Lessons From Practice

The Effects of Positive Pressure Ventilation on Acute Cardiac Tamponade

Muhammad Khidir Mohd Kamil[1], Muhammad Izzat Abdul Halim[2], Khairul Naim Rasman[2], Mohd Anizan Aziz[2], Samsu Ambia Ismail[2]

[1] *Unit of Emergency Medicine and Trauma, Hospital Seri Iskandar, Perak, Malaysia*
[2] *Department of Emergency Medicine and Trauma, Hospital Teluk Intan, Perak, Malaysia*

Abstract

In the resuscitation of acute cardiac tamponade, it is imperative to prioritize circulatory management over airway and breathing interventions. The application of both invasive and non-invasive positive pressure ventilation (PPV) can be detrimental due to the resultant increase in intrathoracic pressure, which adversely affects a heart dependent on preload. Therefore, timely pericardiocentesis is essential to reduce intrapericardial pressure, thereby improving stroke volume and cardiac output. This case elucidates the adverse outcomes, including clinical deterioration, associated with non-invasive PPV in the context of cardiac tamponade.

Keywords: *Cardiac tamponade, pericardiocentesis, intubation, positive pressure ventilation, non-invasive ventilation*

CLINICAL CASE

A 76-year-old male with no known comorbidities presented with acute onset chest pain, shortness of breath, and reduced effort tolerance for one week. He denied experiencing orthopnea, paroxysmal nocturnal dyspnea, fever, and constitutional symptoms. There was no history of contact with pulmonary tuberculosis or family history of malignancy. He did not consume alcohol or smoke.

On examination, his vital signs demonstrated blood pressure 120/80 mmHg, heart rate 106 beats/min, respiratory rate 24 breaths/min, temperature 36.5 °C, and oxygen saturation 94% on room air. Cardiovascular assessment revealed dilated jugular venous pressure and crackles in the left lung with reduced air entry.

His ECG revealed sinus tachycardia, incomplete right bundle branch block (RBBB), and low QRS voltage. Bedside echocardiography showed an irregular pericardial wall with a large pericardial effusion measuring 2.77 cm, right ventricular diastolic collapse, right atrial systolic collapse, and a plethoric inferior vena cava with minimal respiratory variation. [Figure 1] Laboratory investigations indicated a white cell count 15.1 thousand / uL, urea at 34.4 mmol/L, creatinine at 225 µmol/L, and high-sensitivity troponin I at 692.8 ng/L. His chest X-ray showed cardiomegaly, left pleural effusion, and retrocardiac consolidation. [Figure 2] Arterial blood gases revealed type 1 respiratory failure with acute respiratory alkalosis.

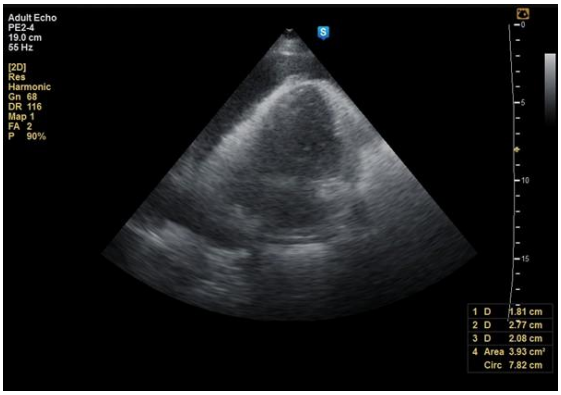


Figure 1: Apical 4 chamber view shows large pericardial effusion measuring 2.77cm with right ventricle diastolic collapse.

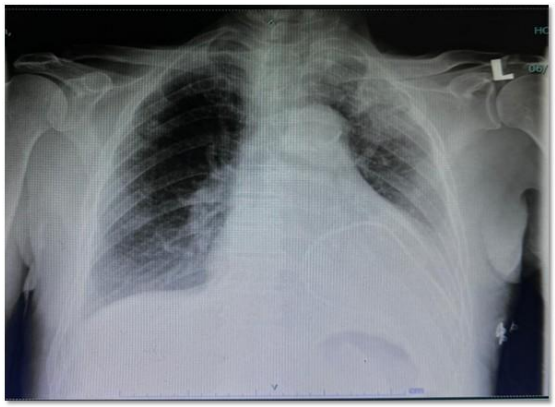


Figure 2: The chest x-ray shows cardiomegaly, left pleural effusion, and retrocardiac consolidation.

