

Tall R Wave in Lead V₁

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Tall R waves in lead V₁ present the life insurance company medical director with a diagnostic dilemma. This ECG pattern may be present in applicants with right bundle branch block, right ventricular hypertrophy, Wolff-Parkinson-White syndrome, posterior myocardial infarction, hypertrophic cardiomyopathy, muscular dystrophy, dextrocardia, misplaced precordial leads, as well as in normal individuals. This ECG case study discusses the ECG features involved in the differential diagnosis.

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Key words: Electrocardiography, prognosis, differential diagnosis.

Received: June 3, 2004

Accepted: June 3, 2004

A 53-year-old female accountant is applying for life insurance. Her past health includes gestational diabetes 25 years ago and a hysterectomy for benign uterine bleeding. Five years prior to her application, she stopped smoking (10 cigarettes per day) and gained 20 pounds in weight. At the time of her application, she was asymptomatic. Both parents have Type 2 diabetes. Two younger siblings are alive and well.

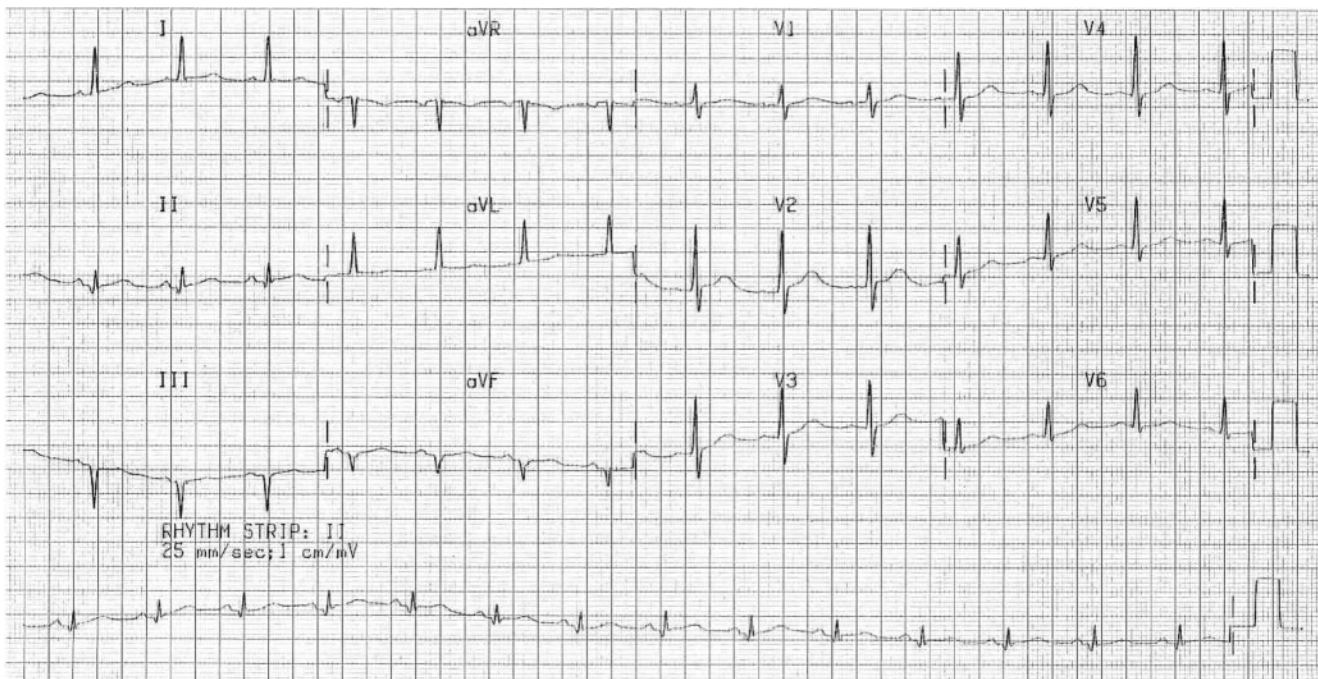
She is 5'5" tall and weighs 156 pounds. Her examination is normal. BP was 140/90. Her routine labwork was normal except for a mildly elevated fasting blood glucose (126 mg/dL), elevated triglycerides (600 mg/dL), an elevated TC/HDL ratio (6) related to a low HDL cholesterol (38 mg/dL).

