

A DETAILED STUDY OF THE DISCUSSION AND MANAGEMENT OF CARDIAC
TAMPONADE IN A 62-YEAR-OLD PATIENT: A CASE STUDY REVIEW

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ABSTRACT

Background: Adherence to quality nursing care standards is essential for ensuring safe and effective healthcare delivery. Though Nurses may possess good knowledge of quality nursing care standard protocols, organizational and contextual factors may influence the extent to which these standards are applied in practice. **Aim:** This study assessed factors influencing adherence to quality nursing care standards among nurse managers in selected tertiary health institutions. **Methods:** A convergent parallel mixed-methods design was employed. Quantitative data were collected from 65 nurse managers using a self-developed structured questionnaire and analyzed using descriptive and inferential statistics. Pearson and Spearman correlation analyses were used to examine the relationship between years of experience and compliance, while Chi-square tests assessed associations between knowledge, staffing adequacy, and utilization of standard protocols. Qualitative data were obtained through one-on-one interviews and open-ended questionnaire items and analyzed thematically. Ethical approval was obtained prior to data collection. **Results:** Quantitative findings showed no significant relationship between years of experience and compliance with nursing care standards ($r = 0.046$, $p = 0.714$), nor between knowledge of standard protocols and their utilization ($\chi^2 = 4.287$, $p = 0.369$). However, a significant association was found between staffing adequacy and utilization of nursing care protocols ($\chi^2 = 14.246$, $p = 0.007$). Qualitative findings revealed that staffing shortages, high workload, limited resources, inadequate institutional support, and insufficient continuous training constrained adherence to standards. Integration of findings indicated that organizational factors are major determinants of adherence irrespective of individual knowledge or experience. **Conclusion:** Adherence to quality nursing care standards among nurse managers is predominantly influenced by organizational and system-level factors rather than individual competencies alone. Improving staffing levels, strengthening institutional support, and ensuring adequate resources are essential for enhancing utilization of quality assurance protocols in tertiary health institutions.

KEYWORDS: Quality nursing care; adherence; quality assurance; Nurse Managers.

INTRODUCTION

Excess fluid accumulation in the pericardial sac causes a rise in intrapericardial and intracardiac pressure, leading to impaired diastolic ventricular filling and a progressive decline in cardiac output, ultimately resulting in cardiac tamponade. A life-threatening condition.^{[1][2][3]}

Cardiac tamponade can be broadly categorized into Acute (penetrating chest trauma/aortic dissection or free ventricular wall rupture followed by MI) leading to severe hemodynamic instability.^[4]

Subacute or chronic causes like infection (TB), autoimmune causes (SLE, RA) / tumors-effusions, chronic pericarditis or even metabolic causes like uremia develop gradually until intrapericardial pressure becomes critical.^{[3][5]}

Iatrogenic complications of invasive cardiac procedures, especially cardiac catheterization, can also cause cardiac tamponade. Recent studies report an incidence of 3.3–8.4%, with risk increasing with age and female gender. Procedure-related tamponade contributes to

approximately 8.5% of in-hospital mortality associated with coronary interventions.^[6] The diagnosis of cardiac tamponade is a clinical diagnosis based on suggestive history and clinical presentation with worsening dyspnea, distended jugular veins, muffled heart sounds and pulsus paradoxus, and should be confirmed by using Doppler echocardiography and pericardiocentesis (a percutaneous route to drain fluid guided by echocardiography or fluoroscopy under local anesthesia) via a subxiphoid approach.^[7]

Due to rapid progression (especially in cancer patients who have a bad short-term prognosis) and high mortality with delayed recognition, understanding the evolving etiological patterns, diagnostic approaches, and clinical implications of cardiac tamponade remains very significant.^[2]

CASE PRESENTATION

This case is centered on a 62-year-old female who presented with a two-week history of shortness of breath and worsening fatigue. She describes a persistent sensation of “lack of air” with dyspnea initially on exertion but now at rest, along with orthopnea.

She was hospitalized as the symptoms worsened. Her past medical history is notable for breast cancer diagnosed 6 years ago, for which she underwent a radical left mastectomy.

On admission, her general conditions were severe, she had afebrile with body temperature of 36.5 degrees, but had impaired skin turgor and overall dryness. Her face was swollen due to increased subcutaneous fat. Sclera was normal, no rash, abdominal distention or neurological symptoms. Her vitals were normal (blood pressure of 115/79 mm Hg, not following the classic hypertensive presentation of becks triad for cardiac tamponade)her but she had tachypnea and oxygen saturation dropped until 85 %, suggestive of mild hypoxemia. Her glucose was 155 mg/dL And slight decline in her urine output.

