

Case Report

Hyperosmolar Cerebral Tissue Contraction: Dehydrated Brain Sign

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Case Report

A 74 year-old male alcoholic patient was brought to hospital with acute confusional state and later stupor, after 2 weeks with asthenia and changes in behavior. A hyperosmolar non-ketotic hyperglycemia (glycemia: 86.5 mmol/L; sodium: 171 mEq/L) by a secondary diabetes due to acute pancreatitis. Vital support measures, i.v insulin infusion, i.v. hypotonic saline, and antibiotics were applied. Two computed tomography (CT) scans of the skull were obtained: when first admitted and after 48 hours of evolution. The former showed slight bilateral striatal hyper density, and the latter revealed a bilateral symmetrical retraction of cerebral tissue in frontal and temporal regions compared to the first one (Figure 1). The patient died of a pulmonary embolism a week later due to acute pancreatitis was detected.

Discussion

Nonketotic hyperosmolar hyperglycemia and diabetic ketoacidosis may produce neurological disorders (hemichorea-hemiballismus, hemiparesis, hemianopia, seizures, and coma) by different ways: intracellular dehydration from hyper osmolality, reactive oxygen species generation, altered neurotransmitter function, ischemia, or altered intracellular enzyme mechanisms [1,2]. They also produce typical imaging findings: focal cortical-subcortical increased magnetic resonance (MR) signal on diffusion-weighted sequences due to edema caused by osmotic dysfunction, T2-weighted MR sequences hypo intensity due to increased plasma ferritin and serum iron with end-

organ iron deposition, and CT scan hyper density of the striates areas due to hypertrophic astrocytes proliferation after small ischemic lesions, which produces chorea and ballismus [1-3].

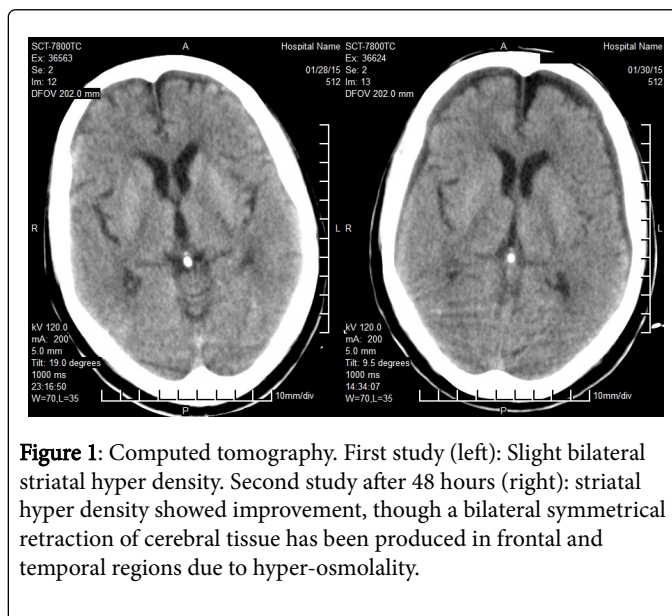


Figure 1: Computed tomography. First study (left): Slight bilateral striatal hyper density. Second study after 48 hours (right): striatal hyper density showed improvement, though a bilateral symmetrical retraction of cerebral tissue has been produced in frontal and temporal regions due to hyper-osmolality.

Conclusion

The CT scan findings in the present case demonstrate that tissue dehydration plays an important role in cerebral dysfunction in patients with hyperglycemia.

References

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