segment elevation in the resting ECG of apparently healthy subjects was first described by Shipley and Halloran in 1936.[3] It has been called by a variety of names including ''unusual RT segment deviation,'' ''premature repolarization,'' ''normal RS-T elevation variant'' and ''early repolarization syndrome.''[8] The term early repolarization *syndrome* has been replaced by some authors[6] with the term early repolarization *pattern.* The rationale is that this ''abnormal'' ECG pattern does not represent organic disease of the heart, is not associated with symptoms, has no effect on longevity and does not require treatment.

The prevalence of this ECG pattern has been estimated between 1%–2% of healthy adults.[9] It is more commonly seen in young individuals, especially males and those who are athletic. Some studies have reported a higher prevalence in blacks, but this has not been confirmed in other studies. Familial occurrence of this ECG pattern has been suggested in some studies.[2] This distribution of the early repolarization pattern suggests both genetic and environmental factors. However, the pattern is not rare in women, older persons, whites and inactive persons.[8]

The benign nature of this early repolarization pattern has been established by several longitudinal follow-up studies, some as long as 26 years. The ST segment elevation often persists for decades but tends to decrease with increased age. All studies have consistently shown no evidence of increased likelihood of fatal or nonfatal cardiovascular events.[8,10]

In conclusion, asymptomatic ST segment elevation raises several prognostically important possibilities for the medical director to consider. By following the traditional adage of interpreting the ECG in light of the clinical setting and paying close attention to the details of the ST segment, conditions with a benign prognosis can be easily separated from those with a less favourable prognosis.

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