

Navigating the Glucose Surge: Painful Trigeminal Neuropathy and Transient Speech Disturbance in a Patient With Severe Hyperglycemia and Rapid Glycemic Correction

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Abstract

Background: Uncontrolled diabetes mellitus can lead to neurological complications such as seizures, altered mental status, and chorea, most commonly in the context of diabetic ketoacidosis (DKA) or hyperglycemic hyperosmolar state (HHS). Treatment-induced neuropathy of diabetes (TIND) is a rare and underrecognized complication associated with rapid improvement in glycemic control. It typically presents with acute-onset neuropathic pain, weakness, or autonomic dysfunction within eight weeks of glycemic improvement, and less frequently with focal or cranial neurological deficits.

Case presentation: We report a 72-year-old man with poorly controlled type 2 diabetes mellitus (T2DM) and hypertension who presented with transient expressive aphasia evolving to persistent dysarthria during severe hyperglycemia with hyperosmolality, in the absence of DKA or HHS. After rapid correction of glycemia over several days, he developed continuous left facial pain in the trigeminal distribution with focal tenderness, a presentation consistent with painful trigeminal neuropathy rather than classic trigeminal neuralgia, accompanied by transient confusion. Computed tomography (CT), magnetic resonance imaging (MRI), vascular imaging, and an extensive diagnostic workup were unremarkable, excluding cerebrovascular and structural causes. The temporal association between the facial pain and rapid glycemic correction raised TIND as a possible, atypical, cranial nerve-predominant explanation; however, this diagnosis remained presumptive, as no baseline HbA1c was available to document the glycemic trajectory required by accepted diagnostic criteria. The facial pain resolved promptly with gabapentin.

Conclusions: This case illustrates two temporally distinct neurological processes: severe hyperglycemia-associated neurological dysfunction preceding treatment and facial neuropathic pain following rapid glycemic correction, rather than a single unifying syndrome, with TIND as a possible, but not confirmed, explanation for the latter. It highlights the differential diagnosis of acute neurological events in patients with diabetes, in whom evaluation for cerebrovascular disease remains essential even when initial imaging is unremarkable, and underscores the potential importance of cautious glycemic correction and neuropathic pain-directed symptomatic management.

Categories: Neurology, Internal Medicine, Endocrinology/Diabetes/Metabolism

Keywords: diabetes treatment, dysphasia, neuropathy, tind, trigeminal neuralgia

Introduction

Diabetes mellitus is one of the most prevalent chronic diseases worldwide and is associated with a broad spectrum of neurological complications, which represent a major source of morbidity in this population. Approximately 50% of patients with diabetes develop peripheral neuropathy over the course of their disease [1]. Diabetic sensorimotor polyneuropathy (DSPN), the most common neurological manifestation, affects approximately 30% of individuals with diabetes and more than 50% over their lifetime, contributing to neuropathic pain, foot ulceration, and lower-limb amputation [2]. Beyond this classic pattern, diabetes can give rise to a range of atypical neuropathic syndromes, including cranial mononeuropathies, radiculoplexus neuropathies, and compressive neuropathies [3-5].

These chronic neuropathic syndromes must be distinguished from two acute neurological processes that occur at opposite ends of the glycemic trajectory. The first is neurological dysfunction arising during severe hyperglycemia. Acute metabolic derangements such as diabetic ketoacidosis (DKA) and the nonketotic hyperglycemic hyperosmolar state (HHS) are well-recognized causes of seizures, chorea-ballismus, hemianopia, and encephalopathy [6-8]. More strikingly, focal cortical deficits mimicking acute ischemic stroke, including dysphasia, hemiparesis, and homonymous hemianopia, have been described in severe nonketotic hyperglycemia even in the absence of a frank hyperosmolar state, and typically resolve with correction of the underlying metabolic disturbance [8-10]. Such deficits are cortical in origin, reflecting a reversible disturbance of language-dominant hemispheric function rather than a disorder of peripheral nerve; dysphasia in this setting is an acquired impairment of language, the ability to produce, understand,

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or repeat words, and not a manifestation of neuropathy. Critically, this presentation occurs while glycemia is markedly elevated and abates as it is corrected.

