

Diabetic striopathy, shortly diagnosed complication in diabetes mellitus II. Case report

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Section: Neuroradiology

Area of Interest: CNS Head and neck Neuroradiology
brain

Imaging Technique: MR

Imaging Technique: MR-Angiography

Special Focus: Endocrine disorders Case Type: Clinical
Case

Authors: Andrés Ruiz-Flores, Diego Bernal, Matías Enrique-Scherer, Ezequiel Piedra, Oscar Montaña

Patient: 86 years, female

Clinical History:

An 86-year-old female patient with clinical history of hospitalization due to diabetic ketoacidosis. The patient refers she refused to use insulin for a year after it was prescribed. She attended clinical control presenting dysarthria, involuntary movements, gait disorder, and confusional symptoms during a 48-hour period.

Imaging Findings:

Non-contrast Brain MRI was performed using a Philips Ingenia 1.5T high-field scanner, with complete protocol scan with T1, T2, FLAIR, FFE and DWI/ADC sequences.

T1 sequence hyperintensity of right basal nuclei with FFE sequence hypointensity.

T2 and FLAIR sequences with slight signal intensity changes in the basal ganglia, predominantly on the right.

Vascular disorders or occlusion signs were not seen in cerebral MRI angiography 3D reconstruction.

Discussion:

Diabetic striatopathy is a rare but potentially lethal complication. There is talk of a prevalence of less than 1 in 100,000, but in recent years it has represented a significant cause of hemichorea, outranked for now by cerebrovascular events [1]. It's defined as a clinical-radiological syndrome with the presence of classic symptoms (chorea) and imaging findings showing hyperdensity (CT) and hyperintensity (MRI), mainly in the basal ganglia [2,3]. This is due to the microenvironment vulnerable to metabolic attacks from the basal ganglia, associated with the characteristic diabetic's atherosclerosis acceleration, which, jointly with hyperosmolar/hyperglycemic alterations, determine an increase in blood viscosity due to damage suffered by the blood-brain barrier [2,4].

The accumulation of gemistocytes due to ischemic events and neuronal dysfunction can partially explain the basal ganglia hyperintensity in T1 sequence and is one of the most accepted theories regarding the pathophysiological mechanism for this entity, although it should be noted that its pathophysiology has not been of the same kind all understood [2,3].

The time interval between the onset of damage at the ultrastructural level and the structural lesion evident from images (MRI/CT) is unknown [2].

In our case, the brain MRI showed T1-weighted sequence hyperintensity of the right basal ganglia with FFE sequence hypointensity, with greater putaminal involvement. This T1 high signal is interpreted as a result of the presence of gemistocytes, which are astrocytes with edematous cytoplasm due to ischemic changes [5].

T2, DWI, FFE/GREE sequences are not characteristic and can present with hyper, iso, hypo, or even mixed intensity. Despite this, with metabolic control of diabetes, these lesions can progressively disappear. The tomographic correlation is described in most of the patients as hyperdensity in the basal ganglia, although some cases have been described where there is no tomographic correlation [4].

The complete reversal of the symptoms with the resolution of the imaging findings is the most frequent result in patients with diabetic striopathy. The mainstay is the metabolic event correction (hyperglycemia), and the symptomatic improvement is achieved by correcting the state of hyperglycemia long before the neuroradiological reversal. Has been said that this can last 3 to 8 months to stop appreciating in the images, but it continues to be a field of study and is individual in each patient [2].

Informed consent was obtained from the patient for publication.

Differential Diagnosis List: Diabetic striopathy, Cerebrovascular event, Wilson's disease, Fahr's disease, Diabetic uremic syndrome

