

HYPOTENSION ON INDUCTION: ANAPHYLAXIS OR CARDIAC FAILURE?

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Abstract

Identification of the cause of hypotension after induction of anesthesia is critical as treatment differs. We describe a case of anaphylaxis in a patient with severe cardiac disease, diagnosed by echocardiography and successfully treated with immediate cardiovascular resuscitation, epinephrine, vasopressors and antihistamines.

Keywords: Cardiogenic shock, anaphylaxis, echocardiography.

Introduction

Hypotension is not uncommon after induction of anesthesia. However, when hypotension is severe, especially in patients with co-morbidities such as coronary artery or valvular disease, identification of the cause may be difficult.

We describe a case of cardiovascular collapse after induction in a patient scheduled for triple valve replacement.

Case

A 69 year-old female with severe valvular disease was admitted for valve replacement. She had moderate right ventricular dysfunction, severe tricuspid regurgitation and marked pulmonary hypertension. An arterial cannula and right pulmonary artery catheter were placed. Anesthetic induction included 4mg midazolam, 250 mg fentanyl, 50mg rocuronium and antibiotics. Fifteen minutes post induction, blood pressure declined to 50-60/30-40. We presumed a diagnosis of pulmonary hypertension crisis with right ventricular failure secondary to underlying valvular pathology and right ventricular dysfunction. However, pulmonary artery pressures remained unchanged, central venous pressure dropped to 3 mmHg and peak airway pressures increased to 38-40mmHg. Transesophageal echocardiography revealed a hyperdynamic heart. Suspecting anaphylaxis, treatment was instituted with epinephrine, vasopressin, norepinephrine, H-1 and H-2 blockers and steroids. Hemodynamic stability was reestablished. Serum tryptase levels exceeded 400 ug/L. We concluded that cardiac collapse was secondary to anaphylactic shock, exacerbated by cardiac pathology.

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Discussion

Anaphylactic reactions are rare intraoperatively and even when treated appropriately have a mortality rate ranging from 3.5-4.7%¹. Anaphylaxis is an immediate hypersensitivity reaction mediated by the interaction of an antigen with IgE causing activation and degranulation of mast cells and basophils. Vasoactive mediators such as histamine, prostaglandins, kinins, leukotrienes and tryptase are released causing severe hypotension, bronchospasm, laryngeal edema, and cardiovascular collapse¹⁻⁴.

Many perioperative agents have been implicated. Sixty to 70% of reactions are secondary to neuromuscular blocking agents (NMDAs), commonly succinylcholine and rocuronium. Latex is also often implicated. Other incriminating agents include antibiotics, hypnotics (thiopental, propofol and midazolam), opioids, local anesthetics, iodinated contrast materials, non-steroidal anti-inflammatory drugs and colloids^{1,4-6}.

Tests to identify allergens include serum tryptase levels, plasma histamine, and specific IgE concentrations. Tryptase and histamine levels should be drawn within one hour of the reaction but can take up to a week for results. Tryptase levels > 25ug/L are strongly suspicious of an anaphylactic etiology and have a positive predictive value of over ninety percent^{5,6}. Radioallergosorbent tests (RASTs) detect IgE antibodies to specific anesthetic drugs such as muscle relaxants and intravenous anesthetic agents^{1,4-7}.

Cardiogenic shock occurs in up to 7% of patients with myocardial infarction. It accounts for over 40-60% of in-hospital mortality within the first 30 days⁸⁻¹¹. Cardiovascular collapse is due to loss of contractile myocardium causing left ventricular (LV) dysfunction, further aggravated by a systemic inflammatory response with refractory vasodilation and myocardial depression. ST segment elevation myocardial infarction (STEMI) accounts for most cardiogenic shock; other causes include acute mitral regurgitation, ventricular septal defect, cardiac rupture/tamponade, dysrhythmias, and type 1 aortic dissection¹²⁻¹⁵.