The Figure is the ECG obtained at the time of application for age and amount. Is it normal or abnormal? If it is abnormal, what is the major abnormality? Could the abnormality be a normal variant? If there is an abnormality, how does it impact your mortality assessment of this case?

ECG INTERPRETATION

The basic rhythm is sinus, at 88 times a minute. Normal duration of the PR interval and QRS complex indicates intact AV and intraventricular conduction. The QT interval is also normal. The QRS axis in the frontal plane is leftward at approximately –15 degrees.

Two major abnormalities are found in the tracing. First, there are tall R waves in V₁-V₃ with R/S ratios greater than 1. These R waves are broad (≥ 0.04 seconds), and they are accompanied by upright T waves. The second abnormality is the presence of Q waves in the inferior leads. A small q wave is present in lead II accompanied by much more prominent Q waves in III and AVF. Together, the tall R waves in V₁-V₃ and abnormal inferior



Applicant's Electrocardiogram.

Q waves, are very suggestive of posterior and inferior myocardial infarctions (MI).

ECG DIFFERENTIAL DIAGNOSIS

Our applicant's ECG raises two issues which may confront the medical director while interpreting ECGs in a life insurance risk assessment context: the significance of prominent R waves in the early precordial leads (V_1 - V_3) and the significance of inferior Q waves. In this case study, we will focus on the tall R waves in V_1 - V_3 .

In the normal heart, ventricular depolarization begins in the middle-left third of the interventricular septum and travels in a downward and left-to-right direction. This initial septal vector of electrical activity will be seen by a right-sided lead such as V_1 as coming directly toward it. Following activation of the septum, the left ventricle, because of its larger mass, dominates the remainder of the depolarization process. Although right ventricular depolarization is occurring simultaneously, right ventricular forces are dwarfed by left ventricular events in the normal adult heart.¹ This results in a characteristic appearance of the QRS complex in V_1 , the rS configuration.

The initial small R wave (symbolized as "r" to denote its small size) occurs because of septal depolarization and the subsequent larger S wave (symbolized as "S" to denote its larger size) occurs because of the dominant effect of the left ventricle.²

The presence of a tall R wave in V_1 , defined as an R/S ratio equal to or greater than 1, should alert the medical director to consider a number of conditions that may impact the applicant's mortality experience. These conditions include: right bundle branch block, right ventricular hypertrophy, Wolff-Parkinson-White syndrome, posterior myocardial infarction, hypertrophic cardiomyopathy, muscular dystrophy, dextrocardia and misplaced precordial leads. In addition, 1%-3% of individuals who are free of clinical disease may also have an ECG with an R/S ratio ≥ 1 in V_1 .

Right bundle branch block (RBBB) is the most common cause of a tall R wave in V_1 .² The diagnosis of RBBB is usually readily apparent when the characteristic triphasic rsR' or rSR' patterns are seen in V_1 . In addition to the triphasic pattern in V_1 , the diagnosis of RBBB requires a QRS duration of 0.12 sec-

onds or more, and a wide terminal S wave in leads I, V₅ and V₆.

Although a R/S ratio >1 is used as a diagnostic criteria for right ventricular hypertrophy (RVH), not all forms of RVH are associated with this pattern. Prominent R waves in V₁ are most likely to be seen in severe right ventricular pressure overload situations such as those that occur in congenital heart disease, eg, pulmonary stenosis, Tetralogy of Fallot and Eisenmenger's syndrome. They are much less likely to occur when the pressure overload situation begins in adulthood such as that seen in mitral stenosis and chronic pulmonary disease. RVH is diagnosed by the finding of a tall R wave or qR pattern in V₁ and a ST-T strain pattern in V₁-V₂. Other supportive findings are right axis deviation (>110 degrees), right atrial enlargement and deep S waves in V₅-V₆ (R/S ratio <1).¹

The accessory conduction pathway(s) that occurs in Wolff-Parkinson-White (WPW) syndrome may alter the pathway of ventricular depolarization and cause ECG patterns that manifest as tall R waves in the right precordial leads and Q waves in the inferior leads. Close attention to the diagnostic features of WPW syndrome should help distinguish it from other causes of tall R wave in V₁. These features include a short PR interval (<0.12 second), QRS prolongation >0.11 second and slurring of the initial portion of the QRS complex (the delta wave). The typical WPW pattern is also often associated with secondary repolarization changes consisting of ST segment and T wave deflection in the opposite direction of the major portion of the QRS complex.² Diagnostic confusion may occur when the pre-excitation pattern is subtle. For example, when the QRS duration is only mildly prolonged, or the PR interval is not absolutely short.

