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## Lesions in basal ganglia in a patient with involuntary movements as a first sign of diabetes – case report and review of the literature

Monika Bekiesińska-Figatowska<sup>1</sup>, Anna Romaniuk-Doroszevska<sup>1</sup>, Marta Banaszek<sup>2</sup>, Arleta Kuczyńska-Zardzewiaty<sup>2</sup>

<sup>1</sup> Department of Diagnostic Imaging, Institute of Mother and Child, Warsaw, Poland

<sup>2</sup> Department of Neurology, Wolski Hospital, Warsaw, Poland

**Author's address:** Monika Bekiesińska-Figatowska, Department of Diagnostic Imaging, Institute of Mother and Child, Warsaw, Poland, e-mail: m.figatowska@gmail.com

### Summary

We present a case of unilateral hyperdensity of the lentiform and caudate nucleus on CT with hyperintensity on T1-weighted images on MRI in a 71-year-old woman with hemichorea-hemiballism and recently diagnosed diabetes.

**Key words:** hemichorea-hemiballism • brain • computed tomography (CT) • magnetic resonance imaging (MRI)

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### Background

Hemichorea-hemiballism may appear in older patients with hyperglycaemia, mostly unilaterally, although it can be bilateral as well. In neuroimaging studies, it is often accompanied by some characteristic lesions. Their descriptions can be found in a few dozens of patients reported on in the literature [1]. Authors of this study present their own material – there were no Polish reports on this subject in Pubmed database.

### Case Report

A 71-year-old woman presented to hospital with involuntary movements of the right limbs. The movements started suddenly, in the morning, on the day of admission. As the blood tests revealed diabetes (serum glucose concentration of 606 mg%), the patient was admitted to the department of internal medicine. She had a three-year history of hypertension treatment.

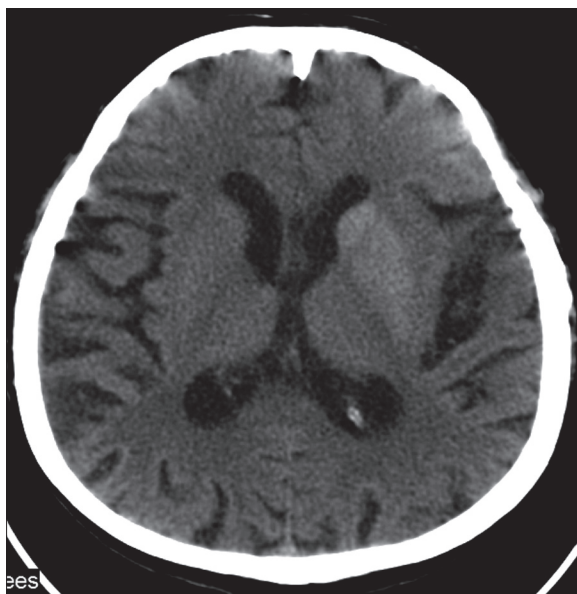
Computed tomography of the head showed hyperdensity of the left lentiform and caudate nucleus (Figure 1). The patient was referred for an MRI which revealed similar results: hyperintensity on SE/T1-weighted images (Figure 2). The lesions were slightly hypointense in other sequences: FSE/T2-, T2flair, DWI and GRE/T2\*- (Figure 3A–D).

Blood glucose level was controlled with an intensive insulin treatment and then with oral antidiabetic medications. Involuntary movements were treated with clonazepam, haloperidol, depakene. Slow regression of movement disorders was observed.

### Discussion

Involuntary movements are mostly present in patients with a previously diagnosed diabetes and are connected with hyperglycaemia (nonketonic one, most of the time). In our case, they preceded the diagnosis of the diabetes. The literature has known such a case already [2]. Involuntary movements are the result of metabolic disorders and tend to subside as soon as glycaemia is controlled.

There are not many causes of a shortened T1 relaxation time of the nervous tissue, expressed by a hyperintense signal on T1-weighted images. They include: iron ions in extravasated blood in the phase of free methemoglobin (in subacute phase of intracerebral haemorrhage), deposits of copper (e.g. in Wilson's disease), of manganese (in hepatic encephalopathy and during parenteral nutrition) and of calcium, fat tissue, melanin (in neurocutaneous melanosis and in melanoma metastases), as well as hamartomas in neurofibromatosis type 1 [3].

