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ROY ADAPTATION MODEL-GUIDED NURSING MANAGEMENT OF STEMI COMPLICATED BY CARDIOGENIC SHOCK: A CASE REPORT

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Abstract

Background: ST-Elevation Myocardial Infarction (STEMI) complicated by cardiogenic shock is a life-threatening cardiovascular emergency associated with high mortality despite advances in reperfusion therapy and intensive cardiovascular care. Comprehensive nursing management is essential not only for physiological stabilization but also for promoting adaptive responses during critical illness. **Case Summary:** A 62-year-old male patient presented with severe chest pain radiating to the left arm, progressive dyspnea, diaphoresis, hypotension, and signs of systemic hypoperfusion. The patient had a history of uncontrolled hypertension, type 2 diabetes mellitus, and long-term smoking. Physical examination revealed hemodynamic instability, respiratory distress, poor peripheral perfusion, and pulmonary congestion. **Investigation and Diagnosis:** Electrocardiography demonstrated ST-segment elevation in the anterior leads consistent with acute anterior STEMI. Laboratory findings showed elevated cardiac troponin and serum lactate levels, while chest radiography revealed cardiomegaly with pulmonary congestion. Echocardiography demonstrated reduced left ventricular systolic function. Based on clinical findings and diagnostic evaluation, the patient was diagnosed with STEMI complicated by cardiogenic shock and acute pulmonary edema. **Management:** Emergency cardiovascular management included oxygen therapy, continuous hemodynamic monitoring, dual antiplatelet therapy, anticoagulation, vasopressor and inotropic support, diuretic therapy, and primary percutaneous coronary intervention (PCI). Nursing management was implemented using Roy Adaptation Model focusing on physiological stabilization, psychological adaptation, therapeutic communication, and family-centered support. **Outcome:** Following integrated cardiovascular management and Roy Adaptation Model-based nursing interventions, the patient's blood pressure improved from 82/58 mmHg to 104/68 mmHg, heart rate decreased from 124 to 92 beats/min, oxygen saturation increased from 88% to 97%, peripheral perfusion and urine output progressively improved within 48 hours, and pulmonary congestion markedly resolved. No recurrent malignant arrhythmias, major bleeding, or other significant in-hospital complications were observed **Conclusion:** This case highlights the importance of rapid recognition, multidisciplinary cardiovascular management, and adaptation-focused nursing care in patients with STEMI complicated by cardiogenic shock. Roy Adaptation Model provided a comprehensive framework for addressing both physiological instability and psychosocial adaptation during critical cardiovascular emergencies.

Keywords: cardiogenic shock; cardiovascular nursing; clinical case report; Roy Adaptation Model; STEMI

INTRODUCTION

ST-Elevation Myocardial Infarction (STEMI) remains one of the leading causes of cardiovascular mortality and morbidity worldwide despite advances in reperfusion therapy and critical cardiovascular management. STEMI occurs due to acute coronary artery occlusion resulting in transmural myocardial ischemia and extensive myocardial necrosis, which may rapidly progress to life-threatening complications, including cardiogenic shock, acute pulmonary edema, malignant arrhythmias, and multi-organ hypoperfusion (Casulli, 2021). Recent epidemiological reports have shown that cardiovascular disease continues to be the primary cause of global mortality, accounting for approximately 17.9 million deaths annually, with acute coronary syndrome contributing significantly to hospital admissions and critical care utilization (WHO, 2022). Cardiogenic shock remains one of the most severe complications of STEMI and is associated with high mortality rates despite improvements in early reperfusion and intensive cardiovascular treatment strategies (Journal et al., 2026).

Cardiogenic shock in STEMI develops as a consequence of extensive myocardial damage leading to impaired ventricular contractility, decreased cardiac output, systemic hypotension, and inadequate tissue perfusion. This condition frequently manifests with hemodynamic instability, respiratory distress, altered peripheral perfusion, oliguria, and metabolic deterioration requiring immediate multidisciplinary management (Baidhowy, 2025). In critically ill cardiovascular patients, rapid clinical deterioration may occur within a short period, emphasizing the importance of early recognition, continuous hemodynamic monitoring, oxygenation management, and evidence-based cardiovascular nursing interventions to prevent further complications and mortality (Alexander et al., 2024). Advanced cardiovascular nursing plays an essential role in supporting physiological stabilization, monitoring adaptive responses, identifying signs of worsening perfusion, and improving patient outcomes during acute cardiac emergencies (Puluhulawa, 2021).

From a nursing perspective, critically ill patients with STEMI complicated by cardiogenic shock experience complex adaptive responses involving physiological instability, psychological distress, impaired functional roles, and increased dependence on healthcare providers and family support systems (Thiele et al., 2019). Therefore, nursing management should not only focus on physiological stabilization but also address psychosocial adaptation and holistic patient responses during critical illness (Ummah, 2019). Roy Adaptation Model provides a comprehensive theoretical framework for understanding patient adaptation through four adaptive modes, namely physiological mode, self-concept mode, role function mode, and interdependence mode. This model enables nurses to assess adaptive behaviors systematically and implement individualized nursing interventions aimed at promoting positive adaptation in critically ill patients (Byrne et al., 2023).

Although several nursing theories, including Orem's Self-Care Deficit Theory, Neuman Systems Model, and Levine's Conservation Model, have been applied in cardiovascular nursing, the Roy Adaptation Model offers distinct advantages for managing critically ill patients with STEMI complicated by cardiogenic shock. Unlike theories that primarily emphasize self-care capacity, system stability, or energy conservation, the Roy Adaptation Model comprehensively addresses patients' dynamic adaptive responses across four interconnected modes: physiological function, self-concept, role function, and interdependence. This holistic perspective is particularly relevant for patients with cardiogenic shock, whose clinical condition is characterized not only by profound hemodynamic instability but also by significant psychological distress, temporary functional dependency, and the need for intensive family support. Consequently, the Roy Adaptation Model provides a more comprehensive framework for guiding nursing assessment, individualized interventions, and continuous evaluation of adaptive responses throughout the acute and recovery phases of critical cardiovascular illness.

