

Images

Aberrant atrial fibrillation simulating ventricular tachycardia as a first sign of Wolf Parkinson White syndrome

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Female patient aged 27 years with no medical history of interest. Athletic constitution, plays sport occasionally and has never undergone a cardiological check-up. Does not take medication and reports no intake of toxic substances.

One hour prior to her admission in Emergency, she presented with an episode of thoracic discomfort which the patient believed to be a palpitation episode unrelated to physical activity. No chest pain or dyspnoea reported. She presented at her Health Centre where an ECG was performed (Figure 1). The patient was referred to UME 112 (emergency service) after application of an electrical defibrillator with suspected ventricular tachycardia.

At the time of admission the BP was 110/60 mmHg, HR 80 bpm, O₂ Sat 96%.

Patient was conscious, well perfused, no with dyspnoea or chest pain.

Physical exploration as of admission time was normal.

ECG monitoring and electrocardiographic recording (Figure 2) were performed, and the patient was admitted for observation.

In April 1928, Wolf, Parkinson and White (WPW) published a full description of this arrhythmia, after having observed it in usually young, patients, presenting with paroxysmic palpitation crises, whose electrocardiograms (ECG) outside such crises presented a short P-R interval (< 100ms) and a widened QRS (> 120 ms) simulating a bundle branch block, together with a slurred upstroke to the QRS, termed delta wa-

ve. The incidence of wpw among the general population is low –around 0.3-0.4% - and generally has a good prognosis.

Both ECG signs as well as their clinical consequences³ are closely linked to the existence of accessory conduction AV pathways. With regard to the incidence of arrhythmias, atrial fibrillation represents 30% of all WPW arrhythmias².

The incidence of arrhythmias in WPW can vary between 12 and 80% depending on the references consulted¹.

Two thirds are secondary to tachycardias due to re-entry circuits, and the rest to atrial fibrillation.

Observation of sudden death in young patients⁴ indicates existence of a group of high risk patients in whom the first sign in adulthood is a pre-excited atrial fibrillation or flutter (Figure 1). Such cases of poor prognosis usually involve accessory pathways with excessively short anterograde refractory periods, requiring the performance of a diagnostic and /or therapeutic electrophysiological study.

REFERENCE

- 1- Wellens HJ, Brugada P, Penn OC. The management of preexcitation syndromes. JAMA 1987; 257:2325-33.
- 2- Boraita Pérez A, Serratosa Fernández S. Revista MONOCARDIO 2000;2:74.
- 3- Lapuerta Irigoyen L. Taquiarritmias cardiacas inducidas por Síndrome de Preexcitación. Importancia de su reconocimiento y su tratamiento de urgencia. Emergencias 1992;4:258-63.
- 4- Pons de Beristain C. La muerte súbita del deportista. Introducción a un protocolo de vigilancia cardiológico. Arch Med Dep 1984;1:37-41.

