

INTRAOPERATIVE CORONARY ARTERY VASOSPASM: A TWIST IN THE TALE!

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Abstract

The cause of variant angina is localized hyperresponsiveness of the vascular smooth muscle cells caused by non-specific stimuli of vasoconstriction. Autonomic imbalance can be one of the mechanisms of spontaneous vasospasm, and sympathetic or parasympathetic stimulation can induce Coronary Artery Spasm (CAS). Although various reports of CAS events have been described, episodes associated with untwisting or manipulation of a visceral structure remains unique. We report one such case of CAS in association with intraoperative untwisting of a torted ovarian cyst treated with intracoronary nitroglycerine in the catheterization laboratory.

Vasospastic or variant angina is a well known clinical condition first described by Prinzmetal and colleagues, characterized by CAS in normal and diseased coronary arteries. General anesthesia can be a triggering event. This case demonstrates unique etiology in that spasm was provoked by surgical manipulation of a torted ovarian cyst. CAS has been implicated as a cause of sudden, unexpected circulatory collapse and death during surgery, cardiopulmonary bypass, and other non-cardiac surgical procedures. There are few reports of coronary vasospasm during regional anesthesia and neuroaxial block.

Many factors are involved in the occurrences of perioperative CAS including activated sympathetic activity, activated parasympathetic activity, cocaine, alkalosis, hypercalcemia, magnesium deficiency, succinylcholine, vasopressors, essential hypertension, Hyperthyroidism, epidural anesthesia, spinal anesthesia, smoking, lipid metabolic disorder, coronary artery aneurysm, commercial weight loss products.

We describe a rare case of CAS during general anesthesia, in a patient with no past history of coronary artery disease, possibly provoked by surgical manipulation of a torted ovarian cyst, which was diagnosed and treated promptly via cardiac catheterization.

Intraoperative coronary artery vasospasm: a twist in the tale!

Introduction

Vasospastic or variant angina is a well-known clinical condition first described by Prinzmetal and colleagues¹ and is characterized by coronary artery spasm (CAS), which may occur in angiographically normal and diseased coronary arteries. If not effectively treated, CAS complications include acute myocardial

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infarction, life-threatening dysrhythmias, and death. Because of its sudden onset and its ability to mimic obstructive coronary ischemia, CAS is difficult to diagnose during general anesthesia.

The cause of variant angina is localized hyperresponsiveness of the vascular smooth muscle cells caused by non-specific stimuli of vasoconstriction². Autonomic imbalance can be one of the mechanisms of spontaneous vasospasm, and sympathetic or parasympathetic stimulation can induce CAS²⁻⁷. Although various reports of CAS events have been described, literature search did not reveal episodes associated with untwisting or manipulation of a visceral structure. We report one such case of CAS in association with intraoperative untwisting of a torted ovarian cyst.

