

ECG CASES

Column Editor: Pedro Colio, PhD, DNP, ENP-C, FNP-C



ECG Changes and High Sensitivity Troponin I in Acute Cholecystitis

Pooja Kumar, RN, BSN

Pedro Colio, PhD, DNP, FNP-C, ENP-C

ABSTRACT

Abdominal pain is a common chief complaint in Emergency departments (EDs) nationwide which presents a wide array of potential etiologies including acute coronary syndrome (Daniels et al., 2020). High-sensitivity troponin I (hs-TnI), a biomarker traditionally linked to cardiac injury, can also be elevated in noncardiac conditions, such as acute cholecystitis. This case study underscores the advantages of hs-TnI over conventional troponin tests, specifically among patients with abnormal ECGs. This case also highlights the hs-TnI superior sensitivity in detecting or excluding subtle myocardial injuries that may arise from conditions like acute cholecystitis (Babic et al., 2012). By integrating hs-TnI into the diagnostic workup, advanced practice registered nurses can more effectively stratify patient risk, thereby enhancing the quality of early diagnosis and management of acute cholecystitis. **Key words:** abnormal ECG, demand ischemia, myocardial infarction

Acute abdominal pain among adult and elderly patients is a common chief complaint in the emergency department, often requiring significant resources and advanced medical decision-making to determine the cause. Myocardial ischemia is frequently included in the differential diagnosis for adult patients with abdominal discomfort. High-sensitivity cardiac troponin I (hs-TnI) is recognized as a gold standard biomarker for assessing cardiac

injury. However, it has also been shown to be elevated in noncardiac conditions, such as acute cholecystitis (Amle, Patil, Sakarde, John, & Mehra, 2022). This case study aims to explore nonspecific electrocardiogram (ECG) changes and the diagnostic value of hs-TnI in an adult patient with known cardiovascular disease who presented with atypical abdominal discomfort.

PATIENT PRESENTATION- HISTORY OF PRESENT ILLNESS

A 55-year-old Hispanic female with a long history of hypertension and hyperlipidemia presented to the emergency department (ED) with dull to severe intermittent abdominal pain radiating to her left shoulder and chest for the past 2 days. She described the pain as severe, rating it 8/10 in intensity, although she noted that

Author Affiliations: DNP, FNP/ENP Student, University of San Diego, San Diego, California (Ms Kumar); Assistant Professor, San Diego State University, San Diego, California (Dr. Colio); and DNP, FNP-C, ENP-C Imperial Cardiac Center Imperial, California (Dr Colio). The authors report no conflicts of interest.

Corresponding Author: Pooja Kumar, RN, BSN, DNP, FNP/ENP Student, University of San Diego, 5998 Alcalá Park Way, San Diego, California 92110 (Poojakumar@sandiego.edu).

DOI: 10.1097/TME.0000000000000566

it did not always worsen with movement or deep breaths. The pain was non-localized to a particular abdominal quadrant. The patient also experienced dyspnea, fatigue, nausea, and vomited twice during this period, resulting in significant distress and tearfulness. She attempted to manage the pain with over-the-counter medications, such as acetaminophen and ibuprofen with no relief. Additionally, she reported no prior history of diagnosed cardiac disease or recent abdominal illness.

PAST MEDICAL AND SOCIAL HISTORY

The patient's medical history includes essential hypertension and hyperlipidemia, for which she has been taking Metoprolol succinate 50 mg and atorvastatin 40 mg daily. Her gynecological and obstetric history includes 2 full-term, uncomplicated pregnancies. She denies any prior surgical history and does not use alcohol, recreational drugs, or tobacco.