The second process occurs after hyperglycemia is corrected. Treatment-induced neuropathy of diabetes (TIND), historically termed "insulin neuritis," is an increasingly recognized iatrogenic complication that follows rapid improvement in glycemic control in patients with chronically elevated glycemia [11–13]. Diagnostic criteria proposed in the literature define TIND by the acute onset of neuropathic pain and/or autonomic dysfunction within eight weeks of a reduction in HbA1c of ≥ 2 percentage points over three months, after exclusion of alternative etiologies [11]. In a retrospective review of 954 patients referred to a tertiary diabetic neuropathy clinic, 10.9% met criteria for TIND, with the absolute risk exceeding 80% when HbA1c decreased by more than 4 percentage points over three months [11]. A literature review encompassing 26 studies and 181 patients confirmed that TIND most commonly presents with severe distal neuropathic pain of burning character, paresthesia, and autonomic dysfunction, with symptoms typically emerging four to eight weeks after treatment intensification [13]. Management remains largely symptomatic, relying on gabapentinoids, serotonin-norepinephrine reuptake inhibitors, tricyclic antidepressants, or opioids [12–13].

These two processes, hyperglycemia-related neurological dysfunction and TIND, are therefore distinct in timing, in the direction of glycemic change, and in the neuroanatomical structures involved. TIND, in its recognized form, is a disorder of peripheral small fibers: the most common presentation is a distal small-fiber neuropathy, and neither cortical deficits nor cranial nerve involvement have been described within its clinical spectrum. Cranial neuropathies in diabetes are instead well-documented as a consequence of longstanding uncontrolled disease, occurring independently of any treatment-related change in glycemia. Oculomotor nerve palsy is the most common cranial mononeuropathy, attributed to microvascular ischemia, and typically presents with acute pain before self-resolving over approximately six weeks [2–3]; the trigeminal and oculomotor nerves have been identified as the most frequently affected [14]. Within the trigeminal distribution, moreover, it is important to distinguish classic trigeminal neuralgia, paroxysmal, electric-shocklike pain triggered by innocuous stimuli and most often attributable to neurovascular compression, from painful trigeminal neuropathy, which produces continuous or dysesthetic pain consequent to a demonstrable lesion, in this context most plausibly microvascular or metabolic.

Cranial neuropathies and cortical deficits occurring specifically in the setting of TIND, rather than as isolated diabetic mononeuropathies or as consequences of hyperglycemia itself, remain poorly characterized. We present a case of TIND accompanied by transient aphasia and trigeminal neuropathic pain following rapid correction of chronically elevated glycemia. We do not propose that aphasia or cranial neuropathy be regarded as established manifestations of TIND; rather, their close temporal relationship to the same glycemic perturbation raises the possibility of a shared mechanism and, more importantly, has direct diagnostic and therapeutic implications. This case underscores the need to consider neuropathic and metabolic etiologies in the differential diagnosis of acute neurological events in diabetic patients whose glucose has been aggressively managed, to distinguish cortical from peripheral and hyperglycemic from post-correction processes, and to pursue controlled, cautious glycemic correction.

Case Presentation

A 72-year-old right-handed man with a history of hypertension, hyperlipidemia, and poorly controlled type 2 diabetes mellitus (T2DM) complicated by multiple digital amputations presented with a three-day history of speech difficulty and generalized weakness. His home regimen consisted of metformin and glipizide; he had not previously received insulin. He reported no headache, fever, facial weakness, limb weakness, or sensory loss. His daughter reported that his speech had become slurred and effortful, with difficulty producing words; comprehension was preserved; and the change was sudden in onset and stable since.

On admission, blood pressure was 212/101 mmHg and heart rate 76. He was afebrile. He was alert and oriented to person, place, and time. There was anisocoria, with a right pupil of 3 mm that was reactive and a left pupil that was irregular and nonreactive; visual fields were intact to confrontation. Facial sensation and strength were intact, and the remainder of the cranial nerve examination was unremarkable. Motor, sensory, and reflex examinations were normal, with no pronator drift, no focal weakness, and preserved plantar responses.

Spontaneous speech was nonfluent, with reduced phrase length, agrammatism, and effortful, telegraphic output. Comprehension of commands was intact. Repetition was intact. Reading and writing were intact. Articulation was additionally slurred, consistent with a coexisting dysarthria. The deficit was classified as expressive aphasia with accompanying dysarthria.

