Management of Cocaine-Associated Chest Pain and Myocardial Infarction

Scientific Statement From the American Heart Association, March 17, 2008

EPIDEMIOLOGY

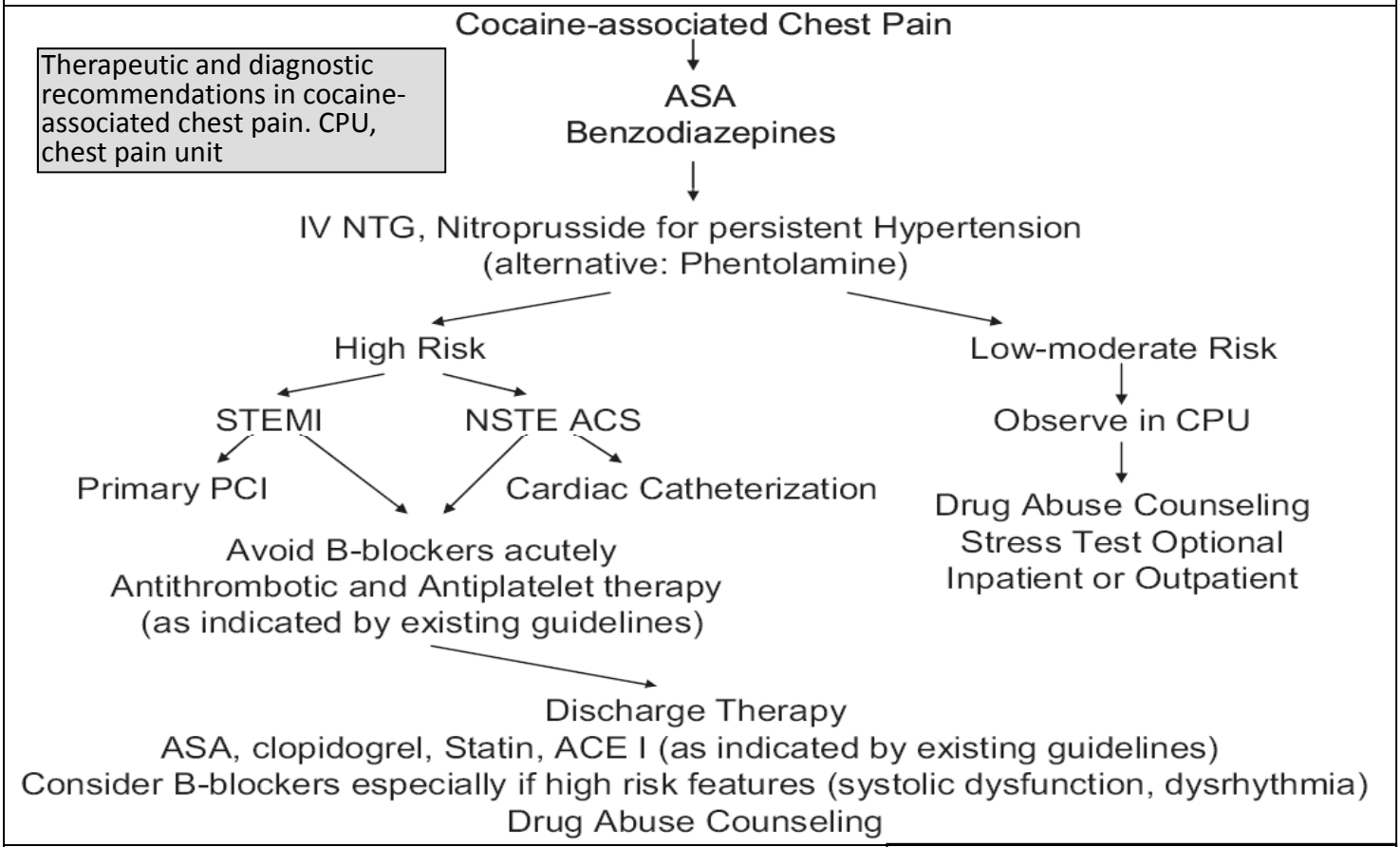
2nd most commonly used illicit drug in US after marijuana. Leads to the most ED visits

PATHOPHYSIOLOGY

Cocaine causes myocardial ischemia or MI in a multifactorial fashion that includes:
(1) Increasing myocardial oxygen demand by increasing heart rate, blood pressure, & contractility;
(2) decreasing oxygen supply via vasoconstriction;
(3) inducing a prothrombotic state by stimulating platelet activation and altering the balance between procoagulant and anticoagulant factors; and
(4) accelerating atherosclerosis

