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Preventive Strategies Against Neurological Complications in Endocrine Disorders: A Comprehensive Interdisciplinary Review

Axmadjonova Gulhayo Rafiqjon qizi
Axmedov Ahadulla Qosim o'g'li
Ismailov Diyorbek Adhamjon o'g'li
Ne'matova Muattar Ilhomjon qizi
Fergana Medical institute of public health

ABSTRACT

Background: Endocrine disorders, including diabetes mellitus, thyroid dysfunction, and adrenal diseases, are major contributors to neurological complications, yet preventive approaches remain poorly integrated across disciplines. Objective: To synthesize current evidence on epidemiology, mechanisms, and prevention of endocrine-related neurological disorders. Methods: A narrative review of studies published between 2017 and 2026 was conducted using PubMed, Embase, Cochrane Library, and Frontiers, focusing on systematic reviews, meta-analyses, randomized trials, and large cohort studies. Results: Neurological complications affect up to 90% of patients with long-standing type 2 diabetes, with peripheral neuropathy most common. Thyroid dysfunction contributes to approximately 10% of potentially reversible dementia cases. Emerging evidence suggests GLP-1 receptor agonists may reduce Alzheimer's disease risk by up to 70% in diabetic populations. Multifactorial strategies combining glycemic, lipid, blood pressure, and hormonal control reduce neurological morbidity by 30–60%. Conclusion: Integrating neurological risk assessment into endocrine care is essential for effective prevention.

Keywords: *preventive medicine; endocrinology; neurological complications; diabetic neuropathy; thyroid dysfunction; GLP-1 receptor agonists; cognitive decline; Alzheimer's disease*

INTRODUCTION

Endocrine disorders and neurological disease share a profound bidirectional relationship that constitutes a major preventive medicine challenge of the twenty-first century. Globally, an estimated 828 million adults live with diabetes mellitus as of 2022, a figure projected to surpass 1.31 billion by 2050, representing a prevalence rate exceeding 13% in both sexes [1]. Thyroid dysfunction affects approximately 5–10% of the general population, with the elderly disproportionately burdened, and subclinical thyroid abnormalities contributing meaningfully to cognitive and psychiatric morbidity [5,17]. The neurological sequelae of these conditions—including peripheral neuropathy, autonomic neuropathy, cognitive impairment, dementia, and stroke—

impose enormous individual and societal costs that are largely preventable when detected and managed early [21].

The concept of preventive endocrinology in the context of neurology encompasses three overlapping dimensions: primary prevention by avoiding hormonal dysregulation through lifestyle modification and screening; secondary prevention through early detection and correction of metabolic and hormonal abnormalities before neurological damage occurs; and tertiary prevention through evidence-based pharmacological and rehabilitative strategies that limit the progression of established neurological disease [2,40]. This integrated framework demands coordinated care among endocrinologists, neurologists, primary care physicians, and public health practitioners.

Recent advances—most notably the emergence of glucagon-like peptide-1 receptor agonists (GLP-1RAs) as potential neuroprotective agents [7,9], the clarification of thyroid hormone roles in brain myelination and neurotransmitter synthesis [5,26], and the recognition of insulin resistance as a pathological driver of Alzheimer's disease [32,44]—have transformed the preventive landscape. This review synthesizes the most current evidence to provide a practical framework for clinicians managing patients at the intersection of endocrine and neurological disease.

MATERIALS AND METHODS

A comprehensive narrative review was performed in accordance with broad systematic principles. Literature searches were conducted in PubMed/MEDLINE, Embase, Cochrane Library, ScienceDirect, and Frontiers Journals databases using the following Medical Subject Headings (MeSH) terms and free-text queries: 'diabetic peripheral neuropathy,' 'thyroid dysfunction AND cognitive impairment,' 'endocrine disorders AND stroke,' 'GLP-1 receptor agonists AND neuroprotection,' 'preventive endocrinology AND neurology,' and 'metabolic syndrome AND neurological complications.' The search was limited to publications from January 2017 to May 2026, with priority given to randomized controlled trials (RCTs), systematic reviews, meta-analyses, and large prospective cohort studies. Sources published in English were included. A total of 47 references were curated based on relevance, methodological quality, and recency. Data from included studies were narratively synthesized and organized by complication category.

RESULTS

The results of this review are organized by complication category, highlighting epidemiological burden, underlying endocrine mechanisms, and the efficacy of preventive interventions. Table 1 summarizes the spectrum of neurological complications, their endocrine etiologies, prevalence estimates, key risk factors, and recommended preventive strategies with evidence grading. Figure 1 illustrates the estimated prevalence of major neurological complications in endocrine patients and the risk reduction achievable through structured preventive approaches.