Although previous studies have extensively discussed the medical management of STEMI and cardiogenic shock, few clinical case reports have specifically demonstrated how a nursing theory can systematically guide assessment, clinical decision-making, intervention planning, and evaluation during critical cardiovascular care. Existing reports predominantly emphasize pharmacological treatment and invasive procedures, while the

application of theory-based nursing management remains underreported. This knowledge gap underscores the need to describe how the Roy Adaptation Model can support holistic nursing care in patients with STEMI complicated by cardiogenic shock (Levine et al., 2022).

To the authors' knowledge, limited clinical case reports have specifically described the integration of Roy Adaptation Model within advanced cardiovascular nursing management for STEMI complicated by cardiogenic shock. This case provides important educational and clinical insight into adaptation-focused cardiovascular nursing care during critical hemodynamic instability.

CASE PRESENTATION

A 62-year-old male patient was admitted to the emergency department with a chief complaint of severe chest pain radiating to the left arm accompanied by progressive shortness of breath approximately six hours before hospital admission. The patient described the chest pain as crushing in nature with a pain intensity of 8 out of 10 and associated with diaphoresis, generalized weakness, palpitations, nausea, and cold extremities. The patient reported that the symptoms initially appeared during physical activity and progressively worsened despite resting at home.

The patient had a previous history of uncontrolled hypertension for approximately 10 years and type 2 diabetes mellitus for five years. The patient also reported a history of smoking one pack of cigarettes daily for more than 25 years. Medication adherence was poor, and the patient admitted irregular use of antihypertensive and antidiabetic medications. There was no previous history of coronary revascularization or documented myocardial infarction. Family history revealed that the patient's father had experienced cardiovascular disease at an older age. Social history showed limited physical activity and poor dietary control.

On admission, the patient appeared critically ill, anxious, diaphoretic, and dyspneic. Initial physical examination revealed a blood pressure of 82/58 mmHg, heart rate of 124 beats/minute, respiratory rate of 30 breaths/minute, body temperature of 36.4°C, and oxygen saturation of 88% on room air. Peripheral perfusion was poor as evidenced by cold extremities, delayed capillary refill time greater than three seconds, and weak peripheral pulses. Jugular venous distention was observed, and lung auscultation revealed bilateral fine crackles in the lower lung fields suggestive of pulmonary congestion. Cardiac auscultation demonstrated tachycardia with diminished heart sounds. Urine output during the initial assessment was decreased.

Initial electrocardiography demonstrated ST-segment elevation in the anterior leads consistent with acute anterior STEMI. Laboratory examination showed elevated cardiac troponin levels, increased serum lactate, hyperglycemia, and mild metabolic acidosis. Chest radiography revealed cardiomegaly with pulmonary vascular congestion suggestive of acute heart failure. Based on clinical presentation, hemodynamic instability, electrocardiographic findings, and laboratory results, the patient was diagnosed with STEMI complicated by cardiogenic shock requiring immediate intensive cardiovascular management and advanced nursing monitoring.

CLINICAL REASONING

The patient presented with acute severe chest pain radiating to the left arm, progressive dyspnea, diaphoresis, hypotension, tachycardia, and signs of systemic hypoperfusion. These manifestations strongly suggested an acute cardiovascular emergency requiring immediate diagnostic evaluation and hemodynamic stabilization. The initial differential diagnoses considered in this case included ST-Elevation Myocardial Infarction (STEMI), Non-ST Elevation Myocardial Infarction (NSTEMI), acute decompensated heart failure, pulmonary embolism, aortic dissection, and severe arrhythmia-associated hemodynamic instability (P. T. A. A. S. B. Z. E. Rizkasari, 2025).

STEMI was considered the primary working diagnosis because the patient demonstrated classic ischemic chest pain characteristics accompanied by electrocardiographic evidence of ST-segment elevation in the anterior leads, indicating acute transmural myocardial ischemia. In addition, elevated cardiac troponin levels further supported the presence of myocardial necrosis consistent with acute myocardial infarction (Aji, Baidhowy, & Rizkasari, 2026). The patient also exhibited hemodynamic instability characterized by systolic hypotension, tachycardia, cold extremities, delayed capillary refill, oliguria, and elevated serum lactate levels, which were strongly indicative of cardiogenic shock secondary to impaired myocardial contractility and reduced cardiac output (Aji, Tania, Ching, et al., 2026).

NSTEMI was excluded because the electrocardiographic findings showed persistent ST-segment elevation rather than ST-segment depression or T-wave inversion typically associated with NSTEMI. Pulmonary embolism was considered due to the presence of dyspnea and hypoxemia; however, the absence of sudden pleuritic chest pain, unilateral lower extremity swelling, and the presence of clear ischemic electrocardiographic findings made pulmonary embolism less likely (Stiexs et al., 2026). Acute aortic dissection was also considered because of severe chest pain and hemodynamic instability; however, the pain was not described as tearing in nature, there was no pulse deficit or mediastinal widening on chest radiography, and the electrocardiographic findings were more consistent with acute myocardial infarction (Aji & Rizkasari, 2021).

Acute decompensated heart failure was identified as a complication rather than the primary diagnosis because pulmonary congestion, bilateral crackles, and oxygenation impairment developed secondary to severe left ventricular dysfunction caused by acute myocardial ischemia. The progression from myocardial injury to impaired ventricular contractility, reduced cardiac output, systemic hypoperfusion, and pulmonary congestion reflected the pathophysiological mechanism of cardiogenic shock complicating STEMI (Aji & Sani, 2021).

From the perspective of Roy Adaptation Model, the patient demonstrated maladaptive physiological responses characterized by impaired oxygenation, hemodynamic instability, reduced tissue perfusion, and metabolic deterioration. Psychological maladaptation was also observed through anxiety, fear, and emotional distress during the acute cardiovascular event. These findings emphasized the importance of comprehensive adaptation-focused nursing management aimed at stabilizing physiological function, supporting psychological adaptation, and improving overall patient responses during critical illness (Hosseini & Soltanian, 2022).

