Patient Case Study

Justin J. Park

School of Nursing, Old Dominion University

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Dr. Sump

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Patient Overview

This case study shows a comprehensive exploration of the care of AB, a 75-year-old African-American male. It will examine the pathophysiology behind AB's condition and identify the signs and symptoms that allowed the medical team to determine his diagnosis. An in-depth analysis will translate those signs and symptoms into relevant nursing problems and review the nursing process that drove AB's care from a nursing perspective. Appropriate outcomes will be identified, and effective interventions will be examined. A thorough review of up-to-date research is included to justify the interventions used. An analysis and evaluation of follow-up assessment data will be used to determine how effective these interventions were for AB. Lastly, a review of knowledge gained from this case study and recommendations for alternative solutions that could have been implemented will be reviewed.

Admitting Diagnosis and Pathophysiology

AB reported to Chesapeake Regional Medical Center emergency department and stated that he had been feeling "poorly" for the previous "few days." Initial assessment of AB indicated bradycardia with a heart rate of 24 beats per minute and significant hypotension with a blood pressure of 63/40. An EKG was promptly performed, and a non-ST elevated myocardial infarction diagnosis with 3rd-degree atrioventricular block was given.

During an MI, cardiac tissue has an imbalance in the supply and demand of oxygen. Cardiac cells cannot receive enough oxygen, and cell death eventually occurs. Multiple factors can lead to MI. According to Harrington et al. (2019), a common cause of MI is atherosclerosis, a cardiac vessel narrowing due to arterial plaque buildup. Sometimes, this plaque buildup blocks enough blood supply to create a deficiency in O2 supply at rest. However, more commonly, plaque

breaks off and forms a thrombus in a cardiac artery, leading to occlusion, which results in infarction. The difference between a STEMI and an NSTEMI is the degree of blockage. During a STEMI, the coronary artery becomes completely occluded, resulting in extensive cellular damage and death. During an NSTEMI, there is only a partial occlusion, and cellular damage and death happen at a slower and are typically less extensive. Both conditions are medical emergencies.

Hinkle et al. (2022) state that the primary risk factor for MI is coronary artery disease (CAD) and its associated risk factors. Atherosclerosis is defined as an abnormal buildup of tissue in the vessel wall. This buildup leads to the narrowing of the coronary vessels, resulting in decreased blood flow to the coronary tissue. It is an insidious process that modifiable and non-modifiable risk factors can trigger. Non-modifiable risk factors include prolonged hypertension, hyperlipidemia, or tobacco use. Non-modifiable risk factors include genetics and family history.

AB was also diagnosed with a third-degree atrioventricular (AV) block. "Third-degree AV block occurs when no atrial impulse is conducted through the AV node into the ventricles" (Hinkle et al., 2022, p.710). The sinoatrial (SA) node is the primary pacemaker to establish heart rate. Electrical impulses are initiated by the SA node, where they travel to the AV node and then out through the bundle of HIS and Purkinje fibers, resulting in the contraction of heart tissue. When conduction is blocked, as with a third-degree AV block, tissues below the block never receive an impulse and, thus, do not contract. When this happens, cardiac tissue throughout the heart can take over and initiate its own impulse as a compensatory measure, albeit at a lower rate. Meloy et al. (2022) state that nearly 10% of myocardial infarctions result in a complete heart block. As myocardial tissue is subjected to prolonged ischemia and begins to necrose, it can no longer conduct electrical signals, resulting in the blockage. Heart rate will depend on

where the blockage is located, with blockages occurring lower in the conduction system, resulting in lower heart rates.

Presenting Signs and Symptoms

Upon admission to the ED, AB presented with signs and symptoms highly indicative of severe cardiac concerns and required urgent and immediate attention and intervention. AB also stated that he had been feeling "poorly" for a few days, diaphoretic, and "very tired" with shortness of breath. He denied having nausea, vomiting, or chest pain. Initial assessment revealed significant bradycardia with a heart rate of 26 beats per minute. Also noted was severe hypotension, evidenced by a blood pressure of 64/40. Both symptoms result in decreased cardiac output. Extreme bradycardia indicates that electrocardiac impulse occurs lower in the conduction symptom and is a hallmark identifier of a complete heart block. Hypotension is another indicator of MI and, combined with bradycardia, creates a vicious cycle. An EKG was performed. ST depression was noted along with a prominent Q wave. ST depression, as opposed to ST elevation, indicates ischemia without infarction. Prominent Q waves are indicative of previous MI. Stat labs were drawn, and the patient's troponin levels were elevated at 1.2ng/mL, whereas a normal troponin level is less than 0.04 ng/mL. Troponin is a protein in the heart that leaks into the blood as heart tissue is damaged. Elevated troponin levels are indicative of MI. Additional labs revealed a hematocrit of 29.9 % and a hemoglobin level of 8.9 g/dL. A normal hematocrit level for males is 42% - 52%, while normal hemoglobin levels are 14 g/dL - 17.4 g/dL (*Lippincott Advisor*, 2019). Low hematocrit and hemoglobin levels reduce the oxygen-carrying capacity of the blood, further contributing to the patient's decreased cardiac tissue perfusion.

