Idiopathic Hypoparathyroidism (IHP) Presenting as “Schizophrenia:” A Case Report

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ABSTRACT

Idiopathic hypoparathyroidism (IHP) with the onset of psychosis is a rare case in the psychiatric clinic. In this case report, we summarize the three facets of IHP, which contains the clinical, biochemical, and radiological features. Besides, the differential diagnosis of this case is supposed to be a highlight that IHP could have the main complaints of psychotic symptoms and featured signs on neuroimaging manifestation.

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Key words: Hypoparathyroidism, schizophrenia, calcification basal ganglia

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Introduction

Hypoparathyroidism is an uncommon disorder of calcium metabolism characterized by hypocalcemia and hyperphosphatemia, which are due to impaired parathyroid hormone (PTH) secretion or action (1). According to the diverse causes, hypoparathyroidism contains several types. Idiopathic hypoparathyroidism (IHP) is diagnosed after exclusion of other known causes and usually has a genetic etiology that is related to the antibodies against calcium-sensing receptor (2). The common clinical manifestations of hypoparathyroidism include tingling and numbness, carpopedal spasm, seizures, and neurocognitive dysfunction (3). So, in this report, we present a relatively rare IHP case associated with psychosis manifestations.

Case

A 29-year-old married female was admitted to the hospital in December 2011 with the symptoms of glibberish, persecution

thoughts, and hallucination. Her medical history could trace back to 2009. At that time, she just felt numb from time to time at the beginning and then had the onset of seizures, which were still under the consciousness. However, she did not pay much attention. Gradually, she told her families that she heard somebody talk to her (when she was alone) and that somebody would kill her. She also combined the behavioral disorders at the same time. Under this kind of condition, she was referred to the hospital for further evaluation and treatment. Nevertheless, her past medical information and past laboratory data were not available.

On the day of admission, there were no abnormal findings in the physical examination, especially no brachydactyly and subcutaneous calcification. Her consciousness was alert, and no mental retardation was noted. The neurological examinations were shown as follows: the excitability of muscle was rising; Trousseau sign was positive; Chvostek's sign was negative, and there was no onset of seizures. Deep tendon reflexes were all decreased and no pathological reflexes were detected. The laboratory data (Table 1) revealed severe hypocalcemia, hyperphosphatemia, and increased creatine kinase (CK). Computed

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Figure 1. Non-contrast brain computed tomography scan showing bilateral symmetrical calcifications located at the cerebellum (dentate nuclei) and in the basal ganglia (putamen and caudate nucleus)

Table 1. The laboratory data of pretreatment and post-treatment

Parameters	Pretreatment	Post-treatment
Ca (mmol/L)	1.31	2.07
P (mmol/L)	2.04	1.91
Mg (mmol/L)	.75	.96
PTH (pmol/L)	<.32	<.32
CK (U/L)	755	116

tomography scan showed extensive brain calcification, especially in the basal ganglia and cerebellum (Fig. 1). According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) about idiopathic hypoparathyroidism, the replacement of calcium and vitamin was then prescribed.

Discussion

The most common manifestations of the central nervous system in IHP are focal or generalized seizures, which occur in as many as 40% (4). The peripheral nervous system presentation includes muscle weakness, positive Trousseau sign or Chvostek's sign, and tetany (5). The other clinical features in this case are consistent with hypocalcemia and neuromuscular dysfunction. However, the various psychotic manifestations observed in other series are not similar to our case (6). In this case, the patient's psychotic symptoms were the main manifestation and easily confused with the diagnosis of schizophrenia. So, the special facet of this case was the differential diagnosis, which makes residents confused the most.

When it comes to the differential diagnosis, this case reminds us to compare it with Fahr's disease (FD), which also presents with progressive neuropsychiatric symptoms and calcifications of basal ganglia. There is some evidence that supports the difference between these two diseases: first, the patient with FD usually has a family history (7), second, FD is typically with an onset age of 30-60 years (8), and third,

FD does not show hypocalcemia and seizures caused by hypocalcemia.

From this case, we ponder three pinning points. First, we consider if the accumulation of calcium induces the irreversible and permanent damage of the brain, especially specific regions. Second, compared with other IHP patients, it is unknown whether the main symptom is under the background of genetics or not (9), such as the CACNA1C genotype, a subunit of the L-type voltage-gated calcium channel (10). Finally, a computed tomography scan allowed earlier diagnosis, with high sensitivity and specificity. The extent of calcification is variable, depending on the stage of the disease, duration of metabolic abnormalities, and volume of calcium deposit.

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