Differentiation of hypotension secondary to anaphylactic versus cardiogenic shock can be difficult intraoperatively. Severe hypotension,

increased airway pressures, pulmonary congestion and cardiovascular collapse are common to both. However, bronchoconstriction, facial edema, and cutaneous manifestations including generalized erythema, urticaria and angioedema are characteristic of anaphylaxis. Hemodynamic consequences and changes in LV loading conditions are secondary to loss of vasomotor tone as opposed to deterioration of myocardial function. Other differences in hemodynamic parameters are shown in Table 1^{1,3,4,7,13-16}.

Table 1
Features of anaphylactic vs. cardiogenic shock

ANAPHYLACTIC	CARDIOGENIC
SIMILARITIES	
Mean arterial pressure - ↓↓ Tachycardia Airway pressures - ↑↑ (Anaphylactic>cardiogenic) Pulmonary edema Dysrhythmias	
DIFFERENCES	
Wheezing/ bronchoconstriction Cutaneous manifestations Facial edema/angioedema Systemic vascular resistance - ↓↓ Pulmonary vascular resistance - ↓↓ Pulmonary capillary wedge pressure - ↓ Left ventricular end- diastolic/end-systolic pressure/area - ↓ Cardiac output/Cardiac index - ↑ Ejection fraction - ↑	EKG changes Systemic vascular resistance - ↑ or ↔ Pulmonary vascular resistance - ↑↑ Pulmonary capillary wedge pressure - ↑ Left ventricular end- diastolic/end-systolic pressure/area - ↑ Cardiac output/Cardiac index - ↓↓ Ejection fraction - ↓ Regional wall motion abnormalities Valvular abnormalities (i.e mitral regurgitation)

Transesophageal echocardiography (TEE) distinguishes between anaphylaxis and cardiogenic shock. Intraoperative assessment of regional wall motion abnormalities and LV end-diastolic and end-systolic diameters (LVEDid and LVESid) can differentiate conditions of decreased LV preload and

afterload versus myocardial dysfunction. Also seen are decreased stroke volume (SV) and ejection fraction (EF), increased LV end-diastolic and end-systolic area (LVEDA and LVESA), increased pulmonary artery pressures, diastolic dysfunction and valvular abnormalities such as mitral regurgitation secondary to ischemia. TEE features of anaphylaxis are increased SV and EF secondary to hyperdynamic LV function^{3,17-19}.

Both conditions require immediate cardio pulmonary resuscitation. Treatment of anaphylactic shock requires interruption of antigen contact by discontinuing causative agents and intravascular volume expansion. Epinephrine is the drug of choice. Inhaled β_2 agonists reverse bronchoconstriction. H1- and H2- blockers and corticosteroids attenuate histamine-related adverse effects and prevent delayed anaphylactic symptoms^{1,3,4,17,20}.

Cardiogenic shock requires improvement of LV function and circulatory support to maintain organ perfusion until coronary blood flow is reestablished via angioplasty, thrombolytic therapy or surgery. Inotropic drugs (epinephrine, dobutamine, dopamine and phosphodiesterase inhibitors) improve myocardial

performance. Vasodilators (nitroglycerin and nitroprusside) alter loading conditions and enhance the effects of inotropes. Diuretics improve pulmonary edema and oxygenation/ventilation^{11,13,15}.

Non-pharmacologic treatment options include intra-aortic balloon pump (IABP) and left ventricular assist device (LVAD) but require residual LV mass. Newer devices (i.e. Impella®) augment cardiac output, improve circulatory support and attenuate the systemic inflammatory response. Although these devices provide superior hemodynamic support, complications such as limb ischemia and bleeding increase^{13,14,21-23}.

Conclusion

Severe hypotension during anesthesia may be due to several causes. Accurate diagnosis is imperative as the treatment required for cardiogenic shock differs significantly from that necessary to reverse anaphylaxis.

Conflict of interest

None.

