


Persistent Hypoglycemia in Critically Ill Non-Diabetic Patients: Uncovering Non-Insulin-Related Causes — A Case Report

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ABSTRACT

Introduction: Hypoglycemia is uncommon in patients without diabetes mellitus, yet critical illness can precipitate it through impaired gluconeogenesis and depletion of hepatic glycogen stores. When this occurs in the absence of insulin therapy or a diabetic history, identifying the underlying cause becomes a diagnostic challenge. **Case:** A 56-year-old male presented with decreased consciousness secondary to severe hypoglycemia (glucose 42 mg/dL). He had a history of suspected metastatic stage IV prostate cancer with a markedly elevated *prostate-specific antigen* (PSA) of 930.8 ng/mL, severe anemia (Hb 4.9 g/dL), transaminitis (*aspartate aminotransferase*/AST 108 U/L), and a normal HbA1c of 4.7% with no prior history of diabetes mellitus. During hospitalization, he was additionally diagnosed with complicated urinary tract infection and hospital-acquired pneumonia. By day three, persistent recurrent hypoglycemia developed despite glucose supplementation. On day five, the patient deteriorated into type 2 respiratory failure requiring ICU admission, and subsequently died. **Discussion:** Persistent hypoglycemia in this patient was likely multifactorial, involving tumor-related mechanisms, critical illness with concurrent infections, hepatic dysfunction, and prolonged malnutrition, whereas normal HbA1c and the absence of glycosuria excluded underlying diabetes mellitus and renal glucose wasting. **Conclusion:** This case underscores that persistent hypoglycemia in critically ill non-diabetic patients is a clinically important and often diagnostically elusive condition. A multifactorial approach encompassing malignancy-related mechanisms, hepatic dysfunction, sepsis, and nutritional depletion is essential for timely recognition and management.

Keywords: Case report, critical illness, hypoglycemia, non-insulin hypoglycaemia.

ABSTRAK

Pendahuluan: Hipoglikemia jarang terjadi pada pasien tanpa riwayat diabetes melitus, namun kondisi kritis dapat memicunya melalui gangguan glukoneogenesis dan deplesi cadangan glikogen di hati. Ketika hipoglikemia terjadi tanpa riwayat terapi *insulin* ataupun diabetes, identifikasi penyebab yang mendasarinya menjadi tantangan diagnostik tersendiri. **Kasus:** Seorang laki-laki berusia 56 tahun datang dengan penurunan kesadaran yang disebabkan oleh hipoglikemia berat (glukosa 42 mg/dL). Pasien memiliki riwayat kanker prostat stadium IV yang dicurigai bermetastasis dengan PSA yang meningkat secara signifikan sebesar 930,8 ng/mL, anemia berat (Hb 4,9 g/dL), transaminitis (AST 108 U/L), dan HbA1c normal 4,7% tanpa riwayat diabetes melitus. Selama perawatan, pasien juga didiagnosis dengan infeksi saluran kemih berkomplikasi dan pneumonia didapat di rumah sakit (*hospital-acquired pneumonia*). Pada hari ketiga, hipoglikemia yang persisten berulang muncul meskipun telah diberikan suplementasi glukosa. Pada hari kelima, kondisi pasien memburuk menjadi gagal napas tipe 2 yang memerlukan perawatan di ICU, dan pasien kemudian meninggal dunia. **Pembahasan:** Hipoglikemia persisten pada pasien ini kemungkinan disebabkan oleh berbagai faktor, yang melibatkan mekanisme terkait tumor, kondisi kritis disertai infeksi, disfungsi hati, dan malnutrisi berkepanjangan, sedangkan nilai HbA1c yang normal dan tidak adanya glikosuria menyingkirkan kemungkinan adanya diabetes melitus dan kehilangan glukosa melalui ginjal sebagai penyebab mendasar. **Simpulan:** Laporan kasus ini menegaskan bahwa hipoglikemia persisten pada pasien kritis non-diabetik merupakan kondisi yang penting secara klinis namun sering luput dari perhatian diagnostik. Pendekatan multifaktorial - mencakup mekanisme terkait keganasan, disfungsi hati, sepsis, dan deplesi nutrisi - sangat diperlukan untuk pengenalan dini dan tata laksana yang tepat. **Kadek Mercu Narapati Pamungkas, I Ketut Suryana. Hipoglikemia Persisten pada Pasien dengan Penyakit Kritis: Telaah Penyebab Tak Terkait Insulin — Laporan Kasus.**

Kata Kunci: Laporan kasus, penyakit kritis, hipoglikemia, hipoglikemia non-insulin.