As a result of septal hypertrophy, hypertrophic cardiomyopathy (HCM) may be associated with a tall R wave in V₁. In these patients, the initial septal QRS forces become unusually pronounced and are directed in a rightward, anterior, and superior direction,

resulting in QRS complexes, in the right precordial leads that are of large amplitude. In addition, deep but narrow Q waves are created from these same forces in the lateral leads (I, AVL, V₅-V₆) and occasionally in the inferior leads. Definitive diagnosis, if not obvious clinically, may require an echocardiogram.²

A tall R wave in V₁ is a common ECG finding in the Duchenne form of muscular dystrophy. The mechanism is presumed to be related to selective myocardial scarring in the posterobasal portion of the left ventricle, resulting in more prominent anterior superior forces of ventricular depolarization. Similar to HCM, these individuals also have characteristic deep but narrow Q waves in the lateral leads. These ECG changes usually occur in those individuals with Duchenne muscular dystrophy who have severe and longstanding disease; therefore, the applicant's history should help identify this etiology when present.²

In the mirror-image form of dextrocardia, the cardiac apex is pointed to the right, and the left atrium and left ventricle are situated to the right side of the right heart chambers. The ECG will generally show QRS complexes with decreasing amplitude across the precordial leads, often starting with a tall R wave in V₁. The other key ECG features are seen in the limb leads. Lead I will have an upside down P wave and QRS complex. Leads AVR and AVL will be reversed, with an upright QRS complex in AVR and a downward directed QRS complex in AVL. Leads II and III will be reversed as well, and AVF will be unaffected. Reversal of the leads allows for proper interpretation of the ECG in these individuals.²

Technical errors resulting in substitution of another precordial lead for V₁ can result in a tall R wave in V₁. Familiarity with the normal P wave, QRS complex and T wave patterns in V₁ can help identify this error.² In addition, "backwards" R wave progression in the anterior precordial leads and isolated negative or biphasic P waves at the site of origin of the

misplaced lead should arouse suspicion of a technical error.

A normal variant should be suspected when an applicant is an asymptomatic young adult. A R/S ratio >1 may be seen in V_2 in up to 10% of normal persons. The occurrence of a tall R wave in V_1 is highly unusual in the absence of cardiac pathology and is estimated to occur in 1%-3% of the normal population. Before a diagnosis of normal variant is made, the other possible causes of tall R wave in V_1 should be ruled out.^{1,2}

In our applicant, there is no history or clinical features suggestive of congenital heart disease, chronic pulmonary disease, hypertrophic cardiomyopathy, muscular dystrophy or dextrocardia. RBBB and WPW can be eliminated from consideration because the QRS complex is not prolonged in this tracing. None of the associated features of RVH (right atrial enlargement, right axis deviation, deep S waves in V_5 - V_6 or ST-T strain pattern) are present.

Before concluding that this is a normal variant, we need to consider posterior myocardial infarction even in the absence of a history of MI. The finding of a tall R wave in the anterior precordial leads especially when found in lead V_1 should always prompt one to think of posterior infarction. Since the inferior and posterior areas of the heart are almost always supplied by the right coronary artery, the additional presence of abnormal Q waves suggestive of inferior MI lends strong support to the diagnosis of posterior MI.

DISCUSSION

Encountering a tall R wave in V_1 in an insurance applicant's ECG presents the medical director with a diagnostic dilemma. The 5 most common conditions that should always be considered in the differential diagnosis are: right bundle branch block, right ventricular hypertrophy, Wolff-Parkinson-White syndrome, posterior MI and normal variant. Additional less common conditions to be considered were discussed earlier.

When the diagnosis is a normal variant, the

prognosis in such an applicant differs markedly from that in other individuals with a tall R wave in V_1 . The diagnosis of a normal variant is made by exclusion. The QRS complex is not widened, excluding RBBB and WPW. There are no associated findings of RVH and no evidence of inferior MI (making posterior MI much less likely). The absence of clinical context and physical findings rules out less common conditions, such as hypertrophic cardiomyopathy, muscular dystrophy and dextrocardia. One is left with the sole remaining possibility—a normal variant.

