Postural changes in T waves

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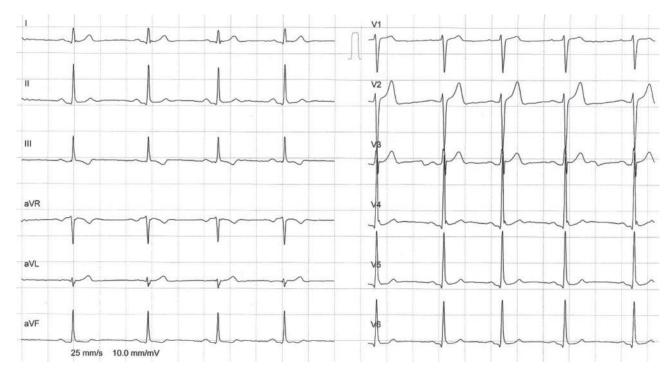
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Case presentation

A 25-year-old patient was referred to our outpatient clinic because of unexplained exercise intolerance and occasional left-side chest pain. The chest pain never occurred during exercise but rather started 30 minutes after the end of the exercise and could last for several days. Because of the exercise intolerance the patient limited his sports activity. The examination by the primary care physician did not reveal any underlying cause for the symptoms. In his past medical history, successful radiofrequency ablation of a right posterior accessory pathway due to Wolff-Parkinson-White syndrome is noteworthy without any recurrence in seven years. Clinical examination and echocardiography showed normal findings. Arterial blood pressure was 134/85 mm Hg and heart rate regular at 60/min. In the resting electrocardiography (ECG), recorded in recumbent position, we found sinus bradycardia with a heart rate of 57/min and previously known T wave inversions in the inferior leads III and aVF (fig. 1). The ECG recorded in upright position before the exercise test showed additional T wave inversions in leads V₄–V₆ (fig. 2) with complete normalisation of all T wave inversions

Figure 1

Resting ECG (recorded in recumbent position) with sinus bradycardia (heart rate at 57/min) and previously known T wave inversions in the inferior leads III and aVF.



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Figure 2

ECG recorded in upright position before starting exercise test with additional T wave inversions in leads V₄–V₆.

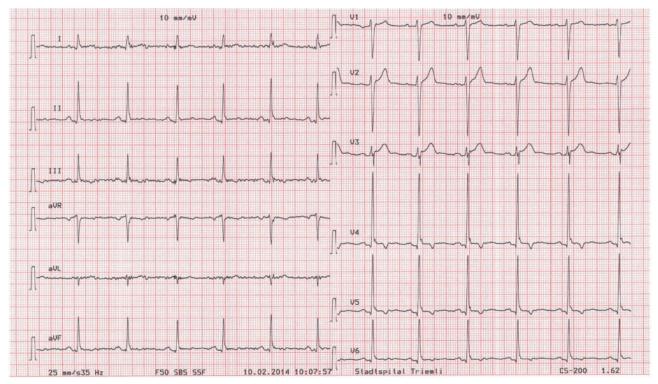
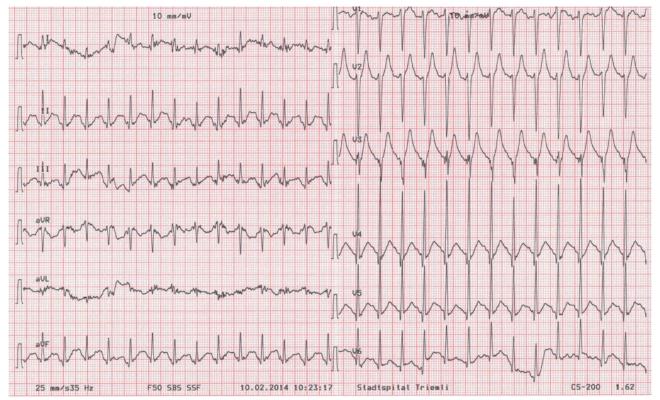


Figure 3 ECG recorded in upright position at maximum exertion with complete regression of all T wave inversions.



sions at maximum exertion (fig. 3). The patient exercised well with a maximum performance of 308 watts (158% of predicted performance). No symptoms occurred during and after exercise testing.

Questions

What do these dynamic T wave inversions indicate? Is there a need for further diagnostic testing for possible coronary artery disease?

Comments

The T wave in the body surface ECG corresponds to the phase of rapid ventricular repolarisation of the ventricular action potential. In adults the normal T wave is inverted in lead aVR, upright or inverted in leads aVL, III and V₁, and is upright in leads I, II, aVF and in chest leads V_3 through V_6 [1]. Abnormalities of the T wave may be secondary to abnormalities of ventricular depolarisation (abnormalities of QRS voltage or duration, e.g., bundle branch block, or pre-excitation syndromes) or primary (unrelated to any QRS abnormality, e.g., ischaemic, pulmonary embolism, hypokalaemia, digitalis effect, takotsubo cardiomyopathy, pericarditis, or memory effect). There are also normal variants of ventricular repolarisation such as persistent juvenile T wave pattern or early repolarisation pattern [2]. T wave inversions may also be induced by changes in body position, as evidenced in our patient, assumedly due to differences in heart position, altered autonomic tone or faster heart rate [3, 4]. Lachman et al. found in a group of healthy young adults that orthostatic changes in ST-T waves of 16 percent were not associated with any symptoms [3]. Mayuga and Fouad-Tarazi found that dynamic T wave changes during tilt table testing are associated with postural orthostatic tachycardia syndrome and vasovagal response [4].

In our patient we interpreted the changes in T waves as postural. Signs for secondary T wave inversions in a patient with former ventricular pre-excitation and successful radiofrequency ablation of the accessory pathway were not evident. We did not perform further diagnostic evaluation for coronary artery disease in absence of cardiovascular risk factors and lack of typical or atypical symptoms of angina pectoris. The inverted T waves in leads III and aVF in the resting ECG were interpreted as a normal variant [5].

References

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