© Am J Case Rep, 2013; 14: 76-79
DOI: 10.12659/AJCR.883837

Received: 2012.10.29
Accepted: 2012.11.12
Published: 2013.03.18

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

Coronary spasm after the topical use of cocaine in nasal surgery

Guy D. Lenders[1ABEF], Philippe G. Jorens[2AEFG], Tim De Meyer[1AE], Tom Vandendriessche[1ABDE], Walter Verbrugghe[2AE], Christiaan J. Vrints[1AEG]

[1] Department of Cardiology, University Hospital Antwerp, Edegem, Belgium
[2] Department of Critical Care Medicine, University Hospital Antwerp, Edegem, Belgium

Summary

Background: Cocaine is a frequently used recreational drug which imposes important health problems with even life-threatening cardiotoxicity. The therapeutic use of cocaine is nowadays restricted to topical anesthesia in ophthalmological and nasal surgery but the possible hazards of this local anesthesia are not always fully appreciated.

Case Report: A 51-year old male patient with moderate cardiovascular risk profile underwent elective nasal surgery and cocaine was used as a local anesthetic. During surgery, ventricular arrhythmias and cardiogenic shock occurred, mimicking an ST-segment elevation myocardial infarction (STEMI) in sinus rhythm. Coronary angiography showed diffuse spasm of the right coronary artery (RCA) which disappeared with intracoronary nitrates. Urine analysis was positive for cocaine. The patient recovered completely with a normal echocardiography and ECG at discharge.

Conclusions: Cocaine cardiotoxicity is not uncommon in the community but a particular situation arises when used in medicine as a topical anesthetic. This is the first case report, to our knowledge, of a cardiogenic shock mimicking a STEMI with documentation of diffuse coronary spasm after cocaine use in nasal surgery. One must be aware of the potential life-threatening complications in this low-risk surgery, moreover when safer alternatives are available.

key words: **cocaine – therapeutic use • nasal surgery • coronary spasm**

Full-text PDF: http://www.amjcaserep.com/fulltxt.php?ICID=XXXXXX18835

Word count: 1323
Tables: 1
Figures: 2
References: 14

Author's address: Guy D. Lenders, Department of Cardiology, University Hospital Antwerp, Edegem, Belgium, e-mail: guy.lenders@gmail.com

Background

Cocaine (benzoylmethylecgonine) is an alkaloid which is obtained from the leaves of the coca plant and is considered to be one of the most frequently used illegal recreational drugs worldwide. Cardiac disease can result from both chronic and first time use with a broad spectrum of clinical manifestations including ischemia with even myocardial infarction, thromboembolic events, malignant hypertension and ventricular arrhythmias with different pathophysiological mechanisms responsible for this cardiotoxicity [1]. While the user himself can be held responsible for this disease in recreational use, a different situation arises when cocaine is used as a therapeutic agent.

Cocaine has been used in medicine since 1884 as a locally applied drug because of its unique combination of anesthetic and vasoconstrictive characteristics [2]. Nowadays it has been largely become obsolete but still is used as a topical agent in nasal and ophtalmological surgery.

We present a case report where cocaine, used as a topical anesthetic agent in nasal surgery, induced a ST-segment elevation myocardial infarction (STEMI) with cardiogenic shock and ventricular arrhythmias secondary to diffuse coronary spasm. The relevant literature is reviewed and the pathophysiology of this cocaine cardiotoxicity is summarized.

Case Report

A 51-year old male obese patient (BMI 31) with no significant cardiovascular history despite some moderate and untreated hypertension underwent elective nasal septoplasty. Pre-operative assessment revealed no contra-indications for surgery. After general anesthesia, a mixture of cocaine (2cc of a solution of 10 g cocaine in 200cc, which is an estimated total of 100 mg) and adrenaline (1:1000) was applied in both nostrils for local anesthesia and vasoconstriction.

Twenty minutes into surgery, multiple ventricular arrhythmias (ventricular tachycardia and ventricular fibrillation) occurred with hemodynamic instability and need for multiple external defibrillations. After administration of 300 mg amiodarone, a stable sinus rhythm was achieved. The ECG in sinus rhythm (Figure 1A) showed diffuse ST-segment elevation in the precordial leads, mimicking an anterior ST-segment elevation myocardial infarction (STEMI). Subsequent treatment for ACS was initiated with heparin (5000 U) and aspirin (250 mg) intravenously (IV) and prasugrel (60 mg) through the nasogastric tube and the patient was urgently transferred to our tertiary center for primary PCI.

