SHORT COMMUNICATION

Special character of atrial paroxysmal fibrillation in coincidence with WPW syndrome

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Abstract

The paper summarizes the knowledge on paroxysmal atrial fibrillation in WPW syndrome. The peculiarities, ECG features, risk markers of sudden cardiac death, pharmacologic and non farmacologic therapy and prevention of this arrhythmia are presented. Potentially dangerous effects of drugs such as digoxin, verapamil, adenosine and betablockers are emphasized. *(Short communication)*

In patients with WPW syndrome, paroxysmal atrial fibrillation (PAF) is characterized by several peculiarities differing from PAF in patients without WPW syndrome. As opposed to PAF without WPW syndrome, which rarely endangers the patient•s life, PAF in the setting of WPW syndrome may have prognostically serious and life-threatening consequences by precipitating ventricular fibrillation (VF) and sudden cardiac death (SCD). While the diagnosis of PAF without WPW is usually not dificult, distinction of PAF in WPW syndrome is very often problematic. In this situation, PAF is relatively frequently misdiagnosed as ventricular tachycardia or as PAF with bundle branch block. The management of PAF in WPW syndrome differs significantly from that without WPW syndrome.

Occurrence

PAF occurs in 10—30 % of patients with WPW syndrome (4). It is more frequent in patients with overt pre-excitation (PE) on a surface electrocardiogram (ECG), thus in patients with anterograde (from atrium to ventricle) conduction over AP. PAF occurs less often in patients with so-called concealed accessory pathway (AP) whit only retrograde (from ventricle to atrium) conduction without manifest PE on surface ECG. In patients with WPW syndrome, PAF may develope de novo as primary arrhythmia, or more often as a consequence of paroxysmal atrioventricular (AV) reciprocal tachycardia. VF and SCD can be the first clinical manifestation of WPW syndrome in patients susceptible to PAF.

Cause of increased susceptibility to paroxysmal atrial fibrillation in patients with WPW syndrome

Cause of increased susceptibility to PAF in patients with WPW syndrome is not exactly explained. It is assumed that presence of a functional AP itself and its ability to excite atrium rapidly and eccentrically are responsible for PAF in the WPW syndrome. The possibility of microreentry within AP or in the proximity of its atrial insertion is considered too as one of the causes. The participation of AP in the development of PAF is proved by elimination of the latter after successful interruption of AP (2). Other hypothesis assumes that PAF in the WPW syndrome is related to electrophysiological abnormalities of atria, which are relatively oft observed in patients with WPW syndrome and which are consider as independent of AP (1).

ECG pattern

ECG during PAF with anterograde conduction over AP show wide abnormally configured QRS complexes, which usually yield

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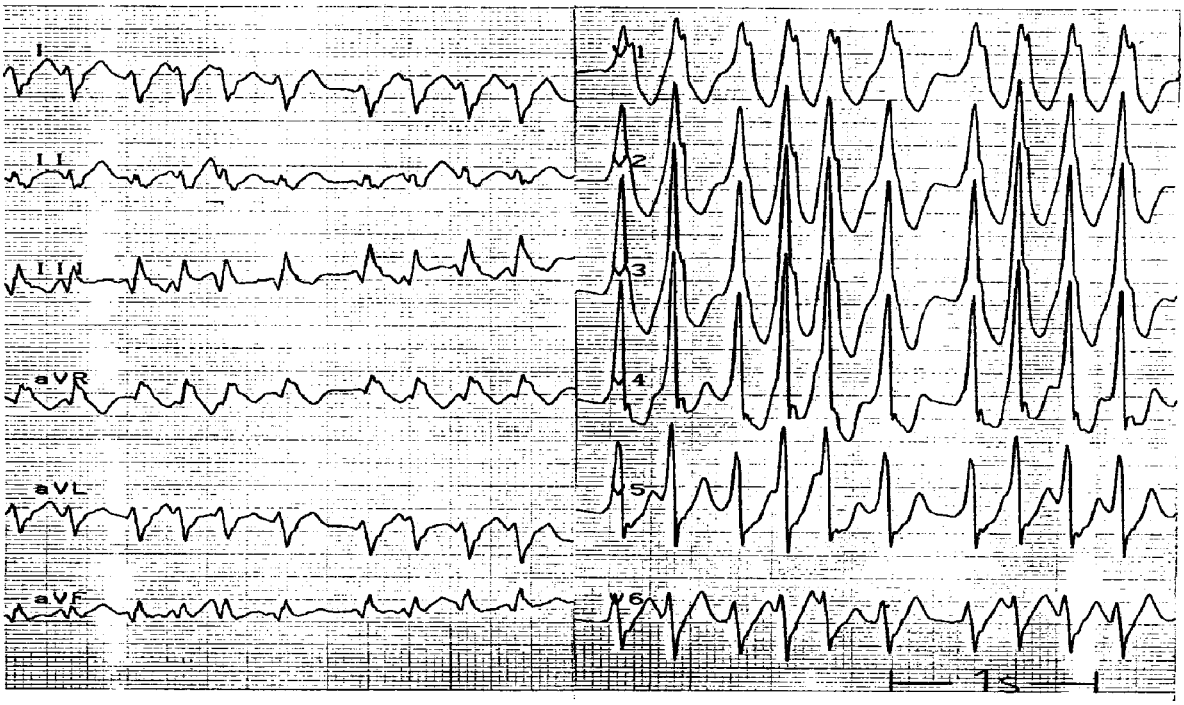