Laboratory evaluation revealed a glucose of 535 mg/dL and an HbA1c of 16.4%. Venous blood gas showed a pH of 7.367 (reference: 7.300–7.400), bicarbonate of 26 mEq/L (reference: 23–30 mEq/L), plasma osmolality of 318 mOsm/kg, and negative serum ketones, excluding DKA and HHS. Noncontrast head CT showed no acute intracranial pathology, and brain magnetic resonance imaging (MRI) with magnetic resonance angiography (MRA) of the head and neck was unremarkable. The electrocardiogram showed normal sinus rhythm with bifascicular block, and transthoracic echocardiography showed a normal left ventricular

ejection fraction with moderate left ventricular hypertrophy. Electroencephalography was not performed, which limited the diagnostic evaluation. Ophthalmology was consulted and attributed pupillary changes to prior cataract surgery and iris defect.

The patient was treated for hypertensive emergency with an intravenous nicardipine infusion and intermittent intravenous labetalol, with subsequent blood pressure control to 150/80. Glycemic management was intensified with basal-bolus insulin (insulin glargine 18 units daily and insulin lispro 8 units three times daily before meals), lowering serum glucose from 535 mg/dL to 120-150 mg/dL over the first four seven days (Figure 1). The speech disturbance was present on admission and therefore preceded any glycemic intervention; it persisted unchanged through the period of glycemic correction and did not worsen as glucose was decreased.

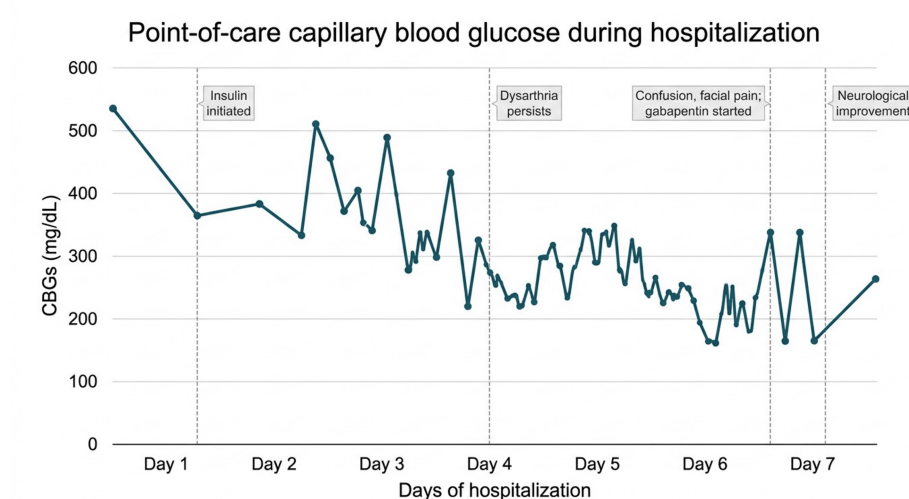


FIGURE 1: Temporal relationship between glycemic correction and neurological events during a seven-day hospitalization

On hospital day 7, by which time glucose had been within 120-150 mg/dL for 1 day, he developed new-onset confusion and left-sided facial pain. The pain was continuous and burning, localized to the V1-V3 distribution, constant with triggers such as chewing, tooth brushing, washing the face, or light touch, and was accompanied by tenderness over the infraorbital nerve. Examination revealed normal sensation in the same distribution, with no allodynia. There was no vesicular eruption, no corneal involvement, no masseter weakness, and no jaw deviation. Repeat CT of the brain was unchanged (Table 1).