Additional assessment data revealed fine crackles bilaterally in the lower lobe of the lungs, poor capillary refill with a time of greater than three seconds in all four extremities, weak

bilateral radial pulses, and pedal pulses that were nonpalpable and difficult to detect via Doppler. Multiple wounds were noted on the lower limbs, indicative of venous stasis ulcers. The low heart rate and blood pressure from a third-degree AV block results in decreased cardiac output, evidenced by the weak peripheral pulses and poor capillary refill of the distal phalanges. This subsequently results in poor venous blood return, leading to a condition known as venous insufficiency. In venous insufficiency, blood stasis, especially in the lower extremities, causes tissue ischemia and necrosis, presenting as ulcers. This blood pooling also causes fluid seepage into the lower limb's interstitial spaces, leading to edema. Poor circulation also causes blood to back up into the pulmonary arteries, thus increasing pulmonary arterial pressure. This increased pressure forces fluid in the blood to perfuse through pulmonary tissue and build up inside the alveolar spaces within the lungs. This fluid buildup causes fine crackles to be heard during the auscultation of lung fields.

Nursing Problems

In assessing AB's case of NSTEMI with third-degree AV block, multiple nursing diagnoses come to the forefront as priorities for care. The following is a priority order of five nursing problems, their interconnectedness, and how they relate to Dorothy Orem's self-care deficit theory.

Decreased Cardiac Output

According to Costa et al. (2023), breathing difficulties, even during mild activities, are key indicators of heart failure's seriousness. Heart rate and rhythm variations, either bradycardia or tachycardia, can indicate further cardiovascular strain. These symptoms are often compounded

by behavioral changes such as anxiety and physical agitation, which can appear as the patient's cardiac output decreases, affecting cerebral perfusion.

AB's alarmingly low heart rate and blood pressure, combined with his dyspnea at rest, signify a heart that cannot meet the body's metabolic demands. The inability to sustain adequate blood flow can quickly lead to severe ramifications in other bodily systems, making this an immediate concern. In this case, the apparent indicators for decreased cardiac output are the low heart rate, low blood pressure, poor capillary refill, and poor peripheral pulses. The signs and symptoms that indicate this problem may have been prolonged are the lower limb edema, venous stasis ulcers in the lower limbs, and fine crackles in the lower lung lobes on auscultation. Further monitoring of AB's mental and psychomotor state will assist with determining if his condition is worsening.

Ineffective Peripheral Tissue Perfusion

Ineffective peripheral tissue perfusion is closely related to decreased cardiac output. Poor cardiac output inevitably leads to inadequate blood flow and return in the peripheral tissues. In AB's case, there is insufficient blood pressure to adequately perfuse distal tissues. Evidence to support this nursing problem is easily evidenced by AB's prolonged capillary refill, lower limb edema, poor peripheral pulses, and venous stasis ulcers. Further Complicating AB's venous stasis ulcers is his history of diabetes mellitus.

Impaired Kidney Function

Ineffective tissue perfusion can cause acute kidney injury in the short term and, if not corrected, chronic kidney disease in the long term. When the kidneys do not perfuse properly, the kidneys will attempt to compensate by activating the renin-angiotensin-aldosterone system

(RAAS). Activation of the RAAS causes vasoconstriction and fluid retention to increase cardiac output. However, vasoconstriction can further reduce the amount of blood reaching the kidneys, further decreasing their perfusion. Ultimately, this can cause ischemia and necrosis of kidney tissue, resulting in damage and functional impairments. AB was beginning to show early signs of decreased kidney function, as evidenced by his blood urea nitrogen (BUN) and creatinine levels. Normal BUN levels are 6 – 20 mg/dL, while normal serum creatinine levels are 0.6 – 1.3 mg/dL (*Lippincott Advisor*, 2019). AB's BUN was 26 mg/dL, and his creatinine was 2.3 mg/dL. AB's impaired kidney function limits available treatment options for his lower limb and pulmonary edema.

