Case Report Detailing an Inferior Myocardial Infarction, Third-Degree Heart Block, and Cardiogenic Shock

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CE Earn Up to 12.0 Hours. See page 675.

Contribution to Emergency Nursing Practice

- What is already known: rapid identification of high-risk cardiovascular clinical conditions and subsequent targeted interventions can improve patient outcomes.
- The main finding: cardiac disease processes can evolve over time and complicate patient care.
- Recommendations for translating the findings of this paper into emergency clinical practice include: remaining up to date on American Heart Association recommendations improve the quality and safety of care for complex, critically ill patients.

Abstract

An infarction in the right coronary artery affects the inferior wall of the heart and can also cause impedance to the cardiac conduction system. The right coronary artery perfuses the sinoatrial and atrioventricular nodes, and a loss of blood flow contributes to a breakdown in the communication system within the heart, causing associated bradycardias, heart blocks, and arrhythmias. This case report details the prehospital and emergency care of a middle-aged man who experienced an inferior myocardial infarction, concomitant third-degree heart block, and subsequent cardiogenic shock, with successful revascularization. This case is informative for emergency clinicians to review symptoms of acute coronary syndrome, rapid lifesaving diagnostics and intervention, and the unique treatment and monitoring considerations associated with right ventricular involvement and third-degree heart block.

Key words: Emergency nursing; Myocardial infarction; Heart block; Percutaneous coronary intervention

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Introduction

This case report details the prehospital and emergency care of a middle-aged male patient who experienced an inferior myocardial infarction, concomitant third-degree heart block, and subsequent cardiogenic shock, with successful revascularization. Facility institutional review board policy was followed, and patient consent was obtained by electronic communication.

Case Report

A 56-year-old man with a past medical history of hypercholesteremia, no documented family history of cardiac disease, and no home medications had a syncopal event while at an outdoor recreation facility. After completing a leisure-time driving activity (go-kart racing), the patient sat down and immediately became pale and unconscious. Owing to bystander concern for cardiac arrest, cardiopulmonary resuscitation (CPR) without defibrillation was initiated, and emergency medical services (EMS) was called. On EMS arrival, the patient was pale and diaphoretic but alert and oriented; in addition, the patient was experiencing chest pain, nausea, and vomiting. The initial vital signs were as follows: heart rate 38 beats per minute (bpm), blood pressure 65/37 mm Hg, and respiratory rate 20 breaths per minute; the patient's blood glucose level was 30 mg/dL. A 12-lead electrocardiogram (ECG) was obtained. EMS identified a third-degree atrioventricular (AV) heart block as well as an inferior myocardial infarction (MI) with ST elevation in leads II, III, and aVF and reciprocal depression in leads I and aVL (Figure 1).

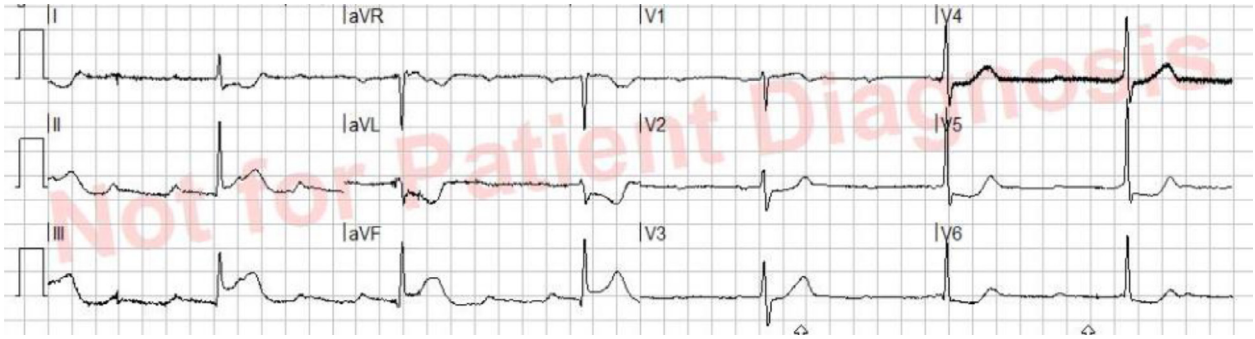

FIGURE 1
Prehospital 12-lead electrocardiogram.

