Emergency Bulletin
Hong Kong College of Emergency Medicine

Cardiogenic shock secondary to HIV-related cardiomyopathy

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A middle-aged man presented to the Accident and Emergency Department with two weeks of shortness of breath and bilateral lower-limb swelling.

The patient had a known history of HIV infection with suboptimal virological suppression on Biktarvy (combination of bictegravir, emtricitabine, and tenofovir alafenamide). Laboratory results two months prior to presentation showed a CD4 count <100 cells/μL and a high HIV viral load.

His vital signs on arrival:
Temperature 36°C
BP: 96/60 mmHg
Heart rate 98 bpm
SpO2 87% on oxygen 6L/min via nasal cannula

Physical examination:

- Speaking in phrases with accessory muscle use
- Elevated jugular venous pressure
- Chest examination: bi-basal crepitations
- Pitting oedema to the knees
- Cool peripheries with purplish discoloration

The patient was in shock and hypoxaemia. Based on the physical examination findings, the initial impression was acute decompensated heart failure with respiratory failure and evolving cardiogenic shock.

What would be the appropriate initial management?

The patient required immediate stabilisation of his critical condition and rapid evaluation for differentiating the cause of his shock. [1,2]

- **Recognize shock and call for help:** involve cardiology, CCU or ICU early.
- **Airway patency:** ensure airway is patent but avoid premature intubation.
- **Breathing:** give high-flow oxygen via non-rebreather mask targeting SpO_2 >92–94% as respiratory failure is a significant predictor for mortality. [1,3] Sit the patient upright if tolerated, assess work of breathing, and prepare for NIV or intubation depending on mental status, fatigue, aspiration risk, and risk of intubation.
- **Circulation:** secure two large-bore IV lines, apply cardiac monitoring, blood pressure and SpO_2 monitoring. Avoid indiscriminate fluid boluses in a congested phenotype, and consider vasopressors and inotropes.
- **Blood tests:** send urgent blood for complete blood count, liver and renal function test, troponin, NT-proBNP, arterial blood gas, lactate and blood culture if infection is possible.
- **Point-of-care ultrasound** (e.g. RUSH protocol): assess LV and RV function,

pericardial effusion, IVC size and collapsibility, lung B-lines, pleural effusion, abdominal free fluid, and abdominal aortic aneurysm to differentiate shock type.[4]

- **Search for reversible causes:** obtain a 12-lead ECG, review serial troponin, and assess for myocarditis, sepsis, opportunistic infection, pulmonary embolism, tamponade and drug or toxin exposure.

Investigation findings of the patient

Point-of-care USG shows:

- Dilated 4 chambers with global hypokinesis. Left Ventricular Ejection Fraction (LVEF) was 5-10% by eyeballing
- Mitral regurgitation and tricuspid regurgitation
- No pericardial effusion
- Dilated inferior vena cava with reduced respiratory variation
- No intraperitoneal free fluid or abdominal aortic aneurysm

Chest radiograph shows cardiomegaly with mildly congested lung fields.

Electrocardiogram shows sinus rhythm with mild T wave inversion over leads V5-V6.

These findings supported severe dilated cardiomyopathy with acute pulmonary oedema and cardiogenic shock.

What are the differential diagnoses of cardiogenic shock?

Cardiogenic shock is a syndrome of inadequate cardiac output causing tissue hypoperfusion and end-organ dysfunction.[1,4] Hypotension may be modest or absent early, so clinicians should look for cool extremities, altered mentation, oliguria, rising lactate, renal or liver dysfunction, pulmonary congestion, and bedside echocardiographic evidence of impaired cardiac performance.

The are many possible aetiologies for cardiogenic shock:

1) Myocardial
   - Chronic heart failure with acute decompensation
   - Acute myocardial infarction (AMI) or
     - Mechanical complications (papillary muscle rupture, ventricular septal rupture, free wall rupture)
   - Acute decompensated heart failure as first presentation (apart from AMI)
     - Dilated cardiomyopathy
     - Stress cardiomyopathy (Takotsubo)
     - Myocarditis (viral, autoimmune)
     - Peripartum cardiomyopathy
     - Endocrine associated (e.g. hyper/hypothyroidism)
   - Myocardial depression in septic shock
2) Valvular dysfunction (e.g. acute mitral regurgitation)
3) Arrhythmia induced
4) Extra-cardiac / obstructive (e.g. cardiac tamponade, massive pulmonary embolism)
5) Drug toxicity (e.g. beta blocker overdose)

How to classify cardiogenic shock by severity?