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INTRODUCTION

Hypoglycemia is defined as a plasma glucose concentration below 70 mg/dL, though clinical symptoms and signs typically emerge when glucose falls below 55 mg/dL.[1] In the context of diabetes mellitus, hypoglycemia is a well-recognized complication of insulin therapy: patients with type 1 diabetes mellitus experience 62–320 hypoglycemic episodes per 100 patient-years, while those with type 2 diabetes mellitus experience a comparatively lower rate of approximately 35 episodes per 100 patient-years.[2–3] Beyond insulin, other antidiabetic agents — including meglitinides and sulfonylureas — are frequent culprits, whereas metformin, glucagon-like peptide-1 (GLP-1) receptor agonists, sodium-glucose co-transporter 2 (SGLT-2) inhibitors, and dipeptidyl peptidase-4 (DPP-4) inhibitors rarely cause hypoglycemia.[1] Profound and prolonged hypoglycemia occurs in 2.7%–5% of cases following insulin overdose.[4,5]

The prognostic significance of hypoglycemia extends beyond its immediate symptoms. A meta-analysis demonstrated that even a single hypoglycemic episode is associated with a 1.4- to 3.2-fold increase in mortality risk.[6,7] In patients without a prior diagnosis of diabetes mellitus, hypoglycemia is considerably rarer but can still arise from diverse etiologies, including alcohol use, critical illness, counter-regulatory hormone deficiencies, and non-islet cell tumors.[8]

Critical illness represents a particularly complex substrate for hypoglycemia. Impaired gluconeogenesis and depletion of hepatic glycogen stores — as seen in heart failure with hepatic congestion, sepsis, renal failure, adrenal insufficiency, malnutrition, and severe infection — can disrupt glucose homeostasis even in the absence of exogenous insulin.[9,10] Severe spontaneous hypoglycemia affects fewer than 1.5% of critically ill patients overall, but is more prevalent in those with fulminant hepatic failure or adrenal insufficiency during septic shock, particularly when compounded by malnutrition, liver cirrhosis, or chronic kidney disease.[4,11,12]

Despite increasing awareness of glucose dysregulation in the ICU setting, hypoglycemia in non-diabetic critically ill patients remains underreported and poorly characterized. The novelty of this case lies in its demonstration of persistent, recurrent hypoglycemia in a patient with suspected metastatic prostate cancer, multiorgan stress, and no history of diabetes mellitus — highlighting the interplay of tumor-related mechanisms, hepatic dysfunction, severe infection, and nutritional depletion as converging drivers of refractory hypoglycemia. This report aims to expand the clinical recognition of non-insulin-related hypoglycemia in critically ill patients and to prompt a systematic diagnostic approach when glucose fails to stabilize.

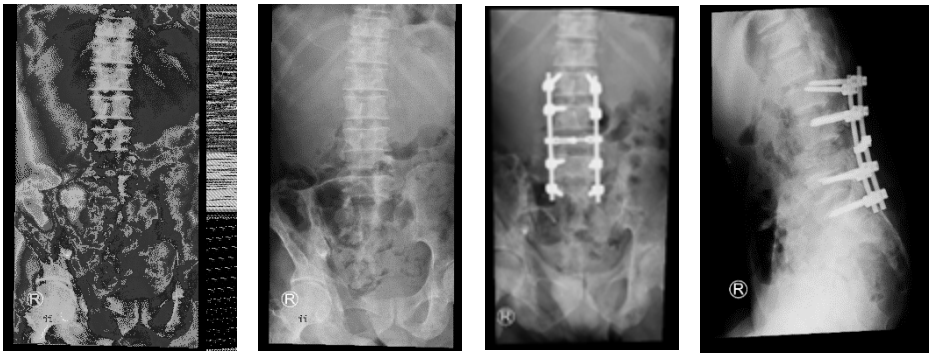
CASE

A 56-year-old male was brought to the emergency department with complaints of generalized weakness and decreased consciousness, with an initial Glasgow Coma Scale (GCS) score of E3V4M5. Vital signs on arrival were within normal limits. Point-of-care glucose testing revealed severe hypoglycemia at 42 mg/dL (reference range: 80–199 mg/dL). An immediate bolus of 50 mL of 40% dextrose (D40%) was administered; repeat glucose measurement 15 minutes later showed recovery to 154 mg/dL, with concurrent improvement in consciousness to a GCS of E4V5M6.

