Intracranial Bilateral Symmetrical Calcifications in Altered Mental Status*

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A 44-year old female was brought to emergency department due to fever and loss of consciousness. Glasgow Coma Score was 4 ($E_1M_2V_1$) upon arrival. Arterial blood pressure was 100/70 mmHg, pulse rate 110 beats/min, respiratory rate 10 breaths/min and body temperature was 38.6 °C. Difficulty in respiration with rough rales in all zones was found. Babinski's sign was bilaterally positive. The patient had no operation or trauma history. She had a habit of drinking boiled ginger water, amount of which could not be elicited. She had presented to various hospitals three times with different reasons such as dyspnea, nausea, dyspepsia, agitation, headache, and altered mental status during past one month. She could not maintain herself and has decrease in perception almost with agitation since she had an ischemic cerebrovascular accident one year ago. She was also diagnosed with nontoxic multinodular goiter and was on L-thyroxin regimen for a long time.

The serum biomarkers showed elevated liver enzyme levels, (SGOT 4300 IU/L; range <31, SGPT 2600 IU/L; range <34), elevated CK (13368 IU/L; range 34-145), hypocalcaemia (5.4 mg/dL; range 8.6-10.2) and hyperphosphatemia (5.0 mg/dL; range 2.3-4.5). On chest X-ray, a suspicious heterogeneous increased density was observed on the left paracardiac area. Abdominal ultrasound showed no pathology. Cranial computed tomography revealed diffuse calcifications in cerebellum, together with symmetrical calcifications in bilateral basal ganglia, and in parenchyma (Fig. 1). She was intubated and hemodynamically stabilized. Urine analysis revealed pyuria but urine and blood cultures were negative. Analysis of cerebrospinal fluid sample was normal. Intact parathyroid hormone level was 120.2 pg/mL (reference range, 15-65 pg/mL).

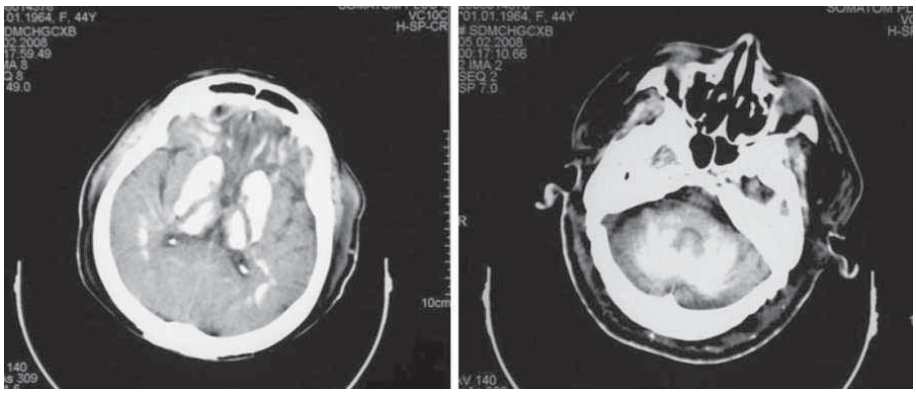

Fig. 1. *Cranial computed tomography revealed bilateral symmetric calcifications in basal ganglia and diffuse cerebellar calcifications.*

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Diagnosis

Pseudohypoparathyroidism

The initially results of laboratory tests were consistent with hypoparathyroidism which was probably secondary to undiagnosed chronic hypoparathyroidism in this case. Varied neurological manifestations are observed in patients with parathyroid hormone disorders. Convulsions and tetany are the most common symptoms.[1] Chronic hypoparathyroid syndrome is characterized by the presence of extra pyramidal signs (dyskinesia, choreiform, and athetoic movements, tremors, and rigidity), cerebellar manifestations (dysarthria, ataxia), psychosis, and epilepsy.[2]

In progress, the patient was empirically treated with ceftriaxone 1 gr/bid plus metranidazole 500 mg/bid, Ca-gluconate 20 mg/bid via intravenous infusion and calcitriol 0.5 mcg/bid via nasogastric tube. Fluid and electrolytes were replaced appropriately together with daily supportive treatment. At the end of fifth day, her GCS was improved to $E_3M_4V_1$ and she began to respond to commands with her eyes. Her fever was controlled and liver enzyme levels approached to upper limit of normal ranges before she was extubated on 6th day.

In this case, the clinical picture was very complex. According to patient history, this clinical picture is chronic. Thus, authors thought acute liver failure, suspicious pneumonia and urinary tract infection diagnosis were the reasons of altered mental status and fever. We also suggested that, chronic pseudohypoparathyroidism may be contributed to the clinical picture.

The purpose of this presentation was to remind that in patients with altered mental status, hypocalcaemia and intracerebral calcification, pseudohypoparathyroidism should be kept in mind as a sole cause or contributor factor (as in our case) of altered mental status.

References

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