The Society for Cardiovascular Angiography and Interventions shock-stage system (SCAI SHOCK) is useful for determining mortality at each stage, as shown in Fig 1. [5]

- Stage A: At risk
- Stage B: Beginning
- **Stage C: Classic**
- Stage D: Deteriorating
- Stage E: Extremis

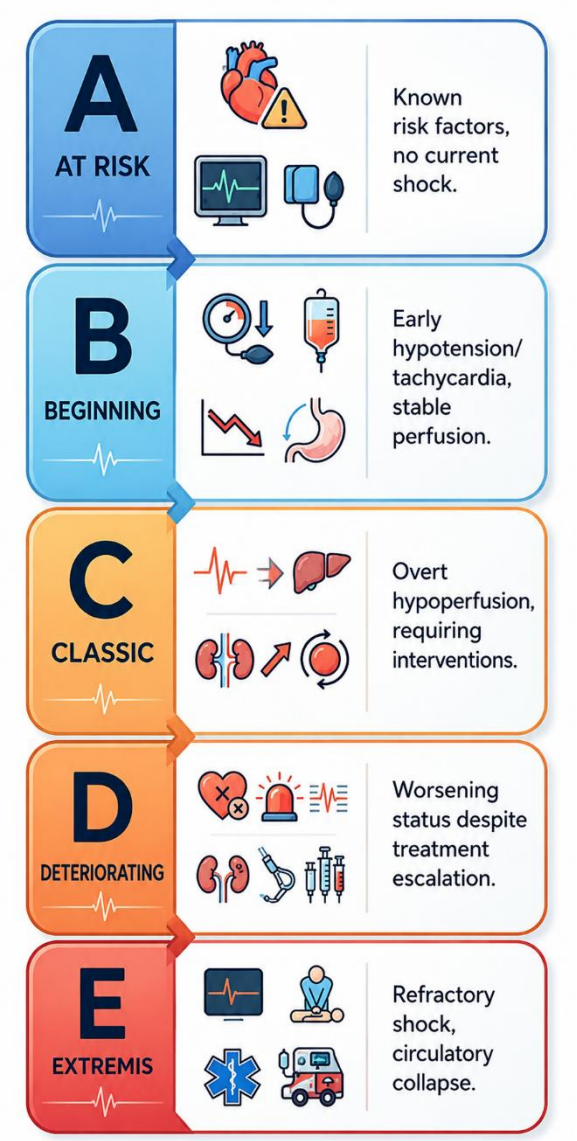

Fig.1 SCAI SHOCK classification

This patient was most consistent with **SCAI Stage C** because he had hypotension, severe pulmonary congestion, cool purplish peripheries and severe LV systolic dysfunction requiring vasoactive support. The stage should be reassessed after each intervention. Failure to improve despite norepinephrine and inotropic support would suggest progression toward Stage D and should prompt escalation to a shock team, invasive haemodynamic assessment, and consideration of temporary mechanical circulatory support. [5,6]

Should the patient be intubated given persistent hypoxia?

Persistent severe hypoxia and cyanosis despite non-rebreather mask make advanced respiratory support urgent.

However, intubation in cardiogenic shock is hazardous. Induction agents, loss of sympathetic tone and positive-pressure ventilation can reduce venous return, impair ventricular preload, and adversely affect right- and left-ventricular loading conditions, potentially precipitating haemodynamic collapse.

A short, closely monitored trial of non-invasive ventilation (e.g. BiPAP) while optimizing haemodynamic with inotropes or vasopressors can be appropriate in milder or early cardiogenic shock with isolated hypoxemia, preserved mental status, and ability to protect the airway. If there is worsening oxygenation despite NIV, deteriorating mental status or respiratory fatigue, definitive airway control should not be delayed. [1,2]

To reduce the risk during intubation:

- Brief the team and prepare for peri-intubation collapse
- Optimise MAP before induction, usually with norepinephrine already running
- Use haemodynamically cautious induction dosing
- Prepare push-dose vasopressor
- Avoid excessive positive end-expiratory pressure after intubation

What is the diagnosis for the patient?

This patient suffers from **severe dilated cardiomyopathy complicated with cardiogenic shock and acute pulmonary edema,** underlying likely **HIV-related cardiomyopathy (HIVAC).**

HIV-RELATED CARDIOMYOPATHY (HIVAC)

What is HIV-related cardiomyopathy?