The patient reported persistent bilateral lower limb weakness lasting approximately two weeks prior to admission. This was in the context of a prior surgical history: he had undergone posterior decompression, stabilization-fusion, biopsy, and culture at the L4 vertebral level (**Figure 1a and b**) for a pathological fracture secondary to metastatic bone disease (MBD) (**Figure 2**), as well as a transrectal prostate biopsy for suspected stage IVB prostate cancer (cT2N1M1) on May 27, 2024. Biopsy results revealed metastatic carcinoma of unknown primary origin. Following disclosure of the biopsy findings, the patient and his family declined further surgical intervention and additional tissue sampling. The clinical suspicion for prostate cancer was reinforced by a markedly elevated prostate-specific antigen (PSA) of 930.8 ng/mL (reference range: < 4 ng/mL). Lumbar MRI demonstrated canal stenosis from a soft tissue mass and a pathological fracture at the L4 vertebral body (**Figure 1**). The patient had been bedridden for approximately three months following spinal surgery at Prof. IGNG Ngoerah Hospital, Denpasar, and reported significantly reduced appetite for the preceding month.

On admission, laboratory investigations revealed severe anemia (Hemoglobin [Hb] 4.9 g/dL), transaminitis (AST/SGOT 108 U/L, ALT/SGPT 37 U/L), blood urea nitrogen (BUN) 37 mg/dL, and serum creatinine (SCr) 0.4 mg/dL. HbA1c was 4.7% (reference range: 4.4%–5.4%), and the patient had no history of diabetes mellitus. A packed red cell (PRC) transfusion of one unit (320 mL) daily was planned over four consecutive days. Post-transfusion complete blood count (CBC) showed leukocytosis (10.19 × 10^3/µL), Hb 9.9 g/dL, neutrophil predominance at 87.9%, and a neutrophil-to-lymphocyte ratio (NLR) of 8.87 (normal < 3.13). The initial diagnoses were severe hypoglycemia and severe anemia.

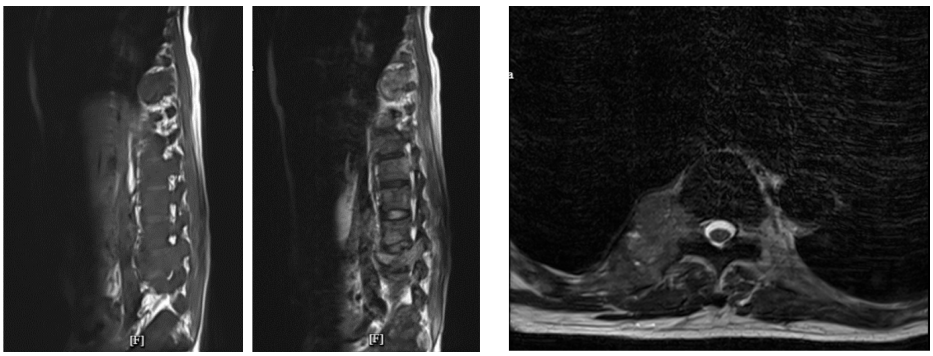
On day three of hospitalization, the patient developed hospital-acquired pneumonia (HAP) based on clinical and radiological findings. More critically, recurrent hypoglycemia emerged with three distinct episodes within a single day. The first episode was detected as 'Low' on point-of-care glucometry; a 50 mL D40% bolus was administered, raising glucose to 254 mg/dL. Five hours later, the patient again registered 'Low' glucose; a repeat 50 mL D40% bolus improved levels to 184 mg/dL at 15 minutes. Later that evening at 19:20, a third hypoglycemic episode occurred — glucose 81 mg/dL with associated altered consciousness — resolving to 171 mg/dL after another D40% bolus. Maintenance therapy was initiated with three scheduled D40% boluses of 50 mL per day, along with daily glucose monitoring. Urinalysis performed at this time revealed a cloudy appearance, leukocyte esterase 75 Leu/µL, nitrite positive, protein 2+, blood 2+, erythrocyte sediment 7–9/HPF (normal: 0–2), leukocytes 15–17/HPF (normal: 0–1), coarse granular casts 1/µL, squamous epithelial cells 5–7/HPF, and bacteria positive — consistent with complicated urinary tract infection (UTI). The absence of glycosuria on urinalysis effectively excluded renal glucose wasting as a contributing cause of hypoglycemia. The diagnoses were updated to include complicated UTI in addition to persistent hypoglycemia and HAP.



