Young Patient With Chest Pain? Suspect Cocaine

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SAN FRANCISCO — Consider cocaine use when diagnosing chest pain, especially in young patients, Dr. Priscilla Hsue said at a meeting sponsored by the California chapter of the American College of Cardiology.

In 2004, 2 million Americans were cocaine users, and cocaine was the most frequently used illicit drug among patients seeking care in emergency departments. Yet one study found that only 13% of patients who presented with chest pain to an emergency department were assessed for cocaine use.

"I was covering the cardiology service a couple of weeks ago, and almost every day 50% of our admissions were for some kind of side effect from cocaine," said Dr. Hsue of San Francisco General Hospital. "This is something we see so often."

About 6% of patients with cocaine-associated chest pain seen in emergency departments develop MI. Patients with cocaine-related chest pain, unstable angina, or MI tend to be younger than 40 years old, male, and cigarette smokers who have no other risk factors for coronary artery disease. Chronic and first-time cocaine users have the same risk for MI. Symptoms can appear within minutes or hours after exposure to any dose of cocaine via any route—smoking, snorting, or ingesting.

Cocaine increases the risk of MI 24-fold within 1 hour of use, with the risk decreasing over time after that. The overall likelihood of MI is seven times higher in chronic cocaine users compared with nonusers. Combining cocaine with alcohol use increases the risk of sudden death by more than 20 times compared with nonusers, studies suggest.

Cocaine use increases the risk for MI in three ways: by increasing the heart rate and blood pressure in a setting of limited oxygen supply; by vasoconstriction (which is a danger especially in patients who smoke cigarettes or who have preexisting cardiovascular disease), and by promoting inflammation (possibly due to increases in C-reactive protein and platelet levels), she said at the meeting, also sponsored by the University of California, San Francisco.

Ischemic chest discomfort from cocaine use can be indistinguishable from unstable angina or non–ST-segment elevation MI due to coronary atherosclerosis.

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DR. HSUE

If cocaine use is suspected or known in a patient with chest pain who has ECG changes, treat with oral nitroglycerin and a calcium antagonist, in accordance with 2002 guidelines from the ACC and American Heart Association. If ST-segment elevations persist, perform coronary arteriography immediately. Give thrombolytic therapy if a thrombus is detected, and consider it if catheterization is not available, the guidelines state.

Those class I recommendations are backed by evidence for or general agreement about their effectiveness and usefulness. The guidelines include several class IIa recommendations based on conflicting evidence or opinions that tend to favor efficacy. These include giving β-blockers for patients with hypertension or sinus tachycardia, and giving intravenous calcium antagonists if the ECG changes suggest ischemia (J. Am. Coll. Cardiol. 2002;40:366-74).

Thrombolytic therapy is controversial because of case reports of complications. There is no way to clinically differentiate cocaine-related MI from non–cocaine-related MI, and 50%-80% of cocaine users with chest pain have abnormal ECG results that can persist for weeks, complicating the diagnosis.

On the other hand, many clinicians believe that β-blockers should not be given to patients with cocaine-induced chest pain, but this view is based on a study of normotensive patients with no prior cocaine use, she noted. The evidence for use of calcium channel blockers likewise comes from a few studies of patients not representative of cocaine users.

"These studies were small, and it's hard to base conclusions on them," Dr. Hsue commented. ■

The
SEARCH FOR SELECTIVITY
in Atrial Fibrillation

Atrial-selective ion channel blockade may reduce the risk of ventricular complications in atrial fibrillation.

Ion channels play a crucial role in cardiac electrophysiology.[1,2] Sodium channels control cell depolarization, the beginning of an action potential.[1] A variety of potassium channels then return the cell to its resting state through repolarization.[2]

In atrial fibrillation, electrical remodeling of the atria occurs such that repolarization is accelerated and the atrial action potential duration and refractory period are shortened.[3-6] This results in the disruption of the normal depolarization/repolarization cycle of atrial cells.[7]

Among the many different potassium channels in the atria and ventricles, only **Kur (ultra-rapid delayed rectifier potassium channel)** is predominantly active in the atria.[1,5,8-11] The Kur channel has not been found to be expressed in the ventricles[1,5,8-11]; therefore, selective action on this channel in the atria may reduce the risk of ventricular proarrhythmias.[8,10]

Astellas Pharma US, Inc., is exploring the *selective* blockade of Kur in the atria in order to gain a better understanding of the different pathways involved in atrial fibrillation.

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