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Back from Irreversibility: Use of Percutaneous Cardiopulmonary Bypass for Treatment of Shock from Refractory Anaphylaxis during Coronary Intervention

ABSTRACT: Anaphylaxis during diagnostic catheterization and coronary intervention is a rare and potentially life-threatening complication. Fortunately, with standard intervention, fatality is rare. We report a case of medically refractory anaphylaxis during a coronary intervention that ultimately responded to percutaneous cardiopulmonary bypass (CPB). The proposed mechanics and use of CPB for shock are discussed.

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Anaphylaxis is a systemic hypersensitivity reaction that results from the release of vasoactive and inflammatory mediators from degranulation of mast cells causing the classical symptoms noted for anaphylaxis: urticaria, hypotension and respiratory compromise.^{1–3} The reactions can be quite severe, and without proper and immediate treatment, may result in death.

Anaphylaxis accounts for 84,000 cases and 500–1,000 deaths annually in the United States.^{1–5} Though most of these deaths occur in instances where medications are unavailable, many deaths occur because conventional measures fail. It is suggested that prolonged cardiopulmonary resuscitation (CPR), even longer than 1 hour, may be necessary for longer-acting mediators, leukotrienes and prostaglandin to be metabolized.⁴ Most patients who have anaphylactic reactions have a history of a reaction in the past and have IgE antibodies that can signal mast cell degranulation.¹ Anaphylactoid reactions are similar reactions where there is direct degranulation of mast cells without IgE involvement. Intravenous (IV) contrast exposure is commonly associated with this direct pathway. Though severe reactions to contrast are rare, premedication with steroids and H1 blockers can decrease severe reactions by 90%.³ Premedication is suggested for those patients with a history of any reactions to IV contrast.^{1–5}

We report here a case of a patient who developed refractory anaphylaxis/anaphylactoid reaction during a percutaneous coronary intervention (PCI). After 45 minutes of exhaustive resuscitation efforts and apparent failure of conventional therapy in the treatment of anaphylaxis, the patient was emergently placed on right atrial-to-femoral artery bypass (cardiopulmonary bypass [CPB]). Remarkably, within 5 minutes of initiation of CPB, the patient regained cardiac contractility and hemodynamic stability. Ultimately, he was discharged from the hospital without major sequelae.

The following case will discuss the utilization of emergency CPB as a life-saving option for medically refractory anaphylactic/ anaphylactoid shock.

Case Report. A 67-year-old male with a past medical history of hypertension, non-insulin-dependent diabetes mellitus and hyperlipidemia presented to a local community hospital emergency room with new-onset nocturnal angina. Evaluation in the emergency department was unremarkable. Electrocardiography revealed non-specific ST changes and a right bundle branch block. Cardiac biomarkers (troponin and CK-MB), however, were mildly elevated and this prompted early initiation of therapy for an acute coronary syndrome. The patient was given aspirin 325 mg, clopidogrel 600 mg, was started on IV heparin and transferred to a tertiary care center for urgent angiography. Administration of beta-blockers was held secondary to bradycardia.

Soon after receiving the aspirin and clopidogrel, the patient complained of itchiness and developed a diffuse urticarial rash over his torso. He received diphenhydramine 50 mg orally, which led to full resolution of signs and symptoms. Of note, 6 months prior to this hospitalization, the patient had a similar reaction (hypotension, near-syncope, urticarial rash and itchiness) that developed shortly after taking an oral preparation for colonoscopy. The polyethylene glycol that is present in the preparation was felt to be the offending agent at that time. The patient had no other history of atopy or allergic reaction, and had never experienced a reaction to IV contrast media.

After the patient was urgently transferred to the tertiary center for cardiac catheterization, and because of his aforementioned history, he was empirically premedicated with oral prednisone 40 mg on the morning of the procedure, followed by an on-call dose to be given 6 hours later. IV histamine-2 receptor blocker 20 mg and oral diphenhydramine 25 mg were also given before the patient was transferred to the catheterization laboratory.