EMS administered a 500-mL normal saline (0.9%) intravenous (IV) bolus and a dose of EPINEPHrine 5 mcg through IV push. After the first dose of EPINEPHrine, the patient's heart rate did not increase, and he became hypertensive to 212/160 mm Hg owing to the vasopressor. Despite an attempt to perform transcutaneous pacing, capture was not achieved, and ultimately pacing was stopped owing to patient intolerance. During transport, repeat vital signs were obtained with a blood pressure reading of 96/68 mm Hg and a heart rate of 38 bpm. Per protocol, EMS administered a second dose of EPINEPHrine 3 mcg IV push for persistent bradycardia and hypotension. Additional medications given by EMS were ondansetron 4 mg IV push, aspirin 324 mg by mouth, fentaNYL 50 mcg IV push, and a dextrose 10% IV infusion for hypoglycemia.

On arrival to the emergency department, the patient complained of left-sided chest pain that radiated to his left arm. His blood pressure was 111/64 mm Hg, and his heart rate was 42 bpm. A second 12-lead ECG was acquired, and the interpretation was unchanged from the initial ECG (Figure 2). Blood laboratory specimens were obtained in the emergency department, and the results outside of normal limits were as follows: potassium 5.9 mmol/L (3.5-5.1 mmol/L), low-density lipoprotein 139 mg/dL (<100 ng/dL), and troponin 684 ng/L (<20 ng/L; high-sensitivity troponin). The patient received heparin 4000 units IV push before transfer to the cardiac catheterization laboratory.

Emergent cardiac catheterization demonstrated mild nonobstructive disease in the left anterior descending system and 100% occlusion of the proximal right coronary artery (RCA). The interventional cardiologist placed a temporary pacing wire and stented the culprit lesion in the RCA with a single drug-eluting stent. Subsequently, the patient's heart block and signs of cardiogenic shock resolved after successful vessel reperfusion of the RCA with restoration of a normal sinus rhythm (Figures 3 and 4).

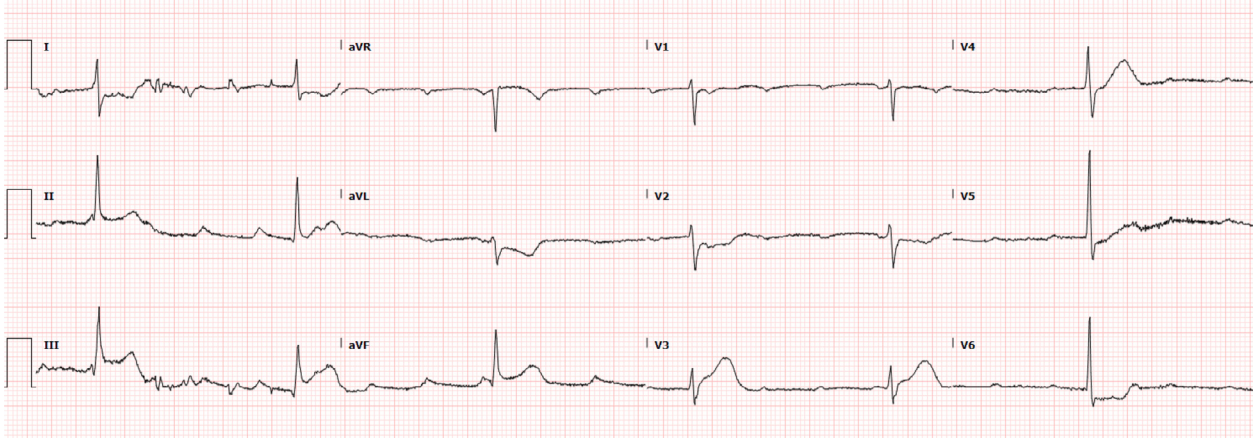

FIGURE 2
ED 12-lead electrocardiogram.

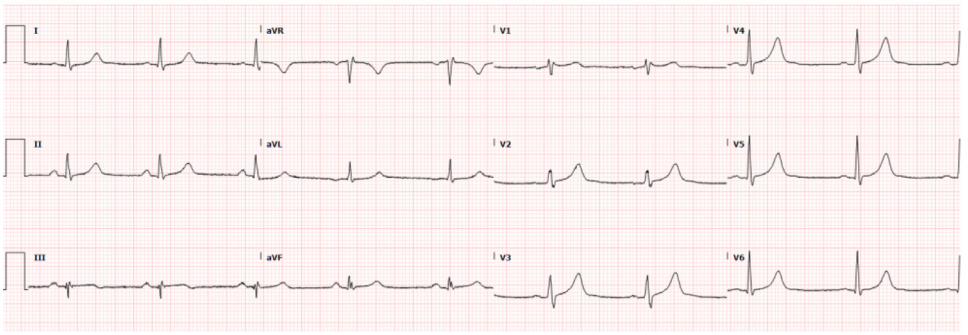

FIGURE 3

Postcardiac catheterization 12-lead electrocardiogram.