Based on the overall clinical presentation, electrocardiographic findings, laboratory examinations, hemodynamic parameters, and pathophysiological deterioration, the final diagnosis established was acute anterior STEMI complicated by cardiogenic shock and acute pulmonary congestion requiring intensive cardiovascular management and advanced nursing care.

DIAGNOSTIC ASSESSMENT

Comprehensive diagnostic evaluation was performed immediately after admission to identify the underlying cause of hemodynamic instability and determine the severity of myocardial injury. Initial electrocardiography (ECG) demonstrated significant ST-segment elevation in the anterior leads, indicating acute anterior ST-Elevation Myocardial Infarction (STEMI). Laboratory examination revealed markedly elevated cardiac troponin levels confirming myocardial necrosis. Elevated serum lactate levels, metabolic acidosis, and impaired peripheral perfusion further supported the diagnosis of cardiogenic shock secondary to extensive myocardial ischemia (Wang et al., 2023).

Chest radiography demonstrated cardiomegaly accompanied by pulmonary vascular congestion and bilateral diffuse infiltrates suggestive of acute pulmonary edema due to left ventricular dysfunction. Echocardiographic examination showed reduced left ventricular ejection fraction with impaired anterior wall motion abnormalities, indicating severe systolic dysfunction contributing to reduced cardiac output and systemic

hypoperfusion (Wang et al., 2024). Hemodynamic assessment revealed persistent hypotension, tachycardia, hypoxemia, oliguria, and signs of peripheral vasoconstriction consistent with cardiogenic shock.

The Sequential Organ Failure Assessment (SOFA) score and Killip classification were also considered during the initial assessment to evaluate the severity of cardiovascular compromise and risk of mortality. The patient was classified as Killip class IV due to the presence of cardiogenic shock and pulmonary congestion, indicating a high-risk clinical condition requiring intensive cardiovascular management and close hemodynamic monitoring (Yeşilyurt, 2023).

Table 1. Summary of relevant diagnostic findings

Examination	Result	Clinical Interpretation
Electrocardiography (ECG)	ST-segment elevation in anterior leads (V1–V4)	Acute anterior STEMI indicating transmural myocardial ischemia
Cardiac troponin	Significantly elevated	Myocardial necrosis and acute myocardial infarction
Blood pressure	82/58 mmHg	Hemodynamic instability consistent with cardiogenic shock
Heart rate	124 beats/minute	Compensatory tachycardia due to reduced cardiac output
Respiratory rate	30 breaths/minute	Respiratory distress and increased oxygen demand
Oxygen saturation	88% on room air	Hypoxemia secondary to pulmonary congestion
Serum lactate	Elevated	Tissue hypoperfusion and anaerobic metabolism
Arterial blood gas analysis	Mild metabolic acidosis	Impaired tissue perfusion and oxygenation
Chest radiography	Cardiomegaly with pulmonary vascular congestion and bilateral infiltrates	Acute pulmonary edema associated with left ventricular failure
Echocardiography	Reduced left ventricular ejection fraction with anterior wall hypokinesia	Severe systolic dysfunction secondary to STEMI
Peripheral perfusion assessment	Cold extremities, delayed capillary refill, weak peripheral pulses	Systemic hypoperfusion due to cardiogenic shock
Urine output monitoring	Decreased urine output	Renal hypoperfusion associated with low cardiac output
Killip classification	Killip class IV	STEMI complicated by cardiogenic shock with high mortality risk
SOFA score	Increased SOFA score	Early organ dysfunction related to systemic hypoperfusion

The combination of electrocardiographic findings, elevated cardiac biomarkers, radiological evidence of pulmonary congestion, impaired ventricular contractility, and systemic hypoperfusion strongly supported the diagnosis of acute anterior STEMI complicated by cardiogenic shock and acute pulmonary edema requiring immediate intensive cardiovascular intervention and advanced nursing management.

THERAPEUTIC INTERVENTION

The patient received comprehensive emergency cardiovascular management immediately after the diagnosis of acute anterior ST-Elevation Myocardial Infarction (STEMI) complicated by cardiogenic shock was

established. Initial stabilization focused on improving myocardial perfusion, restoring hemodynamic stability, optimizing oxygenation, and preventing further organ hypoperfusion. All therapeutic interventions were performed according to evidence-based cardiovascular emergency management and patient safety principles (Mughal, 2022).

Upon admission, supplemental oxygen therapy was initiated using a non-rebreathing mask at 10 L/minute due to hypoxemia and respiratory distress. Continuous cardiac monitoring, pulse oximetry, urine output monitoring, and serial hemodynamic assessments were performed to detect early deterioration and evaluate treatment response. Two peripheral intravenous lines were established to facilitate rapid administration of fluids and emergency medications.

Pharmacological management included dual antiplatelet therapy consisting of aspirin 300 mg loading dose and clopidogrel 300 mg loading dose to reduce platelet aggregation and prevent further coronary thrombus formation. Intravenous unfractionated heparin was administered as anticoagulant therapy to reduce the risk of ongoing thrombosis and recurrent ischemic events. Intravenous norepinephrine infusion was initiated at a titrated dose to maintain adequate mean arterial pressure and improve systemic perfusion due to persistent hypotension associated with cardiogenic shock. Dobutamine infusion was administered to improve myocardial contractility and increase cardiac output while carefully monitoring the risk of tachyarrhythmia and increased myocardial oxygen demand (Hamzaoui & Boissier, 2023).

Intravenous loop diuretic therapy using furosemide was administered to reduce pulmonary congestion and improve respiratory status. Strict fluid balance monitoring was performed to prevent volume overload while maintaining adequate organ perfusion. Morphine was cautiously administered in low doses to reduce severe chest pain, decrease anxiety, and minimize sympathetic stimulation. Blood glucose levels were closely monitored and controlled using insulin therapy to optimize metabolic stability during the acute cardiovascular event.

