Cardiogenic Shock in An Otherwise Healthy Young Adult

Kirsten Kenny PA-S, Cindy Rossi MHS, PA-C
Quinnipiac University Physician Assistant Program

Introduction

- Cardiogenic Shock (CS) is the condition in which the heart is unable to sustain an efficient cardiac output leading to hypoxia and end-organ hypoperfusion.[1-5]
- There is no clear diagnostic criteria, rather it is based off of a collection of subjective and objective findings as well as clinical suspicion.[1]
- CS is a condition associated with high mortality of ~50%.[5]
- Approximately 80% of all cases have acute myocardial infarction (AMI) as the causative agent.[3,4]
- Other etiologies include myocarditis, acute decompensated heart failure, and thyroid disease.[5]
- Non-MI related cardiogenic shock is associated with higher mortality and 30-day readmission rates compared to MI-associated cases and seen in younger, female patients.[4]
- Management of CS may involve therapies like fluid resuscitation, catecholamines, and mechanical support devices like balloon pumps depending on etiology.[1,3,6,7]
- An obvious gap remains in treatment protocols for non-AMI associated cardiogenic shock.[2,5]

Image 1. Bilateral Pulmonary Edema[8]

https://www.consultant360.com/article/consultant360/postoperative-naloxone-induced-pulmonary-edema

Case Description

Initial Brief History

- A 32-year-old male who denies past medical history presents with nausea/vomiting and headache secondary to alcohol and marijuana use.
- Had been drinking alcohol at friend's house previous night.
- Reports 8 episodes non-bloody, non-bilious vomiting beginning at 3am. Has headache and dizziness with ambulation.
- Drank ½ pint of tequila, 1 "cup" of rum, and smoked 1-2 marijuana joints. Denies other drug and over-the-counter medication use. Drank a Gatorade but no other intake.
- Drinks approximately 1-2 drinks/day around 3-4 times/week.
- Not COVID vaccinated.
- Social history: denies other recreational drug use. Reports only consuming personal supply of alcohol and marijuana.
- Past medical history, surgical history, allergies noncontributory. No daily medications.
- Family history: sister, alive, cardiac surgery at 18 for "clot".

Initial Physical Exam

- **Vitals:** Temperature of 36.6 degrees Celsius, 114 beats per minute, 20 respirations per minute, blood pressure of 118/66mmHg, pulse oximetry of 95% on room air
- **General:** Non-toxic, in no acute distress.
- **Skin:** No rash, warm, dry. Capillary refill < 2 seconds.
  Ears, Nose, Throat: Moist mucous membranes.
- **Respiratory:** No rhonchi, wheeze, rales. Slight tachypnea.
- **Cardiovascular:** Tachycardic. No murmurs, gallops, or rubs.
- **Gastrointestinal:** Soft, nontender, nondistended abdomen. No rebound/guarding. Negative Murphy's.
- **Neurologic:** Alert and oriented to person, place, time. Moves all extremities equally.

Subsequent Pertinent Exam Findings

- **Vitals:** Temperature of 36.5 degrees Celsius, 138 beats per minute, 22 respirations per minute, blood pressure of 95/59 mmHg, pulse oximetry of 83% on room air.
- **General**: Diaphoretic. In respiratory distress.
- **Skin:** Cool, clammy.
- **CV:** Tachycardic. Additional heart sound heard.
- **Respiratory:** Breath sounds diminished with diffuse crackles.
- No changes in findings from initial for other body systems.

Differential Diagnosis

Pulmonary Embolism
Acute Coronary Syndrome
COVID-19
Community Acquired Pneumonia
Aspiration Pneumonia
Acute Decompensated Heart Failure

Pertinent Diagnostic Findings

- **Labs**: troponin 0.51ng/mL, proBNP 332pg/mL, D-dimer 664ng/mL, procalcitonin 0.50ng/mL, lactic acid 4.4mmol/L. Normal TSH level.
- COVID rapid and PCR negative.
- Serial ECGs with no evidence of ACS/STEMI.
- **Echocardiogram:** Left ventricular systolic function was severely decreased with an estimated ejection fraction less than 20%. There was global hypokinesis.
- **Chest X-ray 1-view:** Bilateral pulmonary edema.
- **CT Angiography:** No evidence of acute pulmonary embolism. Scattered nodular/ground glass opacities seen throughout the bilateral lung parenchyma.