VITAL SIGNS

- BP: 150/70 mm Hg
- HR: 110 beats per minute
- Temperature: 37.8°C (100.04°F)
- RR: 22 breaths per minute
- O₂ Sat: 98% on room air
- BMI: 28.9

PHYSICAL EXAM

The patient presented in acute distress, appearing tearful but remained alert and oriented to person, place, date, and time. On cardiovascular assessment, she exhibited tachycardia, though her radial and pedal pulses were strong and equal, with brisk capillary refill. Peripheral edema, without pitting, was observed. Respiratory examination showed clear bilateral breath sounds, with no wheezes, rhonchi, or crackles. Abdominal examination revealed mild tenderness in the right upper quadrant but no guarding, rigidity, or rebound

tenderness. There were no signs of periumbilical tenderness. Neurological assessment found the patient alert with no focal deficits, and her Glasgow Coma Scale score was 15. Musculoskeletal evaluation revealed full range of motion in all extremities with no tenderness or obvious deformities except for mild shoulder discomfort previously described. The remainder of the physical examination, including skin and genitourinary systems, was unremarkable.

DIAGNOSTIC LABS AND IMAGING

The patient's diagnostics test included a complete blood count, complete metabolic panel, urinalysis, lipase, high-sensitivity troponin I (hs-TnI), 12 lead ECG, and abdominal sonogram. For detailed results, refer to Tables 1–6.

The diagnostic evaluation revealed leukocytosis with a white blood cell count of $13 \times 10^3/\mu\text{L}$, suggesting an inflammatory or infectious process. Liver function tests indicated elevated bilirubin levels at 1.8 mg/dL alongside mild increases in AST (45 U/L) and ALT (38 U/L), pointing to hepatobiliary involvement. Lipase was slightly elevated at 60 U/L, effectively ruling out significant pancreatic involvement. hs-TnI levels were initially elevated at 0.100 ng/mL, peaked at 0.284 ng/mL 3 hours later, and decreased to 0.138 ng/mL after 8 hours, consistent with demand ischemia rather than acute myocardial infarction. An abdominal ultrasound confirmed a contracted gallbladder containing gallstones and echogenic bile but showed no evidence of biliary obstruction or acute inflammation. This comprehensive assessment helped narrow the differential diagnosis and guide the subsequent management plan.

DIFFERENTIAL DIAGNOSIS/MEDICAL DECISION MAKING

The differential diagnosis, listed in order of suspicion and clinical probability, included

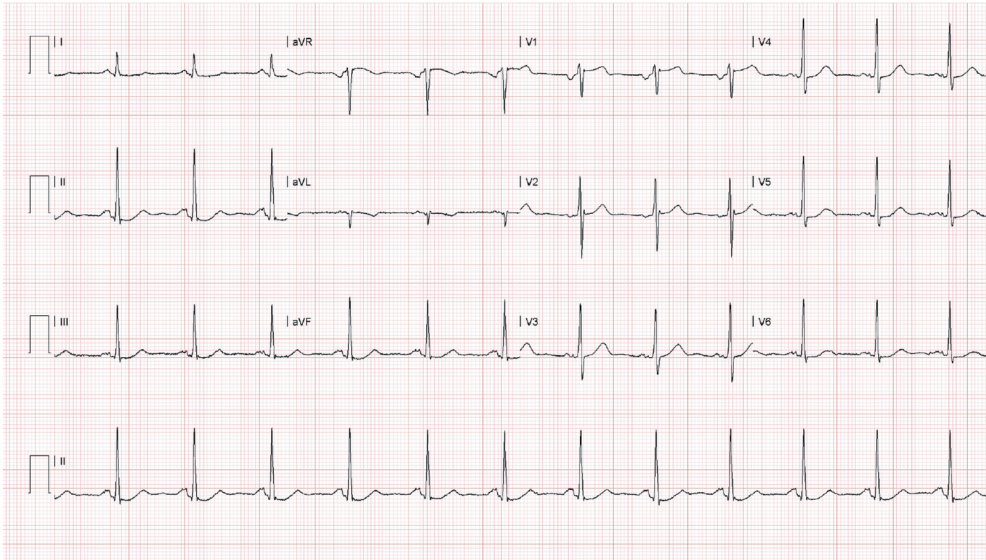


Figure 1. The patient exhibited an abnormal EKG with nonspecific ischemic changes. Imperial Cardiac Center (2024), with permission.

acute cholecystitis, cholelithiasis, pancreatitis, gastritis, and myocardial infarction.

