A 27-year-old man without any previously known health conditions was found unresponsive on the street after he had been exercising. Cardiopulmonary resuscitation was started by bystanders. On arrival of the emergency services, the rhythm strip in Figure 1A was recorded. It shows an irregular wide-complex tachycardia with different degrees of QRS widening, consistent with preexcited atrial fibrillation with very fast conduction to the ventricles. At the end of the strip, QRS complexes become smaller and erratic as atrial fibrillation turns into ventricular fibrillation. After 4 direct-current shocks (Figure 1B), the ventricles are defibrillated but preexcited atrial fibrillation persists. It is only after 17 shocks and amiodarone administration (Figure 1C) that sinus rhythm is restored. The patient was admitted to the hospital where he had an uneventful recovery without neurological sequelae. An echocardiogram ruled out structural heart disease. His baseline ECG showed only minimal preexcitation (Figure 2A). At electrophysiological study, preexcitation over a left lateral accessory pathway became evident while pacing the atrium from the coronary sinus, close to the accessory pathway insertion (Figure 2B). During the electrophysiological study, atrial fibrillation was induced, with very fast conduction to the ventricles over the accessory pathway with a minimal RR interval of 140 ms (Figure 2C), but without hemodynamic collapse. Electric cardioversion restored sinus rhythm. The accessory pathway was mapped in the lateral mitral annulus and ablated successfully.

Preexcited atrial fibrillation with cardiac arrest caused by hemodynamic collapse or degeneration to ventricular fibrillation may be the first manifestation of the Wolff-Parkinson-White syndrome.1 It is of particular interest in this patient that the instant of degeneration to ventricular fibrillation could be registered, and defibrillation was unusually difficult. The first direct-current shocks failed to restore sinus rhythm, but rather converted ventricular fibrillation back into preexcited atrial fibrillation. Multiple shocks at 200J energy were required to defibrillate the atria and restore sinus rhythm. This could be because of an increase in defibrillation threshold in the setting of cardiac arrest and metabolic acidosis.

Of note also, the ECG in sinus rhythm showed only minimal preexcitation that could have been easily overlooked at previous routine evaluations. This highlights the fact that, even in the absence of overt preexcitation or with only minimal preexcitation apparent in the baseline ECG, an accessory pathway may be present that is capable of fast antegrade conduction, with the risk of lethal ventricular arrhythmias should atrial fibrillation occur. This situation occurs typically in young people who have a left-sided accessory pathway, because sinus impulses from the right atrium reach the atrioventricular node before reaching the accessory pathway insertion, and if conduction through the atrioventricular node is fast, ventricular activation occurs mostly by way of the normal conduction system. Adenosine administration can unmask the accessory pathway by blocking conduction through the atrioventricular node. Patients with asymptomatic preexcitation, even if not very apparent in the baseline ECG, should undergo risk stratification with exercise test and be referred for electrophysiological study and ablation, when appropriate, to prevent sudden death.2

Disclosures
None.

References
Figure 1. Rhythm strips obtained during resuscitation. A, Preexcited atrial fibrillation degenerating to ventricular fibrillation. B, Ventricular fibrillation converting back to preexcited atrial fibrillation after a DC shock. C, After multiple DC shocks sinus rhythm is restored. DC indicates direct current.

Figure 2. Tracings obtained at electrophysiological study. A, Baseline ECG with minimal preexcitation. B, Maximal preexcitation while pacing from the left atrium consistent with a left-sided accessory pathway. C, Preexcited atrial fibrillation with very fast conduction to the ventricles (mean ventricular rate >300 bpm).
From Atrial Fibrillation to Ventricular Fibrillation and Back
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