Case report

Potentially misleading manifestation of a ventricular pre-excitation

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A B S T R A C T

We present the case report of a 56-year-old man with an unusual manifestation of an accessory pathway. Failure to detect, or incorrect diagnosis, of this anomaly could have put the patient at high risk of sudden cardiac death. The accessory pathway described in this case report was located at the left posteroseptal area and presented initially with a broad QRS complex tachycardia. Despite being pre-excited atrial fibrillation, it could have been misinterpreted as ventricular tachycardia. Once the rhythm had changed to sinus, a Q-wave in the inferior ECG leads became apparent. This finding could have been misdiagnosed as an old myocardial infarction and treated as such, including prescription of betablockers which might, in theory, increase the risk of sudden cardiac death. The treatment of choice for the patient was radio frequency ablation of the accessory pathway.

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Introduction

Accessory atrioventricular (AV) pathways represent a presence of abnormal conductive tissue between atria and ventricles. They can be clinically completely asymptomatic, but on the other hand the abnormal accessory AV conductivity may result in narrow or broad complex tachycardia. In the latter case the diagnosis may be less obvious and the electrocardiogram (ECG) can be misinterpreted. We present the case report of one of these manifestations to stress out the need for carefully set diagnosis.

Case description

A 56-year-old man was admitted to the cardiology department with a 2-day history of exertional shortness of breath, weakness and fatigue. He also developed central chest discomfort (pressure) and presented to the accident and emergency (A&E) department 2 hours later. He denied having any palpitations or collapse. He was an ex-smoker who stopped smoking 5 years ago and his past medical history comprised of arterial hypertension and chronic allergic alveolitis. His medications included losartan 50 mg/D,
prednisol 20 mg/D and salicylic acid 100 mg/D. He was on a low fat diet due to hypercholesterolaemia. He had a positive family history of a premature ischaemic heart disease (his father died from myocardial infarction at 60). On admission, he was haemodynamically stable with no signs of heart failure, BP was 120/70 mmHg and heart sounds were irregular 230 beats per minute with the peripheral pulse deficit. The pressure chest pain eased off completely with sublingual nitrate. ECG on admission (Fig. 1) showed irregular broad-complex tachycardia (BCT) with positive concordancy in precordial leads, left axis deviation (LAD) and a ventricular rate of approximately 250 beats/min. The tachycardia was initially slowed down by i.v. amiodarone 300 mg but, as he remained tachycardic he was cardioverted to sinus by synchronized electrical cardioversion. The subsequent ECG (Fig. 2) showed sinus rhythm with varying QRS width and Q wave pattern in leads III and aVF. No previous ECG was available for comparison. Blood results were unremarkable with no electrolytes abnormalities, normal thyroid function test and normal creatine kinase (including MB fraction). Troponin T was slightly elevated. Transthoracic echocardiography was carried out and revealed well maintained left ventricular systolic function with mild multifocal regional wall motion abnormalities and moderate mitral regurgitation.

What is the diagnosis?

On detail ECG analysis we can see a broad complex tachycardia which could be misinterpreted as ventricular tachycardia (VT), but the obvious irregular rhythm indicates that the correct diagnosis is pre-excitated atrial fibrillation with an antegradely conducting accessory pathway (Fig. 1). Shortest pre-excited RR interval 200 ms indicates high-risk accessory pathway (very fast antegrade conduction via accessory pathway with risk of degeneration to ventricular fibrillation). The varying QRS width on ECG after electrical cardioversion is caused by pre-excitation including Q wave pattern in the inferior leads which is in fact a negative delta wave. Pre-excitation is more expressed with atrial premature beats (Fig. 2).

This patient underwent an electrophysiology study which confirmed the presence of the atrioventricular accessory pathway in the left posteroseptal region (antegrade conduction – atrial effective refractory period/ERP/240 ms, accessory pathway ERP < 260 ms, AV re-entry tachycardia non-inducible). The pathway was successfully ablated (Fig. 3) with no further ECG evidence of pre-excitation (Fig. 4).

Discussion

Accessory atrioventricular pathways are congenital abnormalities involving the presence of abnormal conduction tissue between the atria and the ventricles which can cause early depolarisation of the ventricular myocardium: pre-excitation.

