Life Threatening Ventricular Arrhythmia in a Patient after Cocaine Binge Presented with Life Threatening Arrhythmias

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Short Communication

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ABSTRACT

Cocaine is the most commonly used recreational drug which has striking adverse effects like Cerebrovascular accidents and cardiovascular events ranging from myocardial ischemia to myocardial infarct and life threatening arrhythmias, which has a potential to effect even younger adults with normal cardiac functional status. Here, we present an interesting case who came with chest pain with ST elevation on EKG and had multiple episodes of life threatening ventricular tachycardia after snorting cocaine, was then found to have normal coronaries on cardiac catheterization and was resuscitated successfully.

Keywords: Cocaine, Arrhythmias, ST elevations.

INTRODUCTION

The use of cocaine as a recreational drug has reached epidemic proportions as its availability has increased in recent years G. Das, (1993). The psychiatric and social sequel of cocaine abuse has been known for a long time. Cocaine may also cause serious medical disorders like seizures and acute cardiovascular events, even in young, otherwise healthy individuals G. Das, (1993); G. Das, (1990); Kloner et al, (1992). Although the basic pharmacological properties have been extensively studied, the precise mechanisms of cocaine cardio-toxicity are complex and in part still unknown.

Case Report

A 45 year old Hispanic male with no significant past medical history was brought to ER by EMS due to severe chest pain which started couple of hours later after he snorted cocaine. EMS initially found him hemodynamically stable; with ST elevations in V1, V2 and V3 on 12 lead EKG. Patient was immediately loaded with ASA and statins, and ER was informed to activate “Code Heart”.

Fig 1: Ventricular tachycardia
Patient became unresponsive on the way to ER, the rhythm on the monitor was found to be ventricular tachycardia (Fig 1). Patient was cardioverted with 360 J. After that he reverted back to normal sinus rhyme and regained his consciousness. A 12 lead EKG done right after this, revealed normalization of ST elevations that were present previously.

Patient entered the ER stable, in sinus rhythm and with an EKG not showing any ST elevations but soon became unresponsive and the rhythm was found to be ventricular tachycardia again. Patient was defibrillated again, converted to sinus rhythm, which quickly reverted back to Ventricular tachycardia with Torsades like pattern (Fig 2).

Patient was reverted to sinus rhythm after defibrillation for 9 times in total. Patient’s electrolyte status was within normal limits and patient was stable and remained in normal sinus rhythm after nine episodes of fatal ventricular tachycardia. Patient’s three cardiac biomarkers remained negative eight hours apart. Patient was rushed to the cardiac catheterization suite for an emergency cardiac catheterization, which revealed non-occlusive coronaries. Patient’s subsequent EKG showed normal sinus rhythm.

Patient’s further course was stable; he did not complain of any further chest pains and stayed in sinus rhythm for next 4 days in CCU.

DISCUSSION

The clinical association between cocaine abuse and ischemic coronary events has been recognized for a long time, and Young and Glauber already reported electrocardiographic changes related to cocaine in 1947 Young et al, (1947). Since then, there have been increasing reports of angina pectoris and acute myocardial infarction in cocaine users even in the absence of substantial coronary artery disease Young et al, (1947); Kossowsky et al, (1984). Acute ischemic events induced by cocaine can be primarily related to an enhanced sympathetic stimulation of the cardiovascular system. By enhancing heart rate, blood pressure and the after-load of the heart by peripheral vasoconstriction, cocaine increases myocardial oxygen demand. Cocaine also increases coronary vascular resistance, reducing blood supply to the myocardium Lange et al, (1989).
Fig 3: Acute effect of Cocaine. Cocaine affects the cardiovascular system through 2 major pathways: Increased sympathetic output and a local anesthetic effect. Through increased sympathetic tone and catecholamine levels, cocaine increased heart rate, blood pressure and myocardial contractility, all of which increase myocardial oxygen demand. Myocardial oxygen supply is decreased through coronary vasoconstriction and enhanced thrombosis. Myocardial oxygen demand may exceed myocardial oxygen supply, leading to ischemia or infarction. Cocaine affects cardiac myocytes directly by blocking sodium channels, which decreases left ventricular and is arrhythmogenic.