Acute Cholecystitis

The patient’s clinical presentation included moderate to severe abdominal pain,

especially localized to the right upper quadrant, along with nausea and vomiting. This was highly indicative of acute cholecystitis or cholelithiasis. The pain radiating to the shoulder further supported this diagnosis. An abdominal ultrasound, the preferred imaging modality for suspected cholecystitis (Lammert et al., 2016), confirmed the

Table 1. Complete blood count

Complete blood count (CBC)	Normal	Results
WBC	4.0–11.0 10 × 3/μL	13
RBC	4.00–5.50 10 × 6/μL	4.64
HGB	12.0–16.0 g/dL	13.9
HCT	37.0%–47.0%	40.7%
MCV	80.0–99.0 fL	87.7
MCH	27.0–34.0 pg	30.0
MCHC	30.5–36.5 g/dL	34.2
RDW	11.0%–16.0%	12
Platelet Count	140.0–440.0 10 × 3/μL	255.0

The bolded value highlight abnormal findings, enhancing readability and emphasizing key deviations that underscore the clinical significance of the study’s results Imperial Cardiac Center, 2024, with permission.

Table 2. Chemistry 10 panel

Chemistry 10 panels	Normal	Results
Sodium	135–145 mmol/L	140
Potassium	3.5–5.3 mmol/L	4.0
Chloride	98–110 mmol/L	107
CO2	22–28 mmol/L	27
Anion gap	8–16 mmol/L	6
Calcium ionized	4.48–5.28 mg/dL	4.66
Glucose	70–99 mg/dL	82
BUN	6–20 mg/dL	12
Creatinine	0.5– 1.1 mg/dL	0.6
Calc osmolality	282–300 mOsm/kg	289
BUN/creat ratio	15–20 ratio	20

Imperial Cardiac Center, 2024, with permission.

Table 3. Liver panel

Liver panel	Normal	Results
Protein	6.4–8.3 g/dL	7.7
Albumin	3.4–4.8 g/dL	4.6
Globulin	2.0–3.9 g/dL	3.1
A/G Ratio	1.1–2.2 ratio	1.5
Bilirubin total	0.3–1.2 mg/dL	1.8
Bilirubin dir	0.0–0.4 mg/dL	0.5
Bilirubin ind	0.0–1.0 mg/dL	0.5
Alkaline phos	25–100 U/L	43
ALT (SGPT)	7–35 U/L	38
AST (SGOT)	8–40 U/L	45

The bolded values highlight abnormal findings, enhancing readability and emphasizing key deviations that underscore the clinical significance of the study's results. Imperial Cardiac Center, 2024, with permission.

presence of gallbladder distention and inflammation. The combination of the patient's clinical presentation, imaging findings, and

Table 4. Urinalysis

Urinalysis, culture if indicated (UACIF)	Normal	Results
Color	Yellow	Yellow
Appearance	Clear	Clear
Specific gravity	1.000–1.035	1.020
pH	5.0–9.0	5.0
Leukocyte esterase	Negative	Negative
Nitrites	Negative	Negative
Protein	Negative	Negative
Glucose	Negative	Negative
Ketones	Negative	Negative
Urobilinogen	Normal	Normal
Bilirubin	Negative	Negative
Blood	Negative	Negative
WBC	0–5/[HPF]	<1
RBC	0–3/[HPF]	1
Bacteria	None seen/[HPF]	None seen
Sq. Ep. cells	0–16/[HPF]	1

Imperial Cardiac Center, 2024, with permission.

