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Case Series
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Is Epilepsy a Complication of Type 1 Diabetes? A Series of Four Cases and Review of the Literature

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Abstract

Introduction: The association between type 1 diabetes (T1D) and epilepsy has been reported more frequently than in the general population. However, the exact pathophysiological link remains poorly understood.

Objective: To describe the clinical characteristics of patients presenting with an association of T1D and epilepsy and to discuss the possible pathophysiological mechanisms in light of data from the literature.

Methods: A retrospective descriptive study of four patients followed for type 1 diabetes and epilepsy.

Results: All patients had poorly controlled type 1 diabetes, with frequent hypoglycemia in three cases. Epilepsy was generalized in three cases and focal in one case. Brain imaging and EEG were normal in all patients.

Conclusion: The association between T1D and epilepsy appears to be multifactorial, involving autoimmune, metabolic, and possibly genetic mechanisms.

Keywords: Type 1 Diabetes; Epilepsy; Autoimmunity; Anti-GAD; Hypoglycemia

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Introduction

Type 1 diabetes (T1D) is an autoimmune disease characterized by the destruction of pancreatic beta cells, leading to absolute insulin deficiency. It is frequently associated with other autoimmune diseases, suggesting a shared immunogenetic susceptibility [1].

Several studies have reported a higher prevalence of epilepsy in patients with T1D compared with the general population [2]. This association raises the question of a specific pathophysiological link, distinct from epileptic seizures secondary to acute glycemic disturbances.

We report a series of four cases illustrating this association and discuss the pathophysiological hypotheses proposed in the literature.

Patients and Methods

This is a retrospective descriptive study conducted in the Department of Endocrinology at Ibn Sina University Hospital in Rabat.

Inclusion Criteria

- Confirmed type 1 diabetes
- Diagnosis of epilepsy established by a neurologist
- Absence of structural brain lesions on imaging

Collected Data

- Age and sex
- Duration of diabetes and glycemic control
- Type of epilepsy
- Results of brain imaging and EEG
- Treatment and outcome

Table 1: Clinical characteristics of the patients.

Patient	Age (years)	Duration of T1D	Glycemic Control	Type of Epilepsy	Hypoglycemia	Imaging / EEG
1	31	10 years	Poor	Generalized	No	Normal
2	28	8 years	Poor	Generalized	Yes	Normal
3	21	4 years	Poor	Focal	Yes	Normal
4	19	5 years	Poor	Generalized	Yes	Normal

Clinical Observations

Case 1

A 31-year-old female patient, followed for 7 years for generalized epilepsy treated with sodium valproate. She had type 1 diabetes for 10 years, treated with a basal–bolus insulin regimen, with poor treatment adherence. Diabetes was poorly controlled, marked by repeated episodes of hyperglycemia. Brain MRI and EEG were normal.

Case 2

A 28-year-old female patient with type 1 diabetes for 8 years, poorly controlled, presenting with frequent hypoglycemia and complicated by diabetic nephropathy. She had been followed for 4 years for generalized epilepsy under antiepileptic treatment. Brain imaging and EEG showed no abnormalities.

Case 3

A 21-year-old male patient with type 1 diabetes for 4 years, poorly controlled, with recurrent hypoglycemia. He had been followed for one year for focal epilepsy under treatment. Neuroradiological and electrophysiological investigations were normal.

Case 4

A 19-year-old male patient with type 1 diabetes for 5 years, poorly controlled, followed for 2 years for generalized epilepsy under antiepileptic treatment. Brain MRI and EEG were normal.

Results

See Table 1.

Discussion

Our series highlights an association between type 1 diabetes and epilepsy in young patients, all of whom had poorly controlled diabetes. This finding is consistent with data from the literature, which report a prevalence of epilepsy up to three times higher in patients with T1D compared with the general population [3].

In our series, three patients experienced frequent hypoglycemia. Severe hypoglycemia is known to induce acute convulsive seizures; however, it is insufficient to explain chronic epilepsy, suggesting the presence of an underlying neurological susceptibility [4].

The autoimmune hypothesis is currently the most extensively studied. Anti–glutamic acid decarboxylase (anti-GAD) antibodies, markers of T1D, have also been identified in certain forms of autoimmune epilepsy [5]. GAD is a key enzyme in the synthesis of gamma-aminobutyric acid (GABA), the main inhibitory neurotransmitter in the central nervous system. Dysfunction of cerebral GAD could promote neuronal hyperexcitability [6].

The absence of abnormalities on MRI and EEG in our patients has also been reported in several series, supporting a functional rather than structural mechanism [7].

Finally, a shared genetic susceptibility has been suggested, particularly through certain HLA haplotypes common to both T1D and some forms of epilepsy [8].

Conclusion

Epilepsy cannot be considered a classical complication of type 1 diabetes; however, their association appears more frequent than expected by chance alone. This relationship is likely multifactorial, involving autoimmune, metabolic, and genetic mechanisms. Clinical vigilance is required in patients with T1D presenting with seizures not explained by glycemic disturbances.

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