Nonketotic hyperglycemia with involuntary movements

Dear Editor,

A 61-year-old woman who had been using insulin irregularly for the treatment of type II diabetes presented with hemichorea-hemiballism that had appeared suddenly in the left arm and left leg two weeks prior. Blood tests showed a blood glucose level of 450 mg/dL, a creatinine level of 0.9 mg/dL, and a urea level of 38 mg/dL. The complete blood count showed no abnormalities. The cerebrospinal fluid glucose concentration was 350 mg/dL. Magnetic resonance imaging (MRI) revealed a right-sided lesion, showing a hyperintense signal on T1-weighted images and a slightly hyperintense signal on T2-weighted images, located in the region of the caudate nuclei and putamen, with no enhancement, no evidence of bleeding in the magnetic susceptibility-weighted sequences, and no restricted diffusion on diffusion-weighted imaging (Figure 1). These imaging findings, together with the clinical and biochemical history, confirmed the diagnosis of hemichorea–hemiballism due to nonketotic hyperglycemia.

Nonketotic hyperglycemia, also known as diabetic striatopathy, is a rare cause of involuntary movements as a primary manifestation of diabetes mellitus; it mainly affects elderly individuals, presenting as the triad of hemichorea-hemiballism, hyperglycemia, and a lesion in the basal nuclei showing a hyperintense signal on T1-weighted images[1]. Clinical and imaging findings are typically unilateral, although they can be bilateral in up to 11.4% of cases[2], being potentially reversible and usually resolving within 2–12 months after the treatment of hyperglycemia[3,4].

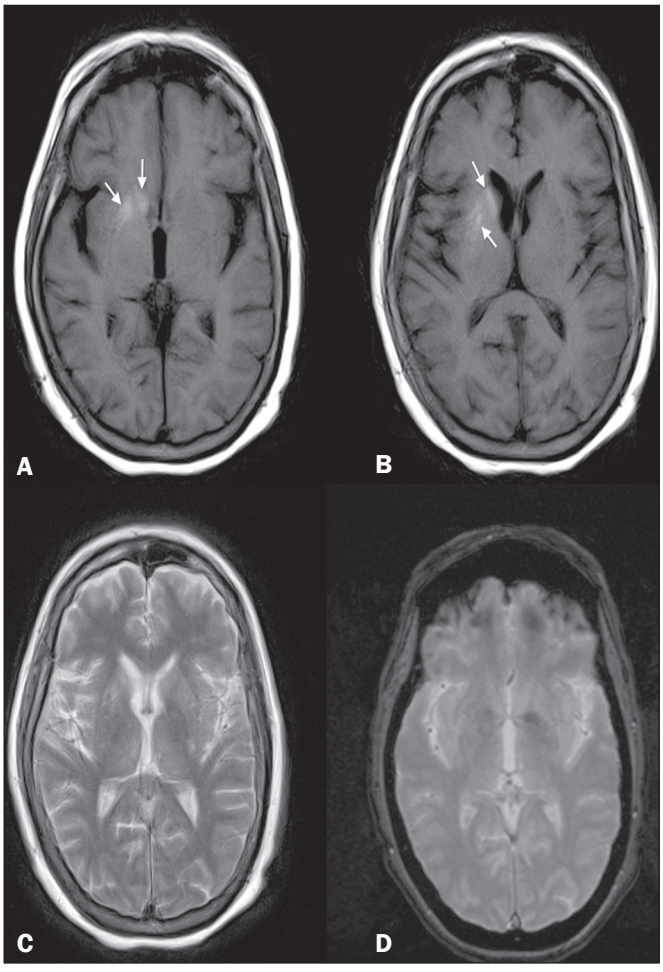

Figure 1. A,B: T1-weighted MRI showing a right-sided lesion with a hyperintense signal in the caudate nuclei and putamen (arrows). **C:** T2-weighted MRI showing a slightly hyperintense signal in the same regions. **D:** T2*-weighted MRI showing that there was no blood deposition in those regions.

Although the pathophysiology of nonketotic hyperglycemia is unknown, potential mechanisms include metabolic changes such as the deposition of proteins and of degradation products of myelin, blood, calcium, or other minerals, which tend to decrease as serum glucose is controlled[5]. Another accepted theory is that a hyperglycemia-induced change in perfusion results in reduced Krebs cycle activity, inducing anaerobic metabolism, causing the brain to use alternative sources of energy, and metabolizing the gamma-aminobutyric acid (GABA) inhibitory neurotransmitter. In nonketotic hyperglycemia, GABA and acetate levels drop rapidly, leading to a decrease in acetylcholine synthesis. It has therefore been speculated that the reduced levels of acetylcholine and GABA in the basal nuclei leads to dysfunction of those nuclei, thus producing involuntary movements such as those seen in chorea-hemiballism[6,7].

For the evaluation of central nervous system diseases, the imaging method of choice is MRI[8–14]. In hemichorea-hemiballism due to nonketotic hyperglycemia, MRI findings are characterized by lesions in the region of the caudate nucleus or lenticular nucleus, showing hyperintense signals in T1-weighted sequences and discretely hyperintense signals in T2-weighted sequences, without enhancement or diffusion restriction, such lesions typically being unilateral[1,3,4], as in the case presented here. The diagnosis of lesions with high signal intensity in T1-weighted sequences of the region of the basal nuclei is broad; the following can be cited as some of the main causes[1,4]: hepatic encephalopathy; prolonged exposure to manganese; prolonged parenteral nutrition; Wilson's disease; subacute intracerebral hemorrhage; exogenous carbon monoxide toxicity; and exogenous methanol toxicity. Correlation with the clinical and biochemical data is fundamental to making the definitive diagnosis[1,4].