Emergency reperfusion therapy through primary percutaneous coronary intervention (PCI) was planned as the definitive management to restore coronary blood flow and minimize myocardial damage. Prior to the procedure, the patient underwent intensive hemodynamic stabilization and continuous reassessment of perfusion status. Post-procedural monitoring included continuous electrocardiographic observation, assessment of recurrent chest pain, peripheral perfusion evaluation, monitoring for arrhythmias, bleeding complications, and reassessment of oxygenation and hemodynamic parameters (Aji, Rizkasari, & Rahmawati, 2026).

From the perspective of Roy Adaptation Model, nursing interventions focused on promoting adaptive responses in four adaptive modes. In the physiological mode, nursing management emphasized oxygenation support, tissue perfusion monitoring, hemodynamic stabilization, pain management, and fluid balance regulation. In the self-concept mode, therapeutic communication and emotional support were provided to reduce anxiety and fear related to the life-threatening condition. In the role function mode, nurses assisted the patient in adapting to temporary physical dependence during critical illness. In the interdependence mode, family involvement and psychosocial support were facilitated to strengthen the patient's coping mechanisms and emotional adaptation (Chen, 2025).

Continuous reassessment was conducted throughout hospitalization to evaluate treatment effectiveness, monitor complications, and ensure patient safety. The integrated combination of pharmacological therapy, reperfusion management, intensive cardiovascular monitoring, and Roy Adaptation Model-based nursing care contributed significantly to gradual hemodynamic stabilization, improved oxygenation, reduction of pulmonary congestion, and enhanced patient adaptation during critical cardiovascular care.

Table 3. Application of the Roy Adaptation Model Throughout the Nursing Care Process

Adaptive Mode	Focal Stimulus	Adaptive Problem	Nursing Intervention	Adaptive Outcome
Physiological	Acute STEMI with cardiogenic shock	Impaired oxygenation, decreased tissue perfusion, hemodynamic instability	Oxygen therapy, hemodynamic monitoring, vasopressor and inotropic support, preparation for PCI	Improved BP, oxygenation, tissue perfusion
Self-Concept	Fear of death and hospitalization	Anxiety and emotional distress	Therapeutic communication, reassurance, psychological support	Reduced anxiety and improved emotional adaptation
Role Function	Physical dependence during critical illness	Inability to perform usual roles	Gradual mobilization, self-care support, discharge preparation	Progressive recovery of functional role
Interdependence	Family concern and dependence	Need for psychosocial support	Family education, shared decision-making, discharge counseling	Enhanced family involvement and treatment adherence

Note: BP = blood pressure; ECG = electrocardiography; PCI = percutaneous coronary intervention; SpO_2 = peripheral oxygen saturation. The table summarizes the implementation of the Roy Adaptation Model across the four adaptive modes throughout the patient's nursing care.

THERAPEUTIC SEQUENCE

The patient arrived at the emergency department approximately six hours after the onset of severe chest pain accompanied by progressive dyspnea, diaphoresis, and generalized weakness. Initial assessment revealed hemodynamic instability characterized by hypotension, tachycardia, hypoxemia, cold extremities, delayed capillary refill, and decreased urine output. Electrocardiography demonstrated ST-segment elevation in the anterior leads consistent with acute anterior ST-Elevation Myocardial Infarction (STEMI). Immediate emergency cardiovascular stabilization was initiated to prevent further myocardial damage and systemic hypoperfusion.

During the initial stabilization phase, oxygen therapy using a non-rebreathing mask at 10 L/minute was administered to improve oxygenation and reduce myocardial oxygen demand. Continuous cardiac monitoring,

pulse oximetry, blood pressure monitoring, urine output monitoring, and serial hemodynamic evaluations were initiated due to the high risk of cardiovascular deterioration. Dual antiplatelet therapy consisting of aspirin and clopidogrel loading doses was administered promptly to inhibit platelet aggregation and reduce ongoing coronary thrombosis. Intravenous anticoagulant therapy with unfractionated heparin was also initiated to minimize further thrombotic complications (Navarese et al., 2021).

Despite initial stabilization efforts, the patient continued to experience persistent hypotension and signs of tissue hypoperfusion, including elevated serum lactate and worsening peripheral perfusion. Therefore, vasopressor therapy using norepinephrine infusion was initiated and titrated carefully to maintain adequate mean arterial pressure and preserve organ perfusion. Dobutamine infusion was subsequently added because persistent low cardiac output and impaired ventricular contractility suggested severe cardiogenic shock. The addition of inotropic support aimed to improve myocardial contractility and optimize systemic circulation while continuously monitoring for tachyarrhythmia and increased myocardial oxygen consumption (Juzar et al., 2022).

As pulmonary congestion and respiratory distress worsened, intravenous furosemide was administered to reduce fluid overload and improve pulmonary vascular congestion. Strict fluid balance monitoring was performed to avoid excessive fluid administration that could exacerbate pulmonary edema while still maintaining adequate tissue perfusion. Arterial blood gas analysis and oxygenation parameters were reassessed regularly to evaluate respiratory improvement and acid-base balance. Morphine was cautiously administered in low doses to reduce severe chest pain, sympathetic stimulation, and anxiety associated with acute myocardial infarction.

Following partial hemodynamic stabilization, the patient was prepared for reperfusion therapy through primary percutaneous coronary intervention (PCI) as definitive management to restore coronary blood flow and minimize ongoing myocardial ischemia. Prior to PCI, serial reassessment demonstrated gradual improvement in oxygen saturation and slight stabilization of blood pressure following vasopressor and inotropic support. Post-PCI monitoring included continuous electrocardiographic surveillance, assessment of recurrent ischemic symptoms, bleeding monitoring, peripheral perfusion reassessment, and evaluation of urine output and mental status to detect early complications and treatment response.

From the perspective of Roy Adaptation Model, therapeutic adjustments were continuously guided by the patient's adaptive responses. Physiological adaptation was addressed through oxygenation support, hemodynamic stabilization, perfusion monitoring, and fluid regulation. Psychological adaptation was facilitated through therapeutic communication and anxiety reduction interventions during critical illness. Interdependence adaptation was supported by involving family members in emotional support and patient care communication. Continuous reassessment of adaptive responses allowed nursing interventions to be modified according to the patient's evolving clinical condition and hemodynamic status (Lawton et al., 2022).