Table 5. Lipase

Lipase	Normal	Result
	12–53/μL	60

The bolded value highlight abnormal findings, enhancing readability and emphasizing key deviations that underscore the clinical significance of the study's results. Imperial Cardiac Center, 2024, with permission.

elevated biomarkers strongly supports the diagnosis of acute cholecystitis. Elevated white blood cell levels and hs-TnI levels underscore the inflammatory and stress response associated with this condition (Amle et al., 2022; Lammert et al., 2016). The need to exclude acute coronary syndrome (ACS) further highlights the diagnostic complexity in cases involving overlapping symptoms (Patel, Ariyarathenam, Davies, & Harris, 2011). This case demonstrates the additional utility of hs-TnI as a biomarker not only for cardiac events but also for significant systemic inflammatory responses in the correct settings.

Cholelithiasis

Acute cholelithiasis was also considered as part of the differential diagnosis. Gallstones usually present with biliary colics that may resolve spontaneously, without an acute inflammatory response or gallbladder thickening. Patients may also present with elevated liver enzymes and localized tenderness

Table 6. High sensitivity-troponin I

(Hs-TnI)	Normal	Result
Initial encounter	0.0–0.04	0.100
3 hours later	0.0–0.04	0.284
8 hours after initial encounter	0.0–0.04	0.138

The bolded values highlight abnormal findings, enhancing readability and emphasizing key deviations that underscore the clinical significance of the study's results. Imperial Cardiac Center, 2024, with permission.

along the right upper quadrant (Lammert et al., 2016). The ultrasound findings indicated gallbladder contraction and inflammation which thus supports the diagnosis of acute cholecystitis rather than uncomplicated cholelithiasis (Knab, Boller, & Mahvi, 2014).

Acute Pancreatitis

Acute pancreatitis typically presents with vague upper abdominal pain that can radiate to the back, along with fever, nausea, vomiting, and toxic appearance. Normal or low serum amylase and lipase levels can help rule out acute pancreatitis (Daniels, Griffiths, & Fisher, 2020). In this case, the patient's lab results did not indicate elevated amylase or lipase, effectively ruling out pancreatitis.

Myocardial Infarction

Myocardial infarction can typically present with acute or dull chest pain and dyspnea; however, it can also manifest with atypical symptoms such as vague abdominal discomfort, fatigue, and pain in the back or neck (Pillai et al., 2020). Considering the patient's age, medical history, and clinical presentation, both ST-segment elevation myocardial infarction (STEMI) and non-ST segment elevation myocardial infarction (NSTEMI), including Type II NSTEMI, were significant concerns. The patient exhibited an abnormal EKG with nonspecific ischemic changes and elevated high-sensitivity troponin levels (see Figure 1). However, the pattern of troponin elevation and the clinical presentation, characterized by predominantly abdominal discomfort rather than persistent chest pain, did not conform to typical ACS or myocardial infarction (MI) (Amle et al., 2022; Patel et al., 2011). Furthermore, the EKG findings and the downward trend in troponin levels suggested that the elevated troponin was indicative of demand ischemia, a condition

commonly associated with systemic inflammation or stress, rather than a primary cardiac event (Espinosa et al., 2023).

ELEVATED TROPONIN IN ACUTE CHOLECYSTITIS PATHOPHYSIOLOGY

Acute cholecystitis is characterized by the inflammation of the gallbladder, which is usually due to an obstruction of the common bile duct by gallstones. This obstruction can often lead to gallbladder distension, inflammation, and bacterial infection. The increased intraluminal pressure from the bile stasis can cause ischemia of the gallbladder wall, causing tissue damage. The damaged mucosa then becomes susceptible to bacterial invasion from intestinal flora exacerbating inflammation (Kimura et al., 2007; Lammert et al., 2016). Patients with acute cholecystitis may exhibit symptoms such as right upper quadrant pain, fever, and leukocytosis. However, atypical presentations, such as referred pain to the shoulder or nonspecific abdominal pain, can also occur, making diagnosing the ED challenging (Demarchi et al., 2012). Elevated hs-cTnI levels in acute cholecystitis may result from systemic inflammation and localized ischemic events caused by increased intra-abdominal pressure. This biomarker is sensitive enough to detect minor myocardial injuries, aiding in the diagnosis of ischemia in noncardiac conditions (Lin, Cardelli, Marino, Lim, & Di Somma, 2022; Patel et al., 2011).