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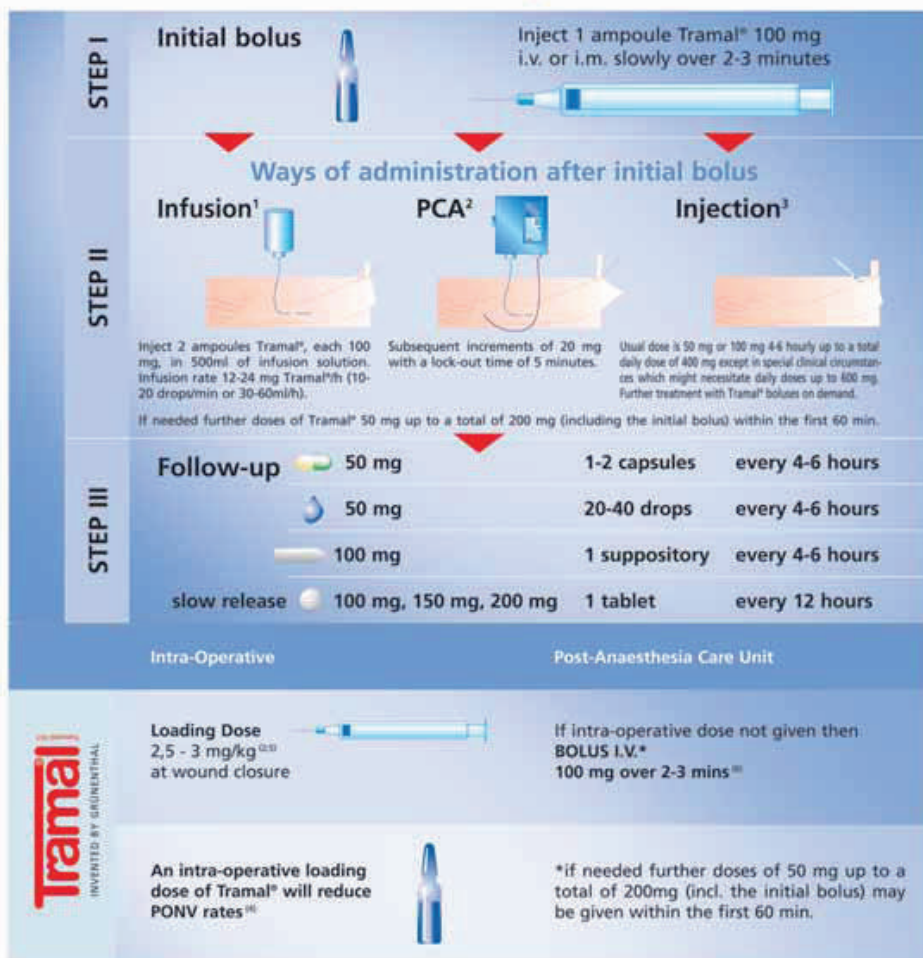


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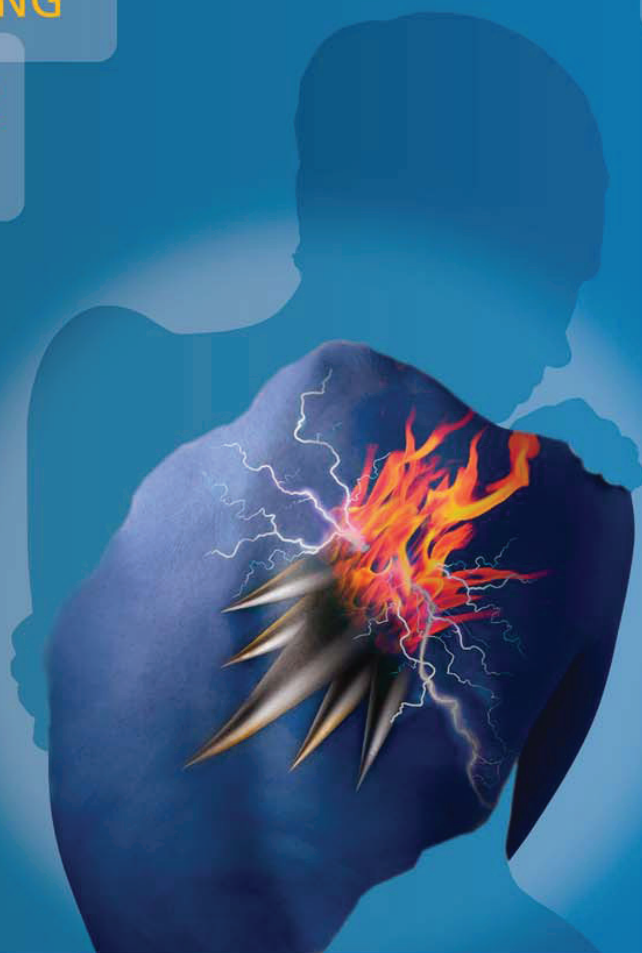
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- ❖ BRIDION rapidly reversed patients from reappearance of T₂[†] in 1.4 minutes²
- ❖ BRIDION rapidly reversed patients from 1 to 2 PTCs[‡] in 2.7 minutes³

