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# A Successful Resuscitation of a Young Patient with Persistent Life Threatening Ventricular Fibrillation during a Minor Surgical Procedure

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***Authors' contributions***

*This work was carried out in collaboration between all authors. Author JS designed the case study, prepared the first draft of the manuscript and revised the manuscript. Author JR involved the data collection and wrote the first draft of case report. Authors VJ and SR performed the literature search, wrote the first draft of the introduction and abstract, as well as revised and edited the manuscript. All authors read and approved the final manuscript.*

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***Case Study***

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## ABSTRACT

**Aims:** A discussion about the treatment options for cardiac arrest due to cocaine toxicity.
**Presentation of Case:** This is a case report of a young man who underwent general anesthesia for a urologic procedure. He suffered a ventricular fibrillation arrest and required over an hour of Advanced Cardiac Life Support (ACLS). Team Strategies & Tools to Enhance Performance and Patient Safety (TeamSTEPPS) techniques were utilized. Within one hour from the initiation of the dysrhythmia he received femoral vessel cannulation in preparation for extracorporeal membrane oxygenation (ECMO). The hypothermia protocol was instituted early for brain protection. The patient completely recovered with no neurological or cardiovascular sequelae from this life threatening cardiac arrest. Upon further investigation, it was discovered that cocaine was consumed one day prior to surgery.

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**Discussion:** The use of cocaine is associated with multiple cardiovascular complications including ventricular fibrillation which was the probable etiology of this patient's dysrhythmia. Recent studies elicited that there are underlying anatomic substrate alterations and changes in the molecular structure when cocaine is used. Cocaine stabilizes membranes in the nervous and myocardial tissue by antagonizing sodium and potassium channels. Multiple interventions were utilized which resulted in a successful resuscitation.
**Conclusion:** The implementation of ACLS, ECMO, hypothermic brain protection and team training all aided in the complete neurologic recovery in this patient.

*Keywords: Cocaine abuse; arrhythmia; cardiac arrest; extracorporeal circulation.*

## 1. INTRODUCTION

Intraoperative cardiac arrests during anesthesia occurred approximately once per 10,000 cases. It is associated with a relatively high mortality of 61% [1]. A study analyzing the cases from 1994 to 2006 concluded that cardiac arrest associated with cocaine users were more likely to survive with neurologic recovery in comparison with an age-matched non-cocaine user control [2].

We are reporting a case of the sudden onset of ventricular fibrillation in a young cocaine using patient who underwent a minor surgical procedure. Our aim is to highlight the importance of knowledge in combining advanced cardiopulmonary life support (ACLS), extracorporeal membrane oxygenation (ECMO), hypothermic brain protection and team training for this particular patient group.

## 2. CASE REPORT

A 40 year-old man presented to the hospital for a cystoscopy, left ureteroscopy, laser lithotripsy and ureteral stent insertion. He had only a past medical history of nephrolithiasis and hydronephrosis. He was a daily smoker and an alcohol drinker but denied a history of illegal drug abuse. Physical examination revealed a normal chest auscultation and heart sounds with a heart rate of 89 beat/min and blood pressure of 143/92 mmHg. All his preoperative laboratory data including electrolytes and hemoglobin were within normal range.

Upon arrival in a small procedure room, a 20 Gauge angiocatheter was inserted for intravenous access. General anesthesia was induced with 200 mg of propofol, 100 mg of lidocaine, 100 µg of fentanyl, 2 mg of midazolam, and 100 mg of succinylcholine after 2 minutes of pre oxygenation. The trachea was intubated with a 7.5 cm tube. Anesthesia was maintained with 2% of Sevoflurane, and muscle relaxation was achieved with 30 mg of rocuronium. The patient received 80 mg of gentamicin and 1 g of ceftriaxone for antibiotic prophylaxis before the procedure started. Intraoperatively, there were several episodes of labile blood pressures from to 68/35 to 167/101mmHg with an accompanying heart rate from 66 to 140 beats / minute in response to the level of surgical stimulation. He remained in sinus rhythm throughout. The hypotension was treated with phenylephrine 100 µg with a good response. Close to the conclusion of the procedure, the rhythm acutely converted to ventricular fibrillation associated with a dramatic drop in the amplitude of end tidal carbon dioxide and loss of a palpable pulse.