TYPE II NSTEMI

Type II NSTEMI represents a mismatch between the metabolic demand of myocardial oxygen and the available supply or increased demand for oxygen. This mismatch is seen in various conditions such as hypertension, diabetes, and anemia (Stein et al., 2014). This was a relevant finding in this case due to the underlying pathophysiology of acute cholecystitis involving inflammation of the gallbladder. Conditions such

as bile stasis and obstruction can also increase the risk of ECG changes, indicating ischemia and an increased need for oxygen supply, leading to an increase in Hs-cTnI. (Meelu et al., 2016).

ELECTROCARDIOGRAPHIC CHANGES IN RELATION TO ACUTE CHOLECYSTITIS

The immediate use of ECG is primarily aimed at ruling out subsets of ACS, such as STEMI. However, it is important to note that subtle ECG findings do not always indicate acute myocardial injury that necessitates immediate intervention. Type 2 NSTEMIs typically present with mildly elevated hs-TnI levels and may exhibit nonspecific ECG changes. These findings are associated with demand ischemia that is not linked to ACS, coronary injury, or plaque rupture (Pillai al., 2020).

Acute cholecystitis can cause irritation and inflammation in adjacent structures, such as the pancreas and liver, potentially leading to pathways that restrict blood supply. This resulting demand ischemia may manifest as changes on the ECG (Patel et al., 2011). Importantly, nonspecific ECG changes, such as ST segment flattening or T-wave inversion, can occur when demand ischemia or Type 2 NSTEMI is suspected (Pillai al., 2020). Given that this patient was over 50 years old and presented with epigastric symptoms, troponin testing was warranted to rule out MI. Early recognition and diagnosis are key in preventing delays in coronary intervention (Patel et al., 2011).

HS-TNI AS A BIOMARKER

High-sensitivity troponin assays have revolutionized the diagnostic approach to ACS and other conditions associated with myocardial injury. These assays effectively detect troponin at lower levels, enabling rapid recognition of myocardial damage and underlying ischemic patterns (Lazar et al., 2022). Biomarkers such as hs-TnI and high-sensitivity troponin T can identify minimal levels of

cardiac troponin in the blood, facilitating earlier diagnosis and intervention (Espinosa et al., 2023; Lin et al., 2022). Hs-cTnI demonstrates greater precision in detecting myocardial injury; however, it does not inherently differentiate between cardiac and noncardiac causes of troponin elevation. Its utility lies in its higher specificity in ED settings, which enables more accurate measurements that must be interpreted in conjunction with clinical context and additional diagnostics (Espinosa et al., 2023; Lin et al., 2022)

CASE MANAGEMENT AND OUTCOME

After a thorough physical and diagnostic evaluation, the patient received intravenous antibiotics, fluids, pain medication, and antiemetics. Given the patient's elevated hs-cTnI and abnormal ECG, the emergency team consulted with cardiology before going into surgery. Cardiology recommended repeating the hs-cTnI test, which indicated decreased and trending levels consistent with demand ischemia likely caused by the inflammatory process associated with cholecystitis. Consequently, no coronary angiogram was deemed necessary for evaluating coronary intervention. The cardiology team cleared the patient for surgery and advised outpatient follow-up for echocardiography and functional stress testing to rule out any underlying structural heart disease and nonobstructive coronary artery disease.

The patient was subsequently evaluated by general surgery for a planned cholecystectomy the following day. To ensure patient safety, the individual was admitted to the medical-surgical unit for cardiac monitoring, further observation and decreased inflammation of the gallbladder before going into surgery. During this period, the patient's troponin levels trended downward, likely reflecting a noncardiac etiology such as demand ischemia secondary to the inflammatory process. However, given the patient's cardiac risk factors,

including hypertension, hyperlipidemia, and high BMI, underlying atherosclerotic disease cannot be excluded without further evaluation. Outpatient cardiology follow-up is essential to assess for potential structural heart disease or nonobstructive coronary artery disease. This careful management illustrates the importance of interdisciplinary collaboration in addressing complex cases where potential cardiac issues intersect with acute abdominal conditions.