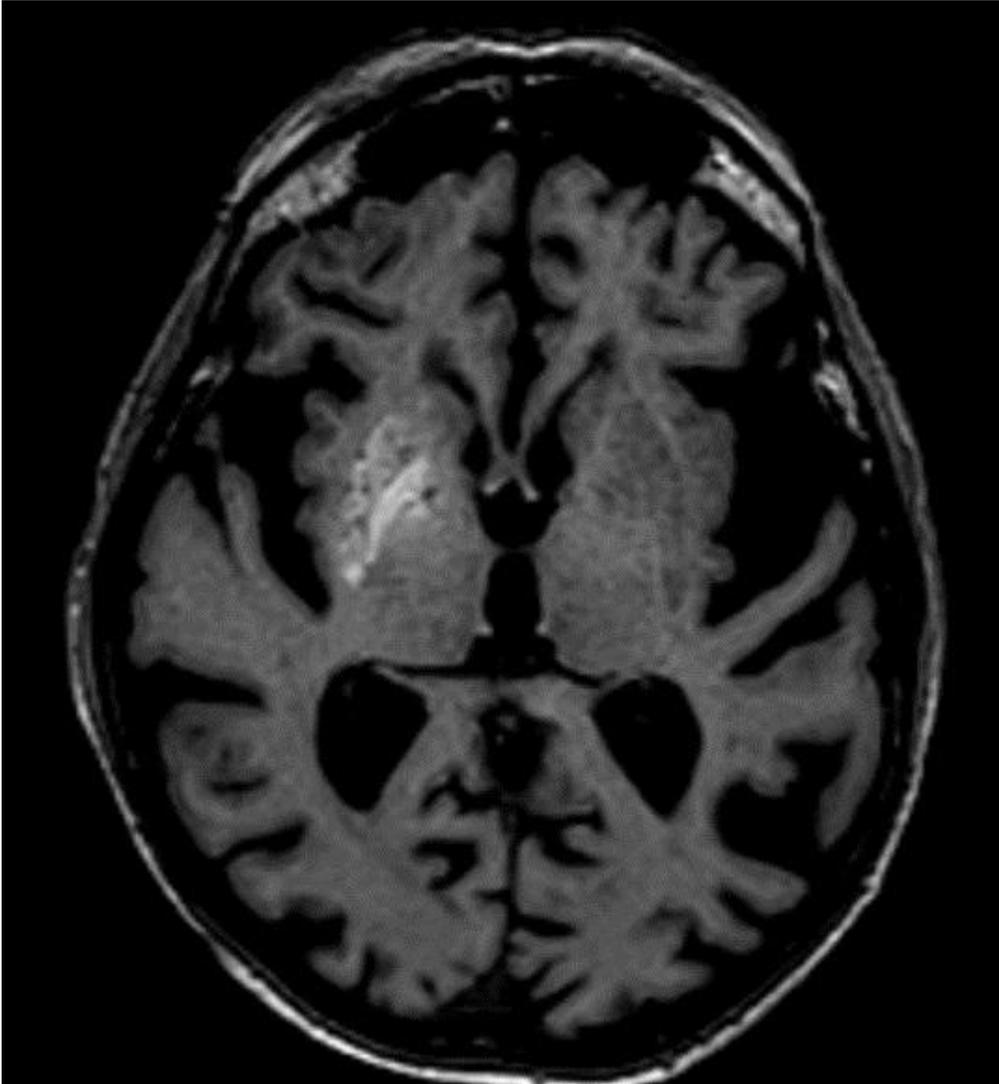
Final Diagnosis: Diabetic striopathy

References:

