E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.02.11

Case Report

# Insomnia as an Atypical Presentation of Acute Circumflex-Territory Transmural Myocardial Infarction with Persistent Sinus Tachycardia: a Case Report

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Received: 19.09.2026, Revised: 16.10.2026, Accepted: 12.11.2026

**ABSTRACT**
**Background:** Acute myocardial infarction (AMI) may be present without classical chest pain, particularly in patients with multiple cardiovascular risk factors. Atypical and nonspecific symptoms may obscure acute myocardial infarction, particularly when classical ischemic symptoms are absent. In this case, persistent insomnia was the atypical presenting symptom, while persistent sinus tachycardia was an important clinical sign that prompted further cardiovascular assessment. Cardiac magnetic resonance (CMR) provides comprehensive assessment of myocardial function, oedema, infarct distribution, late gadolinium enhancement, and myocardial viability.
**Case presentation:** A 51-year-old man presented to his general practitioner with a several-week history of persistent insomnia. He denied chest pain, dyspnea, palpitations, syncope, and constitutional symptoms. His medical history was otherwise unremarkable. He was a current smoker and had a strong family history of premature coronary artery disease; his father had undergone coronary artery bypass grafting at 50 years of age. Clinical examination identified persistent tachycardia, an objective clinical sign that prompted referral to the emergency department. Laboratory evaluation demonstrated markedly elevated cardiac troponin, reported as 2,285, and he was managed initially as acute coronary syndrome. Thyroid function testing was within normal limits. The persistent tachycardia was investigated diagnostically with adenosine; the rhythm remained consistent with sinus tachycardia. Bisoprolol subsequently commenced, resulting in good rate control. Transthoracic echocardiography demonstrated mildly impaired left ventricular systolic function with an estimated ejection fraction of 40–45% and regional wall-motion abnormalities. CT coronary angiography demonstrated severe coronary artery disease involving the circumflex territory, with likely 70–90% stenosis of the mid circumflex/obtuse marginal segment. The coronary calcium score was 350.4, while the LAD and RCA showed non-obstructive disease. CMR demonstrated regional akinesia involving the basal-to-apical lateral segments, extensive myocardial oedema, and a recent transmural myocardial infarction in the conventional circumflex territory. Biochemical testing during admission also established a new diagnosis of type 2 diabetes mellitus.
**Conclusion:** This case highlights that acute myocardial infarction may present with an atypical symptom such as persistent insomnia in the absence of classical ischemic symptoms. In this patient, persistent sinus tachycardia was an important clinical sign that prompted escalation of cardiovascular assessment. Multimodality cardiac imaging was instrumental in establishing the extent and territory of myocardial injury. Normal thyroid function testing helped exclude thyroid dysfunction as a potential reversible cause of persistent tachycardia.

**Keywords:** Acute Myocardial Infarction, Sinus Tachycardia, Circumflex Artery, Cardiac Magnetic Resonance, Myocardial Oedema, Heart Failure With Mildly Reduced Ejection Fraction, Case Report.

## INTRODUCTION

Acute myocardial infarction remains an important cause of morbidity and mortality and does not invariably present typical ischemic symptoms. Although chest discomfort is a classical manifestation, some patients may present with atypical, nonspecific, or relatively subtle symptoms. Consequently, clinicians should maintain a high

index of suspicion when objective findings suggest myocardial injury, particularly in individuals with established cardiovascular risk factors [1].

The diagnosis of myocardial infarction integrates evidence of acute myocardial injury with evidence of myocardial ischemia. Cardiac troponin is central to the assessment of myocardial injury, while regional wall-motion abnormalities and tissue-characterization findings on cardiac magnetic resonance can provide important evidence of ischemic myocardial damage [2,3].

CMR is particularly valuable following acute myocardial infarction because it can simultaneously characterize ventricular function, myocardial oedema, infarct distribution and transmurally, late gadolinium enhancement, and myocardial viability [4].

We report a 51-year-old man who presented with several weeks of persistent insomnia, an atypical presenting symptom, without chest pain or other classical ischemic symptoms. Clinical examination identified persistent sinus tachycardia as an important objective sign. Markedly elevated cardiac troponin and subsequent multimodality cardiac imaging demonstrated an acute/recent transmural circumflex-territory myocardial infarction with regional ventricular dysfunction [5,6].

## CASE PRESENTATION

### Patient information

A 51-year-old man presented to his general practitioner with a history of persistent insomnia lasting several weeks. He did not report chest pain, dyspnea, palpitations, syncope, or constitutional symptoms.

His previous medical history was otherwise unremarkable. He was a current smoker and had a significant family history of premature coronary artery disease. His father had required coronary artery bypass grafting at the age of 50 years. There was no previously documented history of diabetes mellitus, heart failure, coronary artery disease, or significant cardiovascular disease.

