EM CASES SUMMARY

Episode 164 Cardiogenic Shock Simplified

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Understanding the reduced contractility and assessment of end-organ perfusion is key to managing patients with cardiogenic shock

Reduced contractility is the keystone of cardiogenic shock

Cardiogenic shock is defined as systolic blood pressure (SBP) < 90mmHg or the need for pharmacological or mechanical support to maintain a SBP > 90mmHg **and** evidence of end-organ perfusion. Chronic heart failure progresses into cardiogenic shock when the reduced contractility of the ventricle impairs mean arterial pressures and cardiac output which results in decreased end-organ perfusion.

Assessment of end-organ perfusion is central to identifying occult cardiogenic shock

Patients with heart failure may have a lower baseline SBP due to heart-failure related pharmacotherapy which can make the diagnosis of cardiogenic shock difficult. A "soft" SBP may be the patients baseline or it may represent occult shock. Assessing for impaired end-organ perfusion in these patients can significantly aid in the identification of occult cardiogenic shock. On the other hand, a patient can be in a pre-cardiogenic shock, hypertensive state such as SCAPE (see Part 1). Again, assessment for impaired end-organ perfusion can be very helpful in diagnosis and management.

Assessment of end-organ perfusion involves assessment of the skin, mental status, urine output and PoCUS parameters. Assess the **skin** for 1. mottling 2. cool temperature 3. prolonged capillary refill time. Altered **mental status** and **oliguria/anuria** are often present. An elevated **lactate** is suggestive or poor end-organ perfusion although the specificity is poor. Advanced **doppler PoCUS** may aid in assessment of end organ perfusion (portal vein pulsatility index, renal doppler resistive index, splenic doppler resistive index). A **central venous-arterial gap** >6mmHg is an indicator of decreased systemic blood flow. It is the difference between PCO_2 in central venous blood and PCO_2 in arterial blood.

Clinical Pearl: *The skin is a readily observable end-organ. Low cardiac output usually results in impaired perfusion to the skin which leads to mottled, cold extremities with a prolonged capillary refill time. In contrast, patients with sepsis, usually have warm extremities due to vasodilation.*

PoCUS in the diagnosis and management of cardiogenic shock: Lung US, LV function & volume status

In addition to lung ultrasound looking for B-lines for pulmonary edema, PoCUS can be used to assess the overall **LV function** and **volume status**. Global LV function includes mitral valve movement (<1cm is normal), LV size (<5cm is normal) and LV contractility (≥ 1/3 the diameter is normal). Remember that the keystone for diagnosis of cardiogenic shock is reduced contractility. When it comes to volume status patients in cardiogenic shock may be hypovolemic, euvolemic or hypervolemic which will guide whether or not they require diuresis or volume replacement. Volume status assessment includes measuring LV size, jugular venous distention, and IVC size and collapsibility (diminished respiratory variation).

***Clinical Pearl**: It is important to determine the volume status of a patient in cardiogenic shock using clinical and PoCUS parameters because management differs based on volume status. For example, careful fluid bolus/infusion may be required if a patient is in cardiogenic shock and deemed to be intravascularly volume depleted.*

Treatment of the heart failure patient in cardiogenic shock

Goals of management in cardiogenic shock

The overall goal in the management of the heart failure patient in cardiogenic shock is to stabilize them by **maintaining oxygenation** with NIPPV, **maintaining sufficient cardiac perfusion** with vasopressors, **improving cardiac contractility** with ionotropes and **optimizing volume status** so that they can be safely transported to the cath lab or operating room for definitive management of the underlying mechanical lesion, if any. This usually involves 4 simple steps:

1. Optimize **oxygenation** with **NIPPV**
2. Optimize **blood pressure** with **vasopressors** (eg. norepinephrine) to maintain cardiac/end-organ perfusion targeting a MAP of 65-80
3. Optimize **contractility** with **ionotropes** (eg. dobutamine, milrinone)
4. Optimize **volume status** (crystalloid or diuretics)

Determining the cause of cardiogenic shock is essential to optimize management

Think of the causes of cardiogenic shock in 4 categories (keeping in mind that #1 and #2 require emergent mechanical repair):

1. Acute coronary syndromes

2. Mechanical (ie. severe aortic stenosis, endocarditis, ruptured valve, free wall rupture)
3. Myocarditis
4. Progressive non-ischemic chronic heart failure

See Part 1 for the full differential diagnosis of acute heart failure
Clinical Pearl: *assess for papillary muscle rupture/severe mitral regurgitation/free wall rupture in patients with acute coronary syndromes who are in shock; listen for new cardiac murmur and look on PoCUS for obvious papillary muscle rupture/mitral regurgitation/free wall rupture as they require emergent cardiac surgery to replace the mitral valve or repair the free wall*