Over the subsequent hospitalization period, the patient demonstrated gradual clinical improvement characterized by improved oxygenation, stabilization of blood pressure, reduced pulmonary congestion, improved peripheral perfusion, and increased urine output. Continuous multidisciplinary cardiovascular management and intensive nursing monitoring contributed significantly to the patient's recovery and prevention of further hemodynamic deterioration.

CASE TIMELINE

Table 2. Clinical Case Timeline

Time	Clinical Event	Investigation	Treatment	Outcome
Day 0 (Emergency	Patient presented with severe chest	Initial vital signs assessment showed BP	Oxygen therapy via non-rebreathing mask 10	Initial stabilization

Department Admission)	pain radiating to the left arm, progressive dyspnea, diaphoresis, hypotension, and generalized weakness	82/58 mmHg, HR 124 beats/minute, RR 30 breaths/minute, SpO_2 88%. ECG demonstrated ST-segment elevation in anterior leads. Laboratory examination revealed elevated troponin and serum lactate levels	L/minute, continuous cardiac monitoring, aspirin loading dose, clopidogrel loading dose, intravenous heparin initiation	achieved with partial improvement in oxygenation but persistent hypotension and poor peripheral perfusion
Day 0 (First 6 Hours)	Persistent hemodynamic instability with signs of systemic hypoperfusion and worsening respiratory distress	Serial hemodynamic monitoring, arterial blood gas analysis, urine output assessment, chest radiography showing pulmonary congestion and cardiomegaly	Norepinephrine infusion initiated and titrated, dobutamine infusion added for inotropic support, intravenous furosemide administered, strict fluid balance monitoring	Slight improvement in blood pressure and oxygen saturation, pulmonary congestion remained present
Day 1	Continued monitoring in intensive cardiovascular care setting due to cardiogenic shock	Repeat ECG, serial troponin evaluation, peripheral perfusion reassessment, echocardiography demonstrating reduced left ventricular ejection fraction and anterior wall hypokinesia	Preparation for primary percutaneous coronary intervention (PCI), continuation of vasopressor and inotropic therapy, ongoing oxygenation support, pain management, anxiety reduction interventions	Hemodynamic status gradually stabilized, chest pain intensity decreased, peripheral perfusion slightly improved
Day 1 (Post-PCI)	Patient underwent successful reperfusion therapy through primary PCI	Post-procedural ECG monitoring, bleeding assessment, continuous hemodynamic observation	Continued antiplatelet therapy, anticoagulation monitoring, adjustment of vasopressor dosage according to blood pressure response	Improved coronary perfusion, stabilization of cardiac rhythm, reduced ischemic symptoms
Day 2	Progressive clinical stabilization with improvement in respiratory status and tissue perfusion	Repeat chest radiography, arterial blood gas reassessment, urine output monitoring, serial vital sign evaluation	Gradual reduction of oxygen support, tapering of norepinephrine dosage, continuation of cardiovascular medications and nursing monitoring	Improved oxygen saturation, reduced pulmonary congestion, increased urine output, improved

				peripheral perfusion
Day 3-4	Ongoing recovery phase with improvement in adaptive responses	Continuous cardiovascular monitoring and reassessment of hemodynamic parameters	Early mobilization support, patient education, psychological support based on Roy Adaptation Model, family involvement in care planning	Reduced anxiety, improved adaptation to illness, stable hemodynamic condition
Follow-Up (Post-Discharge)	Outpatient cardiovascular follow-up and rehabilitation planning	Clinical reassessment, blood pressure monitoring, symptom evaluation, medication adherence evaluation	Continuation of antiplatelet therapy, antihypertensive medication, diabetic management, smoking cessation education, lifestyle modification counseling	

OUTCOME AND FOLLOW-UP

The patient demonstrated gradual clinical improvement following intensive cardiovascular management and Roy Adaptation Model-based nursing interventions. During the first 24 hours of hospitalization, hemodynamic instability remained the primary clinical concern due to persistent hypotension, impaired peripheral perfusion, and respiratory distress associated with cardiogenic shock. However, continuous monitoring and therapeutic adjustment contributed to progressive stabilization of the patient's cardiovascular status.

Following initiation of vasopressor and inotropic support combined with oxygen therapy and reperfusion management, blood pressure gradually improved from 82/58 mmHg to 104/68 mmHg within the subsequent 48 hours. Heart rate decreased progressively from 124 beats/minute to 92 beats/minute, while oxygen saturation improved from 88% on room air to 97% with reduced oxygen support. Respiratory distress and pulmonary congestion also showed significant improvement as evidenced by reduced dyspnea, decreased bilateral crackles on lung auscultation, and improved arterial blood gas parameters. Peripheral perfusion improved gradually, demonstrated by warmer extremities, improved capillary refill time, stronger peripheral pulses, and increased urine output.

Serial electrocardiographic monitoring showed no recurrent malignant arrhythmias following primary percutaneous coronary intervention (PCI). Repeated laboratory evaluation demonstrated gradual reduction in serum lactate levels and stabilization of metabolic parameters, indicating improved systemic tissue perfusion. Follow-up echocardiographic evaluation revealed persistent but improving left ventricular systolic dysfunction without evidence of further mechanical complications. No major bleeding complications or recurrent ischemic events were identified during hospitalization.

From the perspective of Roy Adaptation Model, the patient demonstrated progressive adaptive responses in all four adaptive modes. Physiological adaptation improved through stabilization of oxygenation, perfusion, and hemodynamic status. Psychological adaptation was observed through decreased anxiety levels, improved emotional control, and enhanced patient cooperation during treatment. In the role function mode, the patient gradually regained independence in performing simple daily activities during the recovery phase. Interdependence adaptation was strengthened through active family involvement, emotional support, and effective communication between healthcare providers and the patient's family.

During short-term follow-up after discharge, the patient remained hemodynamically stable without recurrent chest pain or worsening heart failure symptoms. The patient demonstrated improved adherence to cardiovascular medications, dietary recommendations, smoking cessation counseling, and scheduled outpatient follow-up visits. Education regarding symptom recognition, lifestyle modification, medication adherence, and cardiovascular risk reduction was reinforced during discharge planning. The patient was advised to continue regular cardiology follow-up and participate in cardiovascular rehabilitation to optimize long-term recovery and prevent recurrent cardiovascular events.