INCIDENCE OF MYOCARDIAL INFARCTION

Although the overall incidence of cocaine-associated MI varies between studies (0.7% to 6% of those presenting w/ CP after cocaine), cocaine appears to be an important contributor to MI among the young. In a study of 130 pts w/ cocaine associated MI, the average age was only 38 yrs



Therapeutic and diagnostic recommendations in cocaine-associated chest pain. CPU, chest pain unit

Scientific Strength for Treatment Recommendations for Initial Management of Cocaine-Associated Myocardial Ischemia or Infarction

Therapy	Classification of Recommendation/Level of Evidence
Benzodiazepines	I/B
Aspirin	I/C
Nitroglycerin	I/B
Calcium channel blocker	IIb/C
Phentolamine	IIb/C
β-Blockers	III/C
Labetalol	III/C

Because recidivism is high among patients with cocaine-associated chest pain, chronic β-blocker use should be reserved for those with the strongest indications, including those with documented MI, left ventricular systolic dysfunction, or ventricular arrhythmias, in whom the benefits may outweigh the risks even among patients at risk for recurrent use of cocaine. This decision should be individualized on the basis of careful risk– benefit assessment and after counseling the patient about the potential negative interactions between recurrent cocaine use and β-blockade.

DIAGNOSTIC STRATEGIES

1. *Qualitative immunoassay* detection of the cocaine metabolite benzoylecgonine in urine is the most commonly used laboratory method; it can also be detected in blood and hair

2. Benzoylecgonine has a urinary t½ of 6-8 hrs, it can be detected for ~ 24-48 hrs after use

3. In a study of 18 pts who ingested cocaine intranasally, the mean time to the first negative specimen was 43.6±17.1 (range 16 to 66) hrs

4. Individuals with long-term cocaine use (ingesting up to 10 g/d), benzoylecgonine has been detected 22 days after last ingestion

5. COCHPA (COcaine associated CHest PAin) study: sensitivity of ECG revealing ischemia or MI to predict true MI is 36%. Specificity, positive & negative predictive values of ECG were 89.9%, 17.9%, & 95.8%, respectively

6. Troponins are most sensitive & specific for diagnosis of cocaine-associated MI. Cocaine may cause rhabdo & ↑ myoglobin & CK

7. Long-term cocaine use is associated with higher LV mass index and concentric LVH. This may decrease the utility of echo, as LVH often masks regional WMA.

8. In a study of 734 patients (mean age 43±7 yrs) evaluated for ischemia symptoms after cocaine use, 90 underwent coronary angiography. In this selected, higher risk group, *50% had no significant stenosis*, 32% had single vessel disease, 10% had 2-vessel disease, and 5.6% had 3-vessel disease. Of patients with proven MI, 77% had significant coronary artery disease. Of patients without MI, only 35% had significant coronary artery disease

ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction

6.6. Cocaine and Methamphetamine Users

CLASS I

1. Administration of sublingual or intravenous NTG and intravenous or oral calcium antagonists is recommended for patients with St-segment elevation or depression that accompanies ischemic chest discomfort after cocaine use. *(LOE: C)*

2. Immediate coronary angiography, if possible, should be performed in patients with ischemic chest discomfort after cocaine use whose ST segments remain elevated after NTG and calcium antagonists; PCI is recommended if occlusive thrombus is detected. *(LOE: C)*

3. Fibrinolytic therapy is useful in patients with ischemic chest discomfort after cocaine use if ST segments remain elevated despite NTG and calcium antagonists, if there are no contraindications, and if coronary angiography is not possible. *(LOE: C)*

CLASS IIa

1. Administration of NTG or oral calcium channel blockers can be beneficial for patients with normal ECGs or minimal ST-segment deviation suggestive of ischemia after cocaine use. *(LOE: C)*

2. Coronary angiography, if available, is probably recommended for patients with ischemic chest discomfort after cocaine use with ST-segment depression or isolated T-wave changes not known to be previously present and who are unresponsive to NTG and calcium antagonists. *(LOE: C)*

3. Management of UA/NSTEMI patients with methamphetamine use similar to that of patients with cocaine use is reasonable. *(LOE: C)*

CLASS IIb

Administration of combined alpha- and beta-blocking agents (e.g., labetalol) may be considered for patients after cocaine use with hypertension (systolic blood pressure greater than 150 mm Hg) or those with sinus tachycardia (pulse greater than 100 beats per min) provided that the patient has received a vasodilator, such as NTG or a calcium antagonist, within close temporal proximity (i.e., within the previous hour). *(LOE: C)*

CLASS III

Coronary angiography is not recommended in patients with chest pain after cocaine use without ST-segment or T-wave changes and with a negative stress test and cardiac biomarkers. *(LOE: C)*