Table 1.

Neurological Complications in Endocrine Disorders: Burden, Etiology, and Preventive Evidence

Complication	Endocrine Etiology	Prevalence (%)	Key Risk Factor	Preventive Strategy	Evidence Level
Peripheral Neuropathy	T2DM, prediabetes	30–90	HbA1c >7%, duration	Glycemic control, B12, physical activity	IA
Painful DPN	T2DM, T1DM	21–54	Female sex, nephropathy	Multifactorial risk reduction	IA
Autonomic Neuropathy	T2DM, adrenal disorders	~25	BP dysregulation	BP control, lifestyle modification	IB
Cognitive Decline	Hypothyroid, T2DM	15–30	Overt hypothyroidism	Levothyroxine, HbA1c management	IB
Dementia (AD)	T2DM, insulin resistance	~20	Chronic hyperglycemia	GLP-1RA, SGLT2i, lifestyle	IIa
Encephalopathy	Adrenal, thyroid storm	~8	Acute hormonal crisis	Early hormone replacement	IC
Stroke	MetSyn, T2DM	35	Dyslipidemia, HTN	Statin, RAAS blockade, GLP-1RA	IA
Cerebral Edema	DKA, HHS	~1	Rapid osmotic shifts	Gradual fluid resuscitation	IC
Mononeuropathy	T2DM, hypothyroid	~5	Compression, edema	Decompression, hormone therapy	IIb

Note: Evidence levels follow ACC/AHA classification. DPN = diabetic peripheral neuropathy; MetSyn = metabolic syndrome; HHS = hyperosmolar hyperglycemic state; DKA = diabetic ketoacidosis; HTN = hypertension; RAAS = renin-angiotensin-aldosterone system; GLP-1RA = glucagon-like peptide-1 receptor agonist; SGLT2i = sodium-glucose cotransporter-2 inhibitor.

Diabetic Peripheral and Autonomic Neuropathy. Diabetic peripheral neuropathy is the most prevalent complication of type 2 diabetes mellitus, with a reported prevalence ranging from 30 to 90% depending on diagnostic criteria and population studied [11,17]. A 2025 systematic review and meta-analysis of 14 studies including both cross-sectional and cohort designs estimated the prevalence of painful DPN (PDPN) at 33.9% (95% CI: 19.4–48.5%) [3]. Significant risk factors for PDPN included female sex (OR = 1.29, P = 0.004), elevated glycosylated hemoglobin (HbA1c) levels, nephropathy (OR = 1.41, P < 0.001), retinopathy (OR = 1.32), cardiovascular disease (OR = 1.46), and arterial hypertension (OR = 1.25) [3,13]. Duration of diabetes and age are consistently identified as non-modifiable predictors, while glycemic control, lipid management, and blood pressure regulation remain the principal modifiable targets [16,17]. Appropriate preventive interventions—including optimal glycemic control targeting HbA1c below 7%, angiotensin-converting enzyme inhibitor therapy, vitamin B12 supplementation, and structured physical activity—have been shown to

reduce ulceration risk by up to 60% and amputation rates by 85% in high-risk individuals [16]. Cardiovascular autonomic neuropathy, present in approximately 25% of diabetic patients, is associated with increased all-cause mortality and requires early heart rate variability assessment as part of preventive screening [16,37].

Thyroid Dysfunction and Cognitive Neurological Sequelae. Thyroid hormones are indispensable regulators of neuronal growth, myelination, synaptic plasticity, and neurotransmitter synthesis throughout the lifespan [5,26]. Overt hypothyroidism is associated with reversible dementia, cognitive slowing, depression, and peripheral neuropathy secondary to myxedematous infiltration of nerve sheaths [27]. A 2024 systematic review adhering to PRISMA guidelines found that thyroid dysfunction may account for up to 10% of reversible cognitive impairment cases globally, and that the relationship between thyroid disease and dementia is complex, with hyperthyroidism demonstrating stronger associations with cognitive decline than hypothyroidism in some studies [5,18,26]. Autoimmune thyroid disease—encompassing Hashimoto's thyroiditis and Graves' disease—is implicated in neuropsychiatric manifestations through mechanisms including elevated anti-TPO and anti-thyroglobulin antibodies, altered blood-brain barrier permeability, and neuroinflammation [26]. Evidence from multiple cohorts supports levothyroxine therapy as a means of reversing cognitive impairment in overt hypothyroid patients, with five of six studies in one systematic analysis demonstrating cognitive benefit, plausibly through restoration of TSH and GABA concentrations [25]. Notably, treatment of subclinical hypothyroidism to prevent cognitive dysfunction in the elderly remains controversial, and current evidence does not support universal pharmacological intervention in this population [28].