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Figure 1

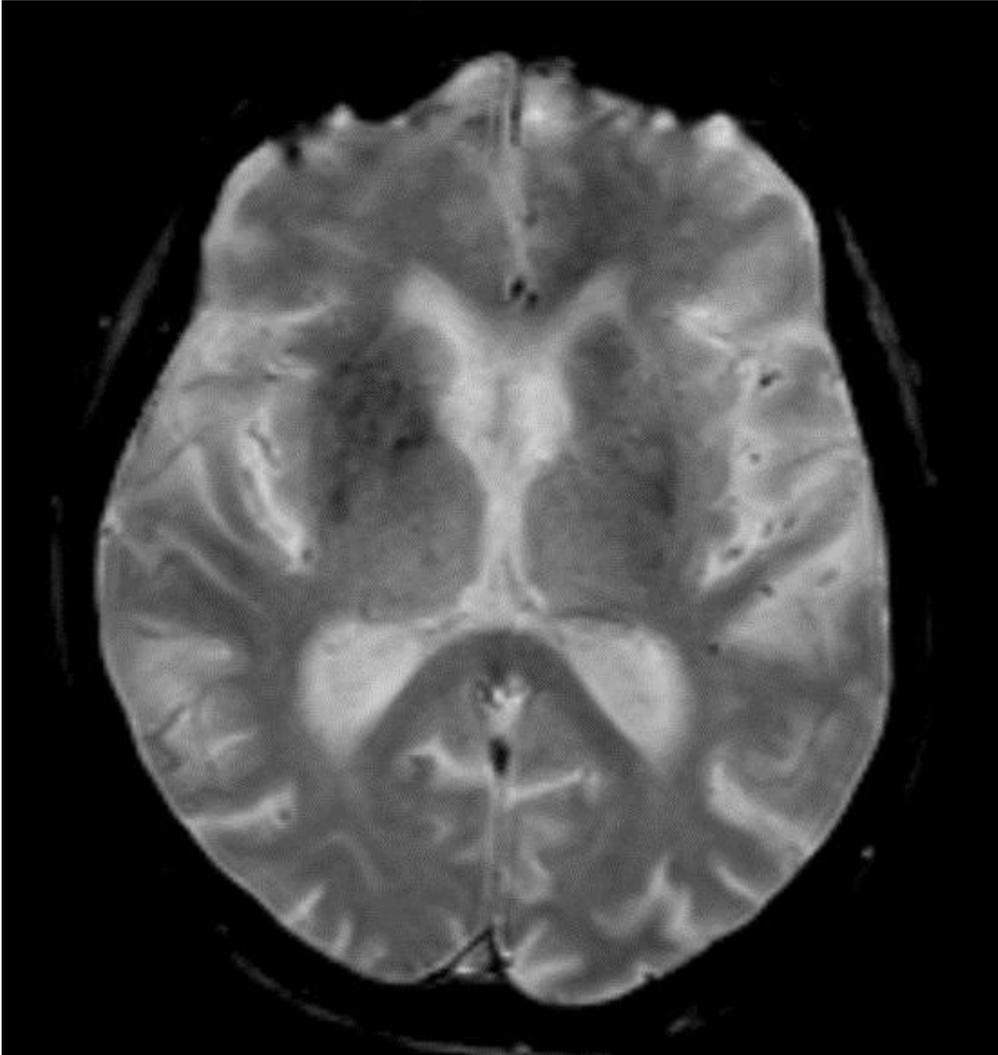
a



Description: T1 sequence hyperintensity of right basal nuclei. **Origin:** © Imaging Diagnostic Department, DIM Clinic, Buenos Aires, Argentina, 2022

Figure 2

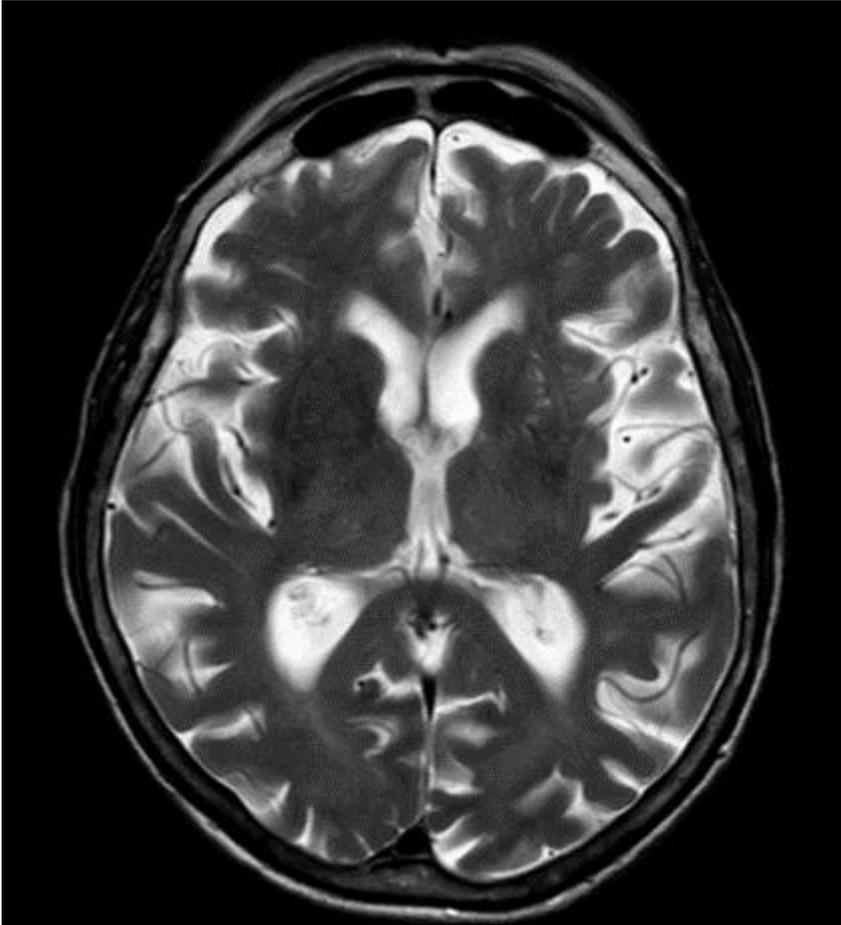
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Description: Magnetic susceptibility sequence (FFE) hypointensity of the right basal nuclei. **Origin:** © Imaging Diagnostic Department, DIM Clinic, Buenos Aires, Argentina, 2022