A CXR chest was performed which revealed a large pericardial effusion & flattened roots in middle and lower lungs fields suggesting pleural effusion. Ultrasound confirmed the presence of bilateral effusion of approximately 400-450 ml on the right lung and 300-350ml on the left lung. Clinically, wet wheezing, unresponsive to percussion and respiratory compromise was also identified. Laboratory examination revealed thrombocytosis and neutrophilia, suggesting inflammation.

Cardiovascular examination percussive dullness (due to fluid displacement). The ECG (figure 1.0) shows sinus rhythm with mild tachycardia HR of 98 bpm, low-voltage QRS complexes, electrical alternans and poor R-wave progression, supporting the presence of a pericardial effusion; echocardiogram(figure 2.0) was performed for confirmation.

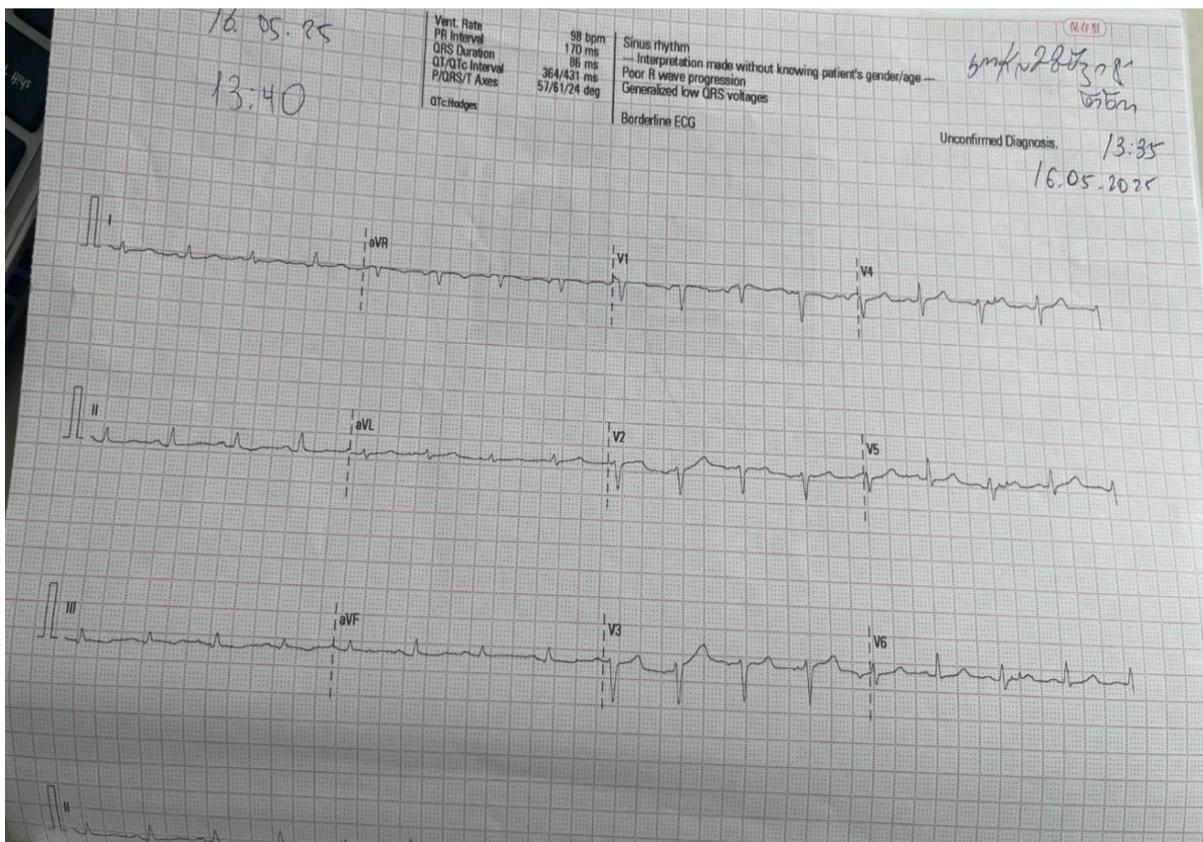


Fig. 1.0: ECG of the patient.