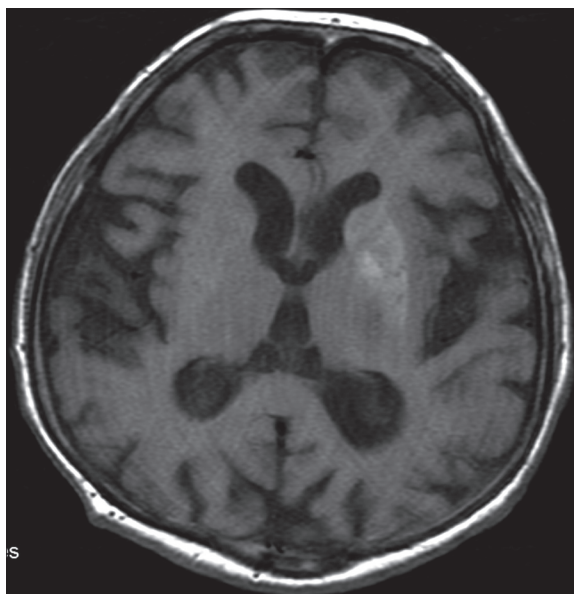


**Figure 1.** CT on the second day of symptoms. Hyperdense lentiform and caudate nucleus of the left hemisphere.

Extravasated blood is also hyperdense on CT, similarly to calcifications. However, they show a different attenuation rate. Fat tissue, on the other hand, reveals negative values of attenuation coefficient and is hypodense on CT.

The presence of manganese depositions in the brain results in movement disorders similar to the ones in our case. Manganese is deposited in the globus pallidus, in pars reticulata of the substantia nigra and, to a lesser degree, in corpus striatum, not only in the course of hepatic encephalopathy and parenteral nutrition but also in professionally-exposed individuals, e.g. miners. The lesions are bilateral, symmetrical. Movement distortions in this case are of extrapyramidal origin. They include hypokinesia, stiffness and tremor [4].

At the beginning, the increased density of lesions within basal ganglia found on CT in patients with hemichorea-hemiballism was ascribed to calcifications [5,6] or hemorrhage to those structures [7]. However, due to the fact that subsequent follow-ups revealed decreased hyperdensity of the involved structures, up to its total resolution, it was concluded that the cause of lesions must be other than calcifications. Hemorrhage to basal ganglia was decided to be the cause of clinical symptoms [7]. After observing discrepancies in the evolution of lesions on CT and MRI, this theory was questioned. Shan pointed to the fact that lesions hyperintense in T1-weighted images were present for years and did not follow a standard evolution of extravasated blood. Brain biopsy in such a patient revealed gliosis with multiple gemistocytes, which, according to the author, is enough to explain the shortened T1 relaxation time [8]. There was also a theory that this is not the bleeding but a short-lasting ischaemia that leads to such an MRI image [8,9]. Finally, it was proved that ischaemia which does not lead to a full stroke results in the deposition of manganese ions in reactive astrocytes and causes a paramagnetic effect [10]. Some authors claim that this explains the diffusion restriction but not the hyperdensity on CT [11].



**Figure 2.** The MRI performed 4 days later. SE/T1 – Hyperintense signal from the lentiform nucleus and a less clear but also hyperintense caudate nucleus.