HIVAC is myocardial dysfunction occurring in people living with HIV. Myocardial disease in HIV is multifactorial and may arise from HIV-related myocardial injury, opportunistic infections, other viral infections, autoimmune responses, drug-related toxicity, nutritional deficiency and prolonged immunosuppression.[7] In regions with widespread ART, the prevalence of HIVAC has been reported to be roughly 4% among people living with HIV.

The classical presentation of HIAVC is symptomatic heart failure with dilated cardiomyopathy, typically featuring decreased ejection fraction.[1] While historically presenting with severe systolic failure, modern cases often show subclinical left ventricular diastolic dysfunction, resembling heart failure with preserved ejection fraction (HFpEF).[8]

Presentation includes heart-failure symptoms such as dyspnoea, oedema, fatigue, and in the late presentation, arrhythmias or sudden death. In advanced untreated disease, the classical phenotype was severe dilated systolic cardiomyopathy. In the modern ART era, the spectrum has broadened, and patients may present with more diverse or even subclinical myocardial dysfunction rather than florid dilated cardiomyopathy alone.

What is the management of HIV related cardiomyopathy and cardiogenic shock?

The management includes resuscitation of cardiogenic shock, management of heart failure, treating the underlying causes and optimising HIV control.

Haemodynamic support:

Norepinephrine is generally preferred as the first-line vasopressor when hypotension threatens coronary and end-organ perfusion.[1,2,6,9] In the SOAP II trial, dopamine caused more arrhythmic events and there was increased 28-day mortality rate with dopamine in the cardiogenic shock subgroup.[9]

Practical vasoactive approach (Fig.2):

- **Norepinephrine:** first-line vasopressor for hypotensive cardiogenic shock to restore perfusion pressure.
- **Dobutamine:** add if cardiac output remains low after MAP is supported; monitor for tachyarrhythmia and hypotension.[2]
- **Milrinone:** consider when beta-blockade, pulmonary hypertension or RV dysfunction is relevant

Target MAP is commonly at least 65 mmHg but should be individualised according to mentation, urine output, lactate clearance, skin perfusion, renal function and chronic blood pressure.[1,2]

Respiratory support:

High-flow oxygen should be given via a non-rebreathing mask initially.Non-invasive ventilation (NIV) can be used as bridge for selected patients with cardiogenic pulmonary oedema.

Early intubation is appropriate for refractory hypoxaemia, worsening respiratory fatigue, deteriorating mental status, or failure of NIV, but the haemodynamic risk must be anticipated.[1,2]

Mechanical circulatory support (MCS):

Temporary MCS, including intra-aortic balloon pump, Impella or VA-ECMO, should be considered for refractory cardiogenic shock.[1,2,4]