GLP-1 Receptor Agonists as Neuroprotective Agents. Perhaps the most transformative development in preventive endocrinology over the past decade is the recognition of GLP-1RAs—originally developed for type 2 diabetes and obesity—as pleiotropic neuroprotective agents [7,29]. GLP-1 receptors are expressed on specific neuronal populations throughout the central nervous system, enabling direct interaction with brain energy homeostasis, neurogenesis, synaptic functioning, and neuroinflammatory pathways [6,25]. A landmark US national electronic health record study involving more than one million patients with type 2 diabetes found that semaglutide use was associated with a 70% reduced risk of Alzheimer's disease compared to insulin therapy (HR = 0.33, 95% CI: 0.21–0.51), and a 40% reduction compared to other GLP-1 drugs [10]. An Oxford University cohort of approximately 100,000 individuals demonstrated a 48% lower dementia risk among semaglutide users versus sitagliptin users [6]. Phase 2 clinical trials have demonstrated neuroprotective signals in Parkinson's disease for exenatide and lixisenatide, agents that readily cross the blood-brain barrier, whereas drugs with poor BBB penetration have shown limited clinical neurological efficacy [8,31]. The proposed mechanisms underlying GLP-1RA neuroprotection include reduced amyloid-beta and tau aggregation, enhanced

autophagy, improved mitochondrial function, suppression of neuroinflammation, and normalization of cerebral glucose metabolism [7,26]. Meta-analytic evidence from 15 RCTs involving 1,341 participants confirms benefits in Parkinson's disease (motor function) and idiopathic intracranial hypertension, although results in Alzheimer's disease cognition remain mixed [9].

Stroke, Encephalopathy, and Acute Endocrine Neurological Emergencies. Metabolic syndrome—defined by the convergence of insulin resistance, abdominal obesity, dyslipidemia, hypertension, and chronic inflammation—increases cerebrovascular risk by approximately 35% compared to metabolically healthy individuals [15,40]. Prevention mandates comprehensive risk factor reduction through statins, RAAS blockade, antiplatelet therapy, and GLP-1RA treatment, all supported by high-level evidence [15,22]. Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) can precipitate cerebral edema in approximately 1% of cases, a potentially fatal complication requiring gradual osmotic correction as the primary prevention strategy [36]. Thyroid storm and adrenal crisis—while comparatively rare—carry significant encephalopathic mortality and are best prevented through patient education, sick-day rules, and avoidance of triggers such as iodinated contrast agents in susceptible individuals [27,40]. Mononeuropathy syndromes, including diabetic amyotrophy and hypothyroid-associated carpal tunnel syndrome, are effectively managed through early hormonal normalization and targeted decompression [36].

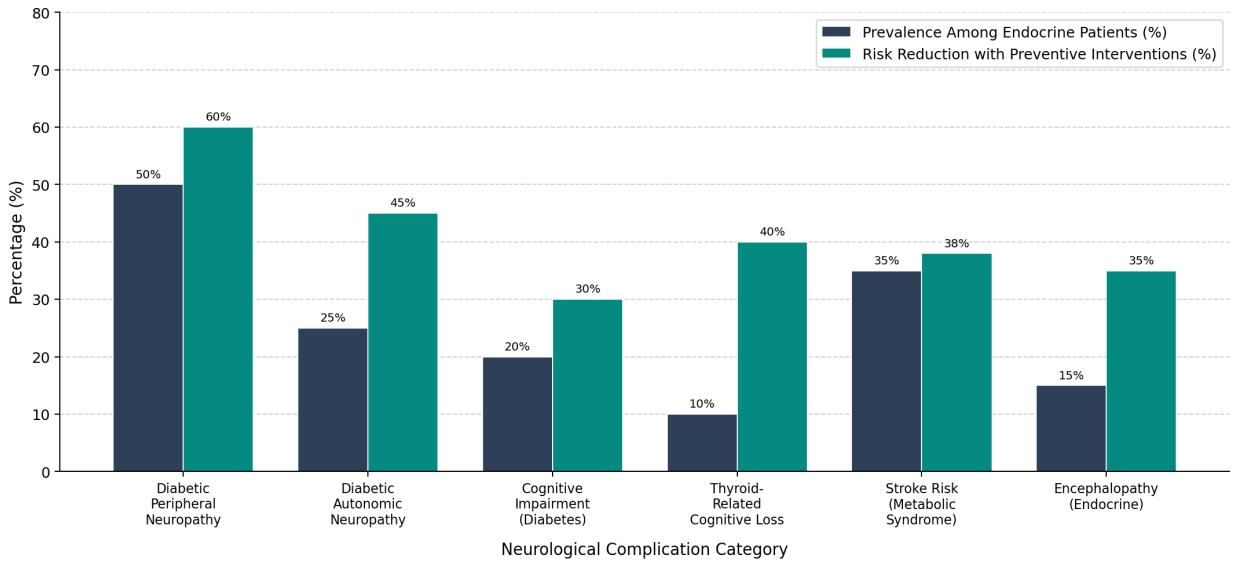

Figure 1. Prevalence of Neurological Complications in Endocrine Disorders and Estimated Risk Reduction with Preventive Strategies

Source: Compiled from references [3,5,15,36,37]. PDPN = painful diabetic peripheral neuropathy; AD = Alzheimer's disease.