(a) (b)

*Photo documentation by Kadek Mercu Narapati Pamungkas.

Figure 1. a. X-ray lumbosacral AP/lateral (May 2024) and b. X-ray lumbosacral AP/lateral post-operation (July 2024).



(a) (b)

*Photo documentation by Kadek Mercu Narapati Pamungkas.

Figure 2. Non-contrast MRI lumbosacral. a. sagittal T1, b. sagittal T2, and c. axial T2. Showing the compression fracture of L4 and paravertebral soft tissue mass on posterior aspect of CV Th9-10 and soft tissue mass on posterior aspect of CV L4.

By day five, the patient's clinical condition deteriorated significantly. He presented with decreased consciousness, oxygen saturation of 89% on low-flow supplemental oxygen, and hypotension with blood pressure of 70/50 mmHg. Emergency fluid resuscitation was initiated with a 200 mL 0.9% NaCl bolus, and high-flow oxygen was applied via non-rebreather mask (NRM) at 15 L/min. Arterial blood gas (ABG) analysis confirmed type 2 respiratory failure: pH 7.16 (reference 7.35–7.45), pCO_2 65 mmHg (reference 35–45), pO_2 149 mmHg, $cHCO_3$ 23 mmol/L, actual base excess (ABE) -6 (reference -2 to +2), standard bicarbonate (SBC) 25 mmol/L, and SpO_2 99%. This pattern was consistent with uncompensated mixed respiratory and metabolic acidosis with severe hypoxemia. A pulmonology specialist was consulted and recommended ICU admission; antibiotic therapy was escalated to meropenem 3 × 1 g IV and levofloxacin 750 mg IV once daily. Additional supportive treatment included N-acetylcysteine 200 mg IV once daily and nebulized ipratropium bromide/salbutamol (0.52 mg/3.0 mg) three times daily. Despite vasopressor support with norepinephrine (titrated from 0.05 mcg/kg/min), hemodynamic status remained refractory. The clinical picture was consistent with septic shock as the terminal driver of multiorgan dysfunction. The patient was declared deceased on the fifth day of treatment.

DISCUSSION

This case presents a 56-year-old male with suspected stage IVB prostate cancer (cT2N1M1), a PSA of 930.8 ng/mL (reference: < 4 ng/mL), vertebral metastasis at L4, and no history of diabetes mellitus (HbA1c 4.7%), who developed persistent and recurrent hypoglycemia in the context of critical illness. The case illustrates the diagnostic complexity of non-insulin-related hypoglycemia when multiple mechanisms converge simultaneously.

Hypoglycemia in non-diabetic patients — often termed non-diabetic hypoglycemia — encompasses a spectrum of etiologies. The most well-characterized endogenous cause is insulinoma, the most common pancreatic neuroendocrine tumor, with an estimated incidence of 4 cases per million annually, typically presenting with endogenous hyperinsulinemia.[13–16] Importantly, non-islet cell tumors (NICTs) can induce hypoglycemia through the secretion of insulin-like growth factor II (IGF-II), which activates insulin receptors and suppresses glucose production — a condition known as non-islet cell tumor hypoglycemia (NICTH).[16,17] In some instances, hypoglycemia may precede the diagnosis of malignancy and serve as its initial clinical manifestation.[18]