Coronary angiography with non-ionic, low-osmolar contrast, demonstrated a subtotal occlusion of the mid-left anterior descending (LAD) artery with thrombolysis in myocardial infarction (TIMI) 2 flow and an estimated ejection fraction of 50% on ventriculography. Coronary intervention was performed with administration of an IV bolus of a glycoprotein IIb/IIIa antagonist (eptifibatide) prior to balloon inflation. At the time of balloon inflation, the patient complained of chest pain, itching and lightheadedness. After approximately 30 seconds of inflation, he became hypotensive with a systolic pressure of 80 mmHg. A diffuse urticarial rash was noted on his chest. Repeat angiography of the LAD showed what appeared to be TIMI 3 flow, though the patient's systemic hypotension limited our ability to adequately assess coronary perfusion. The etiology of the patient's shock was felt to be secondary to anaphylaxis. An immediate dose of IV epinephrine 0.1 mg was given via a central venous line and repeated less than 5 minutes later. The hypotension persisted despite several repeat boluses of IV epinephrine. Aggressive fluid resuscitation was administered with normal saline, and the patient was intubated for airway support. Infusions of both

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diphenhydramine 50 mg IV and methylprednisolone 125 mg IV were also given simultaneously with the initiation of IV norepinephrine and dopamine.

With the refractory nature of the patient's shock, secondary causes were also considered, including profound myocardial ischemia. An intra-aortic balloon pump (IABP) was inserted without improvement, and an emergent two-dimensional (2-D) echocardiogram was performed, revealing severe global hypokinesis and no pericardial effusion. After 20 minutes of resuscitative effort, the patient continued to have refractory hypotension and subsequently developed recurrent ventricular fibrillation. Cardiopulmonary resuscitation (CPR) was again initiated, but his ventricular fibrillation persisted despite multiple defibrillations and the administration of antiarrhythmic therapy. After 45 minutes of CPR with no response to epinephrine, norepinephrine, atropine, glucagon and IV fluids, a repeat 2-D echocardiogram was performed, revealing cardiac standstill. Lastly, because there was still a remote possibility of ischemia causing cardiogenic shock, perhaps due to unrecognized left coronary artery manipulation, surgical revascularization was considered. With the cardiothoracic surgical team at the bedside, it was decided that the patient be placed on CPB to empirically attempt bypass of the left anterior descending artery (LAD).

The patient was quickly transported to the operating room while CPR was ongoing. CPB was immediately instituted via the right femoral artery and vein using Stöckert SIII (Cobe Cardiovascular, Inc., Arvada, Colorado). Flows using CPB were maintained between 4.0–5.2 liters per minute. An intraoperative transesophageal echocardiogram (TEE) revealed a decompressed, yet fibrillating heart. After 5 minutes on CPB, normal sinus rhythm spontaneously returned as well as left ventricular contractility, though it remained globally reduced. High-dose vasopressors were still required to maintain acceptable blood pressure (SBP > 80 mmHg). The heart was rested on CPB for a total of 120 minutes. During this time, TEE showed progressive improvement in left ventricular (LV) function. His LV function and contractility ultimately returned to normal. With such a rapid response to CPB, empiric revascularization of the LAD was not performed. The etiology of the patient's shock was felt to be anaphylactic in nature. This was confirmed by serum tryptase levels. Tryptase is an enzyme released from the mast cells during anaphylactic and anaphylactoid-like reactions, and elevated levels are usually seen in severe reactions.¹ Laboratory results revealed a tryptase level of 50 µg/ml (> 10 µg/ml is suggestive of systemic mast cell degranulation), confirming the etiology of the refractory shock as anaphylactic/anaphylactoid in nature.

After the patient was weaned off the CPB, his cardiac contractility continued to improve, and he remained on hemodynamic support with IV norepinephrine, epinephrine and vasopressin. He was transferred to the surgical intensive care unit, where over the next 48 hours, he was weaned off vasopressors and was extubated. Two weeks later, he was discharged, with no evidence of neurological sequelae and normal LV function.

Discussion. In this case report we present a patient who had severe refractory shock from an anaphylactic/anaphylactoid reaction that was most likely induced by nonionic contrast. Risk of fatal reaction to contrast has been estimated at 0.9 cases per 100,000 exposures. Though he had never had a reaction to IV contrast, his history of allergic reaction to polyethylene glycol in the past, and the more recent reaction to clopidogrel less than 24 hours prior, may have heightened his response to IV contrast. An alternative hypothesis, though less likely, is that this life-threatening event was the manifestation of the late phase of a biphasic reaction to the clopidogrel given the previous day.

By definition, anaphylaxis/anaphylactoid reactions are classically characterized by urticaria, pruritis, angioedema, hypotension (systolic blood pressure [SBP] 30% drop in SBP from baseline), dysphagia, dysphonia, breathing difficulties (stridor, bronchospasm, asphyxiation), syncope, altered mental status, arrhythmia and even chest pain.¹ Epinephrine is the mainstay of therapy, with administered doses being 0.3–0.5 mg intramuscularly or 0.05 mg IV given every 3–5 minutes.^{1–7} IV administration should only be done in monitored settings because of the risks in causing arrhythmia or myocardial infarction.³ Active therapy with beta-blockers, which our patient did not receive, could blunt the response of epinephrine. An IV bolus of glucagon can be given to reverse the effect of beta-blockers. However, it is unlikely in a patient not receiving beta-blockers that glucagon will have any effect on the efficacy of epinephrine.

