

Intraoperative Cardiac Arrest: A Rare Case of Anomalous Coronary Arteries in a Previously Apparently Healthy Young Man

Dalhat Salahu¹ , Misbahu Ahmad² , Atiku Mamuda¹ 

¹Department of Anaesthesia, Aminu Kano Teaching Hospital Kano, Nigeria

²Department of Surgery, Aminu Kano Teaching Hospital Kano, Nigeria

Correspondence to: Dr Atiku Mamuda; email: dratiku1963@yahoo.com

Received: 09 Apr 2021; Revised: 01 March 2022; Accepted: 06 Mar 2022; Available online: 11 April 2022

Summary

Sudden cardiac arrest in an apparently healthy patient is an uncommon and distressing experience for clinicians, and a prompt response improves outcome. A previously undiagnosed underlying cardiac anomaly places patients at risk of intraoperative critical incidents that could be fatal. We herein report a rare case of anomalous coronary arteries in a previously asymptomatic 25-year-old male who was rescheduled for a non-cardiac surgery after two previous episodes of intraoperative cardiac arrests.

Keywords: Cardiac arrest, Coronary artery anomaly, Anesthesia, Right coronary artery, Left coronary artery

Ann Afr Surg. 2022; 19(4): 216-220

DOI: <http://dx.doi.org/10.4314/aas.v19i4.10>

Funding: None

© 2022 Author. This work is licensed under the Creative Commons Attribution 4.0 International License.

Introduction

Intraoperative cardiac arrest during elective, non-cardiac surgery is a rare but potentially catastrophic event. The incidence of intraoperative cardiac arrest varies in different hospitals and countries, and studies have shown an incidence rate ranging from 1.1 to 34.6 per 10,000 anesthesia and a survival rate of 35% to 46.6% (1,2).

The risk factors associated with cardiac arrest in patients undergoing non-cardiac surgery include unstable coronary artery disease, cardiac arrhythmias, heart failure, valvular heart disease, pulmonary embolism, electrolyte abnormalities, hemorrhage, and the anesthetic used at the time of arrest (1,2).

Although congenital coronary artery anomalies are relatively uncommon, they are the second most common cause of sudden cardiac arrest among the young (3). Approximately 1% of the general population have been reported to have an anomalous coronary artery, ranging from 0.3% to 5.6% in studies on patients undergoing coronary angiography (3). These are often not diagnosed

until late adolescence or adulthood because of the lack of symptoms, and some teens or adults with unknown anomalies may have an initial episode of chest pain, heart failure, or even sudden cardiac death before the condition is recognized (3).

We herein report a rare case of a patient who experienced recurrent intraoperative cardiac arrest that was associated with a previously undiagnosed anomaly in the coronary arteries.

Case report

The patient is a 25-year-old man weighing 89kg who presented to our facility with an 8-week history of paraplegia following a road traffic accident. There was no loss of consciousness or other major systemic injuries. Neurological examination revealed flaccid paralysis of both lower limbs, with a power of 0/5 and hyporeflexia; sensations were intact. Magnetic resonance imaging was requested, which revealed a traumatic burst fracture of T12 with retropulsion and

compression of the conus medullaris. He was scheduled for a laminectomy and pedicle screw fixation under general anesthesia.

Pre-anesthesia review revealed a young man with no known comorbid conditions, history of prior exposure to anesthesia, or drug allergy. General examination revealed no abnormalities; chest and cardiovascular examinations were normal, with a good volume pulse of 74 bpm and blood pressure of 110/80mmHg; heart sounds were S1 and S2 only. He had good mouth opening with a Mallampati score of 2. Investigations available were urea electrolyte and creatine, full blood count and differential, fasting blood sugar, and clotting profile, and all were within normal limit ranges. Fasting guidelines were given, and a request was made for two pints of blood.

