

# Intra-operative Refractory Hypotension compounded by Chronic Cocaine use

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## Abstract

The increasing use and abuse of cocaine in Western cultures is an issue of great national and international concern. As anesthesiologists taking care of patients with cocaine abuse we have to be aware of the implications of substance abuse on anesthetic management. We report a case of 42-year-old patient with chronic cocaine use who underwent thyroid lobectomy under general anesthesia and developed refractory hypotension needing multiple vasopressor drugs.

## INTRODUCTION

Illicit drug use may deplete catecholamine levels, impairing their function and therefore a number of biological reactions. Cocaine indirectly acts as sympathomimetic drug by inhibiting the reuptake of norepinephrine (noradrenaline) and dopamine at sympathetic nerve terminals. It can also stimulate release of norepinephrine from the adrenal medulla. Chronic use of such drugs, can lead to depletion of catecholamines and also decreased sensitivity of receptors.

Anesthesia itself causes impairment of cardiac autonomic regulation, which is well tolerated in healthy individuals.

We report a case of refractory isolated severe hypotension needing three vasopressors to maintain systolic blood pressure of 100 mmHg. In ruling out other causes of hypotension, this was a case of hypotension most likely resulting from chronic cocaine use and catecholamine depletion.

## CASE REPORT

A 42-year old man was scheduled to undergo right thyroid lobectomy, for right thyroid nodule. His past medical history was significant for hypertension, high cholesterol, moderate aortic regurgitation, hypothyroidism, and chronic cocaine use. His medications included, Aspirin (stopped week before surgery) ramipril, diltiazem, hydrochlorothiazide, synthroid.

His blood tests including thyroid function tests were within normal limits.

Patient was 6 feet tall and weighed 248 pounds. His

preoperative blood pressure on the day of surgery was 145/75 mmHg, and heart rate was 75/min. The induction of anesthesia was performed with preoxygenation and intravenous administration of 2 mg midazolam, 150 mcg of fentanyl, 200 mg of propofol and 20 mg of cisatracurium. Trachea was intubated with size 7 endotracheal tube in first attempt. Anesthesia was maintained with air oxygen mixture of 50 % each and one MAC of Desflurane.

Surgery was commenced and approximately 20 minutes after the procedure was begun, patient's blood pressure started to decrease to systolic in 70mm Hg, needing boluses of phenylephrine 40 mcg. each. Since there was no satisfactory response to phenylephrine, causes of hypotension were considered. ECG was normal and had no ST segment changes, there was no change in cardiac rate and rhythm, oxygen saturation was normal. Anaphylactic reaction was considered and serum tryptase levels were sent. Intravenously steroids and benadryl and 1 liter of lactated ringers solution were given. However this was not classic anaphylaxis as there were no skin rashes, bronchospasm. This was case of isolated hypotension. The patient continued to be hypotensive and was started on epinephrine drip at 2-3 mcg/min and volume replacement. An arterial line was placed and arterial blood gas values were pH 7.25, pCO<sub>2</sub> 43 mmHg, pO<sub>2</sub> 119mmHg, Na 136 mEq/L, K 3.4mEq/L, iCa 1.2, Hematocrit 50%, glucose 251 mg/dL. Sodium bicarbonate was given to correct metabolic acidosis. Blood was sent for measurement of cardiac troponin enzyme levels. Despite phenylephrine and epinephrine drip the systolic blood pressure continued to be lower than 100 mmHg, and

patient was started on vasopressin drip 2-4 U/hr. The patient showed relatively better response to vasopressin with systolic blood pressures above 100 mmHg, so epinephrine drip was weaned and discontinued by the end of surgery. Surgery was completed in one and half hour. Since the patient had good hemodynamic parameters on only vasopressin drip of 2U/hr. patient was emerged from anesthesia and was extubated on meeting extubation criteria. Transthoracic Echocardiography performed in the recovery room showed left ventricular hypertrophy, aortic regurgitation but no wall motion abnormalities and good left ventricular function. Vasopressin drip was weaned off and discontinued within one hour in the recovery room.

