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Case Report

Sudden Cardiac Arrest during Cervical Fusion Surgery Due to Severe Coronary Artery Spasm

Priya Nair[1], M V Kumar[2]

[1]Junior Consultant, Department of Anaesthesia and Pain Medicine, Columbia Asia Referral Hospital, Bengaluru, Karnataka, India, [2]Senior Consultant, Department of Anaesthesia and Pain Medicine, Columbia Asia Referral Hospital, Bengaluru, Karnataka, India

Abstract

Background: Coronary vasospasm can lead to myocardial ischemia and can cause significant hemodynamic instability, arrhythmia, and cardiac arrest intraoperatively. Coronary artery disease can be overlooked especially in cases in which pre-operative cardiac investigations are within normal limits. Coronary vasospasm can occur even among patients without a prior history of angina, and surgery or anesthesia itself may be strong triggers for vasospasm. Cigarette smoking can cause silent ischemic disturbances of coronary flow mediated by vasoconstriction. Early recognition can allow institution of timely and life-saving intervention.

Case Presentation: A rare case of cardiac arrest due to coronary vasospasm during general anesthesia in a patient undergoing non-cardiac surgery with no pre-existing cardiac comorbidities is presented.

Conclusion: Coronary artery spasm can occur even among patients without significant cardiac comorbidities.Smoking can lead to significant disturbances in coronary blood flow.Early recognition and timely intervention can allow institution of life saving intervention.

Key words: Coronary artery spasm, Intraoperative Ventricular tachycardia, Sudden cardiac arrest

INTRODUCTION

Coronary vasospasm can lead to myocardial ischemia, potentially causing significant hemodynamic instability, arrhythmia, and cardiac arrest.[1] Coronary artery spasm (CAS) plays an important role in the pathogenesis of variant angina as well as ischemic heart disease.[2] Early recognition can allow institution of timely and life-saving intervention and potential crisis can be averted. Without appropriate treatment, coronary vasospasm is also a potentially fatal condition due to ischemia-induced ventricular fibrillation,[3] A rare case of cardiac arrest due to coronary vasospasm during general anesthesia in a patient undergoing non-cardiac surgery with no pre-existing cardiac comorbidities is presented.

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CASE REPORT

A 62-year-old male patient was scheduled for cervical spine fusion (C3-4 and C5-C6) levels, anterior approach. The patient was a long-standing diabetic and hypertensive on medications and regular follow-ups and a good glycemic and blood pressure (BP) control for the past 12 years. He had no history of myocardial infarction, or angina pectoris or any cardiovascular abnormalities in the past. He had a smoking history of 30 pack-years. His physical examination was unremarkable. His pre-operative workup was negative for ischemic heart disease on history, examination, electrocardiogram, and echocardiogram. Pre-operative blood analysis revealed no abnormalities. No premedication was given. After the patient's arrival in the operating room, intravenous access was established. Standard intraoperative monitoring was done with lead II and V5 of the ECG was monitored. BP was 140/70 mmHg and heart rate72 beats/min. After peripheral vein cannulation, anesthesia was induced with midazolam 1 mg, fentanyl 100 mcg i.v, propofol 140 mg i.v, and muscle relaxant atracurium 40 mg i.v which were used and the patient was intubated with size 8 endotracheal

Corresponding Author: Dr. Priya Nair, Department of Anaesthesia and Pain Medicine, Columbia Asia Referral Hospital, Bengaluru - 560 055, Karnataka, India. Contact: +91-9611975964. E-mail: priyanairace@gmail.com