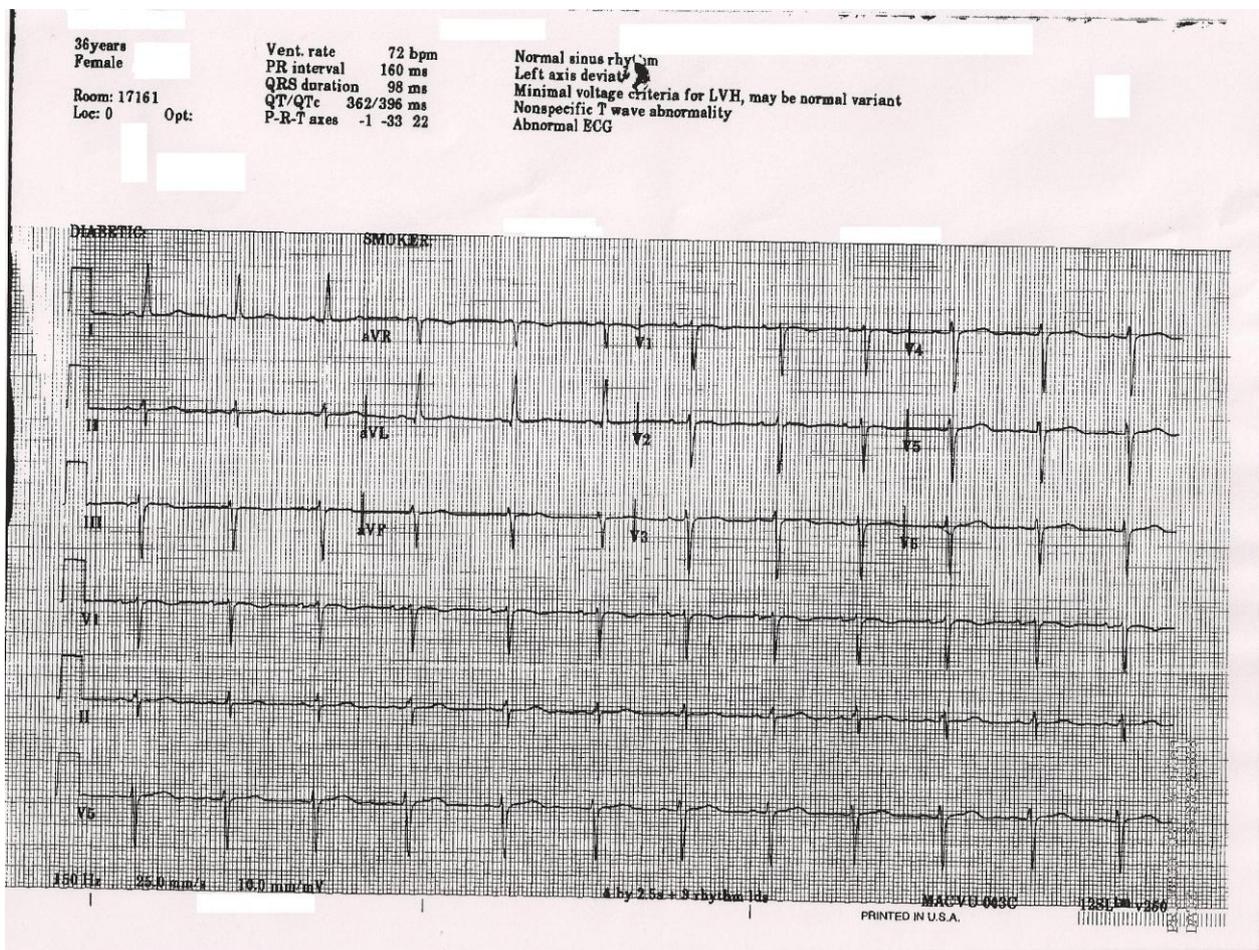
Case report

A 36-year-old Caucasian female, 5 feet 11 inches, 260 pounds, presented to the emergency department with a 2 day history of left lower quadrant pain. Ciprofloxacin treatment from her primary care physician for 24 hours with no improvement preceded presentation. On admission the pain was severe (10/10), sharp, constant, and radiating to her back and vagina.

Asthma, hypertension treated with enalapril 2.5 mg once daily, and 15 pack-year smoking defined past medical history. She had no prior history of ischemic heart disease. Past surgical history included a cholecystectomy and cesarean section. A negative history of sexually transmitted diseases and being multiparous were other pertinent findings. She had no allergies and no other medications.