In our case scenario, the ECG pattern is not a normal variant. The tall R waves in V_1 - V_3 and abnormal Q waves in the inferior leads are most suggestive of old posterior and inferior MI. Isolated acute posterior wall myocardial infarction (MI) is uncommon, occurring in 3%-7% of acute MIs in studies where additional lead electrocardiography using left posterior thorax leads have been utilized. In the past, the terms "inferior" and "posterior" MI were sometimes used interchangeably. Currently, the term *inferior* is used to designate infarctions confined to the diaphragmatic surface of the heart. Whereas, the term *posterior* implies involvement higher up on the posterior surface of the heart.^{1,3}

Detection of posterior MI is difficult in that the standard 12-lead ECG does not adequately image the posterior wall. However, since the anterior wall of the left ventricle lies directly opposite, posterior wall infarctions can be visualized indirectly by observing the anterior precordial leads, which provide an upside-down mirror image of the posterior wall. Alternatively, by extending the precordial leads to include V_7 , (left posterior axillary line), V_8 (at the tip of the left scapula) and V_9 (just to the left of the vertebral column), the posterior wall can be imaged directly and will show the classical changes of myocardial infarction.^{2,4} This is a very useful manoeuvre when doubt remains whether or not there is a posterior MI present.

Since these leads are not obtained routinely, the standard 12-lead ECG diagnosis of posterior wall MI must be made indirectly by

observing the anterior precordial leads (V₁-V₃). In these leads, the ECG findings of acute posterior MI will be reversed. In other words, prominent R waves, ST segment depression and upright T waves in leads V₁ through V₃ represent Q waves, ST segment elevation and T wave inversions. The typical changes of an old posterior MI are tall R waves with R wave duration of 0.04 second or longer. These "reverse" changes can be confirmed by turning the ECG over and holding it up to the light—a Q wave (and if an acute MI, ST segment elevation) will be observed in the anterior precordial leads. This is referred to as a positive mirror test. Although any of the anterior precordial leads (V₁-V₃) may show these findings, a tall R wave in V₁ especially should prompt one to think of posterior MI.

The posterior wall of the heart receives its blood supply from the right (less frequently, the left circumflex) coronary artery. As a result, posterior MIs almost always occur in the presence of a simultaneous inferior MI (or less frequently a lateral). Hence, a virtual prerequisite for diagnosing posterior infarction is evidence of inferior infarction elsewhere on the tracing. In our applicant's tracing, there are indeed inferior Q waves. The Q waves in leads III and AVF exceed 0.04 second in duration and are relatively deep, thus suggesting they are abnormal Q waves. Their presence plus the small q wave in lead II strongly supports the diagnosis of previous inferior and posterior MI.

The ECG diagnosis of posterior infarction has important clinical and prognostic implications. Individuals with an inferior MI who also have a posterior MI have experienced a larger-sized MI. With increasing infarct size, risk of arrhythmia, left ventricular dysfunction and death are proportionally increased. Such risk in the acute phase is also proportional to the total number of ECG leads demonstrating ST segment elevation (\pm depression in the situation of posterior MI). Individuals with isolated posterior MI, if not clinically recognized as a "ST elevation" MI, may not receive appropriate acute therapy including thrombolysis or urgent angioplasty.³

In conclusion, careful analysis of the ECG containing tall R waves in V₁ is required to arrive at a specific diagnosis and allow proper risk assessment. By systematically looking for the usual causes of this pattern, it is possible to separate those who are at low risk from those who are at high risk.

REFERENCES

1. Gauer K, Curry Jr RW. *Clinical Electrocardiography*. Cambridge, Mass: Blackwell Scientific Publications; 1992:90-373.
2. Mattu A, Brady W, Perron A, Robinson D. Prominent R wave in lead V₁: electrocardiographic differential diagnosis. *Am J Emerg Med*. 2001;19:504-513.
3. Brady W. Acute posterior wall myocardial infarction: electrocardiographic manifestations. *Am J Emerg Med*; 1998;16:409-413.
4. Casas R, Marriott H, Glancy D. Value of leads V₇-V₉ in diagnosing posterior wall acute myocardial infarction and other causes of tall R waves in V₁-V₂. *Am J Cardiol*. 1997;80:508-509.