Optimizing oxygenation in cardiogenic shock with carefully titrated NIPPV

Maintaining adequate tissue oxygenation is critical in patients with heart failure and cardiogenic shock, which is usually ideally achieved with NIPPV. It has the added benefit of decreasing preload and afterload. (see Part 1 for oxygenation strategies in heart failure)

Clinical pearl: avoid endotracheal intubation whenever possible in the patient in cardiogenic shock as removal of respiratory drive may lead to cardiovascular collapse

Clinical Pitfall: overshooting positive pressure ventilation in the patient with RV failure; positive pressure ventilation can potentially increase RV afterload and therefore should be used with caution in patients with cardiogenic shock resulting from acute RV failure

Optimize cardiac output by optimizing blood pressure, contractility and volume status

1.Optimizing blood pressure with norepinephrine +/- vasopressin: target a MAP of 65-80. This is required to augment end-organ/coronary perfusion. The preferred first line agent is norepinephrine. Vasopressin may be added as a second line agent. While epinephrine and norepinephrine both have been shown to improve MAP and cardiac indices, norepinephrine has a lower incidence of refractory shock compared to epinephrine.
2.Optimizing contractility with dobutamine or milrinone: while dobutamine is a Beta 1 and 2 agonist and milrinone is a phosphodiesterase 3 inhibitor, both agents are inotropes and vasodilators. A recent RCT showed no significant difference in in-hospital survival and major cardiac outcomes with dobutamine versus milrinone in patients in cardiogenic shock.
Our experts recommend starting with dobutamine as it is a shorter acting drug and can be titrated more easily compared to milrinone. However, for patients taking long-acting beta-blockers, milrinone may be the better first option as it works on a different receptor.

Clinical pitfall: *giving an ionotrope before initiating a vasopressor may decrease BP further as they are vasodilators, which may lead to cardiovascular collapse; our experts suggest initiating norepinephrine prior to giving an ionotrope in heart failure patients with cardiogenic shock*

Clinical pearl: for patients taking long acting beta-blockers milrinone may be the ionotrope of choice in patients with cardiogenic shock

3.Optimizing volume status: based on clinical and PoCUS assessment of intravascular volume, patients may require gentle and cautious crystalloid administration or diuresis with ongoing assessment of volume status.

Practical pearl: it is imperative to consult cardiology/CV surgery early in the resuscitation of patients with cardiogenic shock as there may be a need for emergent mechanical interventions

Cardiogenic shock caused by severe aortic stenosis – avoid tachycardia and maintain DBP in these high risk subset of patients

Patients with severe aortic stenosis and cardiogenic shock have an especially high mortality rate. Once identified by history of PoCUS, imminent consultation with both cardiology and cardiac surgery is important as definitive mechanical interventions are often life saving. These include percutaneous valvuloplasty, transcatheter aortic valve replacement (TAVR) or open aortic valve replacement. ECMO may be considered to bridge them to a percutaneous or surgical intervention.

Avoidance of tachycardia and maintenance of diastolic BP are essential in the management of patients with cardiogenic shock caused by severe aortic stenosis. The fixed lesion at the aortic valve causes the left ventricle to chronically generate high pressures to overcome the high afterload. This leads to the LV hypertrophy requiring higher coronary perfusion pressures. Remember that coronary perfusion pressure (CPP) = diastolic blood pressure (DBP) minus left ventricular end diastolic pressure (LVEDP). Therefore, maintaining a higher DBP is important in order to maintain adequate coronary perfusion.

Clinical pitfall: rapidly decreasing the afterload which the aortic stenosis patient depends on for coronary/organ perfusion with high dose nitrates on ACE inhibitors may precipitate cardiovascular collapse; avoid high dose afterload-reducing medications in patients with aortic stenosis who are in cardiogenic shock or occult shock.

These patients will usually require an arterial line. If the blood pressure is very high, evidence from small a small study suggests that nitroprusside may be of benefit. If the blood pressure is low, consider fluids (often preload dependent), inotropes and/or vasopressors. Inotropes will need to be carefully titrated to ensure tachycardia is avoided.

Temporary mechanical circulatory support (MCS): ECMO, IAPBs and PVADs

The common types of MCS available include intra-aortic balloon pump (IABP), percutaneous ventricular assist devices (PVAD – Impella, Tandem Heart), and veno-arterial extracorporeal membrane oxygenation (VA-ECMO). These resources may be

used to bridge a patient to percutaneous or surgical interventions, bridge to recovery (myocarditis), and sometimes a bridge to end of life decision making (a severe cardiogenic shock patient not improving with medical therapy, and no fixable lesion). There is a lack of robust evidence to suggest that utilization of MCS improves survival in cardiogenic shock patients (especially if there is no lesion to fix), however, it is an option considered in patients with cardiogenic shock who are refractory to medical therapy, and a discussion with interventional cardiology/CV surgery is apt in carefully selected patients.