In the operating suite, a cockpit drill was done to check for oxygen supply, anesthesia equipment, and resuscitative gadgets; baseline vital signs taken with a multiparameter monitor were pulse rate, non-invasive blood pressure, and oxygen saturation (SpO₂), which were all within normal limits. An intravenous access was secured with a 16-gauge cannula with 0.9% saline infusion running. He was given a pre-medication of 0.6mg atropine and 30mg pentazocine, and he was pre-oxygenated with 100% oxygen for 3 minutes. Induction was performed with a sleeping dose of sodium thiopentone (500mg), and intubation was aided with 100mg suxamethonium. Isoflurane for maintenance of anesthesia was switched on at 2%, and 6mg pancuronium given. The patient had a witnessed cardiac arrest 5 minutes after induction of anesthesia. The inhalational agent was switched off, and cardiopulmonary resuscitation (CPR) immediately commenced with an intravenous dose of adrenaline (1mg); return of spontaneous cardiac activity (ROSC) was achieved after defibrillation with 200 J. The patient regained consciousness without any neurological sequelae and was observed in the intensive care unit for 24 hours.

The patient was reviewed by a cardiologist who ordered an electrocardiography (Figure 1) and an echocardiography (Figure 2), both of which revealed no anomalies; chest X-ray also revealed a normal lung

field. A repeat of urea, electrolyte, creatinine, full blood count, and differential blood count also showed normal results.

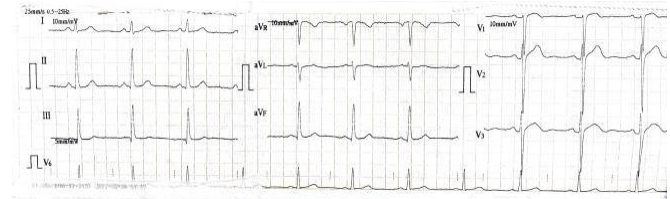


Figure 1. This is an image of the Electrocardiography of the patient with no abnormality detected

A second attempt at anesthesia was made 2 weeks after the first, with resuscitation gadgets and drugs available and ready for use. Pre-medication was performed with 0.6mg atropine and 30mg pentazocine. Induction was performed with 200mg propofol, and 100mg suxamethonium was used to aid intubation. Isoflurane (1.4%) was switched on for maintenance of anesthesia, but the patient again had a witnessed cardiac arrest with sinus bradycardia, and then asystole was seen on ECG monitoring. CPR was commenced, and 1mg adrenaline was administered; ROSC was again achieved after a 200 J defibrillation, and the patient had full recovery on the operating table.

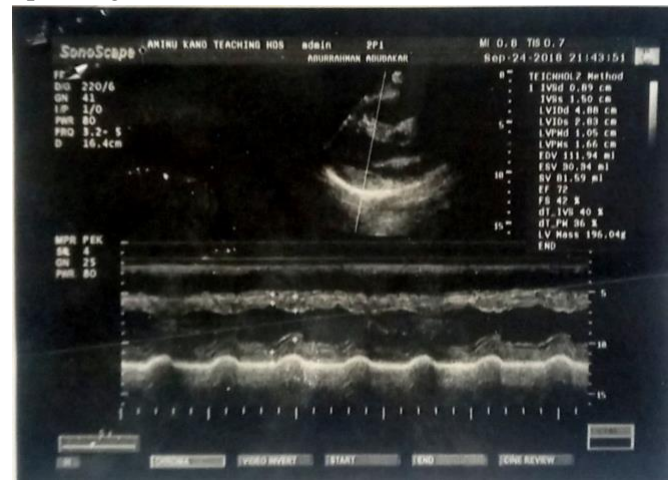


Figure 2. This is an image of the echocardiography of the patient with no abnormality detected

A further review of the patient was then carried out with a request for coronary angiography (Figures 3 and 4), which revealed features of a tortuous right coronary artery, and all coronary arteries were observed to be unusually long and prone to kinking (Figure 5).

