Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Medicine

An Incidental Diagnosis of Silent Myocardial Ischaemia: A Case Report

Dr Shabbar Asharia[1*]

[1]Consultant in Family Medicine, Primary Health Care Corporation, Doha, Qatar

DOI: https://doi.org/10.36347/sjmcr.2026.v14i01.022 | **Received:** 03.11.2025 | **Accepted:** 10.01.2026 | **Published:** 14.01.2026

***Corresponding author:** Dr Shabbar Asharia
Consultant in Family Medicine, Primary Health Care Corporation, Doha, Qatar

Abstract | **Case Report**

Background: Silent myocardial ischaemia (SMI) is a recognised but often underdiagnosed manifestation of coronary artery disease, especially in patients with diabetes mellitus. Autonomic neuropathy can blunt classical anginal symptoms, leading to delayed diagnosis and subsequent poorer outcomes. ***Case Presentation:*** We present the case of a 66-year-old man with type 2 diabetes mellitus who attended a routine primary care review complaining only of fatigue. He denied chest pain or typical anginal symptoms. Initial investigations revealed electrocardiographic changes consistent with a previous inferior myocardial infarction. Subsequent cardiology assessment confirmed a diagnosis of silent myocardial infarction secondary to underlying coronary artery disease. ***Conclusion:*** This case highlights the importance of clinical vigilance for atypical presentations of myocardial ischaemia in patients with diabetes. Early recognition through routine assessment and appropriate investigation is essential to reduce cardiovascular morbidity and mortality in this high-risk population.

Keywords: Silent myocardial ischaemia, Type 2 diabetes mellitus, Autonomic neuropathy, Inferior myocardial infarction, Cardiovascular risk, Fatigue.

INTRODUCTION

Cardiovascular disease remains the leading cause of mortality among individuals with diabetes mellitus, accounting for a substantial proportion of both macrovascular and microvascular complications (Dal Canto *et al.*, 2019). Patients with diabetes are not only at increased risk of developing coronary artery disease but also experience significantly worse outcomes following myocardial infarction compared with non-diabetic individuals matched for age and sex (Lee *et al.*, 2012).

Myocardial ischaemia classically presents with chest pain or angina; however, in patients with diabetes, these symptoms may be absent or markedly reduced. Silent myocardial ischaemia refers to objective evidence of myocardial ischaemia or infarction occurring without typical anginal symptoms (Cohn, 1983). Epidemiological studies suggest that silent myocardial infarction is common in this population, with the PRISM study estimating a prevalence of approximately 23% among patients with type 2 diabetes mellitus (Prasad *et al.*, 2020).

This case report describes a patient with type 2 diabetes who presented with non-specific symptoms and was subsequently diagnosed with a silent inferior myocardial infarction, illustrating the diagnostic challenges and clinical significance of silent myocardial ischaemia.

CASE PRESENTATION

A 66-year-old man attended his general practitioner for a routine diabetes review, reporting increased fatigue over the preceding two months. He denied chest pain, jaw, neck or shoulder discomfort, palpitations, syncope, or dyspnoea beyond his usual baseline. There were no systemic red-flag symptoms such as weight loss, fever, or night sweats.

His past medical history included type 2 diabetes mellitus, hypertension, and hypercholesterolaemia. He was an ex-smoker with a 20 pack-year history. Current medications included ramipril 10 mg once daily, atorvastatin 20 mg once daily, and metformin 1 g twice daily.

On examination, his blood pressure was 127/74 mmHg with a regular pulse of 74 beats per minute. Oxygen saturations were 98% on room air, and respiratory rate was 14 breaths per minute. Cardiovascular and respiratory examinations were

unremarkable. Random blood glucose was 8.1 mmol/L, and urinalysis was normal.

Initial investigations were arranged, including blood tests, a chest radiograph, and a 12-lead electrocardiogram (ECG). The chest radiograph was normal. ECG demonstrated new deep Q waves in leads II, III, and aVF, suggestive of a previous inferior myocardial infarction, with no acute ST-segment changes.

Laboratory investigations showed a haemoglobin level of 135 g/L, HbA1c of 58 mmol/mol, non-HDL cholesterol of 3.8 mmol/L, and an estimated glomerular filtration rate of 78 mL/min/1.73 m².

The patient was urgently referred to cardiology. Further evaluation with echocardiography and coronary angiography confirmed a diagnosis of silent inferior myocardial infarction in the context of underlying coronary artery disease. He was commenced on optimised secondary prevention therapy and enrolled in cardiology follow-up.

DISCUSSION

Silent myocardial ischaemia is a clinically important entity, particularly in patients with diabetes, in whom cardiovascular disease remains the principal cause of death (Dal Canto *et al.*, 2019). The absence of chest pain or classical anginal symptoms often leads to delayed diagnosis, contributing to poorer prognostic outcomes compared with symptomatic myocardial infarction (Lee *et al.*, 2012).

