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KEY WORDS: diabetic striatopathy, hyperosmolar state, Skull CT

ACUTE TOMOGRAPHIC CHANGES OF METABOLIC ORIGIN MIMICKING BASAL GANGLIA HEMORRHAGE, CASE REPORT

Paola Andrea Parra Emergency Department, Instituto Neurológico de Colombia, Medellín, Antioquia, Colombia.

Santiago Vasquez Builes* Emergency Department, Instituto Neurológico de Colombia, Medellín, Antioquia, Colombia. *Corresponding Author

Alejandro Cardozo Emergency Department, Instituto Neurológico de Colombia, Medellín, Antioquia, Colombia.

ABSTRACT

Patients with acute neurological changes, especially with focal neurological deficit, require images for its initial approach, generally skull tomography due to its availability. Here we report a case of an 80-year-old patient with altered state of consciousness and a seizure episode who was admitted on suspicion of cerebral hemorrhage. Her initial blood glucose was high and a hyperdense lesion was found in the right basal ganglion on brain computed tomography, laboratories in which no other metabolic alteration was identified apart from the acute decompensation of Diabetes, however, with a neuroradiology and clinical team, the diagnosis of diabetic striatopathy was made. The patient was treated with a fluid infusion, and serum glucose level was controlled with insulin. The patient gradually recovered consciousness and was alert to his baseline state within 24 hours, without neurological complications.

Patients with risk factors and with findings suggesting stroke, the tomographic high densities, may suggest intracerebral hemorrhage; however, other metabolic and toxic pathologies may have similar tomographic changes.

Our intention, is show to emergency physicians the presence of ganglio basal hyperintensities, mimics for gangliobasal hemorrhage should be studied according to history and clinical context and establish appropriate treatment in a timely manner.

INTRODUCTION

Patients showing acute neurological changes and with the simple skull tomography showing high densities on the basal nuclei, are usually assumed by the emergency department that are passing through an intracerebral hemorrhage; however, some metabolic or toxic emergencies may generate tomographic images similar to hemorrhages that may be misinterpreted.

In diabetes with acute complications, especially in hyperosmolar state, neurological manifestations may be presented, such as altered state of consciousness, focal neurological deficits and abnormal movements being described as hemichorea and hemiballismus.

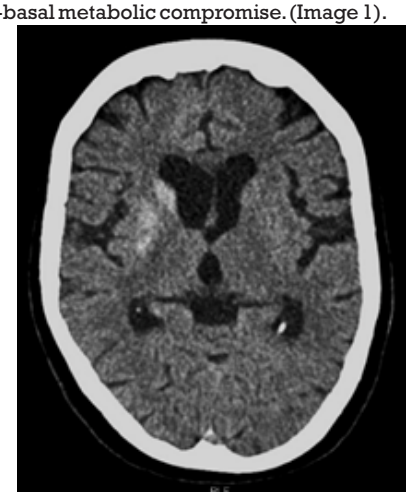
In acute settings, it is logical to consider the stroke as a diagnostic possibility forcing to take images to establish an accurate diagnosis[1].

In tomographic simple or brain resonance studies, it has been documented that patients with hyperosmolar state may have basal ganglion compromise because of the high densities – hyperintensities related to involuntary, continuous and unilateral movement disorders involving the proximal and distal extremities such as Hemichorea or hemiballismus[2]; however, few cases have been described where these movements are absent. Below is documented a case of a patient involved in a consciousness state due to hyperosmolar state and whose image findings suggest diabetic striatopathy.

Case

An 80-year-old, single, childless, female patient, living alone with a history of chronic obstructive pulmonary disease without drug adherence, without frequent medical controls, apparently without any other relevant antecedents, is assessed by domiciliary medicine because her niece found her stuporous at home without knowing the evolution time of her condition, but apparently less than 24 hours. Once she was assessed, it was reported her condition to our institution, so she was admitted to enter in the emergency department. During the transfer, there is evidence of an episode of tonic-clonic movement suggestive of seizure, which is medicated with midazolam.

In the emergency room, her vital signs are interpreted as normal; she is stuporous with Glasgow 7/15, with pinhole pupils slowly reactive and with slight deviation of the lip commissure towards the left. Glucose measurement is taken which resulted in High, so starting 1-liter saline solution and moving to tomography in order to rule out any ischemic or hemorrhagic neurovascular syndrome. The simple skull tomography shows high density at the right striate nucleus territory (caudate and putamen), which respects internal capsule; radiology suggests nucleus-basal intracerebral hemorrhage, but due to our doubts about the respect of the capsule, neuroradiology concept is requested from neuroradiology which considers changes suggestive of nucleus-basal metabolic compromise. (Image 1).