In conclusion, although the occurrence of hemichorea-hemiballism as a complication of uncontrolled diabetes is uncommon, the diagnosis should be considered when the clinical and MRI findings are characteristic of the disease. Thus, delays in the initiation of appropriate treatment can be avoided.

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http://dx.doi.org/10.1590/0100-3984.2015.0253

Intestinal perforation: an unusual complication of barium enema

Dear Editor,

An 83-year-old female patient complaining of constipation was referred to our institution for elective enema with barium contrast, which showed diffuse irregularity in the mucosal folds of the colonic loops and signs of extravasation of the contrast medium into the abdomen and pelvic cavity (Figure 1). After the examination, the patient remained stable, without additional complaints. However, she did not agree to being hospitalized, signing a waiver. Despite being informed of the risks, she remained resolute, promising to return if there were any symptoms. She subsequently returned to the hospital with an acute abdomen, at which time she underwent computed tomography of the abdomen for preoperative evaluation, which demonstrated abdominal wall hernias, diverticulosis of the sigmoid colon, and a large amount of contrast material distributed diffusely throughout the peritoneal cavity and the hernias (Figure 2). The main hypothesis was perforation of the wall of the gastrointestinal tract by the enema. The patient underwent exploratory laparotomy, with an inventory of the abdominal cavity, which confirmed the tomography findings and identified a laceration at the rectosigmoid junction. After 14 days in the intensive care unit, the patient died.

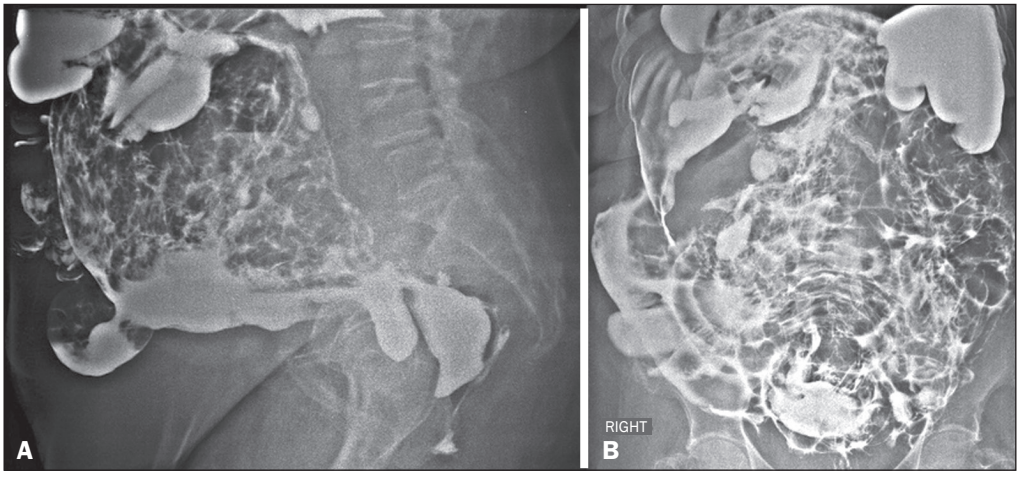

Figure 1. Images acquired during barium enema examination, in lateral (**A**) and anteroposterior (**B**) views.

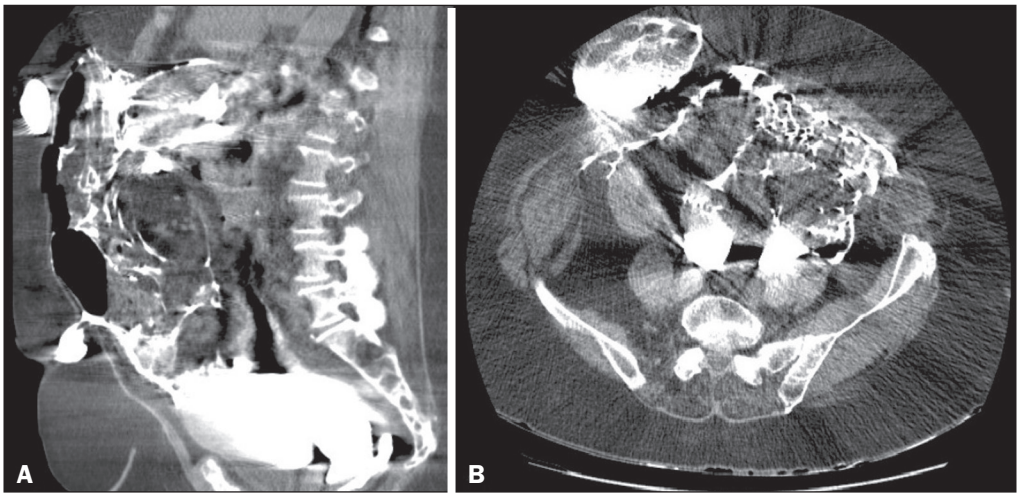

Figure 2. Computed tomography scans of the abdomen, in the axial (**A**) and sagittal (**B**) planes.