He was placed on bilevel positive airway pressure (BiPAP) due to worsening respiratory distress. After 20 minutes, the patient became hypotensive with a systolic blood pressure of 80-90 mmHg, and a diastolic blood pressure of 40-50 mmHg, along with compensatory tachycardia with heart rate ranging 150-160 beats/min. BiPAP was discontinued and replaced with high-flow nasal cannula (HFNC), which the patient tolerated well. Pericardiocentesis was performed via parasternal approach, and a catheter was inserted, draining 250 ml hemoserous fluid. Analysis of the pericardial fluid revealed atypical cells with immature lymphocytes. Contrast-enhanced computed tomography of the thorax showed a left lung mass with distant metastasis to both lungs, the pericardium, and the pleura.

DISCUSSION

We present a case of cardiac tamponade worsened by non-invasive PPV. Unlike Anthony et al., our case was linked to malignancy.[1] Our patient experienced a gradual buildup of pericardial fluid before developing tamponade. The pericardium, a dual-layered sac, normally holds up to 50 ml of serous fluid, which provides lubrication and protection against infection. While the pericardium can stretch, exceeding its elastic limit increases intrapericardial pressure. This rise in pressure affects both left and right atrial and ventricular diastolic pressures, leading to elevated filling pressures and a subsequent decline in cardiac volumes. Reduced diastolic filling decreases stroke volume, and compensatory mechanisms like increased cardiac contractility and tachycardia are insufficient to maintain stroke volume, resulting in decreased cardiac output.[2]

Animal studies have shown the effects of PPV on pericardial effusion.[3-4] Both studies found that fluctuations in pleural pressure during intermittent PPV were transmitted to the pericardial fluid. Cardiac output and right ventricular end-diastolic pressure were higher during spontaneous ventilation compared to intermittent PPV, with or without positive end-expiratory pressure.[3-4]

In resuscitation in the ED, airway and breathing are prioritized over circulation, leading clinicians to use non-invasive ventilation (NIV) to reduce work of breathing. If the patient deteriorates despite on NIV, invasive ventilation may be required. Cardiac tamponade is preload-dependent, and PPV further increases diastolic pressure, reducing stroke volume and cardiac output, worsening the condition.[1-2,5-6] This creates a vicious cycle in acute crashing cardiac tamponade and may result in cardiac arrest if tamponade was not relieved. [Figure 3]

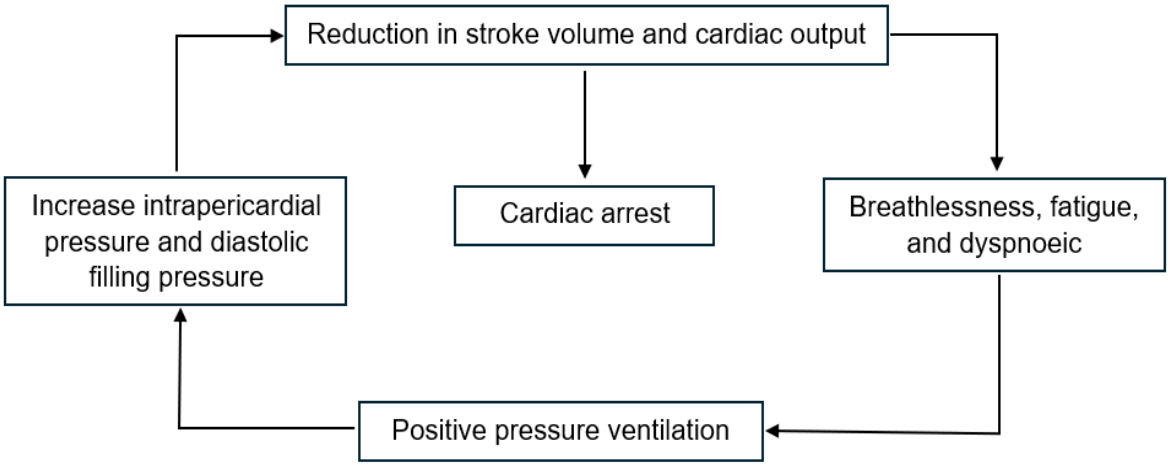
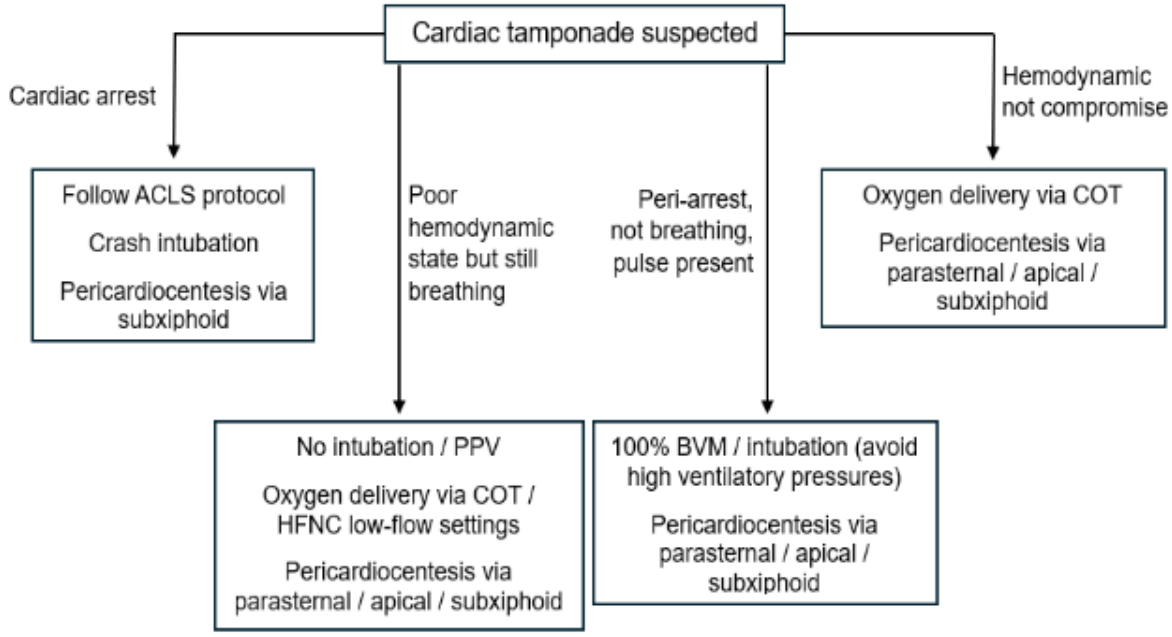