Overall, the integrated combination of evidence-based cardiovascular treatment, intensive monitoring, reperfusion therapy, and Roy Adaptation Model-based nursing management contributed significantly to improved physiological stabilization, adaptive responses, and short-term clinical outcomes in this patient with STEMI complicated by cardiogenic shock.

DISCUSSION

This case report describes a critically ill patient with acute anterior ST-Elevation Myocardial Infarction (STEMI) complicated by cardiogenic shock who required intensive cardiovascular stabilization, reperfusion therapy, and comprehensive nursing management based on Roy Adaptation Model. Cardiogenic shock remains one of the most severe and life-threatening complications of STEMI and continues to be associated with high mortality rates despite advances in reperfusion strategies and intensive cardiovascular care (Elbadawi et al., 2019). The present case highlights the importance of rapid hemodynamic assessment, early recognition of tissue hypoperfusion, timely reperfusion therapy, and integrated nursing management in improving patient outcomes during acute cardiovascular emergencies.

The pathophysiological deterioration observed in this patient was consistent with the progression of extensive myocardial ischemia leading to impaired ventricular contractility, reduced cardiac output, systemic hypotension, pulmonary congestion, and multi-organ hypoperfusion. Previous studies have demonstrated that severe left ventricular dysfunction following STEMI contributes significantly to cardiogenic shock through decreased tissue perfusion and impaired oxygen delivery (Jacob C Jentzer, 2019). In this case, the patient demonstrated classic manifestations of cardiogenic shock including persistent hypotension, tachycardia, cold extremities, oliguria, elevated serum lactate levels, and pulmonary edema, all of which indicated severe circulatory failure and high mortality risk. Early hemodynamic stabilization using vasopressor and inotropic therapy was therefore essential to preserve organ perfusion and prevent further cardiovascular deterioration.

Continuous cardiovascular monitoring and intensive nursing observation played a major role in identifying changes in hemodynamic status and guiding therapeutic adjustments throughout hospitalization. Advanced cardiovascular nursing management in critically ill STEMI patients is essential because subtle changes in oxygenation, peripheral perfusion, urine output, and mental status may indicate worsening shock and impending organ dysfunction (Cenko et al., 2018). In this case, serial assessment of oxygenation, perfusion, fluid balance, respiratory status, and hemodynamic response enabled early detection of clinical progression and optimization of therapeutic interventions. The gradual improvement in blood pressure, oxygen saturation, pulmonary congestion, and peripheral perfusion following integrated cardiovascular management demonstrated the importance of multidisciplinary and evidence-based critical care approaches.

One of the major strengths and novelties of this case report is the implementation of Roy Adaptation Model as the theoretical foundation for cardiovascular nursing management in a patient with STEMI complicated by cardiogenic shock. Most previous case reports and cardiovascular studies primarily focus on medical stabilization and invasive interventions while providing limited discussion regarding theory-based nursing adaptation during critical illness (Prima Trisna Aji; Yunie Armiyati; Elinda Rizkasari, 2026). Roy Adaptation Model offers a holistic framework for understanding patient responses to physiological and psychosocial stressors through four adaptive modes: physiological mode, self-concept mode, role function mode, and

interdependence mode (E. Rizkasari et al., 2025). In this patient, maladaptive physiological responses were evident through impaired oxygenation, decreased tissue perfusion, pulmonary congestion, and hemodynamic instability. Simultaneously, psychological distress and anxiety emerged due to the life-threatening nature of the condition and dependence on intensive cardiovascular care.

The application of Roy Adaptation Model enabled nursing interventions to address not only physiological stabilization but also emotional adaptation, coping mechanisms, and family involvement during critical illness. Therapeutic communication, anxiety reduction, emotional reassurance, and family-centered support contributed to improved psychological adaptation and patient cooperation during treatment. Previous literature has emphasized that holistic nursing care and psychological support may positively influence recovery, coping ability, and adherence in critically ill cardiovascular patients (Hosseini & Soltanian, 2022). Therefore, integrating adaptation-focused nursing management with evidence-based cardiovascular treatment may improve overall patient outcomes beyond physiological recovery alone.

Another important clinical implication of this case is the significance of rapid reperfusion therapy combined with intensive cardiovascular monitoring in reducing myocardial damage and improving survival in STEMI complicated by cardiogenic shock. Current guidelines strongly recommend immediate reperfusion therapy through primary percutaneous coronary intervention (PCI) in patients with STEMI and cardiogenic shock because delayed reperfusion is associated with worsening myocardial necrosis and increased mortality (Bress et al., 2024). In this case, gradual hemodynamic stabilization prior to and following PCI contributed to improved oxygenation, stabilization of cardiac rhythm, reduction of pulmonary congestion, and improved tissue perfusion.

This case also demonstrates the importance of multidisciplinary collaboration involving cardiologists, critical care nurses, emergency teams, and family support systems during acute cardiovascular emergencies. Integrated collaboration facilitated timely decision-making, continuous monitoring, medication titration, complication prevention, and adaptive support during the critical phase of illness. The successful stabilization observed in this patient emphasizes that comprehensive cardiovascular nursing care is not limited to technical monitoring but also includes adaptive, psychosocial, and patient-centered management approaches.

Several limitations should be acknowledged in interpreting this case. First, this report represents a single case and therefore cannot be generalized to all patients with STEMI complicated by cardiogenic shock. Second, long-term follow-up data regarding cardiac functional recovery and long-term adaptation outcomes were limited. Third, invasive hemodynamic monitoring parameters were not comprehensively evaluated due to clinical and resource considerations. Nevertheless, the detailed description of hemodynamic deterioration, adaptation-focused nursing management, and integrated cardiovascular interventions provides important educational value and practical insight for critical cardiovascular nursing practice.

