DIABETHIC STRIATOPATHY:
A Clinical Case and Reflections on Neurological Management of Diabetes

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Diabetic striatopathy (DS), also known as hyperglycemic hemichorea-hemiballism, is a rare neurological complication of diabetes mellitus, characterized by the acute onset of involuntary movements secondary to basal ganglia abnormalities visible on neuroimaging. It is more commonly observed in older women of Asian origin. Prevalence: 1 in 100,000.

Metabolic theory: Anaerobic metabolism + Krebs cycle block → GABA and acetylcholine deplection → Basal ganglia DYsfunction

PATHOPHYSIOLOGY OF DIABETIC STRIATOPATHY

Vascular theory: Hypoperfusion and striatal capillary fragility → Damage to GABAergic neuron → Basal ganglia dysfunction

CASE REPORT

Male, 77 years old, Caucasian;
Heavy smoker; Type 2 diabetes mellitus, untreated;
Myocardial infarction in 2021;
PM implantation in 2022 due to recurrent syncopal episodes.

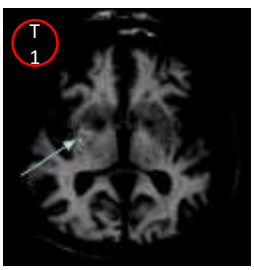
Sudden onset of involuntary, medium-to-large amplitude, jerky movements involving the entire left hemibody, with predominant involvement of the upper limb. The movements had a progressive course over approximately two weeks and diminished during sleep.

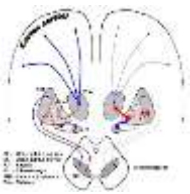
ER:
- Vital signs assessment: nrf.
- Laboratory tests (CBC, renal and liver function, electrolytes, inflammatory markers): nrf.
- Brain CT scan: Within normal limits.

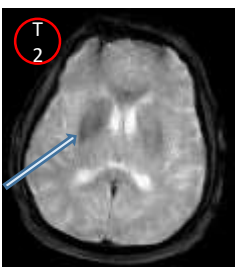
Differential diagnosis:
- ❖ Ischemic or hemorrhagic stroke;
- ❖ Sydenham's chorea;
- ❖ Iatrogenic causes (e.g., L-DOPA, dopamine agonists, antiepileptic drugs);
- ❖ Infections (e.g., encephalitis, toxoplasmosis);
- ❖ Cancer (e.g., metastases, lymphomas);
- ❖ Autoimmune disease (LES);
- ❖ Huntington's disease or other genetic disorders;
- ❖ Endocrine/metabolic disorders (e.g., hyponatremia, hypercalcemia, hyperglycemia).

Inpatient course:
- History: Recent episodes of severe hyperglycemia, with blood glucose levels > 500 mg/dL;
- Autoimmune panel + VES → nrf;
- HbA1c: 14,3% (normal range 4-6%);
- **Brain MRI**: Area of altered signal localized in the posterior-inferior portion of the **right putamen**, characterized by **hyperintensity on T1-weighted** images and corresponding marked signal **attenuation on T2-weighted** sequences.

Therapy:
- Insulin therapy following a basal-bolus regimen;
- Tetrabenazine 25 mg die → 50 mg die.







Follow-up:
Near-complete regression of symptoms observed 45 days after initiation of therapy.

CONCLUSIONS: The diagnosis of non-ketotic hyperglycemic hemichorea-hemiballism requires two key elements: neuroimaging (MRI > CT) and laboratory evidence of uncontrolled diabetes (glycated hemoglobin – HbA1c).

TREATMENT: In addition to strict glycemic control, in over 75% of cases, additional medications (belong to five classes of drugs: antipsychotics, dopamine-depleting agents, benzodiazepines, anticonvulsants, serotonin reuptake inhibitors) are often required to reduce the distressing symptoms of the condition.

REFERENCES:

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2) Arecco, A.,et all. (2024). Diabetic striatopathy: an updated overview of current knowledge and future perspectives. *Journal of endocrinological investigation*, *47*(1), 1–15.