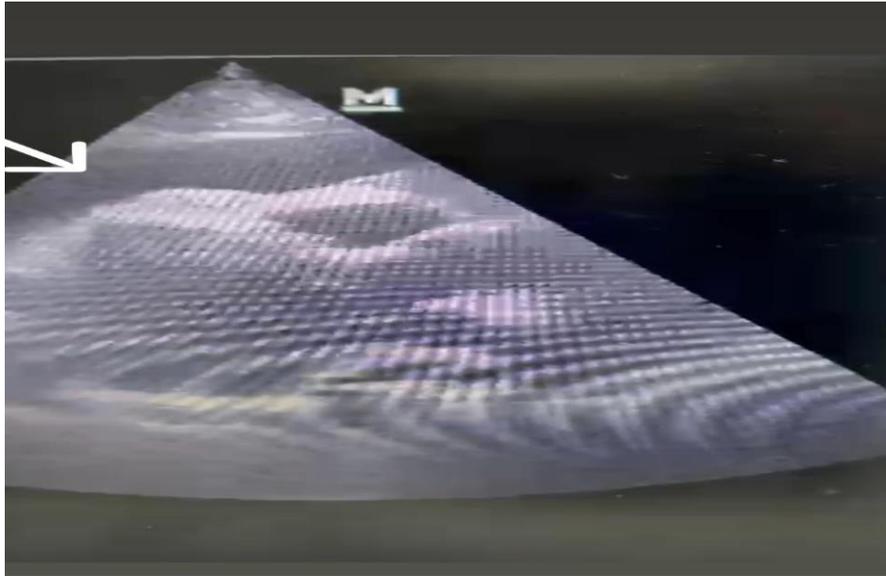


Fig. 2.0: Echocardiogram of the heart, the arrow indicates the pericardial effusion.

Heart sounds were rhythmic but muffled, raising suspicion for fluid build up. No ischemic changes were observed. Interestingly, she showed no signs of a JVD.

Given her symptoms, she was transferred to the cardiac department from the ER. She remained in critical condition, with tachypnea at rest and ongoing respiratory compromise. Oxygen therapy was administered immediately (8L/min) to correct low saturation. Medical management involved administration of IV furosemide (a loop diuretic) to relieve fluid overload and improve respiratory effort. She also was given enoxparin (0.4 mL) for thromboembolism prophylaxis (high risk of clot formation due to immobility). Coraxan was used to support cardiac function. Normal saline (0.9% NaCl, 500ml) IV for stabilizing and hydration.

A definitive diagnosis was made through echocardiogram, showing Pericardial effusion, particularly along the posterior pericardial wall which was causing significant cardiac compression consistent with cardiac tamponade. This prompted an urgent pericardiocentesis, 1000 mL of hemorrhagic pericardial fluid was aspirated. This resulted in rapid improvement. She was relatively hemodynamically stable, respiratory failure decreased. Her overall condition improved sufficiently & she remained under continuous monitoring.

DISCUSSION

Cardiac tamponade is a life threatening medical condition where excessive fluid accumulate in the pericardial sac constricting heart reducing the diastolic filling and cardiac output that can eventually cause cardiogenic shock or death. Pericardial effusion is when fluid accumulate in the sac around the heart, cardiac tamponade is a hemodynamically unstable manifestation of pericardial effusion.

Pericardium is made of two layers the outer fibrous layer made up of an elastic connective tissue and the inner serous layer is also made of two layers - the parietal layer that is in contact with the fibrous outer layer and the visceral layer in contact with the heart also known as the epicardium. The epicardium responsible for production of pericardial fluid. The space between the parietal and visceral layer is where the pericardial fluid exists. The pericardial cavity of a healthy adult is filled with 15 - 50ml of clear, straw coloured fluid. It helps in cushioning, shock absorption and reduces friction.

Infections, inflammation, uraemia and neoplasms produce slow accumulating effusions meanwhile iatrogenic or other traumatic injuries to heart wall, myocardial defects or ventricular wall rupture caused by MI or descending aorta dissection that leaks into the pericardium cause acute cardiac tamponade.^{[8][13]} Slow accumulating effusion is tolerated better than the fast accumulation because when the fluid buildup slowly the fibrocollagenous tissue of pericardium expand to accommodate the growing stress pericardium has limited elasticity therefore the effusion can reach tamponade physiology but it can grow upto 1000ml before it start affecting the diastolic filling.^[11] When there is an acute buildup of fluid pericardial compliance is compromised and 100 - 150 is enough to cause cardiac tamponade.^[9]

The most common cases are iatrogenic traumas like complication of a pacemaker insertion, pericarditis and malignancies followed by idiopathic cases. The fluid can be blood (hemopericardium), pus, chylous or gas (pneumopericardium) singly or in combination.

A 62 year old female came in with dyspnea, orthopnea and fatigue which are common symptoms of cardiac tamponade. The classic sign of cardiac tamponade is the Beck's triad of hypotension, jugular venous distension and muffled heart sounds. Patients may also display

signs like chest pain, palpitations, dizziness, changes in skin colour and altered mental status. Other common signs include pluses paradox, its drop of more than 10 mm hg of systolic pressure during inspiration^[8], decreased electrocardiographic voltage with electrical alternans and tachycardia (fig. 2.0).

Kussmaul's sign, rise in jugular venous pressure during inspiration, is also sometimes seen with cardiac tamponade.