Hospital day	Event	Glucose (mg/dL)	BP (mmHg)	Intervention/diagnostic steps
-3	Onset of speech disturbance, generalized weakness, subjective visual loss	-	-	None; continued metformin and glipizide
1	Admission; dysarthria/aphasia and anisocoria	535 (HbA1c 16.4%)	212/101	CT head; MRI/MRA brain and neck; ECG; TTE; nicardipine, labetalol; basal-bolus insulin started
1-4	Glycemic correction	535 → 120-150	Controlled	Glargine 18 units daily, lispro 8 units TID; speech deficit unchanged
4-6	Speech deficit persists	120-150	Controlled	-
7	New confusion; left facial pain, V1-V3 distribution, with tenderness	120-150	Controlled	Repeat CT head; ophthalmology consultation (pupil attributed to prior cataract surgery and iris defect)
7	Gabapentin 300 mg TID started	120-150	Controlled	-
8-10	Gabapentin titrated to 800 mg TID	120-150	Controlled	-
10	Facial pain resolved; mental status at baseline; speech resolved	120-150	Controlled	-

TABLE 1: Symptom and treatment chronology

TID: three times in a day; MRI: magnetic resonance imaging; MRA: magnetic resonance angiography; CT: computed tomography; ECG: electrocardiogram; TTE: transthoracic echocardiogram

Given the absence of structural, vascular, or metabolic explanation, a neuropathic process was suspected, and gabapentin was started at 300 mg three times daily and titrated to 800 mg three times daily. Over the following three days, his facial pain resolved, his mental status returned to baseline, and his speech disturbance resolved completely.

The final formulation is of two temporally distinct events. The first, which is transient dysarthria/aphasia and generalized weakness with subjective visual loss, occurred during severe hyperglycemia and hypertensive emergency and had resolved without a structural correlate on MRI. The second, which is painful left trigeminal neuropathy, began six days after glucose had been lowered to near-normal levels, a timing consistent with TIND. Hypertensive encephalopathy, posterior reversible encephalopathy syndrome, and a small ischemic event were considered for the first event; trigeminal neuropathy was attributed to the post-correction neuropathic process based on timing, the absence of a structural lesion, and the absence of an alternative cause (herpes zoster, dental or sinus disease, skull-base lesion, demyelinating disease, or vitamin deficiency).

Discussion

Acute neurological complications of diabetes most commonly occur in the context of metabolic decompensation, particularly DKA and HHS, where dehydration, hyperosmolality, and electrolyte derangements contribute to encephalopathy, seizures, focal deficits, and movement disorders [3-5]. Our patient is notable in two respects: he developed acute neurological symptoms in the absence of DKA or HHS, and his course comprised two temporally distinct events. The first, the expressive aphasia, was present on admission, during the phase of severe hyperglycemia (with hyperosmolality short of formal HHS criteria) and untreated hypertension, that is, before any correction of glycemia. The second, the left-sided facial pain in the trigeminal distribution with new confusion, emerged on hospital day seven, after blood glucose had been brought to near-normal levels. This distinction matters diagnostically: because TIND is, by definition, a consequence of glycemic improvement, the admission dysphasia cannot be attributed to it. The case therefore illustrates two distinct but potentially related processes: hyperglycemia-associated focal neurological dysfunction and a subsequent painful trigeminal neuropathy raising the question of TIND.

TIND, historically referred to as "insulin neuritis," is an underrecognized iatrogenic complication characterized by acute, often severe, neuropathic pain and/or autonomic dysfunction following rapid improvement in glycemic control [6-11]. Gibbons and Freeman proposed diagnostic criteria that include (1) a reduction in HbA1c of ≥2 percentage points over a three-month period, (2) development or marked worsening of neuropathic symptoms and/or autonomic dysfunction within eight weeks of glycemic

improvement, and (3) exclusion of alternative etiologies [7]. In their cohort, TIND most frequently presents as a symmetric, length-dependent, small-fiber-predominant neuropathic pain syndrome, commonly accompanied by autonomic features such as orthostatic hypotension, gastrointestinal dysmotility, and sudomotor dysfunction [7,8]. Applying these criteria to our patient, criteria 2 and 3 are satisfied: new neuropathic facial pain appeared within days of glycemic improvement, well inside the eight-week window, and an extensive structural and vascular workup was unrevealing. Criterion 1, however, cannot be formally assessed, as no pre-admission HbA1c was available for comparison; the admission value of 16.4% documents chronic severe hyperglycemia but does not establish its trajectory over the preceding three months, and serial inpatient capillary glucose values, themselves marked by considerable day-to-day variability (Figure 1), are not a substitute for this criterion. TIND therefore remains a possible rather than established diagnosis in this case. The phenotype is also atypical: cranial nerve-predominant pain without autonomic involvement, rather than the classical distal symmetric pattern [7,8].