Diagnostic Assessment

A 12-lead ECG is one of the most important diagnostic tools for the workup of patients with a suspected MI; however, an ECG is insufficient on its own to diagnose acute myocardial ischemia or MI. Elevation of the ST segment in 2 contiguous leads is required to diagnose an ST-elevated MI (STEMI).[1] To diagnose an inferior MI, the trained clinician would look for an ST-segment elevation in leads II, III, and aVF; reciprocal changes may be seen in lead aVL.[2] See Figure 5 for diagnostic criteria suggestive of an inferior STEMI and third-degree AV block. When an inferior MI is identified, a right-sided 12-lead ECG should also be acquired to examine the right ventricle because studies have demonstrated that up to 40% of the inferior wall MIs have associated right ventricular (RV) involvement that predicts a worse outcome.[3]

Additional criteria to inform a diagnosis of an acute MI can be a rise in the cardiac troponin blood levels above the 99th percentile.[1] The high-sensitivity troponin blood test administered (Beckman Coulter hsTnl) has a 99th percentile cutoff of 19.8 ng/L for males.[4] The patient had a maximum troponin level of 14 492 ng/L 12 hours after symptom onset, which represents a significant rise above the 99th percentile.

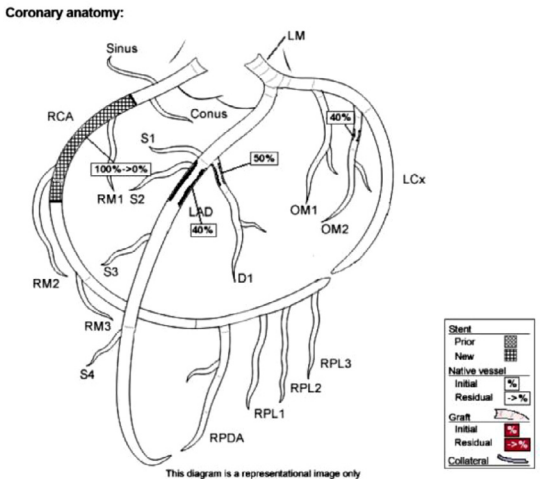

FIGURE 4

Cardiac catheterization laboratory report with depiction of occlusion location and stenting. RCA, right coronary artery; LM, left main; RM1, ramus marginalis 1; RM2, ramus marginalis 2; RM3, ramus marginalis 3; S1, septal perforator 1; S2, septal perforater 2; S3, septal perforater 3; S4, septal perforater 4; LAD, left anterior descending; OM1, left obtuse marginal 1; OM2, left obtuse marginal 2; D1, diagonal artery 1; RPDA, right posterior descending artery; RPL1, posterior left ventricular wall 1; RPL2, posterior left ventricular wall 2; RPL3, posterior left ventricular wall 3; LCx; left circumflex.

Pathophysiology

The blood supply to the heart is provided by the coronary circulatory system. The RCA originates at the anterior aortic sinus and supplies blood to the right atrium, right ventricle, sinoatrial (SA) node, AV node, and posterior aspects of the left ventricle.[5] A blockage in the RCA stops the flow of oxygenated blood to the inferior wall of the heart,[2] leading to myocardial ischemia and eventually MI. When MI occurs, it can also cause impedance to the cardiac conduction system. The RCA perfuses the SA and AV nodes, and a loss of blood flow contributes to a breakdown in the communication system within the heart, causing associated bradycardias, heart blocks, and arrhythmias.[3] With open communication systems and adequate perfusion, the SA node starts an impulse that travels to the AV node. This

ECG Criteria: Diagnosing an Acute Inferior Myocardial Infarction and Third-Degree Heart Block
Inferior ST-segment Elevation MI
- ST-segment elevation of greater than 1 mm (greater than 2 mm in leads V1/V2 in males) in two anatomically contiguous leads: II, III, and aVF. - Reciprocal ST depression is often seen in lead aVL.
Third-Degree Heart Block
- Independent atrial and ventricular activity with no relation between the P wave and the QRS complex. - The atrial rate, demonstrated by the P wave, should be faster than the ventricular rate, as demonstrated by the QRS complex. - *Consideration*: A repeat ECG or rhythm strip may be required to differentiate between second-degree and third degree heart blocks as a second degree heart block may masquerade as a third-degree heart block[7].