### Clinical findings

During clinical assessment, persistent tachycardia was identified as an objective clinical sign. Given the unexplained persistent elevation in heart rate, he was referred to the emergency department for further assessment. The presenting symptom remained persistent insomnia, which was atypical for acute myocardial infarction but provided the initial reason for seeking medical attention. There was no reported clinical history of classical anginal symptoms or heart-failure symptoms.

The persistent tachycardia, rather than the insomnia itself, prompted consideration of a supraventricular tachyarrhythmia. Adenosine was therefore administered diagnostically. The tachycardia persisted and was subsequently characterized as sinus tachycardia rather than a paroxysmal supraventricular tachycardia. Bisoprolol therapy was initiated, with good subsequent rate

### Diagnostic Assessment

### Laboratory investigations

Cardiac biomarkers demonstrated a markedly elevated cardiac troponin level of 2,285. The assay type and units were not available in the supplied laboratory documentation and should therefore be confirmed before journal submission.

In the context of the subsequent imaging findings demonstrating acute transmural myocardial infarction, this marked troponin elevation provided important biochemical evidence of acute myocardial injury. Thyroid function testing, including the thyroid profile, was within normal limits. The normal thyroid profile provided no biochemical evidence of thyroid dysfunction as a contributing cause of the persistent sinus tachycardia. Selected laboratory findings available from the clinical records are summarized below.

Table 1. Selected laboratory findings during the clinical course

| Investigation | Available results | Comment |
|---|---|---|
| Cardiac troponin | 2,285 | Markedly elevated; assay/unit to be confirmed |
| D-dimer | >500 ng/mL | Above laboratory reference threshold |
| WBC | 12.3 → 10.8 → 10.7 → 9.7 $\times 10^9$/L | Initial mild leukocytosis, subsequently improved |

| | | |
|---|---|---|
| Neutrophils | 8.90 → 7.29 → 7.49 → 6.83 $\times 10^9$/L | Initial mild elevation, subsequently improved |
| Haemoglobin | 172 → 151 → 148 → 155 g/L | Initial upper-limit value, subsequently stable |
| Platelets | 386 → 403 → 439 $\times 10^9$/L | Serial values available |
| INR | 1.0 → 1.1 → 1.1 | Within stated reference range |
| Prothrombin time | 11.3 → 11.9 → 11.6 s | Within stated reference range |
| TSH<br>FT3<br>FT4 | 2.5mU/L<br>15pmol/L<br>4.4pmol/L | No biochemical evidence of thyroid dysfunction |

The available biochemical evaluation during admission additionally established a new diagnosis of type 2 diabetes mellitus. The exact diagnostic glucose and/or HbA1c values were not included in the material supplied.

**Transthoracic echocardiography**

Transthoracic echocardiography demonstrated:

- Non-dilated left ventricle
- Mildly impaired LV systolic function
- Estimated LVEF 40–45%
- Regional wall-motion abnormalities
- Impaired diastolic function with normal filling pressures
- Normal right ventricular size and systolic function
- No significant valvular disease

The reduction in LVEF placed the patient within the range generally described as heart failure with mildly reduced ejection fraction (HFmrEF) when interpreted in the appropriate clinical context. Contemporary heart-failure guidance recognizes an LVEF of 41–49% as HFmrEF and supports evidence-based pharmacological management in appropriate patients.

**CT coronary angiography**

CT coronary angiography demonstrated significant coronary artery disease predominantly involving the circumflex distribution.

The report documented:

- Significant motion artefact affecting the circumflex artery
- Likely 70–90% stenosis involving the mid circumflex/obtuse marginal segment
- Non-obstructive disease in the LAD and RCA
- Coronary artery calcium score: 350.4

The report recommended consideration of early catheter angiography, particularly given the strong clinical suspicion of coronary artery disease.

A separate report note dated 26 June 2026 described a high suspicion of severe proximal circumflex disease in the presence of artefact. Because the CT report was technically limited by motion artefact, the precise anatomical location of the most severe circumflex stenosis should ideally be confirmed from invasive coronary angiography if that investigation was subsequently undertaken.

**Cardiac magnetic resonance imaging**

CMR provided detailed tissue characterization and demonstrated:

- Non-dilated left ventricle
- Regional akinesia involving the basal-to-apical lateral segments
- Extensive myocardial oedema
- Recent transmural myocardial infarction involving the conventional circumflex coronary territory
- Late gadolinium enhancement within the affected region
- No evidence of arrhythmogenic right ventricular cardiomyopathy or arrhythmogenic left ventricular cardiomyopathy
- No significant pericardial effusion

These findings were consistent with a recent transmural myocardial infarction involving the circumflex coronary territory.

