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Case Report

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Post-Cardiac Arrest Vasoplegia following Percutaneous Coronary Intervention in a Case with Left Main Ostial Stenosis

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ABSTRACT

Post-cardiac arrest vasoplegia is a rare but potentially fatal complication that can occur following percutaneous coronary intervention (PCI). We presented a case of a 38-year-old female with sinus venosus atrial septal defect (SV-ASD) and left main ostial stenosis who developed refractory cardiogenic shock due to post-cardiac arrest vasoplegia immediately following coronary angiogram (CAG) and PCI of the left main coronary artery (LMCA). Despite aggressive resuscitative efforts, the patient succumbed to the complication 12 hours after the initial event. This report discusses the pathophysiology, management options, and challenges associated with post-CA vasoplegia in patients with underlying cardiac pathologies such as ASD and left main ostial stenosis.

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Introduction

Post-cardiac arrest vasoplegia is an uncommon complication that may arise in individuals undergoing PCI. It presents with various definitions, though a frequently used characterization involves a state of elevated cardiac output (cardiac index, CI > 2.2 L/min/m²) and reduced systemic vascular resistance (SVR < 800 dynes.sec/cm), accompanied by an inability to sustain a mean atrial pressure (MAP) of 60 mmHg despite the administration of high-dose vasopressors (0.5 µg/kg/min) [1,2]. This condition manifests as profound hypotension and diminished SVR, even after receiving adequate fluid resuscitation and inotropic assistance. The precise pathophysiology of post-cardiac arrest vasoplegia remains incompletely elucidated. It is thought to involve endothelial dysfunction due to systemic inflammation, leading to vasodilation, decreased vascular tone, and triggering activation of the coagulation cascade [3,4]. Hypoxia and reperfusion injury following cardiac arrest may lead to damage of the vascular endothelium, resulting in impaired vasomotor tone and increased vascular permeability. We report the diagnosis of post-cardiac arrest vasoplegia in a case of sinus venosus atrial septal defect (ASD) following percutaneous coronary intervention (PCI).

Case Presentation

A 38-year-old female with a known history of moderate-sized sinus venous atrial septal defect (ASD) with normal left ventricular (LV) function, diabetes mellitus (DM), and hypothyroid presented for presurgical evaluation. She underwent a coronary angiogram (CAG) that revealed tight left main ostial stenosis. Post-procedure, she developed ventricular fibrillation (VF) followed by cardiac arrest. Cardiopulmonary resuscitation (CPR) was performed immediately and a return of spontaneous circulation (ROSC) was achieved in five minutes. Her atrial blood gas (ABG) analysis revealed lactic acidosis with PH of 7.2. PCI to the left main coronary artery (LMCA) was performed through the right femoral approach. A drug-eluting stent (DES) (4.0×15mm, Xience Xpedition DES) was deployed with an intra-aortic balloon pump (IABP) support. Despite the patient being started with intravenous (IV) noradrenaline infusion (0.2 mic/kg/min), her blood pressure (BP) remained low (80/40 mmHg). The patient was started on IV bicarbonate and dopamine infusions. Acidosis got corrected in the next 30min with improvement of PH to 7.35. Despite this, the hypotension was refractory. As the echocardiogram showed normal LV function and serum creatinine level (0.9 mg/dl), vasoplegic syndrome was suspected. The patient was administered with methylene blue infusion (1.5 mg/kg) along with vasopressin and steroids, intravenously. There was transient improvement in hypotension for few hours. A reassessment was performed for any bleeding. No haematoma was noted at the groin. Ryles tube and rectal examination did not show any fresh bleeding. The patient was in refractory shock. Patient later developed DIC [disseminated intravascular coagulation] and succumbed 12hours after the initial event.

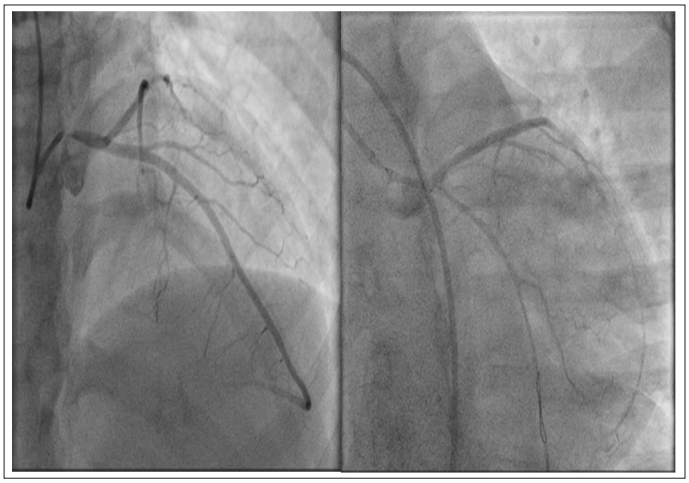

Figure 1a: Coronary Angiogram showing Critical Stenosis of Left Main Coronary Artery, 1b: Post Percutaneous Intervention of Left Main Coronary Artery