The first suggestion of the existence of these pathways was mentioned by Holzmann and Scherf in 1932 [1]. Until 1932 the presence of a broad QRS complex associated with paroxysmal tachycardia was categorized as a bundle-branch block phenomenon [2,3]. First histological evidence of accessory pathway was proved in 1942 by Wood et al. [4]. David Scherf
is thought to be the first person who described our current understanding of the pathogenesis of the Wolff–Parkinson–White (WPW) syndrome in terms of a re-entrant circuit involving both the AV node – His axis and the accessory pathway. This hypothesis was not universally accepted until the 1970s when Durrer and others applied invasive electrical stimulation to the heart to confirm the pathophysiological processes [5–9]. Morady and Scheinman were the first to successfully ablate an accessory pathway (posteroseptal) using high-energy direct-current shocks. Subsequently Jackman, Kuck, Morady, and a number of groups proved the remarkable safety and efficiency of catheter ablation for pathways in all locations using radio frequency energy [10].

Accessory atrioventricular pathways arise during embryonic evolution when atria and ventricles are not adequately divided. These may be either isolated or multifocal with septal or posterior location [11]. The electrical impulse can pass through the pathway either antegrade, towards to ventricle, retrograde, away from the ventricle or in both directions. The majority of pathways allow conduction in both direction, with retrograde only conduction occurring in 17–37% of cases (known as a concealed accessory pathway) and antegrade only conduction rarely seen [12,13]. The electrical activation of the myocardium starts from sinoatrial node and reaches the ventricles through both the accessory pathway and atrioventricular (AV) node. As the accessory pathway conducts the impulse faster than the AV node, the ventricular depolarisation starts from the accessory pathway region and is followed by depolarisation via the AV node-His conduction system. This results in a short PR interval and slurred upstroke of the QRS complex known as the delta wave.

The patients with pre-excitation ECG pattern are often asymptomatic. The antegrade conductivity can spontaneously resolve in about 30% of all cases. Symptomatic patients may experience palpitations, dizziness, shortness of breath or syncope. Of note the first presentation of pre-excitation may be a sudden cardiac death. Fortunately this happens very rarely (0.15–0.4%) [14,15].

Symptoms are caused by atrioventricular re-entry tachycardia (AVRT) which can be either orthodromic or antidromic. The terminology depends on the direction of the electrical impulse going through AV node. The ventricular myocardium is activated through either the normal conduction system resulting in a narrow complex tachycardia (orthodromic AVRT) or through the accessory pathway (antidromic AVRT) with a broad complex tachycardia.

A complex of symptoms due to supraventricular tachycardia with evidence of pre-excitation on ECG is called WPW syndrome [16]. The presence of an accessory pathways is dangerous in patients with atrial fibrillation [17–19]. The AV node provides protection against a fast ventricular response to atrial tachycardia. In the presence of a rapidly conducting accessory pathway fast atrial electrical impulses conducted via the pathway may lead to an extremely rapid ventricular response with degeneration to ventricular fibrillation.

The first line of treatment for symptomatic patients is radio frequency ablation of the accessory pathway with success rate of 90–95% depending of its location [20–22]. There is no clear indication for this treatment in asymptomatic patients but it should be considered with respect to the patient’s profession (e.g. pilot), age (higher prevalence of atrial fibrillation with increasing age) and electrophysiological parameters of accessory pathways. Medical management includes either class I
antiarhythmic drugs (propafenone) or class III (sotalol, amiodarone) which slow down pathway conduction. The use of AV node conduction blocking/slowing drugs (particularly verapamil) may be harmful allowing rapid unopposed conduction down the accessory pathway with the potential of the above-mentioned consequences [23-25].

**Summary**

A careful ECG analysis is vital for obtaining a correct diagnosis of pre-excited atrial fibrillation. This is necessary for further management with respect to the symptoms and prognosis, particularly the risk of sudden cardiac death. The wider differential diagnosis like ventricular tachycardia caused by acute coronary syndrome based on history, ECG and laboratory results could have been considered in brainstorming process, but eliminated as a false route.

We also aimed to show a less common ECG presentation of an accessory atrioventricular pathway due to negative delta wave in inferior leads. This could be potentially misleading for less experienced physician who could be lead to believe that they were dealing with consequences chest pain/acute myocardial ischaemia – ventricular tachycardia and treat the patient accordingly with potentially harmful medication.

We have to keep pre-excitation syndrome in mind especially in young patients with history of palpitations but it may occur at any age, irrespective of the presence of comorbidities or risk factors.

**Conflicts of interest**

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Ethical statement

We hereby declare we have not breached any ethical standards by writing this article.

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