tube, bilateral air entry was confirmed. Anesthesia was maintained using 2–3% sevoflurane in a 45% oxygen/air. Surgery proceeded uneventfully for an hour after which sudden ST elevation [Figure 1] and hypotension were noticed, bolus of ephedrine 6 mg and phenylephrine 100 mcg were administered to the patient, simultaneously, Solumedrol infusion that was on flow was also stopped and BP was restored to normal with an MAP of 70. Rhythm was sinus and there was no ST elevation noted on ECG. After 10 min, rhythm changed to ventricular tachycardia and then to ventricular fibrillation and BP decreased to 32/18 mmHg, surgery was immediately stopped and cardiopulmonary resuscitation started as per ACC/AHA guidelines and electric defibrillation was initiated, invasive arterial line was placed in the right femoral artery, arterial blood gas was sent for analysis. Ventricular tachycardia alternating with VF persisted, noradrenaline and later adrenaline infusions were started. Throughout the resuscitation, the systolic BP was above 140 and saturation was 100 percent. Amiodarone infusion 150 mg was given i.v. Dextrose insulin infusion was started in view of hyperkalemia. Subsequently after six shocks and three bolus doses of adrenaline rhythm reverted to sinus rhythm, pulse rate was 92/min, BP was 130/80, the patient had spontaneous respiration at this time, the patient was sedated and paralyzed. A cardiology consult was obtained, and the patient was transferred from the operating theater to the coronary angiography laboratory. Angiographic images showed normal caliber left main coronary artery, left anterior descending artery, and left circumflex artery with no significant flow-limiting lesions [Figure 2]. A diagnosis of severe coronary vasospasm was made. The patient was shifted to the cardiac care unit and subsequently inotropes were tapered and stopped. Serial ECGs and cardiac enzyme studies showed no abnormalities, thus ruling out myocardial infarction and CAS was diagnosed. The patient suffered no further episodes of VT or ventricular fibrillation during his hospital stay. The patient was extubated the next day with no neurological deficits. The patient recovered well and was discharged. Consent was obtained from the patient to publish this case report.

DISCUSSION

CAS is commonly part of the spectrum of atherosclerotic coronary disease.[4] The degree of vasoconstriction during a spasm ranges from clinically undetectable to complete occlusion. Multiple studies have stated that the lesions at spasm sites have less plaque, no calcification, more diffuse intimal thickening, less lipid and necrotic core, thicker baseline medial width, more prevalent negative remodeling, less thin cap fibrous atheroma, and very small baseline luminal area.[5,6] Coronary angiography with intracoronary spasm provocation testing is the only certain and effective method for the definite diagnosis of significant CAS. ECG monitoring is the only reliable practical method to diagnose vasospastic angina during general anesthesia, with 97% of patients showing ST elevation. Of these, approximately 20% will develop ventricular fibrillation or cardiac arrest. Coronary vasospasm is common, even among patients without a prior history of angina, and surgery or anesthesia itself may be strong triggers for vasospasm. Precipitating factors such as increased catecholamine response and resulting stimulation of a-adrenergic receptors, blood flow redistribution, platelet release of vasoconstrictive substances, and physical handling of coronary arteries have been suggested in the literature.[7,8] Cigarette