At arrival in the cathlab, the patient was still sedated and intubated, hemodynamically stable with a blood pressure of 115/73 mm Hg and an ECG with normalization of the ST-T segment (Figure 1B). Ventriculography in right anterior oblique (RAO) 30° showed an akinetic anterior wall with normokinesia in the other segments, consistent with important anterior ischemia.

Coronary angiography was performed and wedging of the pressure curve occurred when the RCA was cannulated. A gentle injection of contrast revealed a diffuse spasm of the entire right coronary artery (Figure 2A). After administration of intracoronary nitrates, a gradual normalization of the diameter of the lumen occurred with after one minute an angiographically normal RCA without any stenosis or spasm (Figure 2B). Cannulation of the left coronary artery (LCA) was then performed and contrast injection showed a normal LCA without any lesion or spasm in the left anterior descending artery (LAD), which was the suspected culprit artery (Figure 2C).

The patient was subsequently treated medically with nitrates IV and non-dihydropyridine calcium antagonists. After 1 day he could leave the intensive care unit and he could be discharged at home after 5 days. Troponin I level was elevated up to 1.35 µg/L (normal value <0.045 µg/L). Urine analysis was frankly positive for cocaine. Before discharge a transthoracic echocardiography showed a full recovery of the left ventricular function and the ECG was totally normal.

Discussion

Cardiac complications after the use of cocaine are not uncommon. Although they have been described in many case reports [3], they are probably still underreported. Reports of iatrogenic cardiac complications on the other hand, are more rare. A search through literature revealed only 7 case reports of significant cardiotoxicity after the topical application of cocaine in nasal surgery, most of whom presented with myocardial ischemia, non ST-segment elevation myocardial infarction (NSTEMI) or ventricular arrhythmias [4–6,8–10]. Only one case showed ST-segment elevation myocardial infarction (STEMI) [7] but coronary

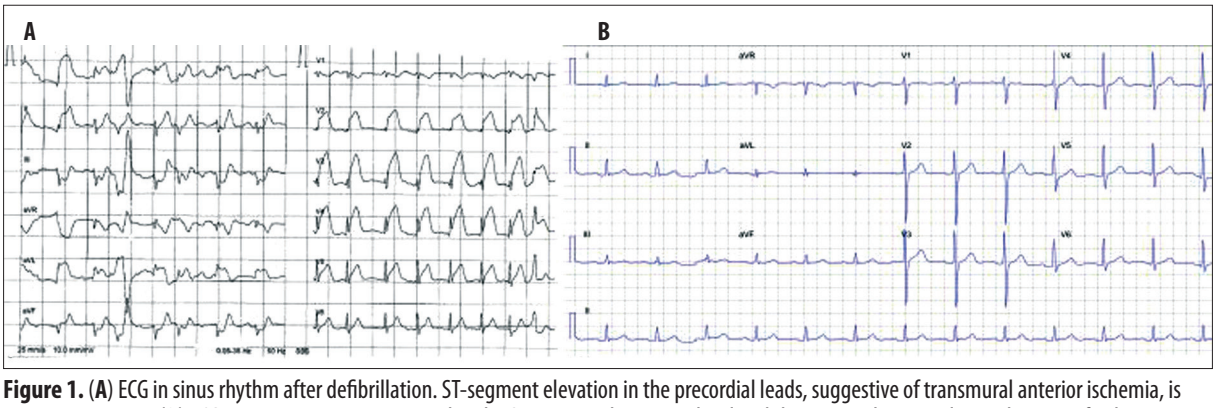

Figure 1. (**A**) ECG in sinus rhythm after defibrillation. ST-segment elevation in the precordial leads, suggestive of transmural anterior ischemia, is present. (**B**) ECG prior to coronary angiography. The ST-segment has normalized and there is no electrocardiographic sign of ischemia.