Malignancy-associated hypoglycemia is broadly categorized into four pathogenic mechanisms: (1) islet cell tumors (insulinoma); (2) non-islet cell tumors producing IGF-II (NICTH); (3) massive tumor burden or hepatic infiltration impairing gluconeogenesis; and (4) tumor-derived autoantibodies targeting insulin or the insulin receptor.[19] In addition to these humoral mechanisms, tumor-derived substances — including cytokines such as TNF-α, IL-1, and IL-6, as well as catecholamines — can directly interfere with glucose metabolism.[19] In this patient, the high tumor burden of suspected metastatic prostate cancer, combined with elevated transaminases (AST 108 U/L) suggesting hepatic involvement, may have contributed to impaired hepatic glucose output through multiple overlapping pathways.

Beyond malignancy-specific mechanisms, the patient's clinical trajectory was complicated by concurrent severe infections. Within the first two days of hospitalization, he developed clinical and radiological features of hospital-acquired pneumonia, and urinalysis confirmed complicated UTI without glycosuria — the latter effectively excluding renal glucose wasting as a contributing factor. The co-occurrence of these infections is clinically significant: the incidence of spontaneous


hypoglycemia in the general population is approximately 1,082 per 100,000 individuals, with infection accounting for 14.3% of cases, and pneumonia specifically associated in 11.2% of cases in a Japanese multicenter study.[20,21]

The critical illness state itself amplifies hypoglycemic risk through several interconnected mechanisms. Multiorgan dysfunction reduces gluconeogenic capacity in the liver and kidneys, increases tissue glucose uptake driven by inflammatory mediators, and disrupts the counter-regulatory hormonal response. Impaired adrenal and pituitary function during critical illness further diminishes the catecholamine and cortisol responses necessary to defend against falling glucose.[22] Superimposed nutritional deficits — this patient had experienced severely reduced oral intake for approximately one month and had been bedridden for three months — further depleted hepatic glycogen stores, eliminating a key short-term glucose reserve.[22]

Studies comparing spontaneous hypoglycemia to iatrogenic hypoglycemia in critically ill patients have consistently shown that spontaneous episodes carry significantly higher mortality, attributable to the underlying severity of illness, elevated inflammatory cytokines, and the absence of a readily reversible cause.[23,24] A review of emergency department data found that in non-diabetic patients presenting with hypoglycemia, the strongest predictors of mortality include advanced liver disease, active malignancy, concurrent sepsis, and the degree of hypoglycemia at presentation.[25] This patient met all four criteria simultaneously.

A comparable case reported by Gonzalez F, *et al*., described a non-diabetic male with metastatic synovial cell sarcoma of the lung who experienced persistent hypoglycemia with blood glucose levels of 20–80 mg/dL, also in the context of malignant pleural effusion and widespread metastatic disease.[26] The parallels with our case — advanced malignancy, absence of diabetes, and refractory hypoglycemia unresponsive to conventional glucose supplementation — highlight a distinct clinical phenotype that warrants systematic evaluation for tumor-mediated and critical-illness-related mechanisms.

In this patient, the likely contributors to persistent hypoglycemia included: (1) tumor-related mechanisms — direct hepatic infiltration or IGF-II–mediated insulin-receptor activation from metastatic carcinoma; (2) sepsis-driven glucose dysregulation from concurrent HAP and complicated UTI; (3) hepatic dysfunction evidenced by transaminitis; (4) severe nutritional depletion with prolonged reduced oral intake; and (5) the overall metabolic stress of critical illness with impaired counter-regulatory responses. The absence of glycosuria and a normal HbA1c reliably excluded renal wasting and underlying undiagnosed diabetes as causative factors.

CONCLUSION

This case highlights that persistent, recurrent hypoglycemia in a critically ill non-diabetic patient is a clinically underrecognized and diagnostically challenging entity. When hypoglycemia fails to resolve despite appropriate glucose supplementation, a systematic search for non-insulin-related causes is imperative — encompassing malignancy-associated mechanisms (hepatic infiltration, IGF-II production), sepsis-driven metabolic dysregulation, hepatic dysfunction, and nutritional depletion. Clinicians should recognize that the convergence of these factors, rather than any single etiology, may underlie refractory hypoglycemia in the critically ill. Early identification of the contributing mechanisms is essential to guide targeted management and may influence prognosis.

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