EKG Series

Foreword

With this case, we introduce a new series of electrocardiography quizzes. We welcome submissions for this series. EKGs should convey a point of interest to the emergency physician, and should be accompanied by a discussion of not more than 1000 words, including references, as appropriate. We ask that the EKGs come from cases that you have first-hand knowledge of, that the facts of the case are true, and the details of any pertinent diagnostic testing are included.

Daniel A. Waxman, MD
Editor, Cardiology Section, IJEM

EKG Series: 1st Case
An unusual tachycardia

Ivan P. Steiner, BSc, MD , MCFP(EM), FCFP

Professor, Director, Studies in Medical Organizations, Department of Family Medicine and Division of Emergency, Medicine, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Canada

Shai Z. Fuchs

Medical Student, Hadassah Medical School, University of Jerusalem, Jerusalem, Israel

Daniel A. Waxman, MD

Attending physician, Department of Emergency Medicine, and Division of Cardiology, Beth Israel Hospital, Assistant Professor of Emergency Medicine and Medicine, Albert, Einstein College of Medicine, New-York, USA

Correspondence:
Ivan Steiner BSc, MD, MCFP (EM), FCFP
10240 Kingsway Avenue,
C.S.C. Room 565,Edmonton,
AlbertaT5H 3V9
Tel: (780) 491-5009
Fax: (780) 477-4916
E-mail: ivan.steiner@ualberta.ca

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- Department of Family Medicine & Division of Emergency, Medicine, University of Alberta, Edmonton, Canada
- Hadassah Medical School, University of Jerusalem, Jerusalem, Israel
- Beth Israel Hospital Medical Center, New-York, USA

Presentation

A 38 year old man without prior medical history complains of a rapid, irregular heartbeat of one hour's duration. The patient was in his usual state of health when the palpitations began. He denies other symptoms. He has had similar bouts infrequently in the past, but has not previously sought medical attention. He is on no medications, and denies drug or alcohol use. On exam, he is somewhat uncomfortable looking, has an irregular apical heartrate of 220 beats per minute (bpm), and a normal blood pressure. The remainder of his physical exam is normal.

Question

The 12 lead EKG of this patient is presented to the attending emergency physician (**Figure 1**).
What do you believe is wrong with this patient?
For the answer to today's case turn the page.

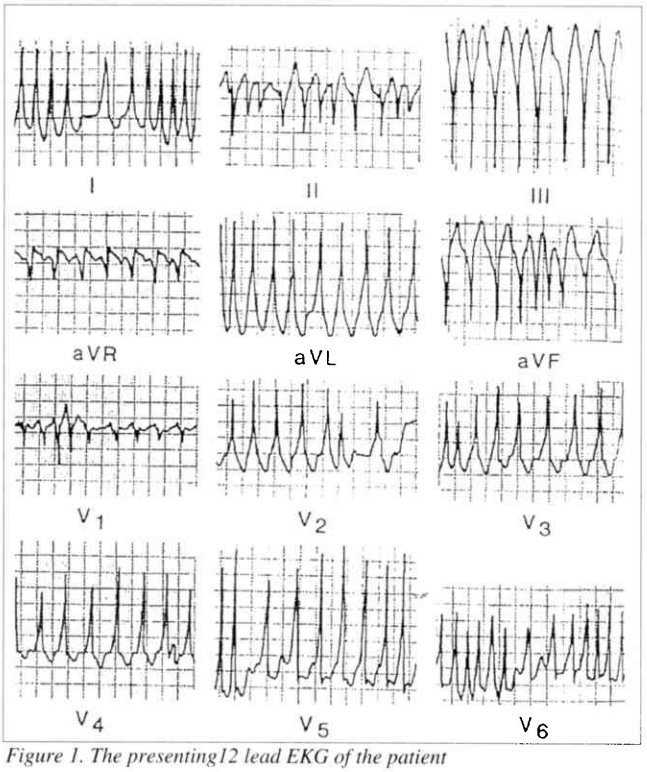

Figure 1. The presenting12 lead EKG of the patient

Interpretation

The EKG shows a wide-complex tachycardia of a markedly varying cycle length. The cycle length is determined by measuring the R-R interval (each small box on the EKG is 40 msec) and in this patient it ranges from 160 msec to 480 msec. Heart rate is calculated at 60,000/cycle length, and the ventricular rate in this case ranges from 126-375 bpm. P waves are absent. Also notable is the marked beat to beat variation in QRS amplitude, duration, and morphology. Such variation strongly implies some sort of fusion. Fusion beats are a composite of impulses which travel down the normal conduction system (AV node → bundle of His → bundle branches) and impulses which arise elsewhere. The 'elsewhere' can be from a ventricular focus such as a pacemaker, from a re-entrant ventricular tachycardia, or, as in this case, from atrial impulses conducted down an accessory pathway. In this patient, the marked beat to beat irregularity, the variable and at time extremely rapid rate rule out the diagnosis of ventricular tachycardia.
The diagnosis is atrial fibrillation, with conduction down an accessory pathway, Wolff-Parkinson-White Syndrome (WPW).

Discussion

The incidence of WPW is difficult to determine, but the prevalence of preexcitation on the resting EKG has been quoted as 0.1% to 0.3% (1). WPW is defined by the presence of ventricular preexcitation, as demonstrated by a delta wave on the resting EKG, along with symptoms to suggest recurrent tachyarrhythmias. Tachyarrhythmias associated with it include AV reciprocating tachycardia and atrial fibrillation (2).
AV reciprocating tachycardias are the most frequently reported arrhythmia. These are 'macroreentrant' tachycardias, involving the AV node for one limb of the cycle and the accessory pathway for the other. Most commonly the cycle goes down the AV node and up the accessory pathway, is therefore narrow complex, and is known as orthodromic reciprocating tachycardia. The alternative, in the other direction, is known as antidromic reciprocating tachycardia, and is wide complex. These arrhythmias will have a constant cycle length, usually at a rate of about 200 bpm, and will be terminated by drugs or maneuvers which block conduction through the AV node (one should always be careful to exclude ventricular tachycardia or WPW with atrial fibrillation or flutter before giving AV nodal blocking drugs to a patient with a wide complex tachycardia).
Atrial fibrillation occurs commonly in WPW, with incidence reported between 10% to 32% of cases (1). Atrial fibrillation is of particular concern, because the accessory pathway may allow very rapid conduction of the fibrillatory waves, as in this case. This rapid ventricular response can predispose to ventricular fibrillation, and this mechanism is thought to be the primary cause of sudden death in WPW syndrome, which has a reported incidence of up to 0.6% per year (2).

Management

Whenever atrial fibrillation is associated with a wide complex ventricular response, the possibility of WPW should be considered. This is crucial, as drugs which block conduction through the AV node (normally a mainstay of therapy for a patient in atrial fibrillation) are contraindicated in the presence of WPW, as they may lead to increased conduction through the accessory pathway. For the hemodynamically unstable patient, treatment is synchronized cardioversion. For a stable patient, an attempt at chemical cardioversion with procainamide or ibutelide is considered acceptable (3).
This patient was treated with procainamide without response. Subsequent synchronized cardioversion with 200J was successful. His EKG post-conversion did not show delta waves, which is atypical for WPW, but subsequent electrophysiologic studies demonstrated several accessory pathways, which were successfully ablated.

References

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