Coronary Vasospasm on Anesthetic Induction in a Patient with Chronic Marijuana Use

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Abstract

Coronary artery vasospasm is a rare complication that may occur in patients undergoing general anesthesia. This case presents a patient with a history of anxiety treated with cannabis and no significant cardiovascular history that had two distinct episodes of coronary vasospasm on anesthetic induction. We propose a mechanism for marijuana as a causative agent in vasospasm as an increase in sympathetic nervous system activity and catecholamine release increasing the risk of acute coronary syndrome due to increased myocardial oxygen demand. Ultimately, this case emphasizes the important of thorough history taking regarding drug use in patients receiving anesthesia and it is imperative to treat these patients as having acute coronary syndrome until it is ruled out.

Introduction

Coronary artery vasospasm is a rare but potentially life-threatening complication that may present in patients undergoing general anesthesia, however there is limited reporting of this occurrence during anesthetic induction. While the exact mechanism of coronary artery vasospasm in the perioperative setting has not been fully elucidated, various risk factors have been identified, one of which is illicit drug use. In recent years, there has been a growing body of literature implicating marijuana use in precipitating coronary artery spasm, but there is limited research or reporting on the relationship between marijuana use and development of coronary artery vasospasm during anesthetic induction.

Case Description

A 51-year-old male with a history of anxiety treated with cannabis presented for robotic inguinal hernia repair. He underwent standard induction with lidocaine, propofol, fentanyl, and rocuronium and intubation without complications, however shortly following insufflation, the patient became hypotensive, bradycardic and developed ST segment elevations. Despite treatment with phenylephrine, epinephrine, and vasopressin and reduction of the amount of inhaled agent, symptoms persisted, and the case was aborted. Cardiology was consulted, and subsequent cardiac catheterization revealed myocardial bridging of the left anterior descending artery and the patient was diagnosed with coronary vasospasm.

Four weeks following the initial episode, the patient presented for rescheduled inguinal hernia repair. After administration of midazolam preceding induction, the patient developed hypotension, bradycardia, and ST segments elevations. Risk stratification yielded the decision to proceed with surgery, which was performed without major intraoperative complications.

Patient Outcome

Postoperative EKG showed no ST segment deviation and transthoracic echocardiogram revealed normal qualitative LV ejection fraction, no wall motion abnormalities and no significant valvular disease.
Patient was kept overnight after aborting the procedure for observation and was discharged the following day in stable condition.
The patient was encouraged to stop marijuana use for one month prior to any future procedures involving administration of anesthesia.
The patient was educated on the importance of strict medication compliance.

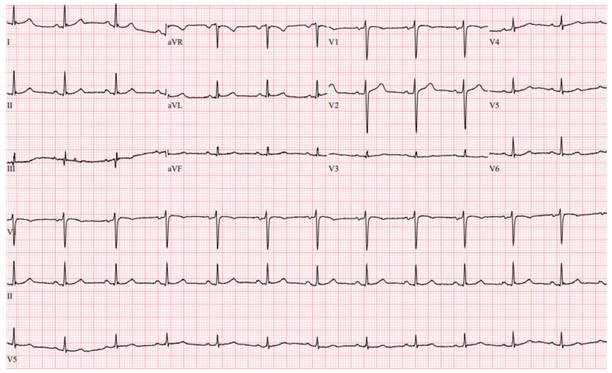

Figure 1. Postoperative Electrocardiogram

Discussion

Coronary vasospasm remains an uncommon cardiac complication during general anesthesia with limited literature detailing vasospasm on induction. Our case details a patient with two episodes of coronary vasospasm on anesthetic induction with no evidence of underlying cardiovascular disease or previous episodes of angina. While illicit substances such as cocaine and methamphetamine have been commonly associated with the pathogenesis of coronary vasospasm through adrenergic stimulation and resultant vasoconstriction leading to demand ischemia, there is limited reporting of cases that implicate marijuana use. One proposed mechanism of marijuana as a causative agent in the development of coronary vasospasm suggests that marijuana increases sympathetic nervous system activity and catecholamine release, leading to increased myocardial oxygen demand and therefore increases the risk of acute coronary syndrome.

Conclusion

While the exact mechanism between marijuana and coronary vasospasm with induction of anesthesia has yet to be defined, the paucity of literature available demonstrates the significance of this study. This case illustrates the importance of thorough history taking regarding any potential drug use in patients receiving anesthesia and regarding coronary vasospasm as a diagnosis of exclusion, it remains crucial to treat these patients as having acute coronary syndrome until ruled out.

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