Unexpected Intraoperative Cardiac Arrest from Coronary Vasospasm

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Background

Intraoperative cardiac arrest, though rare, poses a significant risk during non-cardiac surgery and requires immediate recognition and intervention to reduce morbidity and mortality. Common causes include hypovolemia, myocardial infarction (MI), pulmonary embolism, electrolyte imbalances, and adverse drug reactions. We will present a case of intraoperative cardiac arrest that was ultimately caused by a less common etiology – coronary vasospasm.

Prevalence: Recent epidemiological studies have shown that coronary vasospasm occurs in approximately 2-10% of patients undergoing general anesthesia, with higher prevalence noted in those with predisposing conditions such as coronary artery disease, younger patients, and those with a history of smoking or stress-related cardiomyopathy. It's imperative to recognize at-risk patients to implement preventive measures.

The pathophysiology involves several interconnected mechanisms that contribute to the development of this condition:

1. **Vascular Smooth Muscle Dysfunction**
   - **Endothelial Dysfunction:** The coronary artery endothelium plays a critical role in regulating vascular tone by producing vasodilators like nitric oxide (NO) and vasoconstrictors. In coronary vasospasm, endothelial dysfunction may reduce NO availability
   - **Hyperreactivity of Smooth Muscle Cells:** The smooth muscle within the coronary artery wall can become hyperreactive to various stimuli (e.g., cold exposure, stress, or certain drugs), leading to excessive constriction in response to triggers.
2. **Autonomic Nervous System Imbalance**
   - An imbalance, where sympathetic activity outweighs parasympathetic control, may lead to exaggerated vasoconstrictive responses and precipitate vasospasm.
3. **Inflammatory Response**
   - Localized inflammation within the coronary arteries may contribute to vascular endothelial damage and increased smooth muscle reactivity. Inflammatory cytokines and mediators could enhance the vasoconstrictor response and reduce vasodilatory signaling.
4. **Involvement of Constricting Factors**
   - **Vasoconstrictors:** Factors such as endothelin-1, catecholamines, and angiotensin II can contribute to heightened vasoconstrictor activity. Increased levels of these substances can lead to intense coronary artery constriction.
   - **Cigarette Smoke and Stress:** External stimuli like smoking or emotional stress can elevate catecholamine release, further stimulating vasoconstriction.
5. **Role of Atherosclerosis**
   - Though coronary vasospasm can occur in normal arteries, it is often associated with atherosclerosis. The presence of atherosclerotic plaques may promote local inflammation, endothelial dysfunction, and increased vulnerability to spasm.
6. **Genetic Factors**
   - Some individuals may have a genetic predisposition to vasospastic disorders, where genetic variances affect the function of vascular smooth muscle cells and the endothelium, contributing to spasm incidents.

7. **Triggers**
   - A variety of triggers can lead to coronary vasospasm, including: physical exertion or emotional stress; exposure to cold weather, medication use (e.g., stimulants), drug use (e.g., cocaine; Hormonal fluctuations

References

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A 68-year-old male with cirrhosis, a recent knee replacement, and remote abdominal gunshot wound was admitted for a perforated duodenal ulcer. He underwent emergent exploratory laparotomy and Graham patch repair. Two days later, he returned to the OR for bleeding control.

The patient was given 2 mg Midazolam in pre-op. On arrival to the OR, the patient was hemodynamically stable on no vasopressors, as well as alert and orientated without sedation. Induction occurred at 0541 and the patient was easily intubated with a glidescope. The patient had mild hypotension following induction, which was treated with several boluses of phenylephrine (total 300 mcg). The patient no longer required vasopressors after surgical stimulation. When the surgical team indicated the procedure was nearly complete, the patient was given sugamadex in anticipation of emergence, followed by ondansetron a few minutes later. The sevoflurane was turned off. TOF indicated that paralysis had been reversed.