Discussion

Post-cardiac arrest vasoplegia is a common complication following cardiac bypass grafting (CABG) surgery with an incidence of 5-50% with high morbidity and mortality rates [4-6]. But it occurs rarely following a PCI. The condition's pathophysiology is not well understood, but it is believed to be related to a dysregulation of the nitric oxide (NO) pathway, resulting in vasodilation (Figure 2) and decreased vascular tone [1].

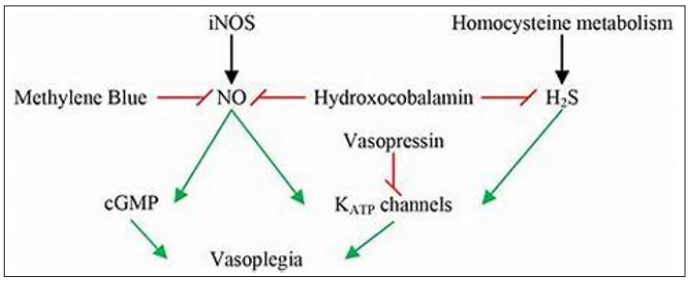

Figure 2: Pathophysiology of Vasoplegia [1]

Pathophysiology of Vasoplegia

Vascular smooth muscle undergoes physiological contraction in response to an increase in intracellular calcium (Ca2+) levels, triggering the phosphorylation of myosin and ultimately resulting in the cross-linking of myosin and actin filaments, leading to vasoconstriction. The elevation of cytoplasmic calcium levels is brought about by the activation of alpha-1 adrenergic (α1), vasopressin-1 (V1) and angiotensin type-1 (AT1) receptors. During cardiopulmonary bypass (CPB), the release of inflammatory mediators can lead to adrenoreceptor desensitization, causing an initial surge in vasoconstrictive mediators followed by their subsequent depletion, as well as the generation of nitric oxide (NO). NO, in turn, induces an increase in cyclic guanosine monophosphate (cGMP), which inhibits the influx of calcium into cells, resulting in muscle relaxation. Additionally, NO activates ATP-sensitive potassium (K-ATP) channels, leading to hyperpolarization and the suppression of vasoconstriction [1].

The condition is associated with a high mortality rate, with reported rates ranging from 30% to 80%. The most likely cause of the refractory cardiogenic shock in our case was post-CA vasoplegia. Management of post-CA vasoplegia is challenging as there is no standardized approach to its treatment. The use of vasopressors such as norepinephrine and vasopressin has been reported [1,6,7]. However, the optimal management strategy remains unclear, and treatment must be individualized based on the patient's underlying cardiac pathology and clinical presentation. The mainstay of therapy is to optimize hemodynamic support, maintain adequate oxygenation, and correct any underlying electrolyte abnormalities. In our case, we initially attempted to treat vasoplegia with a combination of inotropic and vasopressor support. Studies have shown the beneficial use of methylene blue in recommended doses (2 mg/kg IV bolus) in treating vasoplegia [1,8-10]. However, a standard guideline on the therapeutic window for methylene blue should be established as it is time-dependent [11,12]. In our case Inj. Methylene blue infusion was administered along with vasopressors and steroids. Despite aggressive management, the patient's hemodynamics remained unstable, and the patient eventually succumbed to refractory shock. The use of extracorporeal membrane oxygenation (ECMO) has been described as a rescue therapy in patients with refractory post-cardiac arrest shock. However, the decision to initiate ECMO should be individualized and made on a case-by-case basis.

Conclusion

Post-cardiac arrest vasoplegia is a challenging condition to manage and can have devastating consequences. Early recognition and aggressive management using methylene blue with inotropic and vasopressor support are crucial to improving outcomes. However, the optimal management of post-cardiac arrest vasoplegia remains unclear, and further studies are needed to establish standardized treatment protocols.

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