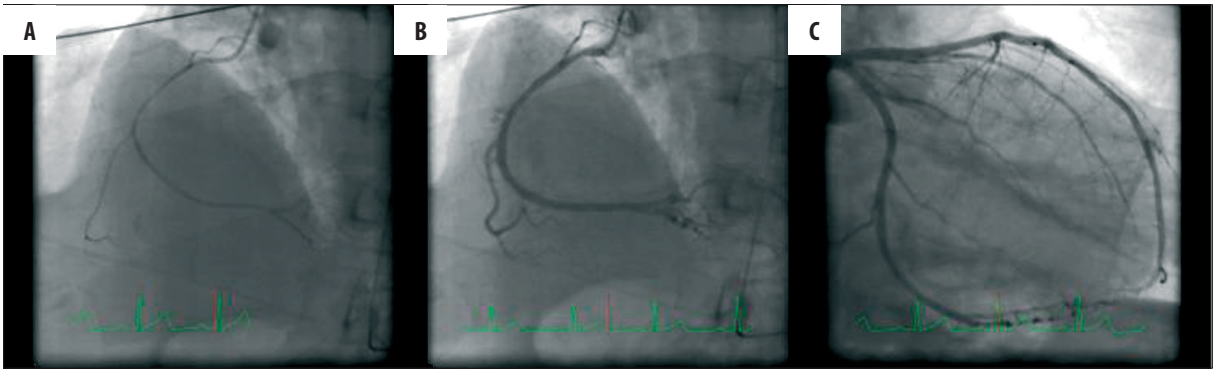

Figure 2. (**A**) Coronary angiography of the right coronary artery (RCA) in left anterior oblique (LAO) 45°. The coronary pressure curve was wedged and a diffuse spasm is present. (**B**) Coronary angiography of the right coronary artery (RCA) in left anterior oblique (LAO) 45° after nitrates. The spasm has disappeared and the RCA is without significant stenosis. (**C**) Coronary angiography of the left coronary artery (LCA) in left anterior oblique (LAO) 45°. The RCX and LAD, which was the suspected culprit artery, show no stenosis.

Table 1. Overview of all case reports with cardiac complications after the topical use of cocaine in nasal surgery and results of coronary angiography.

Case reports	Type of nasal surgery	Cardiotoxicity	Coronary angiography
Chiu YC et al. (1986)	Reduction of nasal fracture	NSTEMI, VT	Not performed
Littlewood SC et al. (1987)	Nasal septoplasty	Myocardial ischemia	Unknown
Lormans et al. (1992)	Nasal septoplasty	VF	Not performed
Ashchi et al. (1995)	Nasal septoplasty	STEMI, VF	Normal
Laffey JG et al. (1999)	Nasal polypectomy	NSTEMI	Normal
Makaryus et al. (2006)	Sphenoidectomy + nasal septoplasty	NSTEMI, cardiogenic shock	Not performed
Torres M et al. (2007)	Nasal septoplasty	NSTEMI, VT	Normal
Lenders G et al. (2012)	Nasal septoplasty	STEMI, cardiogenic shock	Diffuse spasm of RCA

NSTEMI – Non ST-segment elevation myocardial infarction; STEMI – ST-segment elevation myocardial infarction; VT – ventricular tachycardia; VF – ventricular fibrillation; RCA – right coronary artery.

angiography was normal in this patient, as it was in other cases when coronary angiography was performed.

An overview of these reported cases, the cardiotoxicity observed and the results of coronary angiography are presented in Table 1. This report is the first case report, to our knowledge, in which a patient was diagnosed with cardiogenic shock, electrical instability mimicking a STEMI with localized akinesia on ventriculography and a coronary angiography which revealed diffuse spasm of the RCA after the topical application of cocaine.

Cocaine induced cardiotoxicity can have different clinical presentations with different underlying pathophysiological mechanisms to be responsible. The primary action of cocaine is the inhibition of the re-uptake of norepinephrine, serotonin and dopamine (Triple Reuptake Inhibitor, TRI) at preganglionic sympathetic nerve endings, resulting in euphoria and anorexia but even so in hypertension, tachycardia and diffuse vasoconstriction [11]. Acute coronary syndromes can be induced by cocaine mainly by three different pathophysiological mechanisms. The first mechanism is increased myocardial oxygen consumption because of the powerful sympathomimetic effect of cocaine which induces increased heart rate, arterial pressure and ventricular contractility [12]. Cocaine also inhibits the sodium influx into cells, which besides its local anesthetic properties can impair cardiac impulse conduction providing a substrate for ventricular arrhythmias.

The second mechanism involves the prothrombogenic state resulting from a cocaine induced increased platelet reactivity and the third is due to an alpha-adrenergic stimulation of the coronary circulation with subsequent diffuse coronary vasoconstriction. Factors that are implicated to this coronary vasoconstriction are the increased endothelial production of endothelin and the decreased production of nitric oxide [11]. This third mechanism is most likely the one responsible for the ischemia in this case report, knowing that diseased endothelium is more prone to vasospasm. The patient presented here had no significant epicardial coronary disease after the use of nitrates. However, his age, obesity and arterial hypertension, imply a not completely healthy endothelium making him more vulnerable for vasospastic stimuli. Coronary spasm of the RCA is a rather incompatible finding in this case report considering the presentation of an anterior STEMI. The probable explanation is backflow of the intracoronary nitrates from the RCA, which was filmed first, to the LCA. Subsequent imaging of the LCA showed a coronary artery after nitrates. Moreover

we believe that diffuse coronary spasm was present at the time of the hemodynamic instability.

