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Cardiac Tamponade

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Introduction

What is the Topic?

The topic the author chose to research is cardiac tamponade. Cardiac tamponade occurs when fluid builds up in the pericardial cavity, which is the cavity that surrounds the heart. Cardiac tamponade can occur due to various reasons, including myocardial infarctions, pericarditis, traumas, and placement of pacer wires. Palpitations, chest pain, and shortness of breath are common symptoms that occur with cardiac tamponade. Pericardiocentesis and a pericardial window are two interventions that are commonly used to treat cardiac tamponade. Cardiac tamponade is a medical emergency and without any intervention, may lead to cardiac arrest and possible death.

Why was the Topic Chosen?

The author chose cardiac tamponade because she has some past knowledge of the condition. The author previously worked on a cardiac step-down unit and would care for patients that had had pericardial windows performed. Currently, the author works in an emergency department. Two weeks ago, the author assisted another nurse in caring for a patient who was being treated for a heartrate of 18 beats per minute. Transcutaneous pacing was not effective. The cardiologist on call placed a tranvenous pacer while the patient was still in the emergency room. Cardiac tamponade is relevant to the author's nursing practice because the author knows the condition could result from the placement of the pacer wires.

Signs and Symptoms

- Dyspnea
- Chest pain
- Hypotension
- Pulsus paradoxus
- Tachycardia
- Musculoskeletal pain
- Beck's triad- diminished heart sounds, hypotension, & jugular vein distention is classic for cardiac tamponade
- Feeling of anxiety
- Feeling of restlessness
- Fever
- Abdominal distention
- Pericardial effusion on echocardiogram
- Cardiomegaly on chest x-ray

Pathophysiological Processes

Underlying Pathophysiology

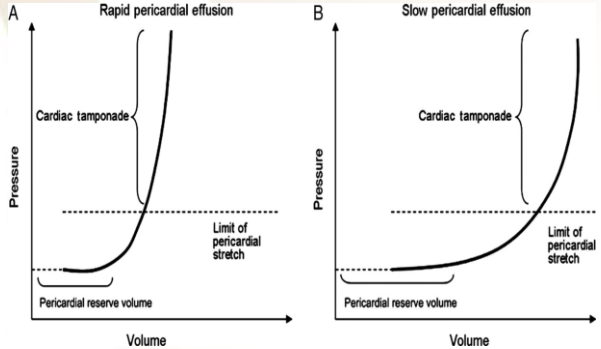
Cardiac tamponade occurs as fluid accumulates in the pericardial sac and causes increased compression on the heart. A reduction in diastolic filling occurs as the pressure of the pericardial fluid increases and thus causes the heart chambers from filling properly. Because the right side of the heart has lower diastolic pressures, the atrium and ventricle are the first to be affected. The improper filling of the right atrium and the right ventricle lead to signs of right sided heart failure, such as hepatomegaly and jugular vein distention. Cardiac arrest can then occur if the left atrium becomes affected due to the minimal filling and circulatory collapse.

Cardiac tamponade has many causes, including infections, trauma, surgery, and placement of pacer wires, a pacemaker, or a central venous line. Cardiac tamponade can be caused by infections, mainly pericarditis. Trauma can cause cardiac tamponade due to perforation of the pericardial sac and resulting accumulation of fluid. Surgery and placement of pacer wires or central lines also cause cardiac tamponade due to perforation of the pericardium. "Perforation by a catheter as well as direct infusion of fluid can cause CT; the right atrium is the most common site of catheter perforation" (Schub & Boling, 2015, p. 1).

Significance of Pathophysiology

The pathophysiology of cardiac tamponade is significant so providers can be proactive instead of reactive. The pathophysiology is important to know how tamponade occurs and for what signs to monitor. Because cardiac tamponade can occur quickly due to the rapid filling of the pericardial sac, it is important to monitor the patient closely. One of the most important diagnostic tests is a chest x-ray after placement of pacer wires or a central venous line. The chest x-ray would show cardiomegaly, which would suggest cardiac tamponade. Daily chest x-rays after cardiac surgeries are obtained for this reason as well. The heart is able to withstand fluid accumulations, as long as the fluid builds up gradually. Echocardiograms would also show the presence and size of a pericardial effusion. Monitoring for heart rate and respiratory rate increases and blood pressure decreases would also alert providers to the possibility of developing tamponade. Because the signs and symptoms of cardiac tamponade are not specific until it becomes acute, it is important to quickly rule out other conditions, including acute myocardial infarctions, hypothyroidism, and cancers. The pathophysiology is significant for providers to recognize the signs and symptoms of cardiac tamponade and to intervene before the condition becomes severe and causes death.

Graph 1. Pressure/volume curve of the pericardium with fast accumulating pericardial fluid leading to cardiac tamponade with a smaller volume (A) compared with the slowly accumulating pericardial fluid reaching cardiac tamponade only after larger volumes (B).



