

COMPLEX PARTIAL SEIZURES AND APHASIA AS INITIAL MANIFESTATIONS OF NON-KETOTIC HYPERGLYCEMIA

CASE REPORT

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ABSTRACT - We describe a case of non-ketotic hyperglycemia (NKH), heralded by complex partial seizures and aphasia of epileptic origin, besides versive and partial motor seizures. This clinical picture was accompanied by left fronto-temporal spikes in the EEG. The seizures were controlled by carbamazepine only after the control of the diabetes. A month later, carbamazepine was discontinued. The patient remained without seizures, with normal language, using only glybenclamide. Complex partial seizures, opposed to simple partial seizures, are rarely described in association to NKH. Epileptic activity localized over language regions can manifest as aphasia.

KEY WORDS: aphasia, seizures, diabetes mellitus.

Crises parciais complexas e afasia como manifestações iniciais de hiperglicemia não cetótica: relato de caso

RESUMO - Descrevemos um caso de hiperglicemia não-cetótica (HNC) cujas manifestações iniciais foram crises parciais complexas e afasia de origem epiléptica, além de crises versivas e parciais motoras. Este quadro clínico foi acompanhado por atividade epileptiforme na região fronto-temporal esquerda ao eletrencefalograma. As crises epiléticas foram controladas com carbamazepina (CBZ) apenas após o controle do *diabetes mellitus*. Após um mês, a CBZ foi suspensa, permanecendo a paciente com linguagem normal, sem novas crises epiléticas, em uso apenas de glibenclâmida. Crises parciais complexas, ao contrário de crises parciais simples, são raramente descritas como manifestação de HNC. Atividade epileptiforme nas regiões relacionadas a linguagem podem manifestar-se como afasia.

PALAVRAS-CHAVE: epilepsia, crises epiléticas, diabetes mellitus, afasia.

Non-ketotic hyperglycemia (NKH) is classically associated to simple partial seizures and partial motor *status epilepticus* (SE) and rarely to complex partial seizures (CPS)^{1,2}. We present a case that was heralded by CPS and aphasia of epileptic origin, before systemic signs and symptoms of diabetic decompensation.

CASE REPORT

A 60 year old black woman, right-handed, was brought by relatives to the neurological emergency room because of altered behaviour for a week. She presented irritation and moments of staring, that seemed to "turn off", stopping domestic work. At other times, she stopped speaking, or forgot the names of her relatives and objects, answering only monosyllables. Besides these episodes, she looked completely well. The patient and relatives denied other neurological and systemic complaints, including fever, polyphagia, polydipsia, polyuria and

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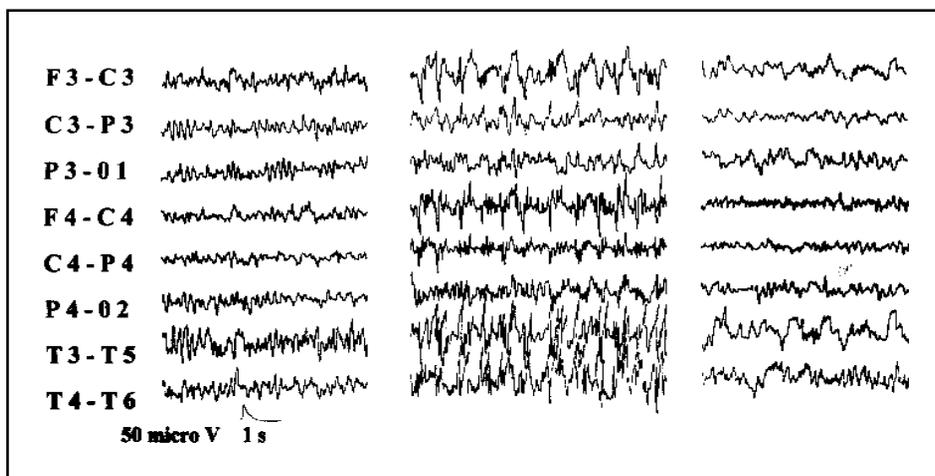


Fig 1. Epileptiform activity in the left fronto-temporal region.

weight loss. She had arterial hypertension, using propranolol irregularly, and diabetes mellitus, with no medication. She showed no abnormalities on general physical examination. Following this, however, she presented indifference and staring, answering only the jargon "eh, eh", attending inconsistently and with a long latency to verbal commands and presented gestural automatisms. She also presented episodes of staring and non responsiveness, followed by a tonic deviation of the head to the right and clonic seizures in the right hemiface. After that, the patient maintained difficult in finding names and a low fluency language for some minutes. She had at least 9 similar episodes, eye-witnessed by physicians. In the interictal period, there were no deficits other than motor aphasia and loss of tempo-spatial orientation.