Fig. 1 a) ECG in paroxysmal fibrillation of atria. QRS complexes are wide, abnormally configured in result of high degree or maximal ventricular pre-excitation, the ventricular rate is 260/minute.

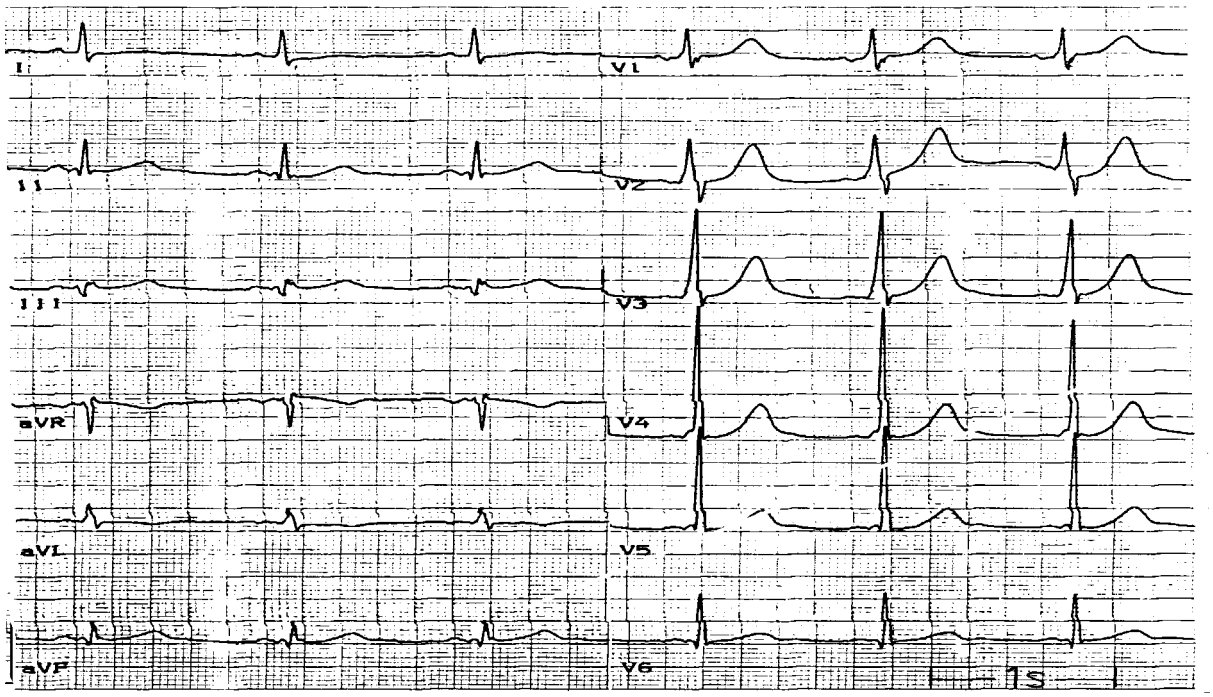

Fig. 1 b) ECG after recovery of sinus rhythm with relatively subtle pre-excitation.

a high or maximum grade of PE (Fig. 1a, 1b). The R-R intervals are irregular. The ventricular rate is usually very high, often over 200—250 beats/minute, in rare cases over 300 beats/minute. The high ventricular rate is responsible for relatively frequent occurrence of symptoms of impaired hemodynamics, such as hypotension or syncope.

