HYPOTENSION ON INDUCTION: ANAPHYLAXIS OR CARDIAC FAILURE?

HIMANI V. BHATT* AND ELIZABETH A. M. FROST**

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 antibiosis. 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%. 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 collapse.

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 ant-inflammatory drugs and colloids.

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. Radioallergosorbent tests (RASTs) detect IgE antibodies to specific anesthetic drugs such as muscle relaxants and intravenous anesthetic agents.

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 days. 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 I aortic dissection.

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.

<table>
<thead>
<tr>
<th>Features of anaphylactic vs cardiogenic shock</th>
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<tr>
<td><strong>ANAPHYLACTIC</strong></td>
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<tr>
<td><strong>SIMILARITIES</strong></td>
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<tr>
<td>Mean arterial pressure - ↓↓</td>
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<tr>
<td>Tachycardia</td>
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<tr>
<td>Airway pressures - ↑↑ (Anaphylactic&gt;cardiogenic)</td>
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<td>Pulmonary edema</td>
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<tr>
<td>Dysrhythmias</td>
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<tr>
<td><strong>DIFFERENCES</strong></td>
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<tr>
<td>Wheezing/bronchoconstriction</td>
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<tr>
<td>Cutaneous manifestations</td>
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<tr>
<td>Facial edema/angioedema</td>
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<tr>
<td>Systemic vascular resistance - ↓↓</td>
</tr>
<tr>
<td>Pulmonary vascular resistance - ↓↓</td>
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<tr>
<td>Pulmonary capillary wedge pressure - ↓</td>
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<tr>
<td>Left ventricular end-diastolic/end-systolic pressure/area - ↓</td>
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<tr>
<td>Cardiac output/Cardiac index - ↑</td>
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<td>Ejection fraction - ↑</td>
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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
HYPOTENSION ON INDUCTION

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 function3,17-19.

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 β2 agonists reverse bronchoconstriction. H1- and H2- blockers and corticosteroids attenuate histamine-related adverse effects and prevent delayed anaphylactic symptoms1,3,4,17,20.

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/ventilation11,13,15.

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 increase13,14,21-23.

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.
References

The key to

Lock-up

Postoperative Pain

Initial bolus
Inject 1 ampoule Tramal® 100 mg i.v. or i.m. slowly over 2-3 minutes

Ways of administration after initial bolus

Infusion
Inject 3 ampoules Tramal®, each 100 mg, in 500ml of infusion solution. Infusion rate 12-24 mg Tramal®/h (160-200 drops/min or 30-60ml/h).

PCA
Subsequent increments of 26 mg with a lock-out time of 5 minutes.

Injection
Usual dose 50 mg or 100 mg 14 hourly up to a total daily dose of 400 mg except in special clinical circumstances which might merit a daily dose up to 600 mg. Further treatment with Tramal® bolas on demand.

If needed further doses of Tramal® 50 mg up to a total of 200 mg (including the initial bolus) within the first 60 min.

Follow-up

<table>
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<th>STEP III</th>
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<tr>
<td>1-2 capsules every 4-6 hours</td>
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<tr>
<td>50 mg</td>
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<tr>
<td>20-40 drops every 4-6 hours</td>
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<tr>
<td>50 mg</td>
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<tr>
<td>1 suppository every 4-6 hours</td>
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<tr>
<td>100 mg</td>
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<td>slow release 100 mg, 150 mg, 200 mg 1 tablet every 12 hours</td>
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Intra-Operative

Loading Dose
2.5 - 3 mg/kg at wound closure

Post-Anaesthesia Care Unit

An intra-operative loading dose of Tramal® will reduce PONV rates

If intra-operative dose not given then BOLUS i.v.*
100 mg over 2-3 mins

For patients with localized BURNING, SHOOTING, STABBING, Neuropathic pain WORKS WHERE IT HURTS
BRIDION®—for **optimal neuromuscular blockade management** and improved recovery

**Predictable and complete reversal**

- 98% of BRIDION patients recovered to a TOF ratio of 0.9 from reappearance of T2 within 5 minutes
- 97% of BRIDION patients recovered to a TOF ratio of 0.9 from 1 to 2 PTCs within 5 minutes

**Rapid reversal**

- BRIDION rapidly reversed patients from reappearance of T2 in 1.4 minutes
- BRIDION rapidly reversed patients from 1 to 2 PTCs in 2.7 minutes

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.

**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 clinical data on exposed pregnancies are available.

BRIDION has not been investigated in patients recovering from surgery or sedation in the intensive Care Unit (ICU) setting. If neuromuscular blockade is required within 24 hours of BRIDION administration, a nondepolarizing neuromuscular blocking agent should be used instead of rocuronium or vecuronium. The most commonly reported adverse reactions were dyspnea (oral or indermocutaneous) and anaphylactic complications (shock, hypotension, difficulty breathing, urticaria or angioedema). In patients treated with BRIDION, a few cases of awareness were reported. The effect of BRIDION on awareness in a few isolated cases of insomnia, anxiety, nightmares (in healthy volunteers) was observed, and no cases of confusion or altered mental status were described following BRIDION administration. Individuals should be prepared for the possibility of allergic reactions and take necessary precautions. In a study of patients with a history of previous complications, those who were reported in 2 patients and a causal relationship could not be fully excluded. Volunteer studies have demonstrated a slight (15-25%) and transient (<15 minutes) prolongation of the prothrombin time of patients treated with BRIDION; however, clinical studies have demonstrated no clinically relevant effect on prothrombin time or PT in patients treated with BRIDION. BRIDION has been demonstrated not to affect pharmacokinetic interactions with prothrombin, carboxypeptidase A, and renin angiotensin-angiotensin-aldosterone system. 25% of cases of severe jaundice reported in clinical trials. Pooled data suggest that clinically significant drug interactions are unlikely to occur with the possible exceptions of cimetidine, furosemide, and hormonal contraceptives.

**REFERENCES**


Please see summary of product characteristics for full prescribing information.

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