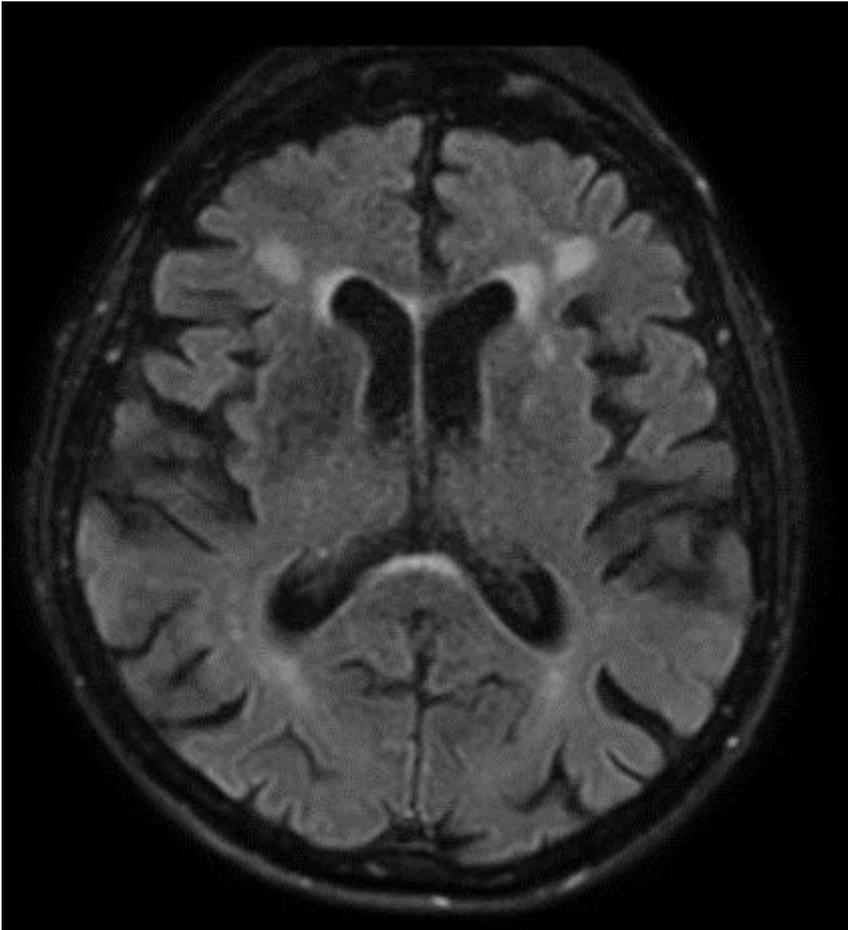
Figure 3

a



Description: T2 and FLAIR sequences with slight signal intensity changes in the basal ganglia, predominantly on the right. **Origin:** © Imaging Diagnostic Department, DIM Clinic, Buenos Aires, Argentina, 2022

b



Description: T2 and FLAIR sequences with slight signal intensity changes in the basal ganglia, predominantly on the right. **Origin:** © Imaging Diagnostic Department, DIM Clinic, Buenos Aires, Argentina, 2022

Figure 4

a



Description: Normal brain MRI angiography 3D reconstruction. **Origin:** © Imaging Diagnostic Department, DIM Clinic, Buenos Aires, Argentina, 2022