FIGURE 5

ECG, electrocardiogram; MI, myocardial infarction; STEMI, ST-elevated MI.

Event	Time	Duration
Call to EMS	6:51 PM	
FMC	7:01 PM	10 minutes Dispatch time
12-lead ECG	7:07 PM	6 minutes FMC to ECG
ED arrival	7:47 PM	40 minutes Transport time
12-lead ECG	7:52 PM	5 minutes ED arrival to ECG
CCL arrival	8:02 PM	10 minutes ED ECG to CCL arrival
Reperfusion	8:20 PM	79 minutes FMC to reperfusion

FIGURE 6

FMC, first medical contact; EMS, emergency medical services; ECG, electrocardiogram; CCL, cardiac catheterization laboratory.
*Total ED length of stay = 15 minutes.

impulse travels through the bundle of His, down the bundle branches, and through the Purkinje fibers causing the ventricles to contract.[6] In the setting of third-degree heart block, the SA node continues to send the impulse, but the AV node does not receive the message. This contributes to a clinical finding of bradycardia where the heart rate may only be 45 to 50 bpm.[7]

Further complicating the patient's clinical course was the cardiogenic shock that was identified in the prehospital setting on the basis of hypotension secondary to the acute MI and third-degree heart block. Cardiogenic shock is the result of a cardiac injury that leads to a decrease in cardiac contractility.[8] This progresses to a cycle of reduced cardiac output and low blood pressure, which further worsens cardiac ischemia.[8] Clinically observed as the inability to maintain a systolic blood pressure of 90 mm Hg or more for 30 minutes or longer, cardiogenic shock may also manifest as altered mental status; oliguria; and cold, clammy skin.[8]

Discussion

REVASCULARIZATION

Treatment of an inferior STEMI and third-degree heart block involves a reperfusion strategy, either mechanical or medicinal, to restore optimal blood flow and cardiac function. The preferred reperfusion strategy is a percutaneous coronary intervention (PCI). Compared with fibrinolytic therapy, PCI has been shown to improve outcomes, including higher rates of vessel patency and lower rates of intracranial hemorrhage and death.[9] In addition, the American College of Cardiologists and the American Heart Association (AHA) recommend a drug-eluting stent with primary PCI so long as the patient is willing to comply with a prolonged course of dual antiplatelet therapy. For a third-degree heart block, initial treatment includes application of transcutaneous or transvenous pacing, which allows for rhythm stabilization; definitive treatment is an implanted pacemaker or PCI if the heart block is caused by an MI.[7]

COMPLICATED BRADYCARDIA

The AHA Advanced Cardiac Life Support 2020 guidelines for the treatment of adult bradycardia recommend atropine 1 mg through IV push every 3 to 5 minutes (maximum dose of 3 mg) as the initial treatment until the desired effect is achieved.[10] However, the guidelines caution clinicians on the use of atropine in bradycardia due to a third-degree heart block because it may not be responsive to the reversal of the cholinergic effects of atropine.[11] Atropine is not recommended as first-line treatment for bradycardia secondary to third-degree heart block because it acts on the AV node, not the SA node, and therefore is rarely effective in raising the heart rate.[12] If a third-degree heart block is identified on an ECG, the clinician should initiate transcutaneous pacing.

For this patient, pacing was not successful; per the EMS report, capture was not achieved, and the patient was unable to tolerate the intervention. In a clinical setting, if pacing is no longer an option for initial treatment, other options for symptomatic bradycardia are treatment of the

underlying medical condition (eg, dialysis), IV medications (either DOPamine or EPINEPHrine), PCI, or pacemaker implantation.[13] Therefore, EMS proceeded to secondary interventions for treatment of third-degree heart block using EPINEPHrine. Because EPINEPHrine is a sympathomimetic catecholamine and acts on both alpha- and beta-adrenergic receptors, it acts on all pacemaker sites of the heart to cause vasoconstriction and can restore hemodynamic stability while awaiting definitive treatment. An additional clinical advantage of the use of EPINEPHrine in this patient's case was the improvement of diastolic blood pressure and coronary artery perfusion pressure

