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CASE REPORT OPEN ACCESS

Atypical Presentation of Acute Myocardial Infarction Mimicking Gastrointestinal Pathology in a Young Adult

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ABSTRACT

Acute myocardial infarction classically presents with chest pain; however, atypical presentations without chest discomfort may lead to diagnostic delay and increased morbidity. Gastrointestinal symptoms such as epigastric pain and vomiting can mimic primary abdominal pathology, particularly in young adults perceived to have a low cardiovascular risk. A 29-year-old male presented to the emergency department with six hours of severe epigastric pain (8/10 intensity) accompanied by recurrent vomiting. He denied chest pain or dyspnoea. Electrocardiography demonstrated 2–3 mm ST-segment elevation in leads II, III, and aVF with reciprocal ST depression in leads I and aVL. High-sensitivity troponin I levels were markedly elevated. Emergent coronary angiography revealed a complete thrombotic occlusion of the proximal right coronary artery. The patient underwent successful primary percutaneous coronary intervention with deployment of a drug-eluting stent, restoring TIMI grade 3 coronary flow. Inferior ST-elevation myocardial infarction may present solely with gastrointestinal symptoms even in young adults. Early electrocardiography remains essential in patients presenting with unexplained epigastric pain to prevent delayed reperfusion and adverse outcomes.

KEYWORDS

Acute myocardial infarction; ST-elevation myocardial infarction; Right coronary artery; Primary percutaneous coronary intervention; Atypical presentation; Young adult

How to cite

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1 | Background

Acute myocardial infarction remains a leading cause of morbidity and mortality worldwide despite advances in reperfusion strategies and guideline-directed medical therapy (Collet et al., 2023). Classical symptoms include retrosternal chest pain or pressure radiating to the arm or jaw, frequently accompanied by diaphoresis and dyspnoea. However, approximately 20–30% of patients with myocardial infarction present without typical chest pain (Canto et al., 2012).

Atypical manifestations may include nausea, vomiting, fatigue, syncope, dyspnoea, or epigastric discomfort (DeVon et al., 2014). These symptoms can mimic gastrointestinal disorders such as gastritis, peptic ulcer disease, or pancreatitis, resulting in diagnostic uncertainty and delays in definitive management.

Patients presenting without chest pain have been shown to experience longer delays to diagnosis and higher mortality rates compared with those presenting with classic symptoms (Pope et al., 2000; Canto et al., 2012). Young adults may be particularly vulnerable to delayed recognition because clinicians may underestimate the probability of coronary artery disease in this population, especially when traditional cardiovascular risk factors are limited (Brieger et al., 2004).

Inferior wall myocardial infarction, most commonly resulting from occlusion of the right coronary artery (RCA), frequently produces gastrointestinal symptoms. This phenomenon is believed to occur due to vagal stimulation and diaphragmatic irritation associated with ischemia of the inferior myocardial wall (Thygesen et al., 2018). For this reason, current European Society

of Cardiology (ESC) guidelines recommend obtaining a 12-lead electrocardiogram within 10 minutes of first medical contact in any patient with suspected ischemic symptoms, even if the presentation is atypical (Collet et al., 2023).

This report describes a case of inferior ST-elevation myocardial infarction presenting as isolated epigastric pain in a young adult, highlighting the importance of maintaining diagnostic vigilance when evaluating patients with unexplained gastrointestinal symptoms.

2 | Case Presentation

A 29-year-old male presented to the emergency department with persistent epigastric pain that began approximately six hours prior to arrival. The pain was described as burning in character, rated 8 out of 10 in severity on a visual analogy scale, and localised to the epigastric region without radiation. The patient reported five episodes of non-bilious vomiting but denied chest pain, dyspnoea, palpitations, diaphoresis, or syncope.

The patient had a 10-pack-year history of cigarette smoking and reported a family history of premature coronary artery disease in his father, who experienced a myocardial infarction at 48 years of age. He had no known history of hypertension, diabetes mellitus, dyslipidaemia, or prior cardiovascular disease.

3 | Methods

3.1 Investigations and Clinical Findings

On initial evaluation, the patient appeared uncomfortable but was hemodynamically stable.



FIGURE 1. Twelve-lead electrocardiogram demonstrating ST-segment elevation in leads II, III, and aVF with reciprocal ST depression in leads I and aVL, consistent with inferior wall myocardial infarction.

Vital signs showed a blood pressure of 148/92 mmHg, a heart rate of 104 beats per minute, a respiratory rate of 18 breaths per minute, oxygen saturation of 98% on room air, and a temperature of 36.8°C. Physical examination revealed mild epigastric tenderness on abdominal palpation without guarding, rebound tenderness, or organomegaly. The cardiovascular examination demonstrated a regular rhythm without murmurs, gallops, or rubs, and the respiratory examination was unremarkable with clear breath sounds bilaterally. Initial treatment with a proton pump inhibitor and antiemetic medication resulted in minimal symptom relief, with the pain remaining at 7/10 intensity.