Hypotension in refractory anaphylaxis should be treated with aggressive, large-volume resuscitation with IV crystalloids and repeated doses or continuous infusion of epinephrine. Support with alpha-adrenergic activity such as norepinephrine or dopamine should be considered.

In our case, the lack of response to conventional measures for anaphylactic/anaphylactoid shock led us to consider other less likely etiologies. The possibility of ischemic injury to the left coronary artery during PCI led us to consider surgical revascularization. The initiation of CPB had an immediate effect on the return of rhythm and hemodynamic stabilization from a former state of cardiac standstill. This patient's refractory anaphylaxis, which was leading to certain fatality, was unintentionally aborted with CPB.

The use of extracorporeal life support has been examined in a myriad of articles and case reports since its inception in the 1960s. The rapidity of initiation and application of CPB has provided possible resuscitative efforts for a likely reversible etiology that remains ineffective with conventional therapy.^{6,7} CPB has many potential cardiac and non-cardiac indications as well as contraindications (Table 1).

In a review of the literature, several large studies on CPB have shown benefit in patients with cardiotoxicity secondary to medication, anaphylaxis during coronary artery bypass graft surgery and sudden cardiac arrest.^{8–10} In a recent study, a comparison was made between conventional CPR and CPR with CPB in patients with in-hospital cardiac arrest.¹¹ The use of rapid and early initiation of CPB showed both short-term benefit with a higher rate in the resumption of spontaneous circulation as well as long-term survival benefits compared to CPR alone. It was noted that CPR that is performed for > 10 minutes has a higher rate of morbidity and mortality. Rapid and early initiation of CPB, as well as its lower complication rate, make CPB a favorable modality in resuscitative efforts for in-hospital cardiac arrest,¹¹ suggesting that CPB should be considered as a potential life-saving intervention.

In some case reports, there is a discussion of the use of CPB as a way of maintaining hemodynamic support and perfusion while an etiology of shock is identified. In addition, there are a few case reports of patients who were nonresponsive to treatment (IV fluids, inotropic support) and stabilized only after initiation of CPB.^{8,10,14} After an etiology of shock is determined and treatment instituted, CPB is effectively weaned with favorable outcomes. Prompt initiation of CPB for patients with refractory shock may improve survival and prevent neurologic injury.

When considering the metabolism of the mediators of anaphylaxis, in theory, CPB would be efficacious. In our case, critical mediators of anaphylaxis may not have been metabolized effectively, given the patient's severe hypotension and ultimate cardiac standstill.^{1,4} In theory, CPB allowed for perfusion and adequate flow with subsequent metabolism of histamine, leukotrienes and prostaglandins. Histamine, with its short half-life of 15 minutes, exerts a potent vasodilatory effect that can be counteracted by epinephrine. With severe anaphylaxis, the overwhelming effect of mediators can be refractory to initial therapy. Mediators such as leukotrienes and prostaglandins with half-lives of 6–8 hours may lead to prolonged ventilator and hemodynamic support for > 24 hours. In our case, CPB provided necessary support for immediate tissue perfusion, and likely aided in metabolism of those shock mediators.

There are several offending agents a patient can be exposed to during a coronary catheterization that can lead to anaphylaxis (Table 2). The most common cause of anaphylaxis/anaphylactoid reactions during coronary catheterization is IV contrast media. Patients with any history of atopy are at increased risk of an allergic reaction to contrast. Previous contrast reactions increase the risk of a subsequent and more severe reaction. Premedication for such patients should include steroids and diphenhydramine.

Conclusion. We describe a case of refractory anaphylaxis despite conventional therapy resulting in electromechanical cardiac standstill. By allowing tissue and organ perfusion, CPB possibly facilitates



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metabolism of anaphylaxis mediators and in the present case, restabilized the patient's cardiac electrical mechanical activity. Thus, CPB should be considered as a potential life-saving measure for patients with refractory anaphylaxis during cardiac catheterization and PCI.

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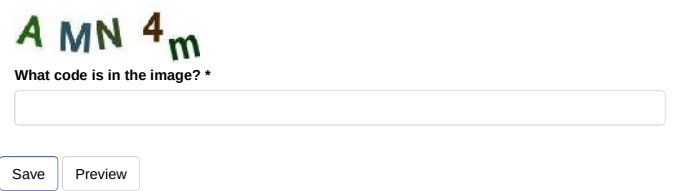
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