Research Topic

"Cardiac tamponade (CT) is a life- threatening condition and a medical emergency characterized by pathologic accumulation of fluid in the pericardial sac that compresses they myocardium, prevents adequate cardiac filling, and reduces cardiac output" (Schub & Boling, 2015, p.1). Fluid builds up in the pericardial sac, which then causes increased cardiac pressure and leads to compression of the heart. Cardiac tamponade results from quick accumulation of even small amounts of fluid. "A rapid accumulation of pericardial fluid will quickly compress the heart, resulting in decreased cardiac output, shock, and (ultimately, if undiagnosed) death" (Ikematsu & Kloos, 2012, p. 264).

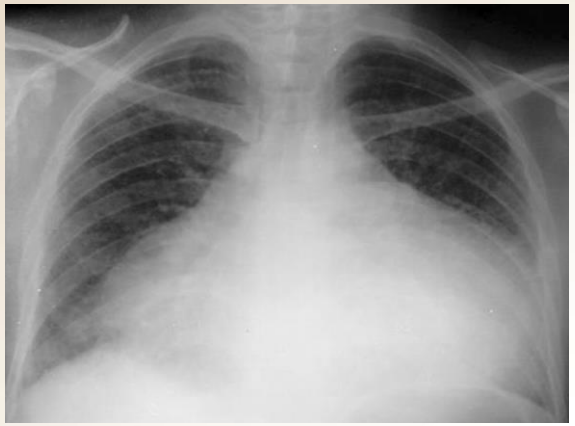


Figure 1. Chest x-ray showing cardiac tamponade.

Case Study

An 83 year old female presented to the emergency room with complaints of generalized weakness. Upon arrival, patient was pale and diaphoretic. Cardiac monitor showed sinus bradycardia with a heart rate of 29 beats per minute. Initial blood pressure was 102/44 and respiratory rate of 24 breaths per minute. Patient was complaining of midsternal chest pressure at a 2 on a 1/10 pain scale. The patient was alert and oriented but lethargic upon arrival, with a GCS of 15. An initial EKG was obtained which showed a junctional rhythm with a left bundle branch block. A portable chest x-ray was obtained which showed cardiomegaly and small bilateral pleural effusions. 1.5 mg of atropine given by medics in squad prior to patient's arrival, another 1 mg of atropine given in ED. No change in HR to either dose. A dopamine drip was started at 30.4 mL/ hr. 1 g of calcium chloride, 50 mEq of sodium bicarbonate, and 500 cc bolus were given. Staff attempting to transcutaneously pace patient, but unable to capture. Patient then being bagged with 100% oxygen due to ineffective respirations. BP now 64/18 and HR 18. Patient given 20 mg etomidate and 50 mg rocuronium to prepare for intubation. Propofol drip then initiated at 10 mcg/kg/min. ET tube 22 cm at the lip, secured with tube holder. Cardiologist now at bedside to place transvenous pacer. Foley catheter and nasogastric tube inserted. Cardiologist remains at bedside to place triple lumen central venous catheter in left subclavian vein due to poor peripheral access and the need for pressers. Vitals then were HR 80 A-V paced on the monitor, RR 16, SpO2 97% on ventilator, BP 123/44. Patient then transported to ICU.

Patient's lab results showed sodium 131, potassium 6.1, chloride 95, BUN 51, creatinine 6.20, calcium 8.4. Troponin was 0.028 and TSH 6.600. Abdominal CT was negative as was head CT. Patient did have history of ischemic cardiomyopathy with preserved ejection fraction. 2D echo from previous admission five months prior showed biatrial enlargement, mild to moderate left ventricular hypertrophy, and enlargement of right ventricle. 2D echo obtained during this admission revealed moderate dilation of left ventricle, left ventricular diastolic dysfunction, and a severely dilated left atrium.

Implications for Nursing Care

Implications for nursing care include close monitoring of the patient. After placement of pacer wires or a central venous line, a chest x-ray needs to be obtained to ensure correct placement prior to using them. The chest x-ray would also rule out cardiomegaly. Vital signs need to be obtained at least every four hours to ensure patient stability. Nurses also need to be knowledgeable of equipment used, such as chest tubes and pleurovacs. The nurse needs to be able to recognize if a clot is present and drainage is decreased, which could also point to developing cardiac tamponade. Nurses need to be knowledgeable regarding cardiac tamponade to ensure they intervene appropriately if a patient's condition deteriorates.

Conclusion

Cardiac tamponade is a life- threatening emergency that can result in death if not treated quickly and appropriately. If a patient presents with hypotension, dyspnea, muffled heart sounds, and jugular vein distention, chest imaging, such as an x-ray should be obtained as soon as possible. An electrocardiogram would also be useful in determining the presence of a pericardial effusion, which usually precedes cardiac tamponade. Nurses, doctors, and other healthcare providers need to be knowledgeable on the presentation of cardiac tamponade to intervene quickly and prevent cardiac tamponade from causing a patient's death.

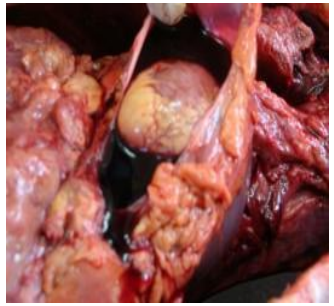


Figure 2. Cardiac tamponade due to hemopericardium

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