HYPOGLYCEMIA IN MI

The patient had an initial fingerstick blood glucose level of 30 mg/dL per the EMS report. During transport, the EMS personnel administered a 10% dextrose infusion per their protocol. Owing to an increased risk of extravasation, the local EMS agency does not stock 50% dextrose in its ambulances. A possible cause of the patient's initial low blood glucose is "silent hypoglycemia," which is defined as a blood glucose level of less than 70 mg/dL without typical symptoms of hypoglycemia.[14] Hypoglycemia was found to be frequently occurring in patients without diabetes diagnosed with an acute MI.[14] On arrival to the emergency department, the patient's blood glucose level was 285 mg/dL. During hospitalization, the patient's hemoglobin A_{1C} level was evaluated and found to be 5.8% (5.7%-6.5%), which is in the prediabetes range.[15] There was no follow-up on discharge for the patient's hemoglobin A_{1C} level.

BYSTANDER CPR IN SUSPECTED CARDIAC ARREST

It is highly probable that this patient did not lose pulses during his suspected cardiac arrest but instead had a syncopal event caused by low cardiac output and decreased peripheral resistance, resulting in hypotension and cerebral hypoperfusion.[16] The patient did receive bystander CPR. Retrospective cohort reviews have revealed the benefit of bystander CPR on 30-day survival rate and overall mortality rate for out-of-hospital cardiac arrests; it is well known that there is a positive correlation between early CPR and survival rates.[10,17] Furthermore, layperson-initiated CPR is endorsed by the AHA because the risk of harm to the patient is low if the patient is not in cardiac arrest.[10] Ultimately, the patient was taken to the cardiac catheterization laboratory for PCI with stent placement in 61 minutes from first medical contact (goal of 90 minutes).[9] Figure 6 outlines the event timeline.

Implications for Emergency Nursing

For an emergency nurse, it is important to be aware of the symptoms of acute coronary syndrome: typical symptoms include chest pain associated with nausea, dyspnea, diaphoresis, or syncope, and atypical symptoms may include fatigue and upper abdominal pain. This patient group classified as high risk needs rapid assessment and intervention, including an ECG within 10 minutes of arrival.[11] The trained emergency nurse is knowledgeable on evidence-based care of patients experiencing an STEMI and associated progression of disease. When an inferior MI is identified on an ECG by an ED provider or cardiologist, the emergency nurse should perform a right-sided ECG by reversing the precordial leads to the right side of the chest (create a mirror image of the traditional precordial leads) to determine RV involvement.[3] If the right ventricle is involved, the patient's treatment pathway should differ in that vasodilators and nitrates should be avoided to maintain RV preload and adequate cardiac output while awaiting intervention.[18] For patients who arrive with symptoms of acute coronary syndrome without ST elevation on their ECG, the emergency nurse should follow their facility protocols for serial cardiac enzyme testing. The emergency nurse should be aware of the time intervals between repeat ECG tracings and serial troponin levels, as well as hospital processes and protocols for the care of cardiac patients.

When a third-degree heart block is identified, transcutaneous pacing is indicated for initial treatment until the cause of the heart block can be reversed or a permanent pacemaker can be inserted. Because temporary transvenous pacing may be done in the emergency department, the emergency nurse should remain up to date on the procedure steps as well as complications that may occur. In addition, the nurse should observe the patient for expected and adverse outcomes as a patient with bradycardia due to a heart block; the patient will likely experience decreased perfusion and decreased cardiac output. A sedative, if ordered, should be administered before the pacing attempt because the patient may experience discomfort. Vital signs, skin color, level of consciousness, and peripheral pulses should be monitored to evaluate the effectiveness of the paced heart rhythm.[5] Potential adverse events include failure to capture, patient discomfort, arrhythmias, and skin breakdown.[5] Documentation of the procedure should include the date and time of pacer initiation, pacemaker settings, and the patient's response. A 12-lead ECG should be obtained before and after pacing is initiated. Finally, the emergency nurse should be knowledgeable about other treatment modalities for third-degree heart block when pacing is not an option, such as pharmacologic management.

Case Conclusion

The patient was admitted to the cardiac intensive care unit and discharged to home after 2 days. As follow-up, the patient had a virtual visit with the cardiology team in which the patient reported no recurrence of chest discomfort. In addition, the patient was able to return to work. This case demonstrates the importance of prehospital communication and notification protocols, especially in the setting of a complicated MI.

Author Disclosures

Conflicts of interest: none to report.

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