Fatigue

Decreased cardiac output directly leads to concerns of fatigue. AB stated that he was tired and had shortness of breath even without exertion. This is expected because AB's heart cannot keep up with his body's basic metabolic demands. Additionally, his poor peripheral tissue perfusion means that his peripheral muscles will likely be deprived of essential nutrients and oxygenation. Further complicating his fatigue is his reduced hemoglobin and hematocrit labs.

Social Isolation

AB's multidimensional health problems create a situation where leaving his home is likely complicated. AB is fatigued even at rest, and his other pertinent medical history of gout and morbid obesity contribute to his home-bound lifestyle. Further exacerbating AB's situation is his lack of family. He has no surviving children, and his wife's various medical conditions prevent her from being able to care for and assist AB. AB's significant venous stasis ulcers in his legs also contribute to his admission of being embarrassed to be seen.

Rosemarie Rizzo Parse's Human Becoming Theory offers invaluable insight into managing AB's overall care. This theory pushes the healthcare team beyond a disease-centric approach to a more person-centered paradigm that focuses on AB as an individual with unique health experiences and goals rather than a person defined by his medical diagnosis. The Human Becoming Theory states that each person's experience is unique and subjective and that the healthcare team's approach should use the patient's perspective to guide quality of life care (Rizzo Parse, 1992). This applies to AB in that, from an outside perspective, his medical condition will tremendously impact his life moving forward. The nurse is responsible for communicating with the patient to determine what aspects of care are a priority to the patient and taking nursing steps to help the patient achieve those goals. For example, decreased cardiac output is not just a hemodynamic problem but affects his overall functioning, including his ability to engage in activities he enjoys. AB's health events will profoundly impact his lived experience and change his interactions with family and friends. It is essential to engage AB to determine his priorities so that his care can be personalized to meet his goals rather than follow rigid protocols.

Patient Outcomes

Transitioning toward identifying patient outcomes, it is imperative to underline the importance of ensuring outcomes correlate to the identified nursing problems. This will establish a framework that can be utilized to identify appropriate nursing interventions and create a mechanism for subsequent evaluations of effectiveness. This discussion will delve into the top two nursing problems for AB's case.

Regarding the first nursing problem of decreased cardiac output, the desired outcome is improving AB's cardiac output by the end of the shift. The patient outcome statement that best reflects this is as follows: The patient will display improved cardiac output by the end of the

shift, as evidenced by improved blood pressure, heart rate, and a decrease in fine crackles in the lower lobe of the lungs.

For the second nursing problem, decreased peripheral tissue perfusion, the expected outcome is for the patient to display improved peripheral tissue perfusion as evidenced by improved peripheral pulses, decreased lower limb edema, oxygen saturation of 94% or greater, and improved cardiovascular output.

Interventions with Rationale

Decreased Cardiac Output

The first intervention for decreased cardiac output will be administering midodrine 10mg orally as ordered. Midodrine acts as a vasopressor and raises the patient's blood pressure, thus increasing cardiac output. However, vasopressors should be used cautiously in AB's situation due to his additional diagnosis of acute kidney injury (AKI). There are potential concerns that vasopressors could further reduce blood flow to his kidneys, exacerbating the AKI. Evidence by Rodrigo Benichel and Meneguin (2018) indicates that using vasopressors poses a significant risk factor for developing and exacerbating AKI. This risk requires heightened vigilance of renal function by monitoring the patient's urinary output and renal labs. Next is to monitor the patient's vitals every four hours. Specific attention should be paid to respiration rate, blood pressure, heart rate, and O2 saturation. Regular monitoring provides real-time data, allowing the healthcare team to adjust medication and interventions to stabilize cardiac output. Along with monitoring vitals, lung sounds will be assessed every 4 hours. During previous assessments, the patient's lung sounds were noted to have fine crackles, indicating increased pulmonary pressure and fluid in the

lungs. Further assessing lung sounds is a quality indicator of current cardiac output. As cardiac output improves, fine crackles will be reduced upon auscultation.

