Challenge at NORA! Emergency Induction for Endotracheal Intubation of a Patient with Acute Myocardial Infarction and Cardiogenic Shock with 10% Ejection Fraction.

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**Introduction**

Maintenance of systemic vascular resistance (SVR) in a patient with severely reduced myocardial contractility is a priority during induction. Decrease in afterload during induction is common with anesthetic agents which is poorly tolerated by patient with global reduction in systolic myocardial function with an ejection fraction of 10%. Selection of anesthetic agents with minimal myocardial depressant effect and minimal effect on SVR are essential during induction. High level of sympathetic activity maintains the SVR in this patient and abolition of such sympathetic effect will have a catastrophic effect as these patients are not able to increase their cardiac output by increasing stroke volume due to global hypokinesia. Preemptive administration of vasopressors prior to induction will prevent reduction in afterload. We describe how effectively we induced such a patient with 10% ejection for emergency endotracheal intubation at Non-Operating Room Anesthesia (NORA) site.

**Case Description**

A 67-year old male, 87 kg with acute myocardial infarction with ejection fraction less than 10% with cardiogenic shock required emergency airway management for coronary angiogram. He was on dopamine infusion to maintain his systolic blood pressure greater than 100 mm Hg. He was given 400 mcg of phenylephrine prior to induction. He was induced with midazolam 2 mg, fentanyl 100mcg and 60 mg rocuronium and was intubated with a size 8 endotracheal tube under direct laryngoscopy with hemodynamic stability during induction and post-intubation period.

**Case Discussion**

**Clinical features of ischemic cardiogenic shock with hypoperfusion:**

1. Hypotension
2. Cold clammy extremities
3. Jugular venous distension
4. Tachycardia
5. Decreased urine output
6. Sweating
7. Mental confusion
8. Narrow pulse pressure
9. Pulmonary edema

**Management of cardiogenic shock:**

a) Inotropes: Dopamine, dobutamine, epinephrine, isoproterenol, norepinephrine infusion
b) Inodilators: Calcium channel blocker levosimendan and phosphodiesterase inhibitor milrinone as infusion
c) Early cardiac catheterization and reperfusion therapy
d) Intra-aortic balloon pump
e) Left ventricular assist device

**Goals of induction for airway management in a patient with low ejection fraction:**

1. Prevent myocardial depression.
3. Maintain SVR.

**Anesthetic agents for induction in a severely reduced ejection fraction:**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effect</th>
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<tbody>
<tr>
<td>Propofol</td>
<td>Significantly decreases SVR and should be avoided. If used, it should be titrated to small doses with simultaneous administration vasopressors to maintain SVR.</td>
</tr>
<tr>
<td>Etomidate</td>
<td>Causes less hypotension than propofol, still it can cause significant hypotension in a patient with cardiogenic shock. Its use in titration with phenylephrine is essential.</td>
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<tr>
<td>Ketamine</td>
<td>Increases sympathetic activity and causes tachycardia at high doses. Small doses (20-30mg) can be used as an adjunct with simultaneous administration of phenylephrine.</td>
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<tr>
<td>Midazolam</td>
<td>Causes less hypotension than other agents. It can be used for induction with pre-emptive administration of phenylephrine to prevent reduction in afterload.</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Causes less hypotension and can be used with simultaneous administration of phenylephrine.</td>
</tr>
</tbody>
</table>

**Our technique for induction in this patient:**

1. Dopamine infusion continued.
2. Administered phenylephrine preemptively to maintain SVR.
3. Fentanyl/midazolam/rocuronium for induction.

**CONCLUSION**

Based on our success story we recommend cardio stable drugs such as fentanyl, midazolam and low dose ketamine for induction when a patient presents with severely reduced myocardial function for airway management and anesthesia. Even such cardio stable drugs can abolish the sympathetic activity and induce afterload reduction. Continuation of inotropic agents and preemptive administration of an adequate amount of vasopressors is essential to maintain afterload.

**References**