IMPLICATIONS FOR PRACTICE

Hs-cTnI is a highly specific tool to rule out cardiac-related conditions and other underlying disease processes in the presence of subtle ischemic ECG changes. However, emergency clinicians must be aware that the elevated Hs-cTnI may present in noncardiac-related illness. Trending Hs-cTnI levels can help mitigate and stratify pertinent positives (Lazar et al., 2022). The case helps to signify the importance of comprehensive assessment as well as using interdisciplinary expertise in improving patient outcomes.

SUMMARY/CONCLUSION

In this case study, the vital role of hs-cTnI in the evaluation of acute cholecystitis is emphasized, particularly in patients presenting with atypical symptoms such as abdominal pain radiating to the chest and left shoulder, which can mimic cardiac events. The patient's clinical presentation and medical history prompted essential diagnostic testing, including an ECG and hs-TnI levels. The abnormal ECG and elevated hs-TnI prompted a cardiology consultation, aiding in the differentiation between cardiac and gastrointestinal differentials.

Importantly, the ECG findings, including ST segment flattening and T-wave inversion, provided critical insights into the patient's condition. These nonspecific changes can indicate demand ischemia, underscoring the complexity of diagnosing abdominal pain in the presence of potential cardiac

issues. An abdominal sonogram ultimately confirmed acute cholecystitis, leading to timely surgical intervention.

This case highlights the importance of considering hs-cTnI not only as a biomarker for cardiac events but also for acute inflammatory processes like cholecystitis. The integration of hs-TnI and ECG findings into the diagnostic workup can significantly enhance clinical decision-making, improving patient outcomes by guiding timely and effective treatment interventions. By recognizing the relationship between elevated troponin levels and ECG changes in the context of acute abdominal pain, emergency clinicians can make more accurate diagnoses and optimize patient management.

REFERENCES

- Amle, D., Patil, N., Sakarde, A., John, D. J., Mehra, B. (2022). Acute cholecystitis leading to elevated high-sensitive cardiac troponin I in a young female without any cardiac ailment: A rare case presentation. *Cureus*, 14(12), e33194. doi:10.7759/cureus.33194
- Babić, Z., Bogdanović, Z., Dorosulić, Z., Basha, M., Krznarić, Z., Sjekavica, I., Marusić, M. (2012). Quantitative analysis of troponin I serum values in patients with acute cholecystitis. *Collegium Antropologicum*, 36(1), 145–150.
- Daniels, J., Griffiths, M., Fisher, E. (2020). Assessment and management of recurrent abdominal pain in the emergency department. *Emergency Medicine Journal: EMJ*, 37(8), 515–521. doi:10.1136/emered-2019-209113
- Demarchi, M. S., Regusci, L., Fasolini, F. (2012). Electrocardiographic changes and false-positive troponin I in a patient with acute cholecystitis. *Case Reports in Gastroenterology*, 6(2), 410–414. doi:10.1159/000339965
- Espinosa, A. S., Hussain, S., Al-Awar, A., Jha, S., Elmahdy, A., Kalani, M., Redfors, B. (2023). Differences between cardiac troponin I vs. T according to the duration of myocardial ischaemia. *European Heart Journal Acute Cardiovascular Care*, 12(6), 355–363. doi:10.1093/ehjacc/zuad017
- Kimura, Y., Takada, T., Kawarada, Y., Nimura, Y., Hirata, K., Sekimoto, M., Gadacz, T. R. (2007). Definitions, pathophysiology, and epidemiology of acute cholangitis and cholecystitis: Tokyo Guidelines. *Journal of Hepato-Biliary-Pancreatic Surgery*, 14(1), 15–26. doi:10.1007/s00534-006-1152-y