Aphasia is an unusual manifestation of diabetic neuropathy and is more typically associated with cerebrovascular disease. However, focal neurological deficits, including aphasia, hemiparesis, hemianopia, and movement disorders, have been described in the setting of severe nonketotic hyperglycemia and hyperosmolar states, even in the absence of frank HHS, presumably due to reversible metabolic or perfusion-related dysfunction rather than infarction [3-5,12-14]. Several case reports and small series have documented transient aphasia or focal cortical deficits in patients with marked hyperglycemia, often resolving with correction of glucose levels and without radiographic evidence of stroke [12-16]. Proposed mechanisms include regional cerebral hypoperfusion, altered neuronal excitability, and hyperviscosity-related microcirculatory changes [4,5,13,14]. In our case, the initial expressive aphasia in the context of profound hyperglycemia, combined with unremarkable CT, MRI, and MRA studies, supports a transient metabolic-neurological effect of hyperglycemia rather than an ischemic event. Two features temper this interpretation and should be acknowledged: the deficit preceded any glycemic correction, and dysarthria persisted for several days after glucose normalization, whereas the deficits reported in the hyperglycemia literature typically resolve promptly with correction. This persistence argues against a purely reversible metabolic phenomenon and kept our differential appropriately broad.

The subsequent development, on hospital day seven, of left-sided facial pain in the trigeminal distribution with focal nerve tenderness, after rapid normalization of blood glucose, aligns more closely with TIND. Although the most common pattern of TIND is a length-dependent, symmetric, small-fiber neuropathy affecting the lower extremities [7-9], cranial neuropathies and focal cranial neuralgias have been reported in association with both diabetic neuropathy and rapid glycemic correction [10,11,17-19]. Painful cranial neuropathies involving the oculomotor, abducens, and trigeminal nerves, sometimes of abrupt onset and severe intensity, have been described in patients with diabetes, often in the context of recent glycemic fluctuations [17-19]. A nosological point deserves emphasis: under ICHD-3, "trigeminal neuralgia" denotes paroxysmal, electric shock-like pain provoked by triggers, whereas our patient's continuous pain with marked tenderness is more accurately designated painful trigeminal neuropathy. The concurrent confusion is more difficult to ascribe; a mild metabolic encephalopathy or unrelated process cannot be excluded, and we do not consider it part of the putative neuropathic syndrome. The focal trigeminal tenderness, absence of structural lesions on repeated brain imaging, and subsequent response to gabapentin favor a neuropathic pain mechanism over compressive, demyelinating, or vascular pathology, although none of these features is specific.

Differentiating TIND or diabetic neuropathy-related focal deficits from more common neurological emergencies such as ischemic stroke, transient ischemic attack (TIA), intracerebral hemorrhage, or complex migraine is critical and mandates prompt neuroimaging. In older patients with vascular risk factors, including longstanding diabetes, hypertension, and hyperlipidemia, acute aphasia and focal deficits must be treated as stroke until proven otherwise [20-22]. Our patient underwent emergent CT imaging followed by MRI and MRA of the head and neck, all of which were unremarkable, effectively ruling out large-vessel occlusion, hemorrhage, and overt infarction. Hypertensive urgency was present at admission; however, blood pressure normalization did not result in symptom resolution, and hypertension alone could not account for persistent dysarthria or the later development of trigeminal pain. Similarly, complex migraine, seizure with postictal deficits, and structural lesions were considered less likely given the clinical course and negative imaging.

The pathophysiology of TIND is not fully elucidated. Several mechanisms have been proposed, including endoneurial ischemia due to rapid microvascular remodeling, oxidative stress from abrupt normalization of chronic hyperglycemia, and immune-mediated small-fiber injury triggered by rapid changes in the glycemic milieu [6-9]. Rapid glucose reduction may also unmask or exacerbate subclinical neuropathy, particularly in patients with long-standing, poorly controlled diabetes and pre-existing complications, as in this case (evidenced by prior digital amputations). It should be noted that our patient's serial glucose values fluctuated substantially rather than declining monotonically (Figure 1), so the precise rate of glycemic change cannot be characterized from capillary measurements alone, a further limitation in applying TIND criteria without a documented HbA1c trajectory. Some studies suggest that a more gradual improvement in glycemic control, limiting HbA1c reduction to <2 percentage points over three months, may mitigate the risk of TIND [7,8]. This must, however, be balanced against the imperative to correct severe acute hyperglycemia and reduce long-term microvascular and macrovascular complications.