Risk stratification, prognosis and disposition of patients with heart failure without cardiogenic shock

There is much regional variation when it comes to the proportion of patients with acute heart failure in the ED who are discharged home. In an attempt to standardize disposition and better risk stratify these patients, various scoring systems have been developed. *The Ottawa Heart Failure Risk Score* (OHFRS) evaluated the risk of 14 and 30-day adverse events among 1100 patients who presented with acute heart failure in 6 tertiary care EDs in Canada.

Patients with a score of < 1 according to the score should be considered for safe discharge home. An OHFRS score of 1 and O2 sat < 90% or any score > 2 should be considered for admission.

Disadvantages of this score are that it includes NT-ProBNP which has little evidence for benefit in the diagnosis of heart failure in the ED, and is not universally available in Canadian EDs; and it assumes that admission will prevent serious adverse events. The score was validated without the BNP, but was less sensitive. The walk test is a commonly used evidence-based test to help in disposition decisions of dyspneic ED patients that our experts find useful.

Items	Points
1. Initial Assessment	
a) History of stroke or TIA	(1) ___
b) History of intubation for respiratory distress	(2) ___
c) Heart rate on ED arrival ≥ 110	(2) ___
d) Room Air SaO_2 < 90% on EMS or ED arrival	(1) ___
2. Investigations	
a) ECG has acute ischemic changes	(2) ___
b) Urea ≥ 12 mmol/L	(1) ___
c) Serum CO_2 ≥ 35 mmol/L	(2) ___
d) Troponin I or T elevated to MI level	(2) ___
e) NT-ProBNP ≥ 5,000 ng/L	(1) ___
3. Walk test* after ED treatment	
a) SaO_2 < 90% on room air or usual O_2, or HR ≥ 110 during 3-minute walk test, or too ill to walk	(1) ___
Total Score (0 - 15):	____

Heart Failure Risk Categories for Serious Adverse Events within 14 days

Total Score	Risk	Category
0	2.8%	Low
1	5.1%	Medium
2	9.2%	Medium
3	15.9%	High
4	26.1%	High
5	39.8%	Very High
6	55.3%	Very High
7	69.8%	Very High
8	81.2%	Very High
9	89.0%	Very High

Image obtained from https://first10em.com/the-ottawa-heart-failure-risk-scale/ohfrs/

Another example of a risk score is the *Emergency Heart failure Mortality Risk Guide* for 7-day (EHMRG7) and 30-day (EHMRG30-ST) mortality. The variables of this score are age, SBP, heart rate, O2 saturation, Cr, K, transport by EMS, troponin positive, active cancer, on outpatient diuresis. The advantage of this score is that it does not require BNP.

Our experts do not routinely use these scores to decide on disposition because the scores do not incorporate the underlying cause of the heart failure which should weigh into

your disposition decision. The patient who presents to the ED with acute heart failure because of dietary or medication indiscretion has an easily reversible cause whereas the patient with an unknown cause or mechanical cause is probably more likely to have a serious adverse event and/or require in-hospital treatments. These scores may help the emergency physician advocate for their patient whom they feel need to be admitted by calculating the score and communicating it to the consultant, or for the patient who would like to go home despite your advice to be admitted, especially for those patients who seem to improve during the ED stay.

It is important for patients and their families to understand the natural history and prognosis of chronic heart failure: it is a progressive disease; ED therapy for may lead to stabilization, however, months to years following the stability phase, the patient's functional status may decline resulting in multiple hospitalizations and eventually the condition may become refractory to treatment.

Take home points for the diagnosis and management of cardiogenic shock

- Understanding the reduced contractility and clinical/PoCUS/lab assessment of end-organ perfusion is key to diagnosing and managing patients with cardiogenic shock
- Patients may present in occult cardiogenic shock with a near normal blood pressure making it challenging to diagnose; this stresses the importance of a thorough assessment of end-organ perfusion
- Effective management of cardiogenic shock requires early recognition of the etiology (eg. ACS, valvular, myocarditis, progressive non-ischemic chronic heart failure)
- Early consultation with cardiology/CV surgery is important to consider, especially in patients with a potentially reversible mechanical cause and in those patients who may benefit from temporary mechanical circulatory support (ECMO, PVADs, IAPBs)
- Resuscitation is a bridge to definitive therapy and includes optimization of: oxygenation with NIPPV, blood pressure with vasopressors (eg. norepinephrine) to maintain cardiac/end-organ perfusion targeting a MAP of 65-80, contractility with ionotropes (eg. dobutamine, milrinone) and volume status (crystalloid or diuretics)
- Patients with severe aortic stenosis in cardiogenic shock have a high risk of mortality, require mechanical definitive management urgently, and careful attention should be paid to maintaining DBP and avoiding tachycardia
- Various risk scores exist to help guide disposition in patients with acute heart failure, but they do not take into account the underlying cause of heart failure; nonetheless they may be useful to calculate and communicate to the consultant and/or patient for the patient who you feel should be admitted

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