The risk of sudden cardiac death

Why is it that PAF in coincidence with WPW syndrome represents such a dangerous disorder of heart rhythm? The cause resides in the fact that the conduction of impulses of high frequency is carried out from atria to ventricle over AP. Hence, the AV node is by-passed together with its task of „filtration" which protects from the conduction of all impulses from atria to ventricles and thus also from high ventricular rate. In contrast to the latter, AP, especially if it has high conduction capacity which can inrease in coincidence with PAF, is able to conduct impulses of high frequency from atria to ventricles. High ventricular rate can result in the development of VF and SCD.

The risk of VF and SCD depend foremost on electrophysiologic properties of AP. The decisive electrophysiologic parameter determining the conduction capacity of AP is anterograde effective refractory period of AP (AERP). It was discovered, that patients with very short AERP (less than 250—270 ms), and thus with its high conduction capacity (1, 5), are at high risk for VF and SCD. AERP AP can be exactly and precisely determined by invasive electrophysiologic investigation. However, there are also several non-invasive markers of short AERP AP, and thus also of high risk of VF and SCD. They include: constant PE and its persistence after administration of ajmaline and during exercise test. Patients with shortest RR interval between preexcited QRS complexes during PAF less than 220—250 ms are too at high risk for VF and SCD (3). The presence of multiple APs represents further risk factor of VF and SCD. The presence of multiple APs increases the probability that at least one of the APs will have a short AERP and high conduction capacity.

Therapy and prevention

Therapy includes both termination of the PAF episode and profylaxis of further recurrence. With regard to marked symptomatology and high risk, PAF in the WPW syndrome should be terminated as soon as posible. Should the situation be urgent, i.e. in cases of high ventricular rate and hemodynamic instability, the therapy of shoice is represented by electrical defibrillation. If arrhythmia is good tolerated or if electrical defibrillation fails, the pharmacological rhythm conversion comes into consideration. Antiarrhythmic drugs of classes I and III, ajmaline, propafenone and amiodarone that have a direct influence on AP and are aimed on the blocking conduction over AP should be given. Ajmaline is administered in a dose of 10 mg/kg of weight, however not exceeding 100 mg, by slow intravenous injection by speed of 10 mg/minute. The therapy of propafenone begins with a bolus of 1—2 mg/kg of weight by slow intravenous dosis during 5 minutes. and continues by continual administration in a dosis of 0,5—1 mg/kg weight, in infusion of 5 % glucose, not exceeding 560 mg/24 hours. Amiodarone is administered in 5 % glucose, initially as a bolus in a dose of 300 mg during 30—120 min and continues to be administered in a dose of 600—900 mg/24 hours.

All drugs that have deprressant effect on AV nodal conduction, such as digoxin, verapamil, adenosine and beta-blockers, are contraindicated in patients with PAF in coincidence with WPW syndrome. These drugs, by means of deterioration or blockade of the AV node conduction, prefer and accelerate conduction of impulses over AP, leading to an incerase in ventricular rate with the risk for occurrence of VF. Besides, verapamil has unfavourable impact upon hemodynamics.

The profylaxis of PAF can be based on oral administration of propafenone, sotalol and amiodarone. Propafenone is effective in higher doses 600—900 mg/24 hours divided into three doses. Sotalol in a dose of 2x80—240 mg is appropriate especially in patients with an assumed participation of increased sympatic activity in the development and maintenance of PAF. Amiodarone, after the achievement of the total saturation oral dosis of 10—12 g or intravenous dosis of 5—6 g is administered in the dosis of 200 mg, i.e. 1 tablet daily or 5 days a week.

The definite causal therapy of PAF and thus also the prevention FK and SCD in coincidence with WPW syndrome is represented by radio-frequency catheter ablation of AP. This intervention should be carried out in all patients with WPW syndrome predisposed to PAF.

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