At 0710 the patient began to develop T wave inversions, widened QRS complexes, severe bradycardia on EKG. 1 g of calcium chloride was administered for possible hyperkalemia with no resolution and the BP continued to fall to systolic in the 40s. 0.5mg of epinephrine was administered without resolution and the patient subsequently lost pulses. CPR was started at 0711. The patient received 2 rounds of CPR, and one debrillation at 0717. The patient opened eyes and had semi-purposeful movements after the first shock with resultant ROSC. At 0721 the patient converted back to V-fib and compressions were resumed. At 0722 the patient was defibrillated again, with resultant ROSC. He had received a total of 3 mg epinephrine, 1500 mg calcium chloride, 2 mg of atropine, and 100 mEQ of bicarbonate. He was transferred to the ICU with new-onset heart failure of unclear cause. Echocardiogram post arrest demonstrated an LVEF of 25% with anterior and apical akinesis concerning for either Takotsubo cardiomyopathy or LAD infarction. Post-arrest EKG and cardiac catheterization ruled out acute myocardial infarction and coronary artery disease. While recovering, he experienced two additional cardiac arrests. Telemetry and exclusion of other potential causes led to a diagnosis of coronary vasospasm. He was started on a nitroglycerine infusion and responded well, with no further cardiac events. He was discharged to an LTAC with a tracheostomy on hospital day 33.

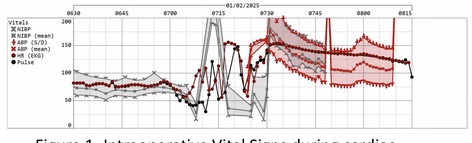

Figure 1: Intraoperative Vital Signs during cardiac arrest

ET Isoflurane (%)					[0.5]	[0.3]	[0.3]
ET Sevoflurane (%)	[1.1]	[0.1]	[0.1]				
O2 (L/min)	[0.8]	[10]	[10]	[10]	[5]	[5]	[5]
N2O (L/min)	[0]	[0]	[0]	[0]	[0]	[0]	[0]
Air (L/min)	[1.4]	[0]	[0]	[0]	[0]	[0]	[0]
piperacillin-t… (g)							
Midazolam (mg)							
FENTanyl (mcg)							
Lidocaine (IV) (mg)							
Propo… (mcg/kg/min)							20
Rocuronium (mg)				100			
PHENylephrine (mcg)							
Albumin human … (g)	*						
Sugammadex (mg)	200						
Ondansetron (mg)		4					
EPINEPHrine (mcg)			[1000]	[2000]			
Calcium Chlor… (mg)			[1000]	500			
Atropine (mg)			1	1			
norepine… (mcg/min)				[6]	[2]		
EPINEPHr… (mcg/min)					2		
Sodium Bicar… (mEq)						[100]	
Multiple Elec… (mL)							
Lactated Ring… (mL)							
Mode Of Ventilation	[Volume Mode]	[Volume Mode]	[Volume Mode]	[Volume Mode]	[Volume Mode]	[Volume Mode]	[Volume Mode]
Tidal (Observ… (mL)	[520]	[440]	[490]	[520]	[520]	[540]	[530]
Resp	[12]	[12]	[18]	[12]	[12]	[24]	[24]
PIP Obser… (cm H2O)	[26]	[44]	[46]	[28]	[29]	[39]	[39]
Plateau Pressure	[21]	[40]	[43]	[23]	[25]	[34]	[33]
PEEP/CPAP (cm H2O)	[5]	[6]	[13]	[5]	[5]	[5]	[5]
FiO2 (ventilat… (%)	[47]	[96]	[97]	[96]	[95]	[96]	[95]