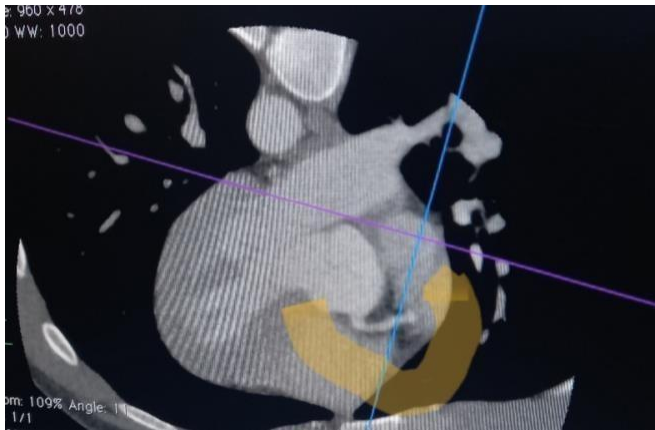


Figure 3. Image of patient's coronary angiography showing tortuous right coronary artery

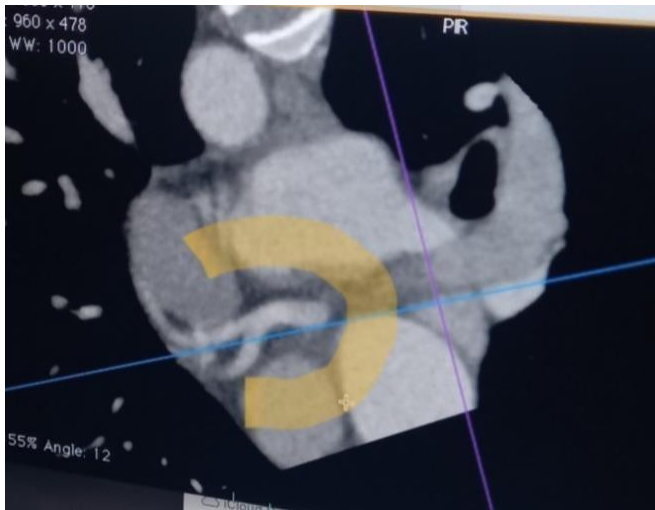


Figure 4. Image of patient's coronary angiography showing tortuous right coronary artery

The patient was then planned and prepared for surgery, after a cockpit drill and ensuring availability of resuscitation gadgets and drugs; baseline vital signs taken were within normal limits, with a pulse rate of 89 bpm, blood pressure of 128/86mmHg, and SpO₂ of 99%. With 0.9% saline running, the patient was pre-medicated with 0.6% atropine and pre-oxygenated with 100% oxygen for 3 minutes; induction was performed with an infusion of a mixture of ketamine 2mg/kg and propofol 2mg/kg given over 7 minutes using an infusion pump. He was then intubated with the aid of suxamethonium (100mg) and connected to the breathing circuit. Total intravenous anesthesia (TIVA) for maintenance of anesthesia was used with a 1:1 mixture of ketamine

(200mg) and propofol (200mg), which was prepared as a 24-mL solution and set at a rate of 10mL/hour.

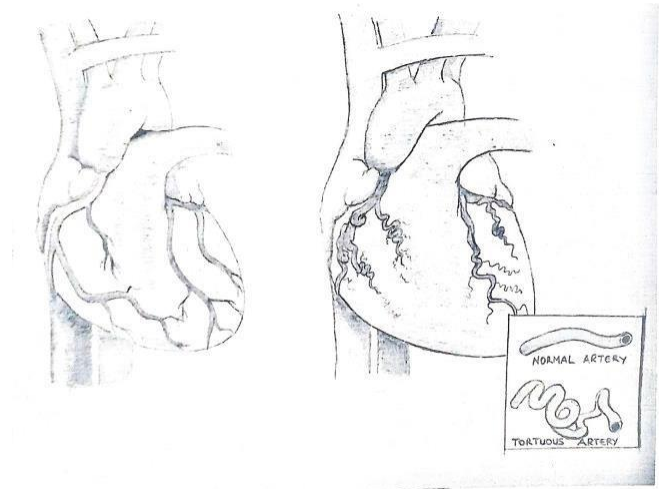


Figure 5. This is a graphical representation showing normal coronary arteries and abnormal coronary arteries which are prone to kinking