High density in right striated nucleus (caudate and putamen), which respects internal capsule

Subsequently, insulin infusion management is started continuously. The laboratory studies reported: arterial blood gases without acidosis or ketonemia but with

hyperosmolarity, 154.5 sodium, 0.1 ketone bodies, CBC not suggestive of infection, adequate kidney function, her glycosylated hemoglobin (HBA1C) was 17.3, urinalysis with ketone bodies, glycosuria, bacteriuria.

The diagnosis of work: hyperglycemic hyperosmolar state (HHS) with image suggestive of metabolic changes; insulin treatment was continued. At 6 hours her Glasgow improved to 13.

At 24 hours the patient recovers her state of consciousness without any alteration to the neurological examination. At 48 hours she is discharged with subcutaneous insulin and at 96 hours after her discharge, she is appointed for revision and simple skull tomography is taken again showing no changes regarding the previous one. At that time, the patient shows no neurological deficit in her basal state, self-reliant and with glycemic control.

DISCUSSION

Diabetic ketoacidosis and hyperosmolar state were recognized as acute complications of hyperglycemia and insulin deficiency[3-4].

The hyperosmolar state may go on with neurological manifestations, such as altered state of consciousness, weakness, hypotonia, pyramid signs, focal neurological deficit, seizures or other less frequent manifestations such as hemichorea or hemiballismus, generally present when there are imaging changes in the basal nuclei, which are suggested by hyperglycemia[3-4]. These tomographic changes are more frequent in postmenopausal women, with non-insulin-dependent diabetes mellitus and from the seventh decade of life[5].

Other causes described about the hyperkinetic movements are the ischemic, hemorrhagic stroke, or brain tumors[6].

Imaging findings associated with HHS can be seen in the simple skull tomography (CT) or in the magnetic resonance imaging (MRI). In the CT is observed a high density area in basal ganglia, especially in putamen and sometimes in the caudate, respecting the internal capsule. The MRI usually shows high density in T1, intensity of variable signal in T2 and diffusion restriction in DWI. Typically, these manifestations are contralateral by the side of clinical presentation of the patient[7].

The image alterations may lead them to be mistakenly interpreted as intracerebral hemorrhage predominantly of basal ganglion, asymmetric calcification of hemoderivatives or mineralization of venous abnormalities. However, unlike the HHS-induce basal ganglia changes, the acute basal ganglion hemorrhage does not respect the anatomic limits, compromising the internal capsule, causing peripheral edema and displacement of brain parenchymal by effect of mass[2,8].

Since the physiopathologic point of view, different hypothesis account for such neuroimaging phenomenon. It has been proposed that the high glucose levels in plasma are directly related to events of secondary cerebral hypoperfusion to hyperviscosity and activation of anaerobic metabolism, with the consequent production of Gamma-amino butyric acid (GABA) as a source of energy[9].

Another possible hypothesis states that due to multiple ischemic changes and metabolic disorders at brain level, there is an excessive formation of gemistocites (reactive forms of astrocytes), gliosis and selective neuronal loss[7,9].

There are no typical neurologic findings in the hyperosmolar state. Although the presence of abnormal movements makes part of the clinic, not always are they present to be suggestive of a metabolic complication[10].

Changes in tomographic high densities of the basal nuclei, in acute context, have also been described in other metabolic conditions or by consumption of toxics, approach of which depends according to the clinic context and the associate conditions. Imaging changes are also described in methanol intoxication, carbon monoxide, hepatic encephalopathy changes and parathyroid compromise[11].

It is important to mention that patients may be clinically recovered with persistence of imaging alterations that may be evident even up to some years[9].

CONCLUSION

Doctors treating metabolic emergencies and neurologic symptoms must be aware that imaging changes in simple skull tomography or brain magnetic resonance, which respect the anatomic limits and are not associated with cerebral edema, may be a consequence of the metabolic emergency, and may persist even if the patient fully improves its symptomatology. In our case, the radiological clinical association suggested a diabetic striatopathy, which if correctly managed in the emergency room, may lead to and early prognosis in spite of the pathologic image persistence.

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