ACLS was initiated promptly. The patient received asynchronous defibrillations at 360 joules fourteen times for 70 minutes. He was administered 150 mg of amiodarone followed by an additional 50 mg doses twice, 1 mg of epinephrine twelve separate times, 1 mg of atropine on four occasions, 40 units of vasopressin on three occasions and 80 mEq of sodium bicarbonate. Meanwhile, a right radial arterial line was placed for blood pressure monitoring. A right internal jugular access was established for continuous administration of vasoactive agents including epinephrine 1 µg/kg/min, norepinephrine 1 µg/kg/min, and 10 units/hour of vasopressin. However, ventricular fibrillation was sustained and refractory to all treatment. There were two occasions in which the ventricular fibrillation reverted to a normal sinus rhythm but each only lasted for 2 to 3 minutes. During that period, an intra-operative transthoracic esophageal echocardiogram (TEE) revealed normal left ventricular contractility, normal valve movement, and no visualized left or right ventricular wall dyskinesis.

Ten minutes after the initiation of resuscitation, the anesthesiologists conducted a huddle with the cardiothoracic surgical team consultation for

emergency venous-arterial extracorporeal circulation was requested. Due to the space limitation of the procedure room and the need for continued chest compressions, cannulation of femoral vasculature was a great challenge for the cardiothoracic surgeon. In the meantime, left subclavian venous access was established for central venous pressure monitoring and for further fluid resuscitation. Hypothermic brain protective therapy was initiated by applying ice packs to the patient's head to ensure cooling of the brain temperatures to 33ºC. Femoral cannulation was complicated by major vessel lacerations and guide wire retention. Situational monitoring and communication principles were used to improve the surgical conditions. Eventually, the patient was transferred to an adequately sized operation room for implementation of fully functional ECMO and repair of the femoral lacerations by vascular surgeons. His hemodynamics were maintained by infusions of epinephrine at 4 µg/kg/min, norepinephrine at 4 µg/kg/min and vasopressin at 2 units/hour while sedation was maintained with 100 µg of fentanyl and 4 mg of midazolamduring the institution of ECMO. The intraoperative resuscitation lasted three hours and the patient received 7550 ml of crystalloid, 2 units of blood and 750 ml of 5% albumin. His total blood loss was 300 mL and urine output was 1500 ml.

An hour after being transferred to the intensive care unit (ICU), the patient converted to normal sinus rhythm. In the ICU he received additional cardiac defibrillations and an amiodarone infusion. He was extubated on the second day in the ICU and completely recovered with no neurological or cardiovascular sequelae. A complete cardiac evaluation was unremarkable. The urine drugs test was inconclusive due to laboratory technical difficulties. Of note, the patient admitted during our postoperative follow-up visit that he consumed cocaine one day prior to his surgery.

## 3. DISCUSSION

Anesthetic-related mortality in healthy patients during general anesthesia for minor surgery is rare [3]. Lethal arrhythmia which is refractory to multiple cardiac defibrillations and prolonged ACLS in these patients is particularly unusual. Nevertheless, it is not uncommon in a young chronic cocaine user [4,5].

Cardiac complications from cocaine intoxication are manifested by dysrhythmia, ischemia, infarction, and sudden death [6]. Recent studies elicited that there are underlying anatomic substrate alterations and changes in the molecular structure [4,7] in the cocaine abusing patient. It has been known this is a consequence of the adrenergic effects and long-term catecholamine toxicity [4,8]. Cocaine increases catecholamines by stimulating the vasomotor center and blocking catecholamine reuptake in the sympathetic nervous system thereby increasing ventricular irritability and lowering the threshold for fibrillation [7]. Cocaine stabilizes membranes in the nervous and myocardial tissue by antagonizing sodium channels and potassium channel [5,8,9]. Therefore, it was not surprising that the sudden onset of life threatening ventricular fibrillation happened in our young patient. A major concern in the clinical anesthetic management of the cocaine abusing patient is the appearance of cardiac arrhythmias due to cation channel blockade [9]. Acute cocaine abusing patients are not good candidates for elective surgery [10]. Since the history of cocaine use was concealed by our patient preoperatively, we initially did not include it in our differential diagnosis. We considered medication error, allergic reaction, pneumothorax, cardiac outflow obstruction or valvular heart disease. Initially, the syringes were reviewed, the patient has signs of rash or bronchospasm and, the breath sounds were unremarkable. Once the structure and functional abnormality of the heart was eliminated by the use of intraoperative TEE, determining the cause of cardiac arrest became more difficult. In considering other possible differentiated diagnoses, the suspicion of drug abuse increased.