The glycemia was 455 mg/dl and the seric Na^+ was 136 mEq/l. There were no clinical or laboratorial evidences of infection. A cranial computed tomography (CT) scan without and with contrast resulted normal. A transthoracic echodopplercardiogram showed no abnormalities. A carotid and vertebral doppler ultrasonography showed a medio-intimal thickening in the carotid arteries, bilaterally, and a plaque in the right carotid. A cerebrospinal fluid (CSF) analysis showed 8 lymphomonocytary cells and a glycorrachia of 130 mg/dl. An electroencephalogram (EEG) (Fig 1) showed epileptiform activity initiated in the posterior left temporal region, with recruiting activity, irradiated to the left frontal region, with a subsequent generalization, with spike and wave complexes. Even using 1,200 mg of carbamazepine (CBZ), the patient maintained very frequent similar episodes, that ceased only after a metabolic control with insulin.

The patient persisted for 2 days with low fluency speech, with difficulty in finding names and omitting prepositions - but maintaining a normal comprehension and attending normally to all verbal commands. She was discharged after 3 days without seizures and with normal speech. An EEG, immediately before the discharge, and another one month later, resulted normal. The patient presented no more seizures nor language alterations and, one month after discharge, the CBZ was withdrawn, using only glybenclamide.

DISCUSSION

We considered the patient's staring and gestural automatisms as CPS. The motor aphasia persisted for two days even after controlling the seizures. The episodic presentation, just as the EEG findings and the normality of the CT scan indicated that the aphasia, together with the CPS, were due to recurring epileptic activity localized near language areas.

Even with a diffuse insult to the central nervous system (CNS), clinical manifestations may originate in a specific region. Hyperglycemia has been associated with symptoms of focal neurological dysfunction such as hemianopsias, hemiballism, hemichorea and choreo-athetosis³⁻⁶. Generally,

patients had previous structural lesions in the areas related to these symptoms, perhaps making them more susceptible. The hippocampal regions CA1, CA3 and CA4 are very susceptible to ischemia and metabolic disturbances. Furthermore, systemic administered agents, like pilocarpine or kainic acid, can produce non-convulsive SE, in the form of "limbic" seizures⁷⁻¹⁰. So, limbic regions could be selectively injured, even in a generalized CNS insult, originating the epileptic activity that can lead to CPS. If it really happens, a much higher frequency of this kind of seizure in NKH would be expected than is actually recognized.

The CPS may present as a confusional state and sometimes with subtle manifestations, being difficult to diagnosis. Usually all the clinical pictures is attributed to the precipitating factor¹¹. We can hypothesize that because of this, the reported frequency of CPS is so low. It is very important to recognize this association, due to considerable morbidity, including neurologic sequelae, and even mortality as a result of lasting, non treated seizures¹¹. The case we present, however, was heralded by neurological manifestations, facilitating its diagnosis. It is very well established that aphasia can be the initial, only and sometimes progressive manifestation of epilepsy and focal SE^{2,11-14}. Other focal signs, like amaurosis, disarthria, speech arrest may manifest localized seizures¹⁵⁻¹⁷.

In this patient, we may think that the aphasia could have resulted from a stroke. The hyperosmolarity, leading to elevation of blood viscosity could facilitate a vascular injury^{6,8}. The patient showed no abnormalities in the pre and post-contrast CT scans. However, magnetic resonance imaging (MRI) would be more precise to identify small vascular lesions. Nevertheless, the initially paroxysmal character of the aphasia, its short time course (two days) in close temporal association to CPS and to clonic seizures in the right hemiface and also the EEG findings over regions related to language, very strongly suggest that the aphasia was of epileptic - not of vascular - origin. Embolization from the atheromatous plaque of the right carotid, might not be associated to the aphasia, since in right-handed persons, the dominant hemisphere for language is the left in 99% of times¹⁸.

The versive component of our patient's seizures, to the right, agrees with the left frontal origin of the epileptic activity, just as the clonic component in the right hemi-face. Besides, the EEG showed epileptic activity over this same region, responsible for language¹⁹.

The moderate CSF cellularity we found in this case can be explained by the very frequent seizures, associated to the high seric osmolarity²⁰.

So, we admit that the aphasia occurred due to a compromise of regions specialized in language because of repeated localized epileptic seizures, precipitated and aggravated by the metabolic dysfunction^{7,8,21}. It had persisted for two days after the seizures control, like a Todd's phenomenon, prolonged because of the unfavorable metabolic local environment. Focal SE produces such a severe localized metabolic dysfunction that may lead to cerebral edema detectable by CT scans and MRI, reversible with seizure control²².

We considered that NKH could have facilitated epileptic activity. In the case described, due to its localization in the left fronto-temporal region, it led to CPS, partial motor and versive seizures and aphasia. CPS and aphasia must be remembered as possible initial clinical manifestations of NKH.

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