Muscle relaxation was achieved with pancuronium (6mg); he was then mechanically ventilated using the synchronized intermittent mandatory ventilation mode. The patient was turned to the prone position; pressure areas were padded, and a free abdomen was ensured before commencement of surgery. Pulse rate, SpO₂, and ECG were continuously monitored, and blood pressure was recorded every 3 minutes, and all remained stable. We however observed a drop in blood pressure 75 minutes after induction of anesthesia to 90/60mmHg; the TIVA infusion rate was then reduced to 6 mL/hour with good effect. The patient received a 2-mg top-up of pancuronium 1 hour after the first. The duration of surgery was 1 hour 45 minutes; at the end of the procedure, the residual effect of muscle relaxant was reversed with a neostigmine atropine mixture, and the TIVA infusion stopped. The patient then continued to breathe spontaneously and was extubated while awake 8 minutes after TIVA was stopped; vital signs remained stable in the immediate postoperative period, and he was transferred to the ward after 1 hour in the recovery room.

Discussion

The coronary arteries, which comprise the right coronary, left circumflex, and left anterior descending arteries, are the arterial blood vessels of coronary

circulation, and they transport oxygenated blood to the heart muscle. The heart requires a continuous supply of oxygen to function and survive (3).

Congenital coronary artery anomalies, with the vessel originating from the contralateral aortic cusp, are one of the most important cardiovascular causes of sudden death among young adults and athletes (4). This is probably related to an acute angulation at the vessel's origin and an anomalous slit lumen of the artery, and this predisposes to arterial compression induced by exercise, resulting in myocardial ischemia and malignant ventricular arrhythmias. Another possible explanation is endothelial damage resulting from chronic compression and turbulent flow, which could lead to vasospasm during stress conditions (4).

An angiograph of this patient showed a tortuous right coronary artery and unusually long coronary arteries; these could have easily resulted into a compression of the arteries, and thus, the arteries became prone to kinking. He experienced episodes of cardiac arrest after induction of anesthesia with both sodium thiopentone and propofol, which are known to have cardiovascular effects. The most prominent cardiovascular effect of propofol during induction of anesthesia is a sudden drop in both systolic and diastolic blood pressures by as much as 30% (5). Propofol is likely to cause hypotension by inhibiting the sympathetic nervous system and by impairing the baroreflex regulatory mechanism. As a result of this sudden drop in blood pressure, our patient probably had a profound decrease in coronary perfusion pressure, and myocardial ischemia was triggered by the compression of the anomalous coronary artery. Ventricular arrhythmias could have occurred as a consequence of ischemia.

However, the use of a mixture of ketamine and propofol as induction agent in this patient offered better cardiovascular stability, with no incident of cardiac arrest. The admixture of ketamine/propofol involves the combination of propofol with its vasodilatory effects balanced by the vasoconstricting properties of ketamine, thus providing cardiovascular stability (6). A meta-analysis by Smischney et al. (6) studied the effect of ketamine/propofol admixture on peri-induction hemodynamics and demonstrated a potentially better

hemodynamic profile with ketamine/propofol admixture-based induction of anesthesia.

The incidence of right coronary artery anomaly ranges from 0.09% to 0.92%, which is higher than that of the left coronary artery (0.02–0.15%), and right coronary artery anomaly is presumed to be the most common type of hemodynamically significant coronary anomaly (7). Our patient had an unusually tortuous right coronary artery. Tortuous coronary arteries may hamper ventricular function and have been proposed as an indicator of ventricular dysfunction; they are associated with reversible myocardial perfusion defects and chronic stable angina (8,9). These suggest that coronary tortuosity may hinder coronary blood supply. In this patient, myocardial ischemia could have occurred over a period of hypotension induced by the anesthetic induction agents sodium thiopentone and propofol because of the anomalous coronary artery anatomy. Furthermore, both events occurred during the induction of anesthesia—a period normally associated with hypotension due to lack of surgical stimuli and high plasma concentrations of anesthetic drugs.