Regardless of the cause, prompt and effective ACLS performance is a key to achieve successful resuscitation. It has been known that ACLS attempts exceeding 15 minutes were seldom effective and those lasting longer than 30 minutes were usually unsuccessful [11,12]. Team training was an additional benefit to this resuscitation. Previously, every member of the perioperative team in our institute underwent team training using the Team Strategies & Tools to Enhance Performance and Patient Safety (TeamSTEPPS) program. We used the leadership skills, situational monitoring, mutual support, communication, and team structure [13]. This allowed us to quickly institute ECMO.

Extracorporeal circulation is not considered as a part of ACLS protocol due to the cost and availability of skillful professions and its related facility. However, when all resuscitation efforts fail, ECMO is a potential option to promote a patient's survival. Research indicated that patients with in-hospital cardiac arrest of cardiac origin who underwent extracorporeal CPR had a short-term and long-term survival benefit over conventional CPR. Better neurological preservation was recognized as well from this approach [14]. It has been suggested to implement extracorporeal CPR for patients who failed from conventional CPR for more than 10 min. With a prolonged CPR ranged from 60 to 180 minutes prior to establishing an extracorporeal circulation, the chance of survival was not promising [14]. In light of the above finding, our early use of ECMO was critical.

In addition, although the oxygen saturation and blood pressure were maintained in the normal range during the course of the resuscitation in our patient, the risk of brain injury might still not be avoidable even with an effective conventional CPR. A study revealed that 1.6%patients who had successful resuscitation had permanent neurological impairment [11]. Despite one study which found that hypothermia did not decrease mortality or improve neurologic function compared to normothermia for out-of-hospital arrests, [15] several other studies have shown that hypothermia improved neurologic outcome [16,17,18]. In fact, the rate of mortality was significantly lower as well in out-of-hospital arrests if mild hypothermia was instituted [18].

Sodium bicarbonate has been shown to be an antidote for sodium channel antagonists [8]. Since metabolic acidosis during the cardiac arrest further enhances sodium channel blockade with cocaine, the use of sodium bicarbonate for the treatment of cocaine-induced wide complex tachycardia and ventricular dysrhythmias has been recommended [8,19]. Large dose of sodium bicarbonate intravenous infusion during the resuscitation used by other medical institutes proved it effectively helping to restore a normal sinus rhythm [8,19]. We opted not to administer sodium bicarbonate infusion for our patient. Perhaps the duration of the ventricular fibrillation would have been shortened if we had used a sodium bicarbonate infusion. A sodium bicarbonate infusion should be considered if there is a high suspicion of cocaine use.

## 4. CONCLUSION

Our patient had a complete recovery with no cardiac and neurologic sequelae despite the fact that conventional CPR/ACLS was performed for more than an hour. Due to the fact that other common causes of cardiac arrest were eliminated, we presumed that cocaine use was the primary cause of the arrhythmia. A number of interventions were carried out to successfully resuscitate the patient. We may estimate that the use of multiple interventions (TeamSTEPPS principles, aggressive ACLS, ECMO and hypothermic brain protection) was helpful for the patient's successful recovery, although we cannot state which one was the predominant factor. Perhaps future studies will investigate if the use of ECMO early in cardiac resuscitation is beneficial when there is a high suspicious of cocaine use.

## CONSENT

All authors declare that written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

## ETHICAL APPROVAL

It is not applicable.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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