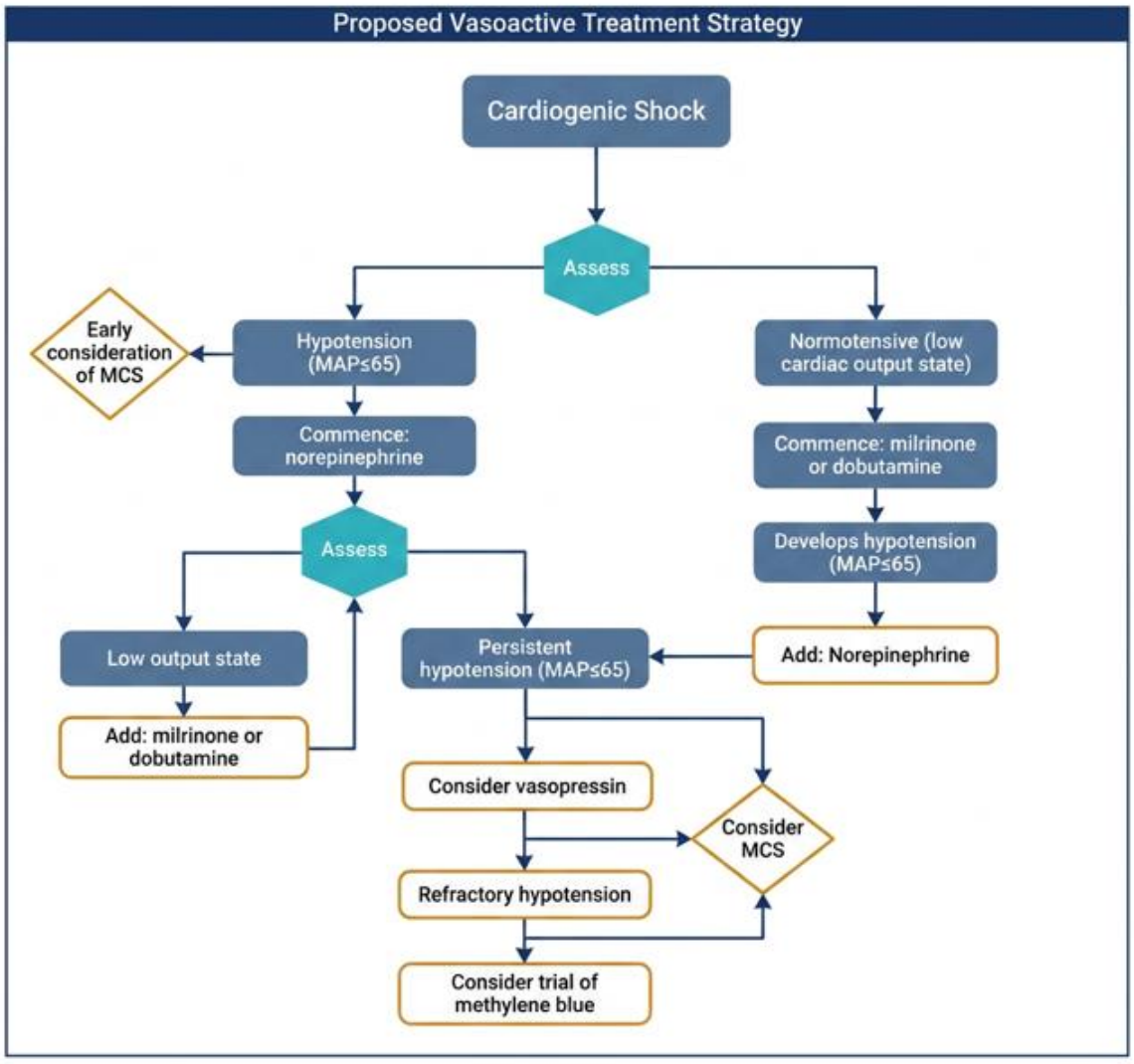

Fig .2 Stepwise vasoactive strategy in cardiogenic shock (Modified from Bloom et al.[6])

Treat the underlying cause:

Treatment for the underlying cause may include:

- Optimise ART (address adherence)
- Rule out ischaemia (angiography if indicated)
- Exclude opportunistic infections
- Evaluate for causes of decompensation such as pulmonary embolism, pulmonary hypertension or pericardial diseases

What is guideline-directed medical therapy (GDMT) for HFrEF?

Once shock has resolved and the patient is haemodynamically stable, treatment should transit from resuscitation to long-term disease modification. HFrEF is defined by LVEF ≤40%, and guideline-directed medical therapy (GDMT) includes four foundational pillars: [10,11]

1. ARNI (sacubitril/valsartan) or ACEI/ARB
2. β-blocker
3. Mineralocorticoid receptor antagonist
4. SGLT2 inhibitor

These are initiated once haemodynamically stable, congestion is improving, renal function and potassium are acceptable, and the patient can tolerate oral therapy. Beta-blockers and vasodilating agents should generally be avoided during active shock or persistent hypoperfusion.

PROGRESS OF THE PATIENT:

The patient was managed in the Coronary care unit (CCU) with vasopressor and inotropic support; mechanical devices were considered but not required. After stabilisation, full guideline-directed medical therapy (GDMT) for HFrEF was started alongside optimised ART.

Learning points

- HIVAC carries high mortality and can rapidly progress to cardiogenic shock.
- Early SCAI staging and multidisciplinary care (cardiology, infectious diseases, critical care) is essential.
- Norepinephrine is the preferred vasopressor agent; inotrope(s) should be added when low cardiac output persists.
- For HIV cardiomyopathy, GDMT for HFrEF and optimisation of ART are essential after stabilisation of cardiogenic shock.

The role of emergency physician:

- Early recognition of occult shock
- POCUS as an extension of bedside examination
- Judicious airway and ventilation strategy
- Rational sequencing of vasoactive therapy
- Early escalation and multidisciplinary coordination

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