

# Postprandial hyperglycaemia and continuous glucose monitoring: a personal journey

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## Abstract

This article outlines my journey through the diagnosis and management of postprandial hyperglycaemia, highlighting the challenges, interventions, and insights gleaned along the way

**Key words:** postprandial hyperglycaemia, continuous glucose monitoring, post-prandial walk

I am a quinquagenarian cardiologist of Indian descent and have a family history of type 2 diabetes (T2DM). In 2018, my HbA<sub>1c</sub> of 43 mmol/mol indicated prediabetes, despite a normal fasting blood glucose (Figure 1). In 2020, an oral glucose tolerance test (OGT) unexpectedly revealed post-load 2-hour glucose of 14 mmol/L, leading to a formal diagnosis of diabetes.

At the time, I implemented lifestyle changes, including modest weight loss (my BMI was 24.5 kg/m<sup>2</sup>) and reduced carbohydrate consumption. Over the next two years, my fasting blood sugar remained normal and my HbA<sub>1c</sub> hovered in the prediabetic range. No further OGTs were performed, and no antidiabetic medication was prescribed, as my HbA<sub>1c</sub> remained well below the UK threshold of 48 mmol/mol for prescribing medication. My isolated postprandial hyperglycaemia did not prompt specific concern.

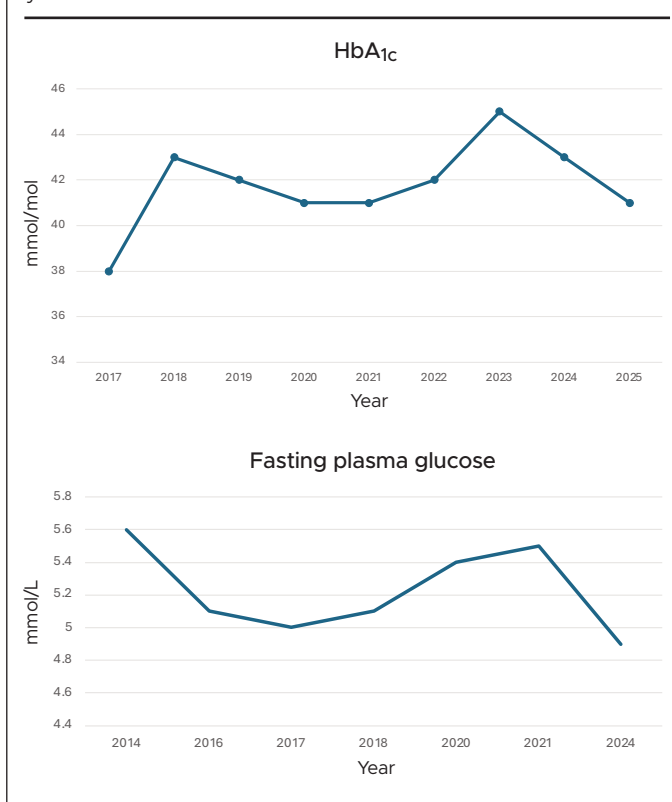
By 2023, I became more aware of the potential of continuous glucose monitoring (CGM) and began using a Freestyle Libre sensor. The data were eye-opening: my postprandial glucose frequently reached 14 mmol/L at 1 hour and 12–13 mmol/L at 2 hours after meals, even with reduced carbohydrate intake.

I found no specific guidelines for treating isolated postprandial hyperglycaemia in the presence of normal fasting glucose and a prediabetic HbA<sub>1c</sub>. However, in the literature there was evidence linking postprandial hyperglycaemia to both microvascular and macrovascular complications.<sup>1</sup>

Determined to address the issue, I lost 5–7% of my body weight and committed to walking 10,000 steps daily. This mildly improved my HbA<sub>1c</sub>, but not my postprandial glucose, which remained stubbornly high on CGM. I began exploring other evidence-based options, including metformin, acarbose and postprandial walking.