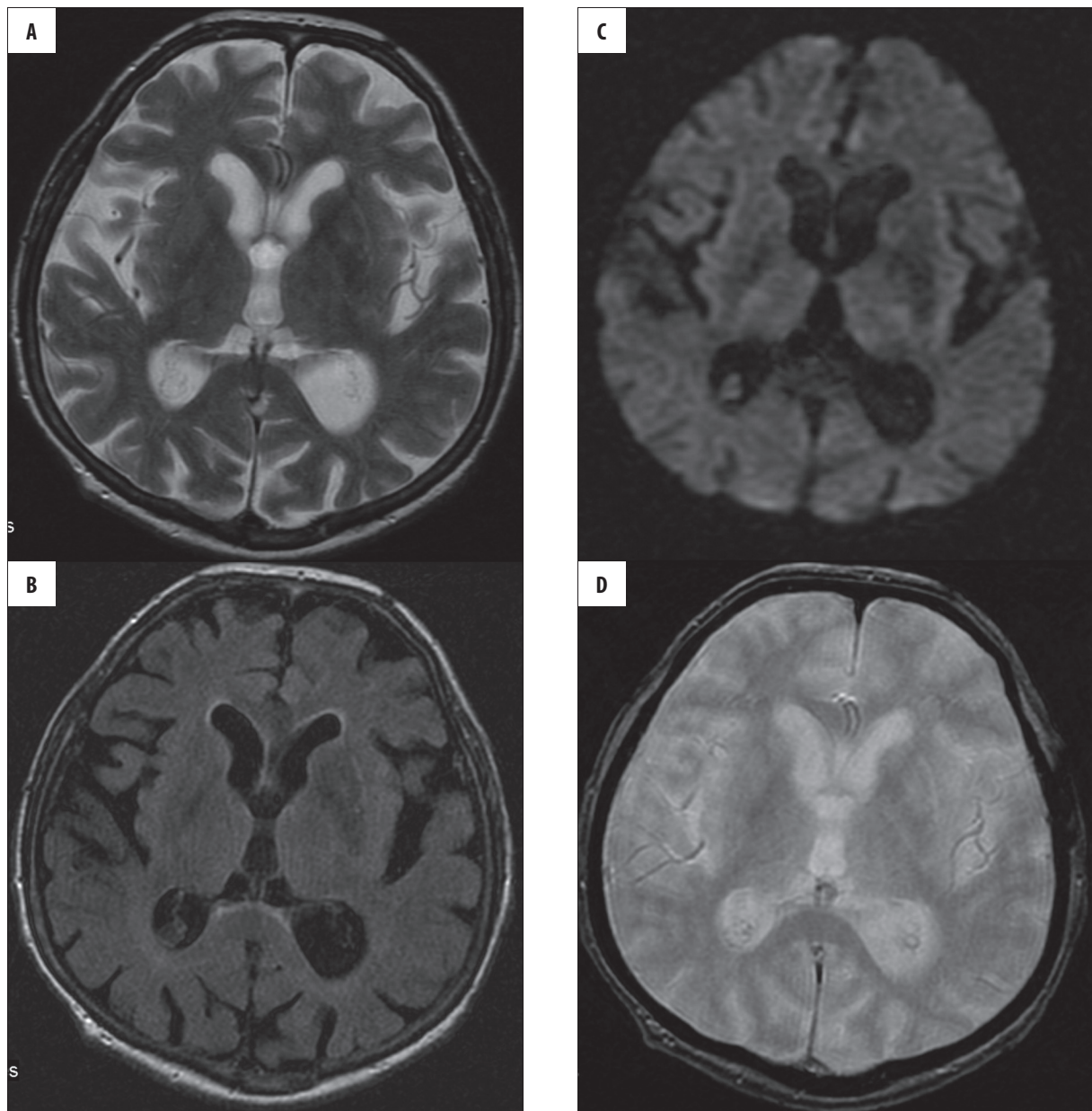
SPECT and PET examinations showed a decreased perfusion/metabolism within the lesions [12,13].

Nath et al. published an extensive report on autopsy findings in one of their patients from which it followed that in case of a classic triad: hyperglycaemia, hemichorea-hemiballism with a sudden onset and hyperintense lesions in the basal ganglia on T1-weighted images, one may find nearly all of the following pathologies in the centralateral hemisphere: a relatively recent infarct, local calcifications, microcalcifications, gemistocytic gliosis [14].

The characteristics of lesions found on CT and MRI are not always the same. All the reports mention an increased signal in T1-weighted images. Some authors report that CT was normal and the lesions were hyperintense in T1-weighted images [12,15]. The lesion is also claimed to have different characteristics in images other than T1-weighted ones. Apart from the cases similar to ours (with a hypointense signal in diffusion-weighted imaging), there are also cases with diffusion restriction [11,12]. There are also publications on hyperintensity on T2-weighted images [7,13]. This may follow from a combination of different anomalies in different ratios in different patients.

An unchanging finding in all patients is the involvement of the lentiform nucleus (putamen). In some of such cases, the lesions can be found in caudate nucleus as well – as it was in our case. Some of the patients present with bilateral lesions and with bilateral involuntary movements. An important feature of this syndrome is the resolution of MRI abnormalities with time and with follow-ups.

Despite multiple attempts, there is still no hypothesis that would unambiguously and coherently account for the type and origin of the lesions: the density on CT with the intensity of signal on MRI at the same time.



**Figure 3.** Discretely hypointense basal ganglia on the left side, within the same layer as in Figure 2. (A) FSE/T2-. (B) T2flair. (C) DWI. (D) GRE/T2\*-.

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