THE CLINICAL PICTURE

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Wolff-Parkinson-White pattern unmasked by severe musculoskeletal pain

A 55-year-old man with no significant medical history presented to the emergency department with left-sided flank pain that had begun 3 days earlier. He described the pain as continuous, sharp, and aggravated by movement. He worked in construction, and before the pain started he had moved 8 sheets of drywall and lifted 5-gallon buckets of spackling compound. He denied any associated chest pain, palpitations, dyspnea, cough, or lightheadedness. His family history included sudden cardiac death in 2 second-degree relatives.

On arrival in the emergency department, his vital signs were normal, as were the rest of the findings on physical examination except for reproducible point tenderness below the left scapula.

Laboratory workup revealed normal blood cell counts, liver enzymes, and kidney function. His initial troponin test was negative.

A routine electrocardiogram (Figure 1) showed normal sinus rhythm with a rate of 65 beats per minute, delta waves (most pronounced in V2), and Q waves in leads II, III, and aVF; the Wolff-Parkinson-White (WPW) pattern. Three subsequent electrocardiograms showed consistent findings.

He was admitted to the hospital and was conservatively treated with nonsteroidal anti-inflammatory drugs for his musculoskeletal back pain.
pain. A follow-up electrocardiogram 24 hours later no longer showed delta waves (Figure 2). Echocardiography showed a normal ejection fraction with no valvular disease. An exercise stress test was negative for reversible ischemia.

The patient was referred to an electrophysiologist for further evaluation, but he returned to his home country (Haiti) after discharge and was lost to follow-up.

■ Wolff-Parkinson-White Pattern vs Syndrome

WPW syndrome is a disorder of the conduction system leading to preexcitation of the ventricles by an accessory pathway between the atria and ventricles. It is characterized by preexcitation manifested on electrocardiography and by symptomatic arrhythmias.

In contrast, the WPW pattern is defined only by preexcitation findings on electrocardiography without symptomatic arrhythmias. Patients with WPW syndrome can present with palpitation, dizziness, and syncope resulting from underlying arrhythmia. This is not seen in patients with the WPW pattern.

A short PR interval with or without delta waves can also be seen in the absence of an accessory pathway, eg, in hypoplastic left heart syndrome, atrioventricular canal defect, and Ebstein anomaly. These conditions are termed pseudopreexcitation syndrome.

Our patient presented with severe musculoskeletal pain that precipitated the electrocardiographic changes of the WPW pattern and resolved with adequate pain control. The WPW pattern can be unmasked under different scenarios, including anesthesia, sympathomimetic drugs, and postoperatively.

Catecholamine challenge has been used to unmask high-risk features in WPW syndrome. Our patient may have had a transient spike in catecholamine levels because of severe musculoskeletal pain, leading to unmasking of accessory pathways and resulting in the WPW pattern on electrocardiography.

Most patients with the WPW pattern experience no symptoms, but a small percentage develop arrhythmias.

In rare cases, sudden cardiac death can be the presenting feature of WPW syndrome. The estimated risk of sudden cardiac death in patients with the WPW pattern is 1.25 per 1,000 person-years; ventricular fibrillation is the underlying mechanism. As our patient had a family history of sudden cardiac death, he was considered at high risk and was therefore referred to an electrophysiologist.
REFERENCES


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