Daher et al. (4) similarly reported a case of cardiac arrest following induction of anesthesia in a patient with an anomalous origin of the left coronary artery scheduled for mastectomy. Their patient received propofol (180mg), midazolam (3mg), and fentanyl (250 µg). The surgery was eventually rescheduled and successfully carried out, and they used etomidate as induction agent, which is more cardiostable, and sevoflurane for maintenance of anesthesia. In another study of 50 previously healthy individuals whose deaths were attributed to anesthesia, Tabib et al. (10) showed anatomical anomaly in most cases. Cardiomyopathy, myocardial diseases, fibrosis of the bundle of His, and anomalous coronary arteries were the most frequently found diseases. Patients who had anomalous coronary arteries had circulatory collapse during induction of anesthesia. This was also seen in our patient, who had cardiac arrest after induction of anesthesia.

With technological advances, diagnosis of anomalous coronary arteries can more easily be made with investigations such as magnetic resonance angiography (MRA) and multislice computed tomography (CT), both

of which surpass coronary angiography. MRA should be the preferred investigation because it offers excellent precision, without exposing the patient to ionizing radiation or iodinated contrast media. These minimally invasive tests should be done after any case of unexpected cardiac arrest (4). In patients with disabling symptoms such as myocardial ischemia, a potentially life-saving treatment for anomalous coronary artery is surgical correction. This involves bypass grafting and reimplantation of the artery to its proper coronary sinus or through an unroofing procedure. The risks and benefits of a surgical procedure for this rare disorder, however, remains challenging. The avoidance of strenuous exercise, use of β -blockers, and coronary angioplasty are the current alternatives to surgery (3,4). A high index of suspicion is important, as patients with an abnormal coronary artery may be asymptomatic and otherwise healthy. Early symptoms might include chest pain, and the use of CT angiography provides an insight. Awareness of this rare cause of sudden cardiac arrest is important, and it is essential to always have resuscitative drugs and equipment available in the operating theater.

Conflict of interest

None to disclose

Author contributions

All authors contributed equally in writing and editing the original draft.

References

1. Fei H, Yufeng W, Yue W. Intraoperative cardiac arrest: a 10-year study of patients undergoing tumorous surgery in a tertiary referral cancer center in China. *Medicine(Baltimore)*. 2017; 96: e6794.
2. Braz LG, Modolo NS, Nascimento P, et al. Perioperative cardiac arrest: a study of 53,718 anaesthetics over 9 years from a Brazilian teaching hospital. *Br J Anaesth*. 2006; 96: 569–75.
3. Villa ADM, Sammut E, Nair A, et al. Coronary artery anomalies overview: the normal and the abnormal. *World J Radiol*. 2016; 8: 537–55.
4. Daher M, Rodrigues A, David B, et al. Sudden cardiac arrest in general anesthesia as the first manifestation of anomalous origin of the left coronary artery. *Rev Bras Anesthesiol*. 2012; 62: 878–84.
5. Ebert TJ, Muzi M, Berens R, et al. Sympathetic responses to induction of anaesthesia in humans with propofol or etomidate. *Anaesthesiology*. 1992; 76: 725–33.
6. Smischney NJ, Seisa MO, Morrow AS, et al. Effect of ketamine/propofol admixture on peri induction hemodynamics: a systemic review and meta analysis. *Anaesthesiol Res Pract*. 2020; 2020: 9637412.
7. Ayalp R, Mavi A, Serçelik A, et al. Frequency in the anomalous origin of the right coronary artery with angiography in a Turkish population. *Int J Cardiol*. 2002; 82:253–7.
8. Turgut O, Yilmaz A, Yalta K, et al. Tortuosity of coronary arteries: an indicator for impaired left ventricular relaxation? *Int J Cardiovasc Imaging*. 2007; 23: 671–7.
9. Gaibazzi N, Rigo F, Reverberi C. Severe coronary tortuosity or myocardial bridging in patients with chest pain, normal coronary arteries, and reversible myocardial perfusion defects. *Am J Cardiol*. 2011; 108: 973–78.
10. Tabib A, Loire R, Miras A, et al. Unsuspected cardiac lesions associated with sudden unexpected perioperative death. *Eur J Anaesthesiol* 2000; 17: 230–5.