Ineffective Peripheral Tissue Perfusion

Regarding the decreased peripheral tissue perfusion, 325 mg ferrous sulfate will be administered three times daily. Ferrous sulfate will increase the patient's iron levels. Iron is an essential component of hemoglobin. Increasing the patient's hemoglobin levels will enhance the oxygen-carrying capacity of the blood, which is essential for adequate tissue perfusion. Additionally, hemoglobin and hematocrit levels should be monitored. Monitoring these levels will indicate if current interventions, such as ferrous sulfate, have the desired effect. If these levels continue to drop, that can indicate the need for further intervention, such as an infusion of packed red blood cells to improve oxygen delivery to peripheral tissue. Application of compression stockings or sequential compression devices (SCD) will enhance venous return and reduce edema, resulting in increased peripheral tissue perfusion. Monitoring edema every four hours with vitals will indicate the effectiveness of SCDs or compression stockings and if additional measures are needed, such as fluid restriction or administration of a diuretic.

Evaluation

Decreased Cardiac Output

By the end of the shift, the patient's blood pressure had significantly improved. Blood pressure readings had risen to 125/60, showing that the administration of midodrine had the desired effect. Heart rate remained stable and within normal limits, ranging between 65 and 75 beats per minute, which was expected given that AB had been previously paced. However, fine

crackles continued to be heard in the lower lobes of the lungs throughout the shift, indicating that further intervention was needed to address the pulmonary fluid buildup.

Ineffective Peripheral Tissue Perfusion

Interventions for the ineffective peripheral tissue perfusion were less successful. The patient's O2 saturation remained acceptable, varying from 98% to 100%. Radial pulses continued to remain weak throughout the shift. Bilateral radial pulses were consistently at a +1, while pedal pulses were nonpalpable and required a Doppler to verify adequate perfusion. Additionally, the lower limbs exhibited +2 edema throughout the shift with no noted improvements despite using SCDs. The patient could not tolerate compression stockings due to the venous stasis ulcers on his legs. The attending physician was notified of the fine lung crackles and the unchanged edema. It was recommended to the physician that furosemide used, and an order was for 20 mg of furosemide twice daily by mouth was ordered. The furosemide was administered too late into the shift to evaluate its effectiveness.

Conclusions

The importance of a multidimensional nursing approach became apparent in caring for AB. Not only did this case require a keen understanding of pathophysiology, but it also illustrates the interrelationship between various systems of the body and how problems in one system can quickly lead to a cascading effect that impacts other systems, further worsening the patient's outcome. The experiences in caring for this patient shed light on the need for a broad set of assessment skills and emphasized the need for a holistic approach to patient care.

In hindsight, there were several areas where the approach to AB's care might have been adjusted to provide better outcomes. For instance, greater attention could have been paid to

earlier interventions for the unchanging lung crackles. The assessment data, combined with the unchanging edema, warranted earlier treatment with a diuretic or collaboration with physical therapy to work towards quicker improvement. Recognizing the psychosocial aspects of AB's care, more immediate steps could have been taken to address concerns about social isolation. Referrals for structured social support or counseling services might have not only improved his psychological well-being but could have positively impacted his physical recovery. A Health-related quality of life (HRQL) in heart failure patients questionnaire could have been done. Research by Johansson et al. (2021) indicates that an HRQL questionnaire accurately indicates mortality and rehospitalization regardless of other factors, including EF percentage. The HRQL survey used in this research study was the Kansas City Cardiomyopathy questionnaire for patients, comprising 12 items. Having a patient such as AB complete this quick questionnaire can be a reliable indicator of the patient's future needs and can guide care by providing insights into what specific quality-of-life areas need to be addressed.

This case accentuated the role of ongoing education and flexibility in nursing practice. Despite applying evidenced-based interventions, AB's case presented unexpected outcomes such as unchanged edema and fine crackles in the lungs, which necessitated revaluation and a change to the plan of care. This was a real-world lesson in the significance of evaluation and the willingness to adapt based on new assessment data and patient responses. Caring for AB was an extensive, practical lesson in the complexity and adaptability required in nursing practice. It validated the importance of a well-rounded skill set, ongoing education, understanding pathophysiology, and the indispensable role of empathetic, holistic care in the modern healthcare environment.

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Appendix A

ODU Honor Code

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"I pledge to support the Honor System of Old Dominion University. I will refrain from any form of academic dishonesty or deception, such as cheating or plagiarism. I am aware that as a member of the academic community, it is my responsibility to turn in all suspected violators of the Honor Code. I will report to a hearing if summoned."

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