Overall, this case highlights the critical importance of early recognition of cardiogenic shock, rapid reperfusion therapy, intensive hemodynamic monitoring, and holistic adaptation-based nursing management in improving patient outcomes during acute STEMI emergencies. The integration of Roy Adaptation Model into advanced cardiovascular nursing management provided a comprehensive framework for addressing both physiological instability and psychosocial adaptation during critical illness, thereby contributing to improved patient stabilization and recovery.

This case emphasizes that advanced cardiovascular nursing extends beyond physiological monitoring and includes adaptive psychosocial support, early recognition of hemodynamic deterioration, therapeutic communication, and individualized patient-centered intervention. Integration of Roy Adaptation Model into cardiovascular critical care nursing may strengthen holistic patient management and improve adaptive responses during acute cardiac emergencies.

DIAGNOSTIC CHALLENGE

Several diagnostic and clinical management challenges were encountered during the early assessment and stabilization of this patient with acute ST-Elevation Myocardial Infarction (STEMI) complicated by cardiogenic shock. One of the primary challenges involved differentiating the cause of severe hemodynamic instability and respiratory distress during the initial emergency presentation. The patient arrived with hypotension, tachycardia, hypoxemia, diaphoresis, pulmonary congestion, and signs of systemic hypoperfusion, all of which could also be observed in other life-threatening cardiovascular and respiratory emergencies such as acute decompensated heart failure, pulmonary embolism, severe arrhythmia, or acute aortic syndrome (Trials et al., 2024).

Another important diagnostic challenge was the rapid progression of cardiogenic shock shortly after symptom onset. The patient exhibited severe circulatory compromise characterized by persistent hypotension, elevated serum lactate levels, oliguria, cold extremities, and impaired peripheral perfusion. In critically ill cardiovascular patients, early shock manifestations may overlap with compensatory sympathetic responses, making early recognition of true hemodynamic deterioration clinically challenging (Darzi, 2025). Therefore, continuous reassessment of perfusion status, serial hemodynamic monitoring, and repeated clinical evaluations were required to identify worsening circulatory failure and guide therapeutic escalation.

The presence of pulmonary congestion and respiratory distress also complicated the clinical decision-making process because oxygenation support and fluid management had to be balanced carefully. Excessive fluid administration could potentially worsen pulmonary edema, whereas inadequate circulatory support might aggravate systemic hypoperfusion and organ dysfunction. This condition required continuous adjustment of vasopressor therapy, inotropic support, oxygen therapy, and diuretic administration based on dynamic hemodynamic responses and ongoing cardiovascular monitoring (Abu-qbeitah & Ahmad, 2026).

Another challenge involved determining the optimal timing for reperfusion therapy through primary percutaneous coronary intervention (PCI). Although early reperfusion is strongly recommended in STEMI management, the patient's unstable hemodynamic condition increased the risk of procedural complications and peri-interventional deterioration. Intensive stabilization using vasopressor and inotropic therapy was therefore necessary prior to definitive reperfusion management. Continuous multidisciplinary coordination between emergency physicians, cardiologists, intensive care nurses, and cardiovascular care teams played a critical role in optimizing patient safety and clinical decision-making during the acute phase of illness.

One of the major diagnostic challenges was distinguishing acute STEMI complicated by cardiogenic shock from other life-threatening causes of chest pain and hemodynamic instability, including pulmonary embolism, acute aortic dissection, NSTEMI, and acute decompensated heart failure. At the time of presentation, overlapping clinical manifestations such as hypotension, dyspnea, hypoxemia, and pulmonary congestion created initial diagnostic uncertainty. Therefore, rapid integration of serial electrocardiographic findings, cardiac biomarker results, chest radiography, bedside echocardiography, and repeated hemodynamic assessments was essential to establish the final diagnosis and exclude alternative conditions (Aji, Rizkasari, & Baidhowy, 2026).

Clinical decision-making prioritized immediate hemodynamic stabilization while simultaneously preparing for definitive reperfusion therapy. Persistent ST-segment elevation in the anterior leads, elevated cardiac troponin levels, evidence of severe left ventricular systolic dysfunction, and progressive systemic hypoperfusion strongly supported the diagnosis of acute anterior STEMI complicated by cardiogenic shock. These findings justified urgent primary percutaneous coronary intervention (PCI) together with vasopressor and inotropic support. Although complete pre-hospital information and invasive hemodynamic monitoring data were limited during the emergency phase, serial clinical reassessment and multidisciplinary collaboration enabled timely therapeutic decisions and successful patient stabilization.

LIMITATION

Several limitations should be considered when interpreting this case report. First, this report describes a single patient with ST-Elevation Myocardial Infarction (STEMI) complicated by cardiogenic shock; therefore, the clinical findings and outcomes cannot be generalized to all critically ill cardiovascular patients. Individual patient characteristics, severity of myocardial injury, comorbid conditions, and variations in hemodynamic responses may influence clinical progression and treatment outcomes differently across cases.

Second, although comprehensive cardiovascular assessment was performed during hospitalization, several invasive hemodynamic monitoring parameters such as pulmonary artery catheterization data and advanced cardiac output measurements were not comprehensively available during the initial stabilization phase. The absence of detailed invasive hemodynamic evaluation may limit deeper interpretation regarding the severity of circulatory compromise and response to vasopressor or inotropic therapy.

Third, the exact duration of coronary artery occlusion prior to hospital admission could not be fully determined because the patient arrived several hours after symptom onset and pre-hospital clinical documentation was limited. Delayed presentation may have influenced the extent of myocardial injury, severity of cardiogenic shock, and subsequent clinical outcomes. Additionally, serial long-term echocardiographic evaluation and advanced imaging follow-up were limited during the post-discharge period, restricting comprehensive assessment of long-term ventricular recovery and cardiac remodeling (Trisna Aji et al., 2025).

Another limitation involves the short-term nature of the follow-up evaluation. Although the patient demonstrated significant clinical improvement during hospitalization and early outpatient follow-up, long-term adaptation outcomes, recurrence risk, medication adherence sustainability, and long-term quality of life could not be comprehensively assessed within this report. Future studies with prolonged follow-up and larger patient populations are needed to evaluate the long-term impact of Roy Adaptation Model-based cardiovascular nursing management in critically ill STEMI patients (Prima Trisna Aji, Marta Tania GCC, 2026).