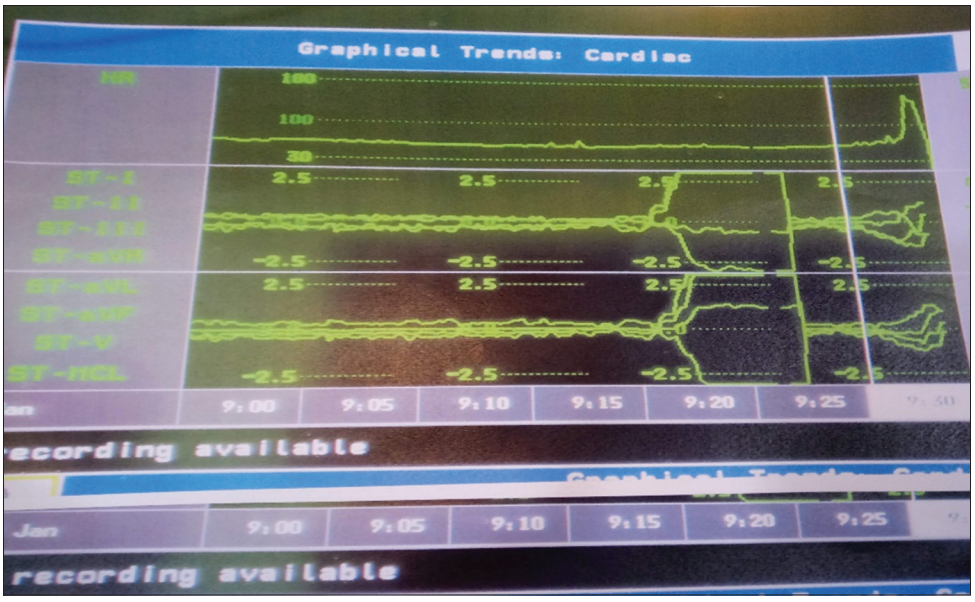

Figure 1: ST trends showing intraoperative ST elevation

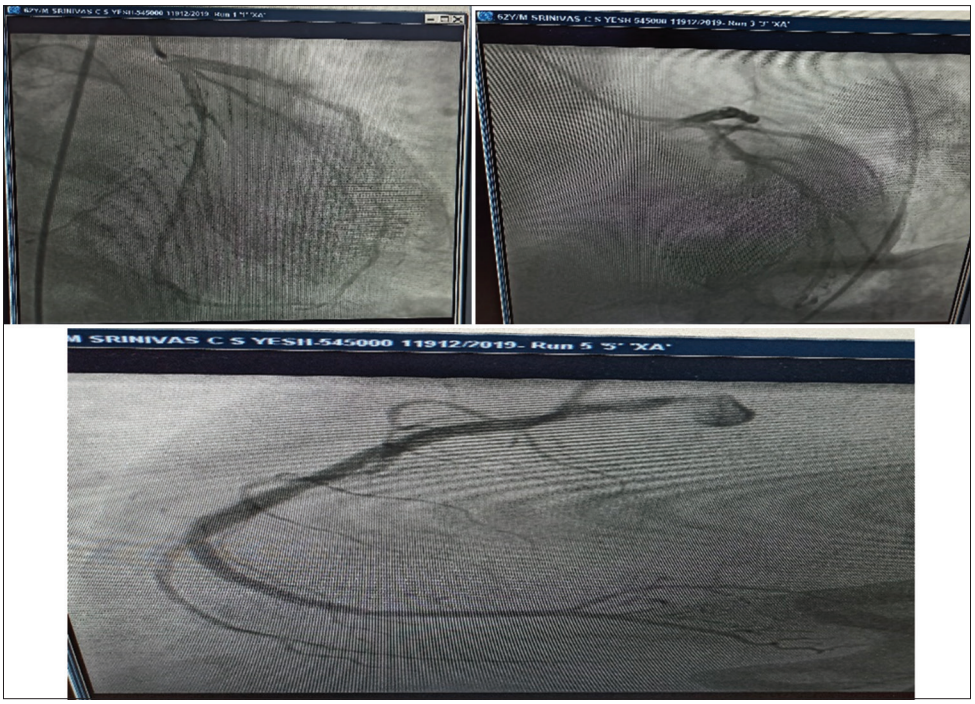

Figure 2: Normal angiogram with minimal coronary artery disease

smoking can cause silent ischemic disturbances of coronary flow[9] mediated by vasoconstriction. The episodes of ST-segment elevation in the present case were most likely due to CAS, because the episodes were not preceded by increases in either HR or BP, and the post-operative ECGs and laboratory data were normal.

CONCLUSION

Coronary vasospasm can occur even among patients without a prior history of angina, and surgery or anesthesia itself may be strong triggers for vasospasm. Cigarette smoking can cause silent ischemic disturbances of coronary flow mediated by vasoconstriction. Early recognition can allow institution of timely and life-saving intervention.

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