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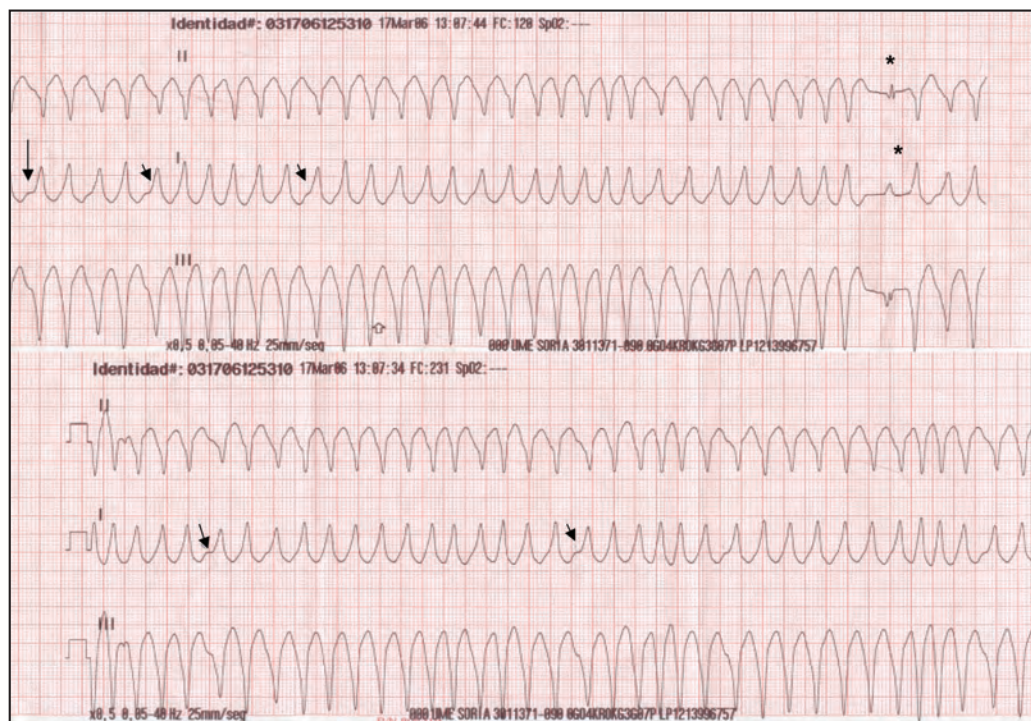


Figure 1. Observe variation of RR complex. Although QRS are wide (aberrance) in some you can see (arrows) a slurring termed delta wave, leading to suspected pre-excited aberrant atrial fibrillation instead of ventricular tachycardia, alternating at some points with narrow QRS (*).

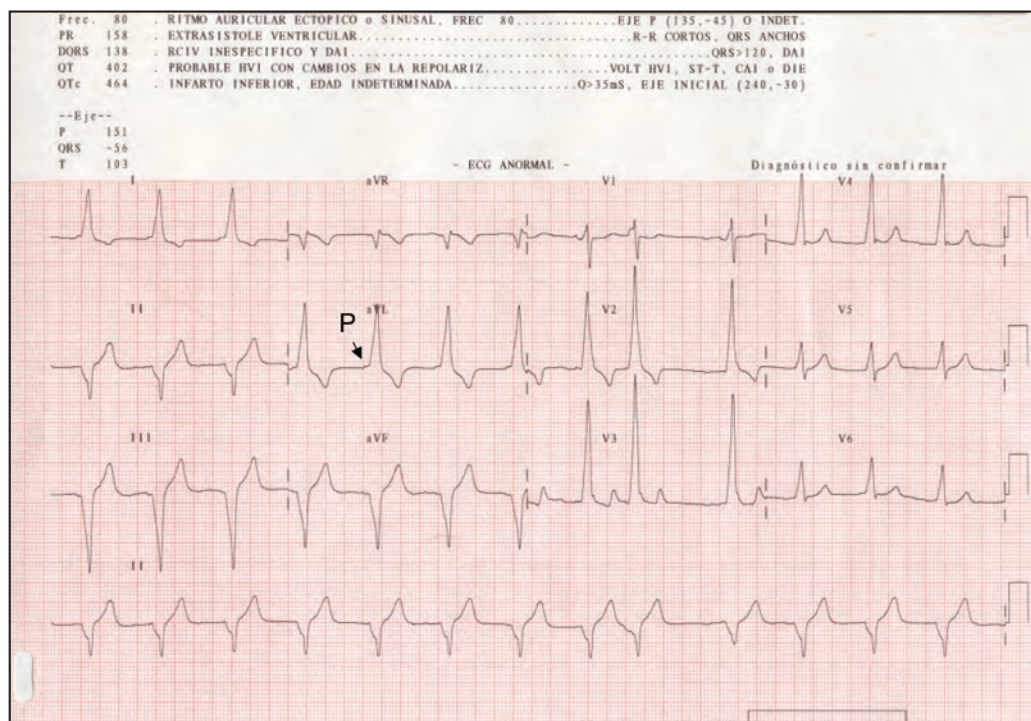


Figure 2. Observe the morphology of QRS over 120 mseg, simulating a bundle branch block with a P-R interval < 100 mseg, erroneously interpreted by the computer system.