Figure 3: A cycle of positive pressure ventilation raises intrathoracic pressure which increases intrapericardial and right ventricle diastolic filling pressures, ultimately reducing stroke volume and cardiac output.

In our case, the patient, though hemodynamically stable but in distress, experienced significant tachycardia and hypotensive episodes after the initiation of BiPAP. Tachycardia served as a compensatory mechanism to maintain cardiac output.[2] The patient's condition improved after being switched to HFNC and undergoing pericardiocentesis. Although HFNC theoretically provide low PEEP (2 – 5 cmH_2O), our patient was well tolerated using HFNC as a mode of ventilatory support.[7]

We present an algorithm for acute cardiac tamponade management in the ED. [Figure 4] For patients who maintain respiration but are hemodynamically compromised or in distress as seen in our case, it is advisable to abstain from intubation or NIV. Oxygen delivery should employ either 100% oxygen therapy or HFNC at low-flow rates. Pericardiocentesis may be executed via subxiphoid, parasternal, or apical approaches. We recommend the parasternal approach for pericardiocentesis under ultrasound guidance due to its proximity to pericardial effusion and decreased risk of hepatic and pulmonary injury.[8]



ACLS, Advanced Cardiac Life Support; BVM, bag-valve mask; COT, conventional oxygen therapy; HFNC, high-flow nasal cannula

Figure 4: A proposed algorithm for the management of acute cardiac tamponade in the emergency department.

CONCLUSION

The principal management of acute crashing cardiac tamponade should be emphasized on circulation rather than standard 'airway and breathing', and timely pericardiocentesis should be performed to improve the outcome of the patients. Oxygen desaturation and hypotension should not be translated automatically into immediate intubation and PPV.

CONFLICT OF INTEREST

We declare that there is no conflict of interest in this study.

AUTHORS' CONTRIBUTION

Every author has made a significant contribution to the publication of this journal.

CORRESPONDENCE

Muhammad Khidir Mohd Kamil,
MB BCh BAO, MEmMed
Unit of Emergency Medicine and Trauma,
Hospital Seri Iskandar,
Perak, Malaysia.
ORCID: 0000 0002 5932 4152
Email: presisoc90@gmail.com

LESSONS FROM PRACTICE

- In an acutely crashing cardiac tamponade, always prioritize circulatory support and rapid cardiac decompression over standard airway and breathing interventions.
- Emergent pericardiocentesis is crucial to immediately relieve intrapericardial pressure, thereby restoring cardiac output.
- The use of both invasive and non-invasive positive pressure ventilation can be lethal due to the resultant increase in intrathoracic pressure in a preload-dependent heart.

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