Figure 2: drugs administered during the code

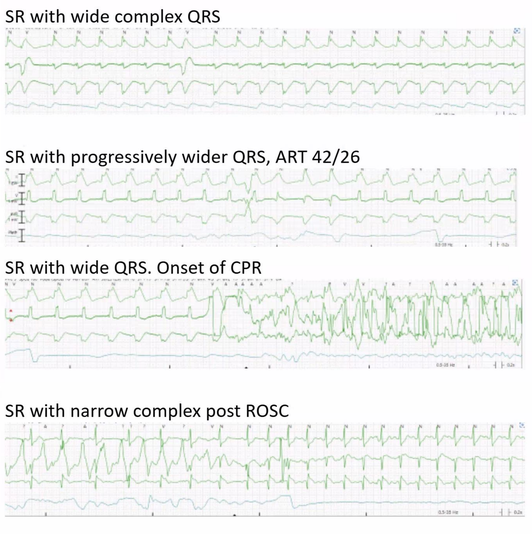

Figure 3: Telemetry during of second episode of cardiac arrest

Discussion and Best Practices

In the described case, the patient was stable throughout surgery, with minimal blood loss and no vasopressor support. Postoperative evaluations ruled out electrolyte imbalance, thrombosis, and MI secondary to coronary artery blockage. However, a subsequent cardiac arrest in the ICU revealed the true cause: coronary vasospasm. Coronary vasospasm involves sudden constriction of coronary arteries due to overactive vascular smooth muscle cells, leading to transient chest discomfort and ST segment changes on an electrocardiogram. Intraoperative triggers include vasoactive medications such as ephedrine, phenylephrine, adrenaline, and possibly ondansetron, which has been associated with cardiac effects, including vasospasm. Although vasospastic angina is more commonly seen in younger patients with fewer cardiac risk factors, it must be considered in all patients. Diagnosing it during surgery is challenging due to masked symptoms, making continuous cardiac monitoring vital. Treatment typically involves nitrates and calcium channel blockers. This case highlights the importance of considering coronary vasospasm in unexplained cardiac arrest during non-cardiac surgery.

1. Preoperative Assessment
Risk Stratification: Identify patients at higher risk for coronary vasospasm, including those with a history of coronary artery disease, vasospastic angina, or significant stress.
Medication Review: Evaluate current medications that could precipitate vasospasm (e.g., stimulants) and consider adjustments or alternative medications when possible.

2. Monitoring
Continuous ECG Monitoring: Implement continuous electrocardiographic monitoring to detect ST-segment changes indicative of ischemia or vasospasm.
Hemodynamic Monitoring: Use invasive monitoring techniques (e.g., arterial line) for continuous blood pressure and cardiac output assessment in high-risk patients.

3. Induction and Maintenance
Choice of Anesthetic Agents: Utilize agents that are less likely to promote vasospasm (e.g., avoid high doses of sympathomimetics or agents that can stimulate the sympathetic nervous system).
Gentle Induction Techniques: Maintain hemodynamic stability during induction to prevent reflex sympathetic activation, which may provoke vasospasm.

4. Medication Management
Administering Vasodilators: Be prepared to use nitrates or calcium channel blockers if vasospasm is anticipated or during episodes of vasospasm. Consider using magnesium sulfate when vasospasm does not respond to initial treatment.

5. Intraoperative Techniques
Fluid Management: Ensure adequate fluid administration to prevent hypotension, which can exacerbate coronary vasospasm.
Avoidance of Hyperventilation: Maintain normocapnia to prevent vasoconstriction associated with respiratory alkalosis.

6. Intraoperative Response
Prompt Recognition: Be vigilant for clinical signs of coronary vasospasm such as changes in heart rate, blood pressure, or ST-segment abnormalities on the ECG.
Timely Intervention: Rapidly initiate treatment with nitrates or calcium channel blockers if vasospasm occurs, and consult with cardiology if needed.

7. Postoperative Care
Close Monitoring in Recovery: Continue monitoring for any signs of cardiovascular instability in the postoperative period, especially in patients with a history of coronary vasospasm.
Patient Education: Provide guidance for patients on managing their cardiac health postoperatively, including medication adherence and lifestyle modifications.