It was demonstrated in a controlled study that a single dose of 100mg cocaine intranasally, that may correspond to a usual recreational dose, induces a mean heart rate increase of 20 beats/min and a mean rise in blood pressure of 20 mmHg Resnick et al, (1976). Fischman et al (1976) showed that 32 mg of cocaine intravenously increased heart rate by 34% and blood pressure by 15%. Heart rate and blood pressure peaked 10 min after injection and returned toward baseline by 46 min. Such hemodynamic effects are within cardiovascular alterations observed during daily life and only endanger subjects with coronary artery disease, rarely seen in younger cocaine abusers. Therefore, the eventual occurrence of intense coronary spasm seems to play an important role in ischemic events induced by cocaine (Fig 3). Howard et al, (1985) has reported a case of myocardial infarction in a 28-year-old woman using cocaine but showing no coronary risk factors and a normal coronary arteriogram, same as in our patient. In some, but not all, patients using cocaine and admitted to hospital for myocardial infarction, a coronary thrombosis could be demonstrated. Therefore, infarction may result from coronary spasm or coronary artery thrombus that lyses spontaneously, or from a combination of both. Although coronary spasm may injure the endothelium and contribute to thrombosis development [Vincent et al, (1983)], vasospasm as the sole cause for thrombosis is questionable. (Fig 3) It was, however, demonstrated that cocaine also exerts a procoagulant effect by increasing platelet aggregation and thromboxane production, but on the other hand decreases the plasma levels of antithrombin III [Chokshi et al, (1989)]. Therefore, myocardial infarction development precipitated by cocaine seems to be a complex multifactorial process: in a state of already impaired myocardial oxygen demand/supply balance, characterized by tachycardia and high blood pressure, as well as by enhanced coronary vascular resistance, an additional unpredictable stimulus might induce intense coronary spasm. Besides further worsening myocardial oxygen supply, vasospasm may contribute to coronary thrombosis formation, reducing blood supply to the ischemic zone below a critical level and thus inducing irreversible cell damage and infarction.

Cocaine abuse is associated with a high risk of developing cardiac dysrhythmias [Nanji et al, (1984); Isner et al, (1986)]. The treatment of ventricular arrhythmias depends on the interval between cocaine use and arrhythmia onset. If an arrhythmia occurs immediately after cocaine use, it will likely benefit from administration of sodium bicarbonate. On the other hand, late onset arrhythmias are probably secondary to myocardial ischemia associated...
with cocaine use and vasodilator therapy should be the mainstay of the treatment Wems et al, (1990). Standard management for ventricular arrhythmias, including lidocaine, is reasonable for persistent or recurrent ventricular arrhythmias McCord et al, (2008). The electrical stability of the heart may be impaired by ischemia as well as by the enhanced endogenous sympathetic activity induced by cocaine. Particularly during ischemia, cardiac arrhythmias may result from an alteration of the autonomic activity and from any intervention eliciting sympathetic nerve activity enhancement. The potentiation of ischemic events and direct pharmacological action of cocaine on myocytes is linked to the synergistic effects on the intracellular calcium homeostasis. During ischemia, intracellular calcium is liberated out of the sarcoplasmic reticulum, enhancing cytoplasmic calcium concentration, although the total calcium content of the myocytes remains unchanged Wems et al, (1990). During reperfusion, sarcolemma membrane undergoes damage and highly reactive oxygen free radicals contribute to the calcium overload of the previously ischemic tissue Wood et al, (2001). Cocaine furthermore contributes to these phenomena through the intracellular calcium enhancement mediated by the alpha and beta-adrenoceptor stimulation G.E. Billman, (1990). Elevation in intracellular calcium can provoke oscillations of membrane voltage occurring after the repolarization of action potentials and is known as late or delayed after-potentials. In the case where the amplitude of the after-potentials reaches the necessary threshold voltage, repetitive action potentials may be generated. This view is also consistent with observations showing that calcium antagonists protect against the cardio-toxic and the arrhythmogenic effects of cocaine. Cardio-protection by calcium channel antagonists must, however, is interpreted cautiously regarding the mechanisms involved: besides preventing calcium overload of myocytes, these agents may act by preventing or releasing coronary spasms, restoring coronary blood flow and a sufficient oxygen supply.

The local anesthetic properties of cocaine may also substantially contribute to the induction of cardiac dysrhythmias by affecting impulse conduction Kabas et al, (1990). Cocaine inhibits sodium channels responsible for the fast depolarization in action potentials and therefore reduces conduction velocity in cardiac tissue that is dependent on the rate of depolarization. This is reflected by a broadening of the QRS-complex in the ECG of whole animals treated with cocaine Przywara et al, (1989). Conduction delays and resulting unidirectional blocks may, however, create a substrate for reentrant circuits. Cocaine has moreover been shown to affect the repolarization process by inhibiting potassium efflux M.N. Levy, (1989). The resulting prolongation of action potential duration and of the effective refractory period may slightly differ in adjacent regions of the heart.

In conclusion, cocaine has multiple effects on myocardium leading to ischemia and deleterious arrhythmias. Arrhythmias are in part due to the direct effect of cocaine on cell membrane by intracellular calcium imbalance and in part secondary to the ischemia caused by the sympathetic over activity, coronary spasm and thrombosis. Calcium channel blockers should be the part of the treatment while addressing the cocaine-induced arrhythmias.

REFERENCES