Management of diabetic neuropathy and TIND is primarily supportive and symptomatic, with a focus on careful glycemic optimization and neuropathic pain control. There is no established disease-modifying therapy for TIND; instead, treatment centers on moderating the rate of glycemic improvement where clinically appropriate, managing pain, and addressing autonomic dysfunction when present [7-9]. Recommended agents for painful diabetic neuropathy, including duloxetine, pregabalin, gabapentin, and tricyclic antidepressants, are commonly used in TIND, and gabapentin has demonstrated efficacy in diabetic neuropathic pain and is frequently used off-label in this context. In our patient, initiation and titration of gabapentin to 800 mg three times daily was followed by resolution of the facial pain within three days. The concurrent improvement in dysarthria and cognition over the same period is more difficult to attribute: gabapentin is not an established therapy for language or encephalopathic phenomena, and spontaneous or multifactorial resolution cannot be excluded given the fluctuating course. The temporal association therefore supports the use of neuropathic pain agents for the neuropathic pain component, including atypical cranial presentations, while the resolution of speech and cognitive symptoms should be interpreted more cautiously.

In summary, this case describes two temporally distinct neurological syndromes in a patient with extreme chronic hyperglycemia: a transient focal deficit during the untreated hyperglycemic-hypertensive phase, best explained as reversible hyperglycemia-associated neurological dysfunction, and a subsequent painful trigeminal neuropathy emerging after rapid glycemic correction, fulfilling two of the three Gibbons-Freeman criteria. TIND remains a possible, atypical, and unconfirmed diagnosis in this patient. The case illustrates the cranial presentations in which TIND should be considered, the necessity of rigorously excluding stroke, and the practical limitation that inpatient glucose data cannot substitute for the HbA1c trajectory central to the accepted diagnostic framework.

Conclusions

This case illustrates two temporally distinct neurological syndromes in a 72-year-old man with long-standing, poorly controlled diabetes, rather than a single unifying process. The first, which is the transient expressive aphasia followed by persistent dysarthria, was present on admission, during severe hyperglycemia and untreated hypertension, before any correction of glycemia, and is best characterized as severe hyperglycemia-associated neurological dysfunction; ischemic and structural causes were excluded by CT, MRI, and MRA. The second, which is the continuous left facial pain in the trigeminal distribution with focal tenderness, emerged on hospital day 7, after rapid glucose normalization. Because the pain was continuous and associated with tenderness rather than paroxysmal and trigger-provoked, it is more accurately designated painful trigeminal neuropathy than trigeminal neuralgia. Its onset after rapid glycemic correction raises TIND as a possible, atypical, cranial-predominant explanation; however, the diagnosis remains presumptive, as no baseline HbA1c was available to document the glycemic trajectory required by accepted diagnostic criteria, and temporal association alone does not establish causation. The accompanying confusion is not attributable to a neuropathic process and remains incompletely explained.

Resolution of the facial pain within days of gabapentin initiation supports a neuropathic pain mechanism for that symptom; the concurrent improvement in speech and cognition cannot be ascribed to gabapentin and may reflect spontaneous or multifactorial resolution.

These observations underscore that both severe hyperglycemia and its rapid correction can be associated with acute neurological syndromes in patients with long-standing diabetes and microvascular complications. In elderly patients presenting with speech disturbance or altered mental status, evaluation for cerebrovascular disease, seizure, and structural pathology remains essential even when initial neuroimaging is unremarkable. Awareness of TIND should broaden, not shortcut, the differential once dangerous causes are excluded and may support more gradual glycemic correction where clinically feasible. Further research is needed to define the incidence, risk factors, and mechanisms of TIND, particularly its cranial presentations, and to refine diagnostic criteria, including the role of documented HbA1c trajectories and optimal rates of glycemic correction.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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