The pathophysiology of silent myocardial ischaemia in diabetes is multifactorial. Myocardial ischaemia typically activates cardiac afferent nerve fibres through mechanical and chemical stimuli, resulting in pain perception mediated by the autonomic nervous system. In patients with chronic hyperglycaemia, damage to autonomic nerve fibres leads to diabetic autonomic neuropathy, which diminishes pain perception and alters cardiovascular reflexes (Shakespeare *et al.*, 1994; Vinik and Ziegler, 2001). Additionally, reduced catecholamine receptor sensitivity may blunt classical autonomic responses such as tachycardia, sweating, and palpitations.

Silent ischaemia may therefore present with non-specific symptoms including fatigue, exertional dyspnoea, dizziness, nausea, or palpitations. In some cases, as illustrated here, it may be detected incidentally through routine ECG or imaging performed for unrelated reasons (Draman *et al.*, 2013).

Current guidelines do not recommend routine screening for myocardial ischaemia in asymptomatic patients with type 2 diabetes, nor the use of antiplatelet therapy for primary prevention (NICE, 2021a). Nevertheless, clinicians play a crucial role in recognising subtle symptoms and aggressively addressing modifiable cardiovascular risk factors. This includes smoking cessation, weight management, blood pressure control with a target below 140/90 mmHg, and optimisation of lipid profiles through statin therapy (NICE, 2021b; NICE, 2021c).

Glycaemic control should be individualised, with consideration given to therapies that confer cardiovascular benefit. Sodium–glucose co-transporter 2 (SGLT2) inhibitors have demonstrated cardiovascular risk reduction in patients with type 2 diabetes and established atherosclerotic disease or elevated cardiovascular risk (NICE, 2023).

Learning Points

- Silent myocardial ischaemia is common in patients with type 2 diabetes and may present without chest pain.
- Non-specific symptoms such as exertional fatigue should prompt consideration of underlying cardiac pathology in high-risk patients.
- Routine ECGs during chronic disease reviews can aid early detection of silent myocardial infarction.
- Aggressive cardiovascular risk factor modification remains central to prevention and management.

CONCLUSION

Silent myocardial ischaemia represents a diagnostic challenge due to its atypical or absent symptomatology, particularly in patients with diabetes mellitus. This case demonstrates how routine assessment and a low threshold for investigation can uncover significant underlying coronary artery disease. Increased clinical awareness and proactive cardiovascular risk management are essential to improving outcomes in this high-risk population.

REFERENCES

- Cohn, P.F. (1983) 'Silent myocardial ischemia', *Annals of Internal Medicine*, 98(3), pp. 406–412.
- Dal Canto, E., Ceriello, A., Rydén, L. *et al.* (2019) 'Diabetes as a cardiovascular risk factor: An overview of global trends of macro and microvascular complications', *European Journal of Preventive Cardiology*, 26(2_suppl), pp. 25–32.
- Draman, M.S., Thabit, H., Kiernan, T.J., O'Neill, J., Sreenan, S. and McDermott, J.H. (2013) 'A silent myocardial infarction in the diabetes outpatient clinic: Case report and review of the literature', *Endocrinology, Diabetes & Metabolism Case Reports*, 2013, 130058.
- Lee, J.H., Park, J.S., Kim, Y.H. *et al.* (2012) 'Prognostic impact of diabetes mellitus and hypertension for mid-term outcome of patients with

acute myocardial infarction who underwent percutaneous coronary intervention', *Journal of Cardiology*, 60(4), pp. 257–263.

- NICE (2021a) *Primary prevention of cardiovascular disease (NG238)*. London: National Institute for Health and Care Excellence.
- NICE (2021b) *Hypertension in adults: Diagnosis and management (NG136)*. London: National Institute for Health and Care Excellence.
- NICE (2021c) *cardiovascular disease: Risk assessment and reduction, including lipid modification (NG238)*. London: National Institute for Health and Care Excellence.
- NICE (2023) *Type 2 diabetes in adults: Quality standard (QS209)*. London: National Institute for Health and Care Excellence.
- Prasad, D.S., Kabir, Z., Devi, K.R., Peter, P.S. and Das, B.C. (2020) 'Prevalence and risk factors for silent myocardial ischemia in asymptomatic type 2 diabetes mellitus patients: The PRISM study', *Indian Heart Journal*, 72(1), pp. 27–31.
- Shakespeare, C.F., Katritsis, D., Crowther, A., Cooper, I.C., Coltart, J.D. and Webb-Peploe, M.W. (1994) 'Differences in autonomic nerve function in patients with silent and symptomatic myocardial ischaemia', *British Heart Journal*, 71(1), pp. 22–29.
- Vinik, A.I. and Ziegler, D. (2001) 'Recognizing and treating diabetic autonomic neuropathy', *Cleveland Clinic Journal of Medicine*, 68(11), pp. 928–944.