LETTER TO THE EDITOR

The sarcopenic trap: New-onset diabetes despite massive weight loss after sleeve gastrectomy

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To the Editor,

Laparoscopic Sleeve Gastrectomy (LSG) is widely recognized for its efficacy in inducing weight loss and resolving Type 2 Diabetes Mellitus (T2DM) (1). However, we report a paradoxical case of new-onset (de novo) T2DM complicated by severe glucotoxicity and sarcopenia, occurring late after successful LSG. A 42-year-old female patient underwent LSG for severe obesity. Pre-operatively, her weight was 113 kg (BMI 41.5 kg/m²). She was metabolically healthy with no history of diabetes (Fasting Glucose 5.0 mmol/L). The post-operative course was initially uneventful with significant weight loss. However, 15 months post-surgery (weight 82 kg), she presented with severe hyperglycemia symptoms. Clinical characteristics and laboratory data are summarized in Table 1. Initial investigations revealed frank diabetes: Fasting Glucose 15.7 mmol/L and HbA1c 9.3% (78 mmol/mol). Initial workup indicated suppressed beta-cell function due to glucotoxicity (C-peptide 0.56 nmol/L), Insulin 33.8 pmol/L).

We initiated intensive insulin therapy (Basal-Bolus) for 5 months to resolve glucotoxicity and preserve beta-cell mass. Currently, 20 months post-surgery, the patient's weight has further decreased to 75 kg (BMI 27.6 kg/m²; Total Weight Loss 38 kg). This represents an excellent total weight loss outcome. After a 3-day insulin washout period, re-evaluation showed significant recovery of beta-cell secretory function: C-peptide increased to 0.83 nmol/L, and fasting insulin rose to 99.6 pmol/L. Initially, Latent Autoimmune Diabetes in Adults (LADA) was suspected due to the rapid onset in a non-obesity phenotype, as described in similar post-bariatric cases (2). However, the significant recovery of C-peptide levels and insulin secretion after glucotoxicity resolution clinically ruled out the progressive beta-cell destruction typical of autoimmune diabetes. Despite this recovery, hyperglycemia persisted (Glucose 10.0 mmol/L, HbA1c 7.4% (57 mmol/mol)), pointing towards severe peripheral insulin resistance. Further investigation identified Sarcopenic Obesity as the underlying cause. Serum creatinine remained persistently low (58 µmol/L), reflecting skeletal muscle depletion (3). Evaluation according to AWGS 2019 criteria confirmed sarcopenia: Calf circumference 32 cm, Handgrip strength 16 kg,

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Table 1. Timeline of clinical and metabolic parameters: comparison between pre-operative, 15-month, and 20-month follow-up.

Parameter	Pre-Op (Baseline)	15 Months Post-Op (Crisis)	20 Months Post-Op (Current)	Interpretation
Weight (kg)	113	82	75	Continued Weight Loss (-38kg)
BMI (kg/m²)	41.5	32.0	27.6	Severe obesity → Overweight
Glucose (mmol/L)	5.0	15.7	10.0	Glucotoxicity → Persistent Diabetes
HbA1c (NGSP %/IFCC mmol/mol)	<6.0(<42)	9.3 (78)	7.4(57)	Improved with therapy, not cured
Insulin (pmol/L)	-	33.8	99.6	Proof of Beta-cell Recovery
C-Peptide (nmol/L)	-	0.56	0.83	Rules out LADA
Creatinine (µmol/L)	Normal	58	58	Marker of Muscle Depletion
Calf Circ. (cm)	-	-	32	Confirmed Sarcopenia

Abbreviations: BMI=Body Mass Index, LSG=Laparoscopic Sleeve Gastrectomy, HbA1c = Glycated Hemoglobin, LADA = Latent Autoimmune Diabetes in Adults, AWGS = Asian Working Group for Sarcopenia.

and five-time chair stand test 12 s (4,5). We hypothesize that the rapid post-surgical weight loss involved disproportionate catabolism of skeletal muscle tissue. Since skeletal muscle is the primary site for insulin-stimulated glucose disposal, its severe depletion likely induced insulin resistance, leading to hyperglycemia even in the absence of massive adipose tissue (6). We conclude that while insulin therapy successfully reversed the glucotoxicity, the patient's underlying sarcopenia-induced insulin resistance prevents the restoration of euglycemia. This case highlights that massive weight loss can be metabolically detrimental if accompanied by severe muscle wasting.

Ethical Approval: All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments. This study was approved by the Ethics Committee of Tashkent State Medical University (Protocol No:11/25, Date: 18.11.2025, Approval duration: 2025-2026).

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Authors Contribution: FB and OT conceived and designed the study. FB and YN acquired the data and drafted the manuscript. OT supervised and critically revised the project. FB and YN reviewed the pertinent literature. YN and OT analyzed and validated the data. All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work.

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