BRIDION is indicated for the reversal of neuromuscular blockade induced by rocuronium or vecuronium. In children and adolescents (aged 2-17 years), BRIDION is only recommended for routine reversal of moderate rocuronium-induced neuromuscular blockade.¹

Important safety information

BRIDION is not recommended in patients with severe renal impairment. Studies in patients with hepatic impairment have not been conducted and, therefore, patients with severe hepatic impairment should be treated with great caution. Caution should be exercised when administering BRIDION to pregnant women as no clinical data on exposed pregnancies are available.

BRIDION has not been investigated in patients receiving rocuronium or vecuronium in the Intensive Care Unit (ICU) setting.

If neuromuscular blockade is required within 24 hours of BRIDION administration, a nonsteroidal neuromuscular blocking agent should be used instead of rocuronium or vecuronium. The most commonly reported adverse reactions were dysgeusia (metal or bitter taste) and anesthetic complications (movement, coughing, grimacing, or suckling on the endotracheal tube). In patients treated with BRIDION, a few cases of awareness were reported. The relation to BRIDION was uncertain. In a few individuals, allergic-like reactions (ie, flushing, erythematous rash) following BRIDION were reported. Clinicians should be prepared for the possibility of allergic reactions and take the necessary precautions. In a trial of patients with a history of pulmonary complications, bronchospasm was reported in 2 patients and a causal relationship could not be fully excluded. Volunteer studies have demonstrated a slight (17%-22%) and transient (<30 minutes) prolongation of the prothrombin time/activated partial thromboplastin time (PT/aPTT) with BRIDION; however, clinical studies have demonstrated no clinically relevant effect on peri- or postoperative bleeding complications with BRIDION alone or in combination with anticoagulants. As BRIDION has demonstrated an in vitro pharmacodynamic interaction with anticoagulants, caution should be exercised in patients on anticoagulation for a pre-existing or comorbid condition. This pharmacodynamic interaction is not clinically relevant for patients receiving routine postoperative prophylactic anticoagulation. Although formal interaction studies have not been conducted, no drug interactions were observed in clinical trials. Preclinical data suggest that clinically significant drug interactions are unlikely with the possible exceptions of toremifene, fusidic acid, and hormonal contraceptives.

* Train of four

† Post-tetanic counts

‡ Second twitch

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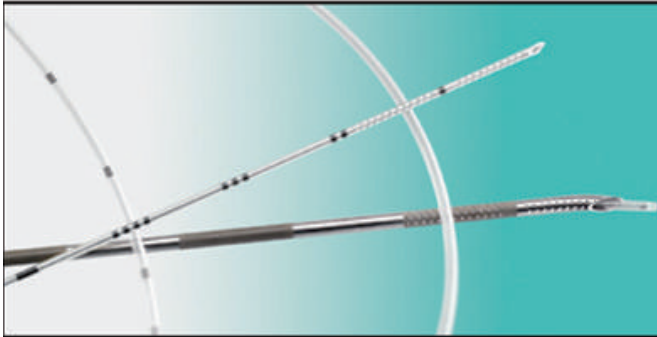
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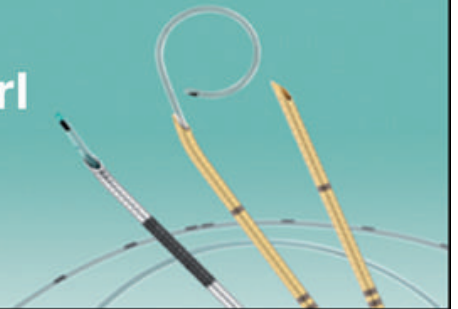
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