### **DISCUSSION**

The 2005 National Survey on Drug Use and Health reported approximately 33.7 million Americans aged 12 and older (13.8% of Americans in that age group) tried cocaine at least once. Cocaine is the second commonest illicit drug used and the most frequent cause of drug related deaths.<sup>2</sup>

Cocaine abuse has crossed social, economic, geographic and international borders and today it remains a major problem (of global proportions) facing our society.

Cocaine is an ester local anesthetic, which acts as a powerful sympathomimetic agent. It blocks the presynaptic reuptake of norepinephrine and dopamine producing high level of these neurotransmitters at the postsynaptic receptors. Cocaine may also increase the release of catecholamines from central and peripheral stores.<sup>3</sup> Cocaine use has been associated with cardiovascular effects such as chest pain, myocardial infarct, arrhythmias, cardiomyopathy, myocarditis, stroke, endocarditis, aortic dissection. Cocaine has been shown to have direct effects to impair baroreflex functions independently of autonomic effects.<sup>4</sup>

Cocaine in initial phase causes increase in intracellular concentration of calcium, but later in course of cocaine use, there is decreased intracellular calcium concentrations resulting in depressed myocardial function. Several studies demonstrate that chronic cocaine use has a direct depressive effect on LV function. This effect seems to be independent of myocardial blood flow and coronary artery diameter. Long term cocaine use has been associated with regional LV diastolic dysfunction when analysed by MRI. Regarding the subacute and chronic cardiomyopathies, a clear association has been made with ischemic cardiomyopathy and cocaine use.<sup>5,6</sup>

Our patient had sudden unexpected severe hypotension. This hypotension was 20 minutes after hemodynamically stable induction, so could not be attributed to induction drugs. There was no surgical factor that could be attributed. Patient had no ST segment changes or arrhythmias to suggest ischemic changes in the heart. Post-operative follow-up on troponin levels was negative. Such isolated hypotension could have been due to anaphylactic reaction, however the hypotension was not immediately preceded by any drug, there were no skin rashes or change in peak airway pressures and bronchospasm. Follow up on tryptase levels was within normal limits.

Hemodynamic instability activates a complex array of interacting neural and hormonal reflexes, typified by two distinct phases mean arterial pressure is maintained. In the first phase, there is a progressive increase in systemic vascular resistance and heart rate, primarily resulting from activation of the sympathetic nervous system and the renin-angiotensin system. The second reflexive phase is accompanied by a withdrawal of sympathetic tone and the importance of renin, angiotensin, and vasopressin in protecting against further reductions in mean arterial pressure.<sup>7</sup> Chronic use of cocaine leads to depletion of catecholamines, in addition to this patient was treated with Renin-angiotensin antagonist (ramipril), which may have suppressed Renin-angiotensin mechanism that can help maintain blood pressure within normal limits.<sup>8</sup> Also this patient was on hydrochlorothiazide and may have had lower intravascular fluid volume, which is reflected by his hematocrit on higher side by blood gas analysis. However later causes of hypotension are amenable to fluid resuscitation and small doses of vasopressors.<sup>9</sup> The fact that this patient had refractory hypotension that needed to be supported with three vasopressors such as phenylephrine, epinephrine, and vasopressin suggests that this patient had catecholamine depletion, which responded to exogenous supply.

In conclusion, anesthesiologists must be aware of the cardiac complications associated with chronic cocaine use. After prolonged cocaine use, body's ability to continually release neurotransmitters for even normal physiologic purposes is hindered. Essentially they are "used up" and even additional cocaine use will not have the same physiologic effect. This results in an overall depletion of synaptic neurotransmitters and a depressed, rather than excitatory, state.<sup>10</sup>

In cases of refractory hypotension differential diagnosis

must be thought of and treatment must be directed accordingly.

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