When fluid accumulates in the pericardial cavity (fig. 1.0), it compresses the thin right heart which impairs the diastolic filling thus reducing the preload and stroke volume. The compensatory increased tachycardia is not enough to maintain stroke volume that results in reduced cardiac output.^[10] The amount of fluid required to affect the diastolic filling depends on the rate of accumulation and the compliance of the heart.

When the cardiac output is reduced several neurohormonal mechanisms get activated like elevated activities of adrenergic nervous system and the renin-angiotensin-aldosterone system, acutely this activation might be able to restore cardiac function but if the condition persists the changes will not be able to maintain the cardiac function and heart will progress into a state of decompensated heart failure.

Diagnosis can be suspected based on clinical findings. ECG showing low voltage or electrical alternans (refer fig. 1.0), X-ray showing enlarged silhouette and echocardiography showing right ventricular collapse or bowing of the interventricular septum to the left help to confirm the diagnosis.

Echocardiography assisted pericardiocentesis was performed on the patient 1000ml of hemorrhagic fluid was aspirated. The causes of hemopericardium include invasive cardiac procedures (accounts for 31% of the cases), thoracic trauma, complications of ischemic heart disease (e.g. left ventricle-free wall rupture), malignancy, infection, autoimmune conditions, anticoagulants, chronic kidney disease, ruptured coronary artery aneurysms and medications.^[14] Non cardiac malignancies are most commonly related to hemopericardium compared to cardiac malignancy. These include lung and breast cancer followed by lymphoma, melanoma and renal cell carcinoma. Prevalence of hemorrhagic pericardial effusion caused by tuberculosis has declined but it is still significant among HIV patients. Extensive diagnostic workup should be conducted to determine the etiology of hemopericardium. It also can be caused by unknown factors which is called the idiopathic hemorrhagic pericardial effusion. There is a lack of epidemiological data about hemopericardium.

Distinctive diagnosis

1. Large pericardial effusion without tamponade
2. Constrictive pericarditis

3. Pulmonary embolism
4. Tension pneumothorax
5. Cardiogenic shock
6. Aortic dissection.

MANAGEMENT

Cardiac tamponade is addressed by removing the fluid surrounding the heart through a procedure known as pericardiocentesis, which involves using a needle to drain the fluid.

Cardiac tamponade can also be treated through surgery in certain situations, especially if it results from an injury, if there is damage needing repair, if a needle cannot access the fluid, or if removal of the pericardium is necessary. In critical cases, such as cardiac arrest due to tamponade, a thoracotomy may be performed at the bedside in the ER. Additionally, it's important to address the underlying cause of the cardiac tamponade, which may involve administering pain relief, antibiotics, or other medications based on the specific situation.^[15]

But before proceeding to either of these methods, it's important to take measures to not aggravate the symptoms by providing the patient with oxygen therapy, volume resuscitation, and be placed on bed rest with the legs elevated. It's also advised to avoid using positive-pressure ventilation as it would reduce the venous return even further making the condition worse.^[4]

Because of its excellent safety profile and dependable access to the pericardial space, the subxiphoid route is still the most widely used technique for pericardiocentesis. This technique is especially appropriate in both emergency and elective situations since it reduces the possibility of harming nearby tissues like the myocardial, lungs, and coronary arteries. It has been seen in all of the trials that the subxiphoid method of pericardiocentesis produced extremely low rates of complications. The rarity and often moderate nature of adverse events, such as arrhythmias, vascular damage, or pneumothorax, further support the efficacy and safety of this strategy.^[3]

CONCLUSION

Cardiac tamponade is a medical emergency that can rapidly progress to circulatory collapse and death if not promptly recognized and treated. This example demonstrates how tamponade can manifest atypically, particularly in individuals with underlying cancer, where conventional symptoms like hypotension or raised jugular vein pressure may not be evident despite significant impairment. Both the volume and velocity of fluid accumulation affect the physiological effects of pericardial effusion; even at modest volumes, fast effusions can cause life-threatening compromise because of restricted pericardial compliance.

Echocardiography remains the diagnostic modality of choice and plays a crucial role in guiding timely

pericardiocentesis, which can lead to dramatic clinical improvement, as seen in this case following drainage of 1000 mL of hemorrhagic fluid.

Given the broad range of possible causes, particularly malignancy and iatrogenic injury, determining the underlying etiology is critical for appropriate long-term management. Early recognition, rapid drainage, and thorough investigation are key to improving outcomes in cardiac tamponade.

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I confirm that I have obtained full consent from the patient to use his/her clinical details for educational and publication purposes. The pictures of the ECG and Echocardiogram was attached with the consent of the patient and consulting physician. This report was prepared with the assistance of artificial intelligence for language enhancement only; all medical content and interpretation are based on the original clinical findings.

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