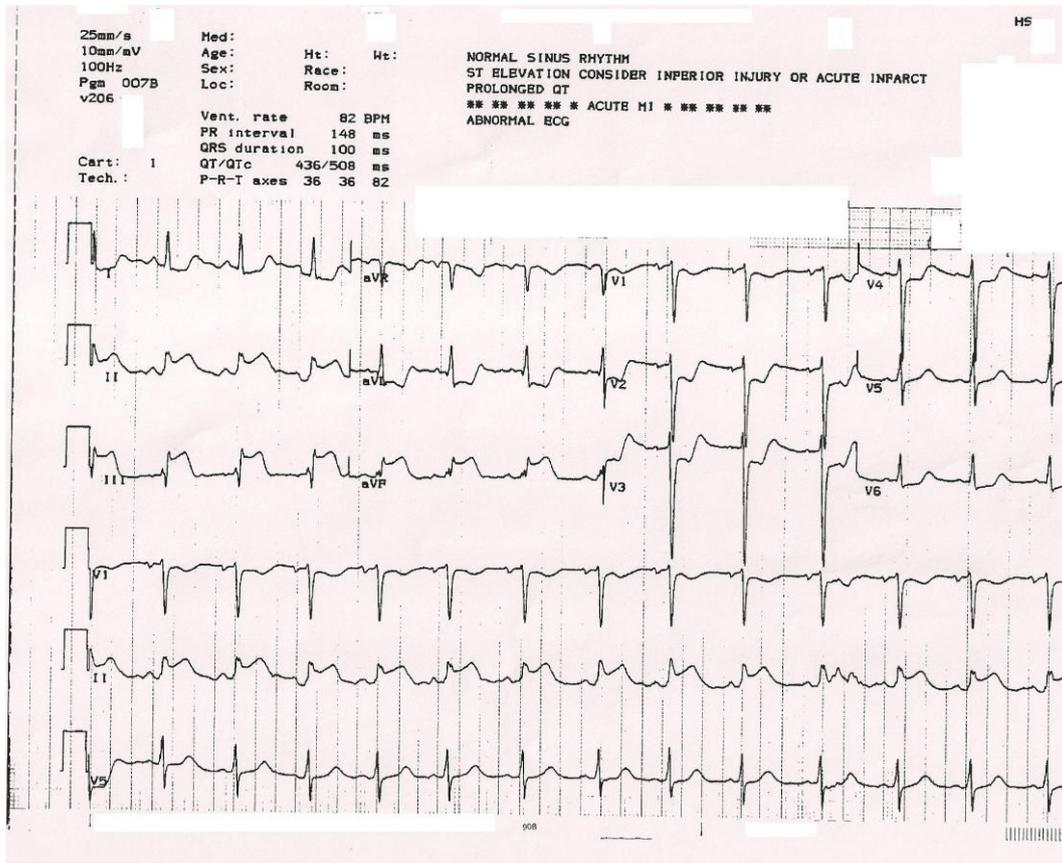
Her initial vital signs were stable with temperature of 98.5 degrees, heart rate 88 beats per minute, blood pressure 150/92 mmHg, and respiratory rate 16 breaths per minute. Tenderness to deep palpation in the left lower quadrant persisted with vaginal examination revealing cervical motion tenderness and bilateral adnexal tenderness. Ultrasound and CT scan discovered a complex, cystic, 10.3 × 13 × 13 cm left adnexal mass. Lab findings were unremarkable and urine pregnancy test was negative. She was admitted to the gynecology service and started on Cefotetan 2g q12h and Doxycycline 100 mg q12h and scheduled for a diagnostic laparoscopy. Pre-operative EKG showed no evidence of ischemia (Fig. 1).

Fig. 1



On day 3 of her hospital stay she underwent a diagnostic laparoscopy with general anesthesia. Rapid sequence induction using propofol 200 mg and suxamethonium 120 mg facilitated securing of the airway with a size 7.0 oral endotracheal tube. No instability was noted on induction. Desflurane maintained general anesthesia. Intravenous fentanyl provided analgesia and cisatracurim provided muscle relaxation. Laparoscopy revealed the left ovarian mass was torsed 3 times. Forty-five minutes following initial incision the ovary was untwisted. At this point significant ST elevation was noted on the telemetry monitor in association with a drop in systolic blood pressure from 120 mmHg to 80 mmHg and hypoxemia. Subsequent 12 lead EKG revealed significant ST elevation in LII, LIII, and aVF with ST depression in V1-4 suggestive of acute inferior-posterior myocardial infarction. (Fig. 2) Arterial blood gas analysis showed pH: 7.33, pCO₂:55, pO₂:69, HCO₃:29, BE:+2, Na:138 mmol/L, K:3.5 mmol/L, Glu:138 mg/dL, Ca:1.1, Hb:13.6 g/dL. The patient was treated with 100% O₂, crushed aspirin 325 mg via orogastric tube and boluses of Ephedrine and Phenylephrine, 30 mg and 300 mcg respectively, in divided doses. A left salpingo-oophorectomy was completed expeditiously. Interventional cardiology was consulted and the patient was transferred to the cardiac catheterization lab within 25 minutes from the onset of EKG changes under propofol anesthesia.

Fig. 2



The patient underwent emergent cardiac catheterization and coronary angiography via the right femoral artery. The right coronary artery (RCA) was a large caliber dominant vessel. Irregular and moderate stenosis was present in the proximal to mid RCA which resolved completely after injection of intra-coronary nitroglycerin. A large RCA with no evidence of acute lesion or thrombus remained, consistent with coronary spasm. Left ventricular function was preserved and pulmonary capillary wedge pressure was normal. No obvious pulmonary embolus was demonstrated on pulmonary arteriogram.

The patient was transferred to the Cardiac Care Unit (CCU) from the cardiac catheterization lab and extubated 5 hours post operatively. Subsequent Troponin studies were mildly elevated on postoperative day 1. She was discharged home on post operative day 3, with no sequelae, treated with a calcium channel blocker and nitrate. Pathology subsequently revealed a serous cystadenoma with no evidence of carcinoma.