Table 1. Timeline

| Event | Time |
|-----------------------|------------------------------|
| Symptom onset | 0 hours |
| First medical contact | 6 hours |
| ECG performed | 8 minutes after triage |
| Troponin drawn | 15 minutes |
| Cath lab activation | 25 minutes |
| Balloon inflation | 68 minutes (door-to-balloon) |

Initial laboratory testing demonstrated mild leucocytosis with a white blood cell count of $12.4 \times 10^9/L$, while liver function tests, serum amylase, and lipase levels were within normal limits, reducing the likelihood of acute hepatobiliary or pancreatic pathology. A 12-lead electrocardiogram obtained shortly after triage revealed ST-segment elevation of 2.5 mm in lead II, 3.0 mm in lead III, and 2.0 mm in lead aVF, along with reciprocal ST-segment depression of 1.5 mm in lead I and 1.0 mm in lead aVL. These findings were consistent with acute inferior ST-elevation myocardial infarction.

TABLE 2. Cardiac Biomarkers

| Time | hs-Troponin I (ng/L) | Reference |
|------|----------------------|-----------|
| 0 hr | 2,450 | <34 |
| 3 hr | 6,780 | <34 |
| 6 hr | 8,920 | <34 |

The marked rise in cardiac troponin confirmed acute myocardial necrosis (Thygesen et al., 2018)

Bedside transthoracic echocardiography demonstrated hypokinesia of the inferior wall with preserved overall left ventricular systolic function and an estimated ejection fraction of approximately 55%.

3.2 Intervention and Treatment

The patient was immediately transferred to the cardiac catheterization laboratory for emergent coronary angiography via right radial artery access. Coronary angiography revealed a complete (100%) thrombotic occlusion of the

proximal right coronary artery with TIMI grade 0 flow before intervention, while no significant atherosclerotic disease was found in the left coronary circulation. Primary percutaneous coronary intervention was subsequently performed with the placement of a 3.5 × 24 mm everolimus-eluting drug-eluting stent. Procedural details included stent deployment at 16 atmospheres and post-dilation using a non-compliant balloon at 18 atmospheres, which resulted in the restoration of TIMI grade 3 coronary flow with no residual stenosis or procedural complications.

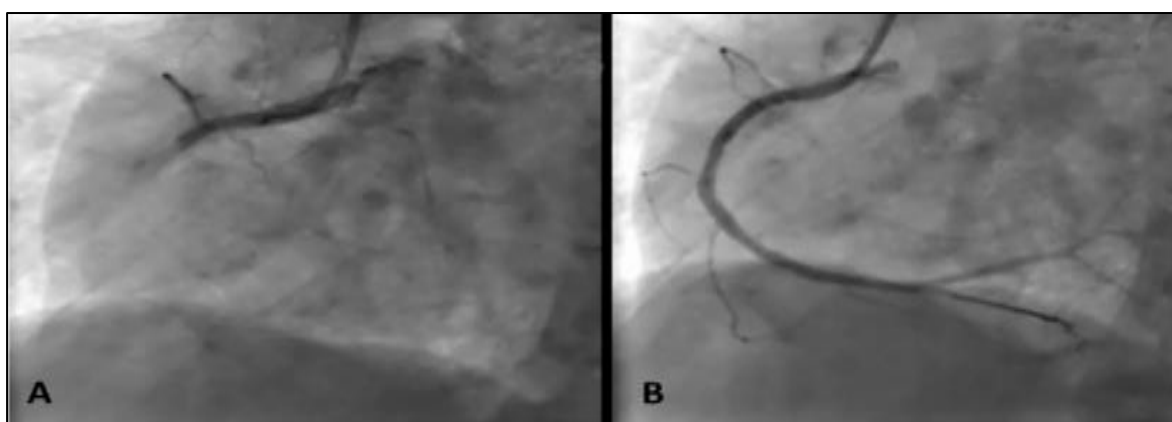


FIGURE 2. Coronary angiography demonstrating proximal right coronary artery occlusion before intervention (A) and restoration of coronary blood flow after drug-eluting stent placement during primary PCI (B).

Following the procedure, the patient was initiated on guideline-directed medical therapy including Aspirin 300 mg loading dose followed by 75 mg daily, Ticagrelor 180 mg loading dose followed by 90 mg twice daily, Atorvastatin 80 mg daily, Metoprolol succinate, and Ramipril. Management was consistent with contemporary ESC recommendations for acute coronary syndromes (Collet et al., 2023).

Follow-Up and Outcomes

The patient's epigastric pain resolved almost immediately following coronary reperfusion. His hospital course was uncomplicated, and he was discharged on dual antiplatelet therapy and secondary prevention medications.

Three-Month Follow-Up

At outpatient follow-up three months later, the patient reported complete resolution of symptoms. Laboratory testing demonstrated a

reduction in low-density lipoprotein cholesterol from 156 mg/dL at admission to 72 mg/dL. Medication adherence was confirmed, and the patient successfully completed a structured cardiac rehabilitation program.