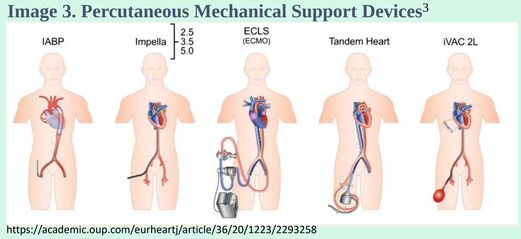
Final Diagnosis

- Cardiomyopathy likely secondary to myocarditis
- Tachycardia
- Acute Reduced Ejection Fracture Heart Failure (HFrEF) (resolved)
- Cardiogenic Shock: New York Heart Association (NYHA) Stage IV, Society for Cardiovascular Angiography and Interventions (SCAI) cardiogenic shock stage C (resolved)
- Acute Hypoxic Respiratory Failure (resolved)

Figure 2. Initial Complete Blood Count Results

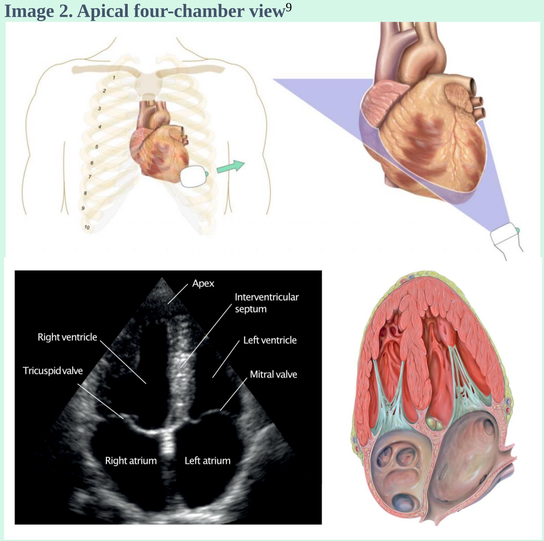
White Blood Cell Count	Hemoglobin	Hematocrit	Platelet Count	Neutrophil Percent	Lymphocyte Percent	Monocyte Percent	Eosinophil Percent	Basophil Percent
23.4 Thou/uL	16.3 g/dL	48.5%	126 Thou/uL	80.4%	9.6%	8.8%	0.0%	0.3%

Image 3. Percutaneous Mechanical Support Devices[3]



https://academic.oup.com/eurheartj/article/36/20/1223/2293258

Image 2. Apical four-chamber view[9]



https://ecgwaves.com/topic/the-standard-adult-transthoracic-echocardiogram-a-protocol-to-obtain-a-complete-study/

Figure 1. Emergency Room Course of Events.

Time	1247	1517	1546	1645	1710
Event	Had significant leukocytosis. Benign abdominal exam and imaging. Plan: Ondansetron, H2 blocker, and 2L IVF. Oral intake.	Passed oral intake trial. Repeated vitals. Now hypotensive, tachycardic, hypoxic of unclear etiology. Reports shortness of breath.	On non-rebreather with SpO2 >90%. Labs pending. Anticipate admission.	Negative COVID test. ICU bed pending. Cultures drawn. IV vancomycin and cefepime started per protocol.	XR with pulmonary edema. ECHO with < 20% ejection fraction. Consult cardiology. Plan: catheter lab & ICU.

Outcome

- Swan-Ganz catheter placed with findings consistent with cardiogenic shock.
- Patient admitted to ICU.
- Started on milrinone drip 0.125mcg/kg/min and furosemide with clinical improvement. Metoprolol tartrate 25mg BID added.
- Work-up for etiology including cardiac MRI and bloodwork had findings consistent with myocarditis.
- Ventricular function improved and patient was discharged home on carvedilol (switched from metoprolol).
- Discharge plan included gentle up titration of 90-day guideline-directed medical therapy with ventricle function re-evaluation and potential ICD placement.

Discussion

- Majority of CS arise as complications of cardiac etiologies, specifically AMI. [1,5]
- Non-AMI CS tends to be seen in younger patients and has poorer clinical outcomes compared to AMI cardiogenic shock. [2]
- CS requires quick clinical management and initial resuscitation to protect end organs from further hypoperfusion and cell death. [1]

Conclusion

- This case demonstrates the time-sensitive nature of cardiogenic shock and how quickly a patient's status can diminish.
- Given that non-AMI cardiogenic shock patients tend to be female and younger, CS should be in the differential for all acutely decompensating patients regardless of cardiac history or age.
- Research must continue to explore the management of initial resuscitation and subsequent stages as there currently are no uniform therapy guidelines.

Resources

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