Finally, although the Roy Adaptation Model provided a comprehensive theoretical framework for guiding nursing assessment and intervention, patient adaptation was evaluated primarily through clinical observation, routine nursing assessment, and multidisciplinary clinical judgment rather than using standardized adaptation measurement instruments specifically developed within the Roy Adaptation Model framework. Consequently, the validity and reliability of adaptation assessment may have been influenced by subjective clinical interpretation and inter-observer variability. Future case reports and clinical studies are encouraged to incorporate validated adaptation assessment instruments to provide more objective, reproducible, and theory-consistent evaluation of patient adaptation outcomes. Nevertheless, the present case demonstrates the practical feasibility of integrating the Roy Adaptation Model into cardiovascular nursing management and offers valuable clinical insights for adaptation-focused care in critically ill patients with STEMI complicated by cardiogenic shock.

LEARNING POINTS

1. Early recognition of cardiogenic shock in patients with ST-Elevation Myocardial Infarction (STEMI) is essential to prevent rapid hemodynamic deterioration, multi-organ hypoperfusion, and increased mortality risk.
2. Continuous hemodynamic monitoring, oxygenation assessment, and peripheral perfusion evaluation are critical nursing responsibilities for detecting clinical deterioration and guiding timely therapeutic interventions in critically ill STEMI patients.
3. Prompt reperfusion therapy combined with evidence-based pharmacological stabilization significantly improves myocardial perfusion and short-term clinical outcomes in patients with STEMI complicated by cardiogenic shock.

4. The Roy Adaptation Model provides a practical framework for critical cardiovascular nurses to systematically assess adaptive responses, prioritize individualized nursing interventions, integrate family-centered care, and evaluate both physiological and psychosocial recovery throughout hospitalization.
5. Successful management of STEMI complicated by cardiogenic shock requires close multidisciplinary collaboration, with cardiovascular nurses playing a central role in early assessment, continuous monitoring, therapeutic coordination, patient education, and long-term adaptation support.

CONCLUSION

This case report highlights the importance of rapid recognition and comprehensive multidisciplinary management of ST-Elevation Myocardial Infarction (STEMI) complicated by cardiogenic shock as a life-threatening cardiovascular emergency. Early hemodynamic stabilization, prompt reperfusion therapy, continuous cardiovascular monitoring, and evidence-based pharmacological management contributed to improved oxygenation, tissue perfusion, and short-term clinical recovery. Beyond conventional nursing care, which primarily focuses on physiological monitoring and treatment support, the Roy Adaptation Model provided additional value by enabling nurses to systematically assess adaptive responses, integrate psychosocial and family-centered interventions, prioritize individualized nursing care, and continuously evaluate both physiological and psychological recovery throughout hospitalization. This holistic, theory-guided approach strengthened comprehensive cardiovascular nursing management and supported the patient's overall adaptation during critical illness. Further studies involving larger patient populations and longer follow-up periods are warranted to evaluate the effectiveness of Roy Adaptation Model-based nursing interventions across diverse cardiovascular critical care settings.

ETHICAL CLEARANCE

Ethical clearance was not required for this clinical case report according to institutional policy because the manuscript describes a single anonymized clinical case without experimental intervention or identifiable patient information. All clinical data were fully anonymized to protect patient confidentiality and privacy in accordance with the ethical principles of the Declaration of Helsinki and publication ethics standards for case reports. Written informed consent for publication was obtained from the patient prior to manuscript preparation and submission.

PATIENT CONSENT

Written informed consent for publication of this clinical case report, including related clinical data and non-identifiable clinical images, was obtained from the patient prior to manuscript preparation and submission. All patient information was anonymized to ensure confidentiality and privacy protection throughout the reporting and publication process. No personal identifiers, medical record numbers, facial photographs, or identifiable information were included in this manuscript.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest related to the writing, analysis, and publication of this clinical case report.

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AUTHORS' CONTRIBUTION]

Prima Trisna Aji contributed to case conceptualization, clinical data collection, cardiovascular nursing assessment, clinical interpretation, manuscript drafting, and preparation of the final manuscript. Muhammad Nur Hasbi contributed to clinical supervision, critical review of cardiovascular nursing management, manuscript revision, and scientific interpretation. Elinda Rizkasari contributed to manuscript editing, academic

review, language refinement, and approval of the final version of the manuscript. All authors read and approved the final manuscript prior to submission.

DATA AVAILABILITY

The data supporting the findings of this clinical case report are available from the corresponding author upon reasonable request. However, certain clinical data cannot be publicly shared due to patient confidentiality, privacy protection, and ethical considerations.

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LIST OF ABBREVIATIONS

ACS = Acute Coronary Syndrome
AHA = American Heart Association
BP = Blood Pressure
CT = Computed Tomography
ECG = Electrocardiography
HR = Heart Rate
MRI = Magnetic Resonance Imaging
NSTEMI = Non-ST Elevation Myocardial Infarction
PCI = Percutaneous Coronary Intervention
RR = Respiratory Rate
SOFA = Sequential Organ Failure Assessment
SpO_2 = Peripheral Oxygen Saturation
STEMI = ST-Elevation Myocardial Infarction

FIGURE AND TABLE LEGENDS

Table 1. Summary of relevant diagnostic findings in a patient with STEMI complicated by cardiogenic shock.

Table 2. Clinical case timeline describing the chronological progression of clinical deterioration, diagnostic evaluation, therapeutic interventions, and patient outcomes during hospitalization and follow-up.

Table 3. Hemodynamic monitoring and clinical response during intensive cardiovascular management.

Variable	Result	Notes
Blood pressure	Improved from 82/58 mmHg to 104/68 mmHg	Hemodynamic stabilization after vasopressor and inotropic therapy
Oxygen saturation	Improved from 88% to 97%	Improved oxygenation and pulmonary perfusion
Peripheral perfusion	Improved capillary refill and extremity temperature	Better systemic circulation after stabilization

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