Six-Month Follow-Up

At six months, the patient remained asymptomatic with no recurrent ischemic symptoms. He reported sustained smoking cessation and adherence to lifestyle modifications including improved diet and regular physical activity. Repeat echocardiography demonstrated improvement in left ventricular function with an ejection fraction of approximately 60%.

4 | Discussion

This case highlights the diagnostic challenges associated with atypical presentations of acute

myocardial infarction, particularly in younger patients who may not initially appear to be at high cardiovascular risk. Cognitive biases, including anchoring and premature diagnostic closure, can lead clinicians to focus on gastrointestinal causes when patients present with abdominal symptoms (Croskerry, 2003).

Inferior wall myocardial infarction frequently produces nausea, vomiting, and epigastric discomfort due to vagally mediated autonomic responses and irritation of the diaphragm (DeVon et al., 2014). These symptoms may overshadow cardiac manifestations, potentially delaying recognition of acute coronary syndrome.

Although risk stratification tools such as the HEART and TIMI scores are widely used in patients presenting with chest pain, their application may be limited in individuals with atypical symptoms (Backus et al., 2013; Antman et al., 2000). Clinicians should therefore maintain a low threshold for obtaining an electrocardiogram in patients presenting with unexplained epigastric pain or persistent gastrointestinal symptoms.

Early recognition is particularly important because timely reperfusion therapy significantly improves survival and reduces the risk of complications such as heart failure and arrhythmias. In the present case, rapid electrocardiographic evaluation allowed prompt activation of the cardiac catheterization team and successful primary percutaneous coronary intervention within guideline-recommended timeframes.

Educational initiatives emphasizing atypical presentations of acute coronary syndromes may help reduce diagnostic delays and improve patient outcomes.

5 | Conclusions

Acute myocardial infarction can present solely as epigastric pain in young adults without classical chest discomfort. Clinicians should maintain a high index of suspicion when evaluating patients with unexplained gastrointestinal symptoms, particularly when cardiovascular risk factors are present. Early electrocardiography and adherence to evidence-based guidelines are essential to ensure rapid diagnosis and timely reperfusion therapy.

6 | References

- Amsterdam, E. A., et al. (2014). AHA/ACC guidelines for ACS management. *Journal of the American College of Cardiology*, 64(24), e139–e228. <https://doi.org/10.1016/j.jacc.2014.09.017>
- Antman, E. M., et al. (2000). The TIMI risk score for unstable angina/non-ST elevation MI. *JAMA*, 284(7), 835–842. <https://doi.org/10.1001/jama.284.7.835>
- Backus, B. E., et al. (2013). A prospective validation of the HEART score. *International Journal of Cardiology*, 168(3), 2153–2158. <https://doi.org/10.1016/j.ijcard.2013.01.255>
- Brieger, D., et al. (2004). Acute coronary syndromes without chest pain. *Chest*, 126(2), 461–469. <https://doi.org/10.1378/chest.126.2.461>
- Canto, J. G., et al. (2012). Prevalence and mortality among patients with MI presenting without chest pain. *JAMA*, 307(8), 813–822. <https://doi.org/10.1001/jama.2012.109>
- Collet, J.-P., Thiele, H., Barbato, E., et al. (2023). 2023 ESC guidelines for acute coronary syndromes. *European Heart Journal*, 44(38), 3720–3826. <https://doi.org/10.1093/eurheartj/ehad191>
- Croskerry, P. (2003). The importance of cognitive errors in diagnosis. *Academic Medicine*, 78(8), 775–780.
- DeVon, H. A., et al. (2014). Typical and atypical symptoms of ACS. *Journal of the American Heart Association*, 3(2), e000586. <https://doi.org/10.1161/JAHA.113.000586>
- Pope, J. H., et al. (2000). Missed diagnoses of acute cardiac ischemia. *New England Journal of Medicine*, 342(16), 1163–1170. <https://doi.org/10.1056/NEJM200004203421603>
- Thygesen, K., et al. (2018). Fourth universal definition of myocardial infarction. *European Heart Journal*, 40(3), 237–269. <https://doi.org/10.1093/eurheartj/ehy462>
- Yusuf, S., et al. (2004). Effect of modifiable risk factors associated with MI (INTERHEART study). *The Lancet*, 364(9438), 937–952. [https://doi.org/10.1016/S0140-6736\(04\)17018-9](https://doi.org/10.1016/S0140-6736(04)17018-9)

Author Contributions

Moses Phiri: conceptualization, data curation, formal analysis, writing – original draft, writing – review and editing.

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Ethics Statement

This case report was conducted in accordance with the principles outlined in the Declaration of Helsinki. Institutional ethical approval was not required for publication of this particular case report, in line with local regulations.

Conflicts of Interest

The author declares no conflicts of interest related to this work.

Data Availability Statement

All data generated or analysed during this study are included within this published article. Further details are available from the corresponding author upon reasonable request, subject to patient confidentiality considerations.