The cardiotoxic effect of cocaine is not a new finding but the occurrence after its therapeutic use is unusual. Cocaine has been used as a therapeutic agent since 1884, mainly as a local anesthetic but nowadays it has been largely become obsolete [2]. Because of its unique potent anesthetic and vasoconstrictive properties however, cocaine still is used by many ENT-surgeons for topical anesthesia because successful anaesthesia in endonasal surgery is of critical importance for patient comfort. It is capable of reducing bleeding and pain, increasing safety during and after the procedure.

Alternative products are available and maybe should be preferred to cocaine. In a study by Tarver et al. [13], cocaine was compared with a mixture of lidocaine and oxymetazoline for its local anesthetic potency. This study showed no difference in efficacy without the risk for cardiac complications with lidocaine and oxymetazoline. A second study of Noorily [14] compared cocaine with the combination of tetracaine and oxymetazoline and showed the latter to be the better and safer anesthetic. These results warrant the use of these alternative agents.

Conclusions

Cocaine remains a dangerous substance with rapid systemic absorption and unpredictable cardiac effects, even when used as a topical agent. Different pathophysiological mechanisms account for this cardiotoxicity. This case report illustrates STEMI and cardiogenic shock caused by diffuse coronary spasm, secondary to topical cocaine use in nasal surgery. The wide use of topical cocaine as a local anesthetic for ENT-procedures necessitates the realization of the adverse effects of this agent, moreover when safe alternatives are available.

Acknowledgements

We thank Peter Gorissen for editing the figures.

References:

1. Minor R, Scott B, Brown D, Winniford M: Cocaine-induced myocardial infarction in patients with normal coronary arteries. Ann Intern Med, 1991; 115(10): 797–806
2. Long H, Greller H, Mercurio-Zapalla M et al: Medicinal use of cocaine: A shifting paradigm over 25 years. Laryngoscope, 2004; 114: 1625–29
3. Mohamad T, Niraj A, Farah J et al: Spectrum of electrocardiographic and angiographic coronary artery disease findings in patients with cocaine-associated myocardial infarction. Coron Artery Dis, 2009; 20(5): 332–36
4. Chiu Y, Brecht K, DasGupta D, Mhoon E: Myocardial infarction with topical cocaine anesthesia for nasal surgery. Arch Otolaryngol Head Neck Surg, 1986; 112: 988–90
5. Littlewood S, Tabb H: Myocardial ischemia with epinephrine and cocaine during septoplasty. J La State Med Soc, 1987; 139(5): 15–18
6. Lormans P, Gaumann D, Schwieger I, Tassonyi E: Ventricular fibrillation following local application of cocaine and epinephrine for nasal surgery. ORL J Otorhinolaryngol Relat Spec, 1992; 54(3): 160–62
7. Ashchi M, Wiedemann H, James K: Cardiac complication from use of cocaine and phenylephrine in nasal septoplasty. Arch Otolaryngol Head Neck Surg, 1995; 121(6): 681–84
8. Laffey J, Neligan P, Ormonde G: Prolonged perioperative myocardial ischemia in a young male: due to topical intranasal cocaine? J Clin Anesth, 1999; 11: 419–24
9. Makaryus JN, Makaryus AN, Johnson M: Acute myocardial infarction following the use of intranasal anesthetic cocaine. South Med J, 2006; 99(7): 759–61
10. Torres M, Rocha S, Rebelo A et al: Cardiovascular toxicity of cocaine of iatrogenic origin. Case report. Rev Port Cardiol, 2007; 26(12): 1395–404
11. Lange R, Hillis D: Cardiovascular complications of cocaine use. N Engl J Med, 2001; 345: 351–58
12. Long H, Greller H, Mercurio-Zapalla M et al: Medicinal use of cocaine: A shifting paradigm over 25 years. Laryngoscope, 2004; 114: 1625–29
13. Tarver C, Noorily A, Sakai C: A comparison of cocaine *vs* lidocaine with oxymetazoline for use in nasal procedures. Otolaryngol Head Neck Surg, 1995; 113: 370–74
14. Noorily A, Noorily S, Otto R: Cocaine, lidocaine, tetracaine: which is best for topical nasal anesthesia? Anesth Analg, 1995; 81: 724–27