**Diagnostic Interpretation**

The overall findings were consistent with an acute/recent transmural myocardial infarction involving the circumflex coronary territory, associated with significant circumflex coronary artery disease and mildly impaired LV systolic function.

The diagnosis was supported by the convergence of:

- Markedly elevated cardiac troponin
- Regional LV systolic dysfunction
- Regional wall-motion abnormalities
- CT evidence of severe circumflex coronary stenosis

- CMR evidence of extensive myocardial oedema
- Transmural late gadolinium enhancement in the circumflex distribution
- Regional akinesia corresponding anatomically to the infarct territory
- Normal thyroid function testing, making thyroid dysfunction an unlikely explanation for the persistent sinus tachycardia

The combination of biochemical evidence, coronary anatomical findings, ventricular dysfunction, and characteristic CMR tissue findings supported the diagnosis of acute/recent circumflex-territory transmural myocardial infarction.

**Therapeutic Intervention**

The patient was initially managed as a case of acute coronary syndrome following identification of marked troponin elevation.

Because persistent tachycardia was demonstrated to be sinus tachycardia rather than a supraventricular tachycardia, bisoprolol was initiated and resulted in good heart-rate control.

Following cardiology assessment, the patient commenced on guideline-directed medical therapy for acute coronary syndrome and heart failure with mildly reduced ejection fraction.

The exact medications, doses, and duration were not available in the supplied documentation and should be inserted into the final manuscript from the discharge prescription. This is important for publication because CARE guidance recommends reporting the type, dose, duration, and changes in therapeutic interventions.

Contemporary ACS guidance emphasizes evidence-based antithrombotic therapy, high-intensity lipid lowering, appropriate revascularization assessment, and secondary prevention following ACS.

The 2022 AHA/ACC/HFSA heart-failure guideline supports evidence-based therapy for patients with HFmrEF, including a recommendation for SGLT2 inhibitor therapy and consideration of other guideline-directed therapies in appropriate patients.

**Follow-up and Outcome**

The patient remained under cardiology follow-up following hospital discharge.

In view of the extensive myocardial oedema, transmural infarction, and mildly impaired LV systolic function, the cardiology team recommended repeat CMR approximately six weeks after discharge.

The purpose of repeating CMR was to:

1. Reassess LV systolic function
2. Evaluate recovery of regional wall motion
3. Assess the evolution of myocardial oedema
4. Reassess infarct characteristics and myocardial viability

The six-week CMR result was not included in the available records and should be added when available. This follow-up is particularly relevant because myocardial oedema and infarct characteristics can evolve substantially over time, and serial CMR may demonstrate changes in myocardial function and tissue characteristics.

**Timeline**

Table 2. Clinical timeline

| Time point | Clinical event |
|---|---|
| Several weeks before hospital assessment | Persistent insomnia developed |
| General practitioner assessment | No chest pain, dyspnea, palpitations, syncope, or constitutional symptoms; persistent tachycardia identified |
| Emergency department assessment | Persistent tachycardia investigated; cardiac troponin markedly elevated at 2,285 |
| Early hospital course | Managed as acute coronary syndrome |
| During assessment of tachycardia | Adenosine administered diagnostically; rhythm characterized as sinus tachycardia |
| Subsequent management | Bisoprolol commenced with good rate control |
| Thyroid assessment | Thyroid profile within normal limits |
| Echocardiographic assessment | LVEF 40–45%, regional wall-motion abnormalities, impaired diastolic function |
| CT coronary angiography | Severe suspected circumflex disease; likely 70–90% mid-Cx/OM stenosis; calcium score 350.4 |
| CMR | Recent transmural circumflex-territory MI, extensive oedema and regional akinesia |

| | |
|---|---|
| During admission | New diagnosis of type 2 diabetes mellitus established |
| Discharge | Guideline-directed therapy for ACS and HFmrEF; outpatient cardiology follow-up |
| Six weeks after discharge | Repeat CMR planned to reassess LV function and myocardial viability |

## DISCUSSION

This case illustrates an important diagnostic challenge: acute myocardial infarction may occur without the classical presentation of chest pain. The patient initially presented with several weeks of persistent insomnia, which represented the atypical presenting symptom. Persistent sinus tachycardia was subsequently identified as an objective clinical sign and helped trigger further cardiovascular evaluation. The absence of chest pain did not exclude significant myocardial injury, particularly given his cardiovascular risk profile [7].

The patient had several important risk factors for coronary artery disease, including active smoking and a strong family history of premature coronary artery disease. His father had required coronary artery bypass surgery at 50 years of age. In addition, previously unrecognized type 2 diabetes mellitus was diagnosed during the admission, providing an additional cardiovascular risk factor [8].