- Knab, L. M., Boller, A. M., Mahvi, D. M. (2014). Cholecystitis. *Surgical Clinics of North America*, 94(2), 455–470. doi:10.1016/j.suc.2014.01.001
- Lammert, F., Gurusamy, K., Ko, C. W., Miquel, J. F., Moreau, R., Portincasa, P., van Laarhoven, C. J. H. M. (2016). Gallstones. *Nature Reviews Disease Primers*, 2(1), 16024. doi:10.1038/nrdp.2016.24
- Lazar, D. R., Lazar, F. L., Homorodean, C., Cainap, C., Focsan, M., ... Olinic, D. M. (2022). High-sensitivity troponin: A review on characteristics, assessment, and clinical implications. *Disease Markers*, 2022, 9713326. doi:https://doi.org/10.1155/2022/9713326
- Lin, Z., Cardelli, P., Marino, R., Lim, S. H., Di Somma, S.; Great Network. (2022). Advantage of using of high-sensitivity troponin I compared to conventional troponin I in shortening time to rule out/in acute coronary syndrome in chest pain patients presenting to the Emergency Department. *Medicina (Kaunas, Lithuania)*, 58(10), 1391.
- Meelu, O. A., Baber, U., Theodoropoulos, K., Mennuni, M. G., Kini, A. S., Sharma, S. K. (2016). Acute cholecystitis and myocardial infarction: A case study with coronary involvement. *Clinical Case Reports*, 4(8), 793–796. doi:10.1002/ccr3.621
- Patel, N., Ariyathenam, A., Davies, W., Harris, A. (2011). Acute cholecystitis leading to ischemic ECG changes in a patient with no underlying cardiac disease. *JSLS: Journal of the Society of Laparoendoscopic Surgeons*, 15(1), 105–108. doi:10.4293/108680811X13022985131534
- Pillai, B., Trikkur, S., Farooque, U., Ramakrishnan, D., Kakkra, J. J., Kashyap, G., ... Vishwanath, J. (2020). Type II myocardial infarction: predisposing factors, precipitating elements, and outcomes. *Cureus*, 12(7), e9254. doi:https://doi.org/10.7759/cureus.9254
- Stein, G.Y., Herscovici, G., Korenfeld, R., Matetzky, S., Gottlieb, S., ... Iakobishvili, Z., Fuchs, S. (2014). Type-II myocardial infarction—patient characteristics, management and outcomes. *PloS One*, 9(1), e84285. doi:10.1371/journal.pone.0084285

Lippincott®
NursingCenter®

NCPD Nursing Continuing
Professional Development

TEST INSTRUCTIONS

- Read the article. The test for this nursing continuing professional development (NCPD) activity is to be taken online at www.nursingcenter.com/CE/AENJ. Tests can no longer be mailed or faxed.
- You'll need to create an account (it's free!) and log in to access My Planner before taking online tests. Your planner will keep track of all your Lippincott Professional Development online NCPD activities for you.
- There's only one correct answer for each question. A passing score for this test is 8 correct answers. If you pass, you can print your certificate of earned contact hours and access the answer key. If you fail, you have the option of taking the test again at no additional cost.
- For questions, contact Lippincott Professional Development: 1-800-787-8985
- Registration deadline is June 4, 2027.

PROVIDER ACCREDITATION

Lippincott Professional Development will award 2.0 contact hours and 0 pharmacology contact hours for this nursing continuing professional development activity.

Lippincott Professional Development is accredited as a provider of nursing continuing professional development by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 2.0 contact hours. Lippincott Professional Development is also an approved provider of continuing nursing education by the District of Columbia, Georgia, West Virginia, New Mexico, South Carolina, and Florida, CE Broker #50-1223. Your certificate is valid in all states.

Payment: The registration fee for this test is \$21.95.

For more than 100 additional nursing continuing professional development activities related to emergency care topics, go to NursingCenter.com/ce.