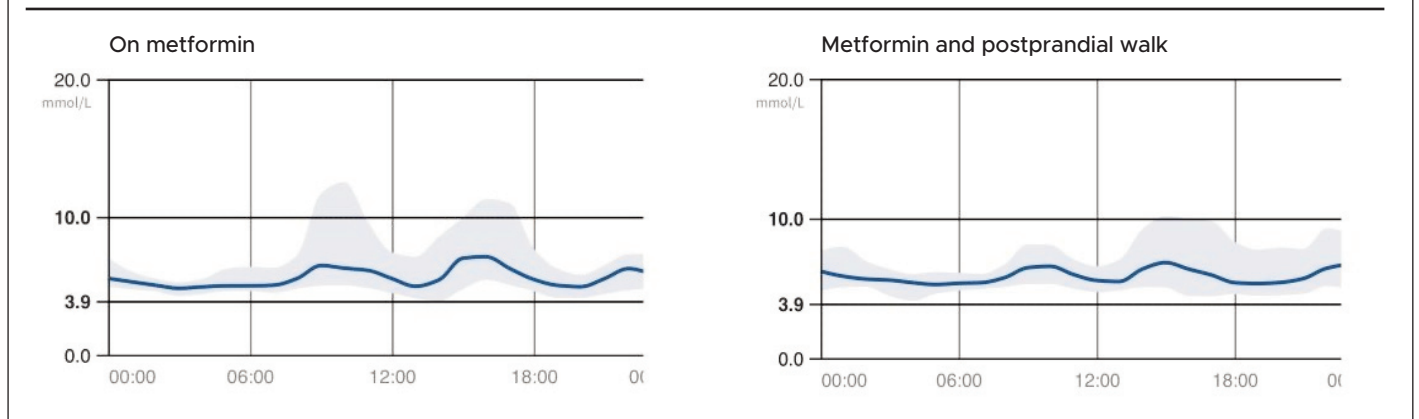
With my GP's agreement, I started metformin, discovering that it was most effective when taken at least an hour before

**Figure 1.** HbA<sub>1c</sub> and fasting blood glucose (FBG) over the years



meals. I incorporated a 20–30 minute walk at my work following lunch and a similar walk after my evening meal. CGM data confirmed the profound impact of the postprandial walk as exercise-induced glucose uptake by muscle tissue operates independently of insulin. On days when walking was not

**Figure 2.** Average of daily graphs of glucose through LibreLink. Blue line = average blood sugar; Light blue shadow = glucose range. Taken from my account LibreView. Postprandial walk reduces the peak after meal



possible (such as during days in the cath lab), I minimised lunchtime carbohydrate intake. During a visit to India to see my family, I trialled acarbose (available over the counter) with meals, finding it also helpful for postprandial glucose. I never combined acarbose and metformin. Smaller, more frequent meals (six per day) helped, though this was challenging given my professional commitments. Adding an SGLT2 inhibitor to metformin did not further reduce postprandial glucose. Throughout, CGM allowed me to observe the effect of each intervention in real time.

Currently, I take metformin twice daily, one hour before main meals, and continue my postprandial walking routine after lunch at my work. I also walk after my evening meal, tailoring the duration to my CGM readings.

This approach has allowed me to keep my postprandial glucose consistently below 10 mmol/L, and mostly below 9 mmol/L (Figure 2). My HbA<sub>1c</sub> is now 41 mmol/mol, with further improvement anticipated as postprandial glucose control continues. CGM-calculated HbA<sub>1c</sub> also shows encouraging trends.

From my personal journey with postprandial hyperglycaemia, I can perhaps conclude the following:

- Without more frequent or routine OGTs, significant postprandial hyperglycaemia and early diabetes may go undetected, particularly when fasting glucose and HbA<sub>1c</sub> are normal or only mildly elevated. My own diagnosis was delayed as a result.

- Continuous glucose monitoring (CGM) offers a much richer understanding of individual glucose patterns and the effects of interventions than traditional fasting blood sugar or HbA<sub>1c</sub>. CGM data also provide additional metrics such as time in range and average glucose. Their long-term significance is still being studied but may better predict complications.
- Isolated postprandial hyperglycaemia, especially in the early stages of diabetes, is an under-recognised phenomenon. More research is needed to define its importance and the most effective strategies for its management, as well as to understand the long-term impact of controlling postprandial glucose on diabetes complications.



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