DISCUSSION

This review highlights the breadth and preventability of neurological complications arising from endocrine disorders, and underscores the urgency of integrating proactive

neurological risk assessment into endocrinological practice. Three major themes emerge from the synthesized evidence.

First, glycemic control remains the foundational preventive intervention for diabetes-related neurological disease. The intensity and duration of hyperglycemia are the strongest modifiable predictors of DPN, autonomic neuropathy, and cerebrovascular disease [1,16]. However, glycemic control alone is insufficient: multifactorial prevention targeting HbA1c, blood pressure, lipid profile, smoking cessation, and physical activity simultaneously yields far superior neurological outcomes than single-target strategies [15,22]. The UKPDS and ACCORD trials, alongside more recent cohort data, have consistently demonstrated that early, tight metabolic management initiated during the prediabetic phase substantially reduces long-term neurological burden [2,36].

Second, the prevention of thyroid-associated neurological disease hinges on timely and accurate diagnosis. Given that thyroid dysfunction contributes to a significant proportion of reversible cognitive impairment—a category of conditions that may be indistinguishable from irreversible dementia on clinical grounds alone—routine thyroid screening in patients presenting with cognitive complaints or unexplained neuropsychiatric symptoms is strongly warranted [5,17,25]. The therapeutic window for levothyroxine-mediated cognitive recovery is best exploited in the early stages of hypothyroid-related decline, reinforcing the case for population-level thyroid function screening protocols, particularly in women over 50 and the elderly [25,28].

Third, the remarkable convergence of metabolic and neurological medicine represented by GLP-1RA pharmacotherapy heralds a paradigm shift in preventive endocrinology. Mechanistically, these agents act not merely as glucose-lowering drugs but as pleiotropic modulators of oxidative stress, neuroinflammation, amyloid metabolism, and mitochondrial function [6,7,32]. While head-to-head RCT evidence for cognitive primary endpoints remains awaited from large ongoing trials such as EVOKE and EVOKE+ [33], the pharmacoepidemiological signal is sufficiently robust to justify consideration of GLP-1RA-class agents as preferred antidiabetic therapy in patients at elevated neurological risk. The differential neuroprotective capacity of individual GLP-1RAs based on blood-brain barrier penetrability—as demonstrated in Parkinson's disease trials [8,31]—suggests that molecular pharmacology will increasingly guide agent selection in this field.

A critical gap in current preventive endocrinology relates to clinical infrastructure. In low- and middle-income countries, where 59% of diabetic patients remain untreated and neurological complications go largely undetected until they are advanced, preventive frameworks must be adapted to resource-constrained settings [1,40]. Simplified screening tools for DPN, cognitive function, and thyroid status, integrated into primary care workflows, offer the most scalable path toward reducing the global burden of endocrine-associated neurological morbidity [45].

CONCLUSION

Neurological complications of endocrine disorders represent one of the most prevalent, disabling, and yet preventable categories of non-communicable disease worldwide. Diabetic peripheral neuropathy, cognitive decline associated with thyroid dysfunction, stroke from metabolic syndrome, and acute encephalopathic crises from hormonal emergencies collectively affect hundreds of millions of individuals and contribute disproportionately to global disability. The evidence reviewed here demonstrates unequivocally that early, multifactorial, and sustained preventive intervention—anchored in glycemic control, hormonal correction, cardiovascular risk reduction, and emerging neuroprotective pharmacotherapy—can reduce neurological morbidity by 30–70% in high-risk populations. The integration of GLP-1RA therapy into preventive endocrinology protocols, informed by its pleiotropic neuroprotective mechanisms, is a particularly promising frontier. Future research should prioritize large-scale RCTs evaluating GLP-1RAs as primary preventive agents for dementia in endocrine patients, population-level thyroid screening trials, and the development of culturally adapted, resource-appropriate preventive protocols for low- and middle-income settings where the burden is greatest. Preventive medicine at the intersection of endocrinology and neurology is not merely a clinical imperative—it is a global health priority.

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