Discussion

Vasospastic or variant angina is a well known clinical condition first described by Prinzmetal and

colleagues, characterized by coronary artery spasm in normal and diseased coronary arteries. CAS is more prevalent in the Japanese than Caucasian population. Typical EKG changes include sudden ST segment elevation in the leads overlying the ischemic region with associated ST segment depression in the reciprocal leads. The resolution phase of coronary spasm is characterized by rapid return to baseline of ST segments.

General anesthesia can be a triggering event although CAS during anesthesia is rare⁸⁻¹⁰. This case demonstrates unique etiology in that the CAS was provoked by surgical manipulation of a torted ovarian cyst. CAS has been implicated as a cause of sudden, unexpected circulatory collapse and death during surgery, cardiopulmonary bypass, and other non-cardiac surgical procedures¹. There are few reports of coronary vasospasm during regional anesthesia and neuroaxial block¹²⁻¹⁴.

Known risk factors for CAS include cigarette smoking, which this patient had, and lipid metabolic disorders. These predisposing factors may contribute to vascular endothelial dysfunction manifested as a propensity to spasm. At the cellular level, this coronary artery hypercontractility has been attributed to a reduced bioavailability of nitric oxide, up-regulation of Rho-kinase, and excessive levels of high-sensitivity C-reactive protein in the region of arterial spasm¹⁵.

The cause of perioperative CAS is unknown. Many factors are involved in the occurrences of perioperative CAS including activated sympathetic activity, activated parasympathetic activity, cocaine, alkalosis, hypercalcemia, magnesium deficiency, succinylcholine, vasopressors, essential hypertension, Hyperthyroidism, epidural anesthesia, spinal anesthesia, smoking, lipid metabolic disorder, coronary artery aneurysm, commercial weight loss products.

Potential general mechanisms to explain the occurrence of coronary vasospasm in this subset of patients include redistribution of blood flow, altered humoral factors, increased catecholamine response secondary to the level of anesthesia, and imbalance of vasoconstrictor-vasodilator forces. More specific factors thought to provoke spasm may interact in the perioperative period, including increases in blood pH, excess α -adrenergic activity, stimulation of the parasympathetic nervous system, physical manipulation of a coronary artery, and release of vasoconstrictor substances by platelets¹¹.

Disruption of the sympathetic-parasympathetic balance has been theorized as a cause of vasospasm¹⁸. Stimulation of the parasympathetic nervous system or acetylcholine administration may contribute to the genesis of the CAS. This causal relationship has been demonstrated in numerous studies^{2-7,16,17}. Acetylcholine is one of the alternatives (such as histamine, dopamine, serotonin, or hyperventilation) to ergonovine, an ergot alkaloid, which is used to stimulate α -adrenergic and serotonergic receptors to specifically provoke vasospasm.

Vagal afferents innervate a diverse range of structures of the thoracic and abdominal viscera. A proportion of these afferents function as mechanoreceptors and respond to changes in intramural tension within the structures innervated¹⁹. Mechanosensitive visceral afferent input is well represented in literature in terms of the genesis of vagal reflexes²⁰⁻²². In this patient, untwisting of the torted ovarian cyst may have resulted in such an autonomic imbalance which triggered CAS.

Prompt coronary angiography, as in this case, is the only definitive modality for early diagnosis and targeted treatment. Pharmacological testing, such as provocation with intravenous ergonovine, should be used only under special conditions and with extreme care. The response of our patient to intra-coronary nitroglycerin was dramatic. If transfer to the cardiac catheterization lab is delayed, intravenous nitroglycerine has also been reported to reverse intraoperative CAS¹¹. However, intravenously administered nitroglycerin is not always effective. Nifedipine, a calcium-channel blocker, is effective in relieving coronary artery spasm²³. Unlike

nitrates, nifedipine prevents smooth muscle contraction by inhibiting the inward calcium current during depolarization, thereby preventing excitation contraction coupling²⁴. Nifedipine may be used if CAS is refractory to nitrates. In cases with a high incidence of suspicion, a preoperative calcium-channel blocker should be administered, and nitroglycerin should be available during surgery.

In conclusion, we have described a rare case of CAS during general anesthesia, in a patient with no past history of coronary artery disease, possibly provoked by surgical manipulation of a torted ovarian cyst, which was diagnosed and treated promptly. Excellent interdisciplinary cooperation combined with favorable timing made this outcome possible without complications.

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