Cocaine use and acute left ventricular dysfunction

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A previously fit 36-year-old caucasian woman was admitted to the coronary care unit, in August, 1999, with a 2 hour history of central precordial chest pain associated with sweating and shaking. She had smoked ½ oz tobacco weekly for three years, and previously smoked cigarettes since age 15, but denied recreational drug use. Her mother had suffered from angina at the age of 56 years. On examination her resting heart rate was 125 beats/min and supine blood pressure was 102/67 mm Hg. There were no clinical signs of heart failure. The resting 12-lead electrocardiogram showed sinus tachycardia with normal electrical axis. There was widespread deep T-wave inversion in the anterior chest leads. Biochemical investigations including initial creatine kinase (116 IU/L) and cholesterol (5 mmol/L), were normal. Serial cardiac enzymes and troponin T measurement were also normal. Her plasma renin activity (PRA) and A-type natriuretic peptide (ANP) on admission were normal at 0–4 ng/L per s (normal range 0.14–0.69 ng/L per s) and 1–3 pmol/L (normal values 0.3–5.2 pmol/L), respectively. In view of the probable diagnosis of an unstable coronary syndrome she was treated with aspirin, oral metoprolol, intravenous nitrates, and subcutaneous low-molecular-weight heparin. Coronary angiography shortly after admission showed a normal epicardial left coronary system. Echocardiography, however, showed extensive anterolateral left ventricular hypokinesia with an estimated ejection fraction of 35–40%. On day 3, PRA increased to 0.89 ng/L per s and ANP to 4·1 pmol/L. On day 7 ANP rose further to 6.0 pmol/L. Because of her normal angiogram a pharmacological cause for her coronary syndrome was sought. A urine sample collected on admission and examined by fluorescence polarisation immunoassay was positive for the primary cocaine metabolite, benzoylecgonine. She was discharged taking aspirin 75 mg daily and carvedilol 3.125 mg at night.

When to consider cocaine as a cause of cardiovascular disease

- history or laboratory evidence of cocaine use
- unexplained symptoms or signs of heart failure
- unexplained global or focal myocardial dyskinesia on echocardiogram
- acute coronary syndrome in a patient with angiographically normal coronary arteries

restlessness, anxiety, and paranoia. Later effects include tremor and delirium, pyrexia, and convulsions. The cardiac effects of cocaine, however, are not well recognised by drug abusers. Like amphetamine, it enhances norepinephrine release and probably causes myocardial ischaemia or infarction by inducing coronary artery vasospasm. At the same time cocaine can cause focal or global myocardial dyskinesia due to reduction in myocardial ischaemia or infarction by inducing coronary artery vasospasm. Furthermore, up-regulation of tissue plasminogen activator inhibitors can predispose to thrombosis because of increased platelet aggregation and decreased fibrinolysis. Thus, myocardial infarction, or sudden cardiac death can occur in association with cocaine use. Our case shows that cocaine use can be associated with acute dilated cardiomyopathy without an increase in conventional cardiac markers of ischaemia. ANP can be raised, which suggests that this should be monitored in these patients. β-blockers are contraindicated in the presence of cocaine-mediated cardiac toxicity. A systematic approach to screening for cocaine-mediated cardiac syndromes would limit inadvertent use of β-blockers. Public health guidance on cocaine should refer to its possible serious cardiac effects. We propose that cocaine abuse should always be considered in the differential diagnosis in young patients admitted with atypical presentation of an acute coronary syndrome.

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References