The persistent tachycardia initially prompted consideration of a supraventricular tachyarrhythmia. Diagnostic administration of adenosine did not result in termination of the tachycardia, and the rhythm was characterized as sinus tachycardia. Importantly, thyroid function testing was within normal limits, reducing the likelihood of hyperthyroidism or other thyroid dysfunction as a reversible cause of persistent tachycardia [9].

In retrospect, the persistent sinus tachycardia was an important objective clinical sign accompanying an otherwise clinically subtle presentation in which insomnia was the atypical presenting symptom of acute myocardial injury. The diagnosis was subsequently strengthened by multimodality cardiac imaging. Echocardiography demonstrated a regional wall-motion abnormality and LVEF of 40–45%, indicating impaired LV systolic function. CT coronary angiography identified likely severe circumflex/obtuse marginal disease, although interpretation was limited by motion artefact. Importantly, the distribution of coronary disease corresponded with the CMR findings [10].

CMR was particularly informative. The combination of regional akinesia, extensive myocardial oedema, and transmural late gadolinium enhancement within the circumflex distribution strongly supported a recent transmural infarction. CMR can provide detailed information concerning infarct location, transmural extent, oedema, and myocardial viability, making it an important modality for myocardial tissue characterization after AMI [11, 12].

### Clinical implications of the atypical presentation

The absence of chest pain can create a diagnostic challenge. In this case, persistent insomnia was the atypical presenting symptom, while persistent sinus tachycardia was the clinical sign that triggered escalation of cardiovascular evaluation. This distinction is clinically important: insomnia alone is nonspecific, but when accompanied by an unexplained persistent tachycardia and cardiovascular risk factors, it should not automatically be attributed to psychological or benign causes. The objective sign of persistent tachycardia therefore provided an important reason to investigate for an underlying cardiovascular cause.

The normal thyroid profile was also clinically important because thyroid dysfunction, particularly thyrotoxicosis, is a recognized reversible cause of persistent tachycardia. In this patient, normal thyroid testing supported the search for an alternative underlying cause and contributed to the eventual identification of acute myocardial injury.

The case also demonstrates the value of integrating biochemical, anatomical, functional, and tissue-characterization data. Troponin established significant myocardial injury, echocardiography

demonstrated regional dysfunction, CT coronary angiography identified significant coronary disease, and CMR established the infarct territory and provided additional information regarding myocardial oedema and transmural infarction.

The 2025 ACC/AHA/ACEP/NAEMSP/SCAI ACS guideline emphasizes integrated assessment and evidence-based treatment of acute coronary syndromes, including pharmacological therapy, invasive assessment/revascularization when appropriate, and secondary prevention.

**Newly diagnosed diabetes mellitus**

The discovery of type 2 diabetes mellitus during admission is also clinically relevant. Diabetes is an established cardiovascular risk factor and substantially influences long-term coronary risk. In this case, it adds to the patient's already important risk profile of active smoking and strong familial predisposition.

Consequently, the case illustrates the importance of using an acute cardiovascular presentation as an opportunity to identify previously unrecognized modifiable cardiovascular risk factors and institute long-term secondary prevention.

**Strengths and Limitations**

A major strength of this case is the use of multimodality cardiovascular assessment, allowing correlation between biochemical myocardial injury, LV dysfunction, coronary anatomy, and myocardial tissue characteristics. Another strength is the atypical clinical presentation, in which persistent insomnia represented the presenting symptom rather than classical ischemic chest pain, while persistent sinus tachycardia served as an important clinical sign that led to further cardiovascular evaluation.

Several limitations should be acknowledged. First, the supplied records do not include the complete ECG findings, serial troponin trajectory, exact troponin assay and units, or the detailed metabolic profile. Second, the CT coronary angiogram was limited by motion artefact involving the circumflex artery. Third, the supplied information does not document the results of invasive coronary angiography or whether percutaneous coronary intervention was performed. Finally, the planned six-week follow-up CMR result is currently unavailable.

These limitations should be addressed by adding the relevant clinical and investigation details when they become available before journal submission.

**CONCLUSION**

This case demonstrates that significant acute myocardial infarction may present without classical ischemic symptoms and may initially manifest through an atypical symptom such as persistent insomnia. In this patient, persistent sinus tachycardia was an important objective clinical sign that prompted further cardiovascular assessment, particularly in the presence of major cardiovascular risk factors.

In this patient, elevated cardiac troponin, regional LV dysfunction, severe circumflex coronary disease, and CMR evidence of extensive oedema and transmural infarction established the diagnosis of acute circumflex-territory myocardial infarction. The normal thyroid profile helped exclude thyroid dysfunction as an alternative explanation for the persistent sinus tachycardia.

Repeat CMR is valuable for evaluating recovery of ventricular function, evolution of myocardial tissue abnormalities, and myocardial viability.

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