Clinical Case Report

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Misdiagnosis of idiopathic hypoparathyroidism

A case report and literature review

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Abstract

Rationale: Idiopathic hypoparathyroidism (IHP) is a rare endocrine condition, which is frequently represented by neuropsychiatric disorders. Hence, the misdiagnosis rate of the disease is rather high, especially for neurologists.

Patient concerns: We reported a case of misdiagnosed, atypical IHP. In addition, the literature on IHP and the misdiagnosis published in China in the past 2 decades has been reviewed and summarized.

Diagnoses: Blood testing confirmed that parathyroid hormone (PTH) = 0 pg/mL and the final diagnosis was IHP.

Interventions and outcomes: With calcium and vitamin D supplementation, the patient's myasthenia improved significantly, and muscle enzymes returned to normal gradually. One-year follow-up demonstrated that the patient's myasthenia disappeared, and the blood calcium and PTH levels were normal. In addition, the literature on IHP and the misdiagnosis published in China in the past 2 decades has been reviewed and summarized.

Lessons: The misdiagnosis rate of IHP in China was high in the past 2 decades, which might be attributed to the misdiagnosis as epilepsy or mental diseases. A clinician should be able to understand the disease and emphasize the screening of high-risk population, especially for those patients with hypocalcemia, hyperphosphatemia, and increased blood creatine kinase with unknown causes or nontypical clinical symptoms.

Abbreviations: CK = creatine kinase, CT = computed tomography, IHP = idiopathic hypoparathyroidism, PTH = parathyroid hormone.

Keywords: hypoparathyroidism, misdiagnosis, neuropsychiatric disorder

1. Introduction

Idiopathic hypoparathyroidism (IHP) is a rare endocrine disease, with highly variable symptoms and signs. It is frequently represented by neuropsychiatric disorders, and hence, is liable to misdiagnosis. Here, we report 1 case of misdiagnosed, atypical hypoparathyroidism. As epidemiological data on a misdiagnosis of this disease are unavailable in China or globally, we reviewed the literature on hypoparathyroidism and its misdiagnosis published in China from 1994 to 2013, in order to aid the clinicians, especially neurologists to accurately diagnose this condition.

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The authors report no conflicts of interest.

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2. Case report

A 37-year-old woman of Han ethnicity was admitted to PLA 44 Hospital on February 5, 2013, because of limb debilitation and predisposition to fatigue for 4 years and aggravation for 2 months. The patient visited another hospital of the city in 2010 for limb debilitation. Routine examinations revealed a blood creatine kinase (CK) of up to 4000 U/L, normal CK isoenzymes, and electromyogram; no muscle biopsy was performed. The level of blood calcium and parathyroid hormone (PTH) was unknown. The patient was diagnosed with polymyositis. Her limb debilitation improved after 1 month treatment with glucocorticosteroids, which was gradually reduced in half a year. Nevertheless, her symptoms worsened thereafter. Although 2 months before admission, her symptoms of myasthenia were aggravated and she experienced fatigue while walking and laboring, but without obvious dead limbs, muscle spasm, myalgia, babbling, or unconsciousness. No body weight change was observed in the course of disease. She had no history of thyroid disease, parathyroid disease, cancer, surgery, and family history of hereditary disease.

Physical examination revealed the data on the following parameters: Temperature: 36.5°C; Pulse: 90 bpm; Respiration: 22 bpm; Blood Pressure: 110/80 mm Hg; normal development, moderate nutrition status, consciousness, voluntary body position, no jaundice of the skin, petechia, or rashes. The skin was not pigmented, as well as the hair and eyebrows were normal. No pharyngeal hyperemia was observed. Her neck was flexible, and the thyroid gland was not enlarged. Her breathing sounds were normal in both lungs, and no rales were heard. The heart rate was

90 bpm with regular rhythm and no murmurs. Her abdomen felt soft, with liver and spleen not palpated beneath the costal margin. Any edema in the lower limbs was not observed. A neurological examination revealed an involuntary tic of the eyelids and lips, normal eyeball movements, 2 pupils of the same size with normal light reflex, symmetrical facial skin folds, normal hearing of both ears, soft palate with normal mobility, the uvula in the neutral position, existence of pharyngeal reflex, and tongue in the neutral position when protruded. No obvious amyotrophy was noted in the limbs, and normal limb muscular strength (of 5), normal superficial and profound sensation being symmetrical in the limbs, physiological reflexes present, and pathologic reflexes absent. Laboratory tests revealed CK: 1070 U/L, CK-MB: 7 U/L, troponin: 1.25 ng/mL, lactate dehydrogenase: 525 U/L, hydroxybutyrate dehydrogenase: 343 U/L, blood calcium: 1.68 mmol/L, phosphate: 1.79 mmol/L, potassium: 3.7 mmol/L, sodium: 143 mmol/L, chloride: 100 mmol/L, magnesium: 0.54 mmol/L. The electrocardiogram suggested prolongation of the Q-T interval. The test results were normal for hepatic and kidney function, blood gas analysis, rheumatic and immunologic panel, blood glucose, C-peptide, diabetes antibodies, and thyroid function. The computed tomography (CT) scans of the head and chest, electroencephalogram, and electromyogram were normal. Muscle biopsy found that the skeletal muscle specimen had muscle fibers of similar size; the most striated muscles were distinctly delineated, a few muscle fibers were thinned and faintly stained, and infiltration of a small number of lymphocytes was seen between muscle fibers. The clinical diagnoses were myasthenia and muscle enzyme elevation with suspected causes such as metabolic myopathy or others. The patient was then transferred to another hospital for muscle-specific staining, but no abnormality was detected. Subsequent testing confirmed that PTH = 0 pg/mL and the final diagnosis was hypoparathyroidism. With calcium and vitamin D supplementation (calcium carbonate D3, oral, 0.6 g/time, three times per day; Alfa collagen soft capsule, oral, 0.5 μg /time, one times per day), the patient's myasthenia improved significantly, and muscle enzymes returned to normal gradually in a year. A recent follow-up in 2015 demonstrated that the patient's myasthenia disappeared, and the blood calcium and PTH levels were normal.

Approval for the study by the Ethics Committees was not required because it was a case report. Written informed consent was obtained from the patient for publication of this case report.

Table 1

Case numbers and misdiagnosis rates of hypoparathyroidism (1992–2012).

	1994–1998	1999–2003	2004–2008	2009–2013	Total
Case number	63	154	373	430	1020
Number of misdiagnosed cases	10	46	130	115	301
Misdiagnosis rate	15.87%	29.87%	34.85%	26.74%	29.51%

3. Literature review

3.1. Materials and methods

3.1.1. Sources and screening of the literature. The literature was retrieved using the China Biology medicine disc (199411–201311) produced by the Institute of Medical Information of the Chinese Academy of Medical Sciences, with hypoparathyroidism as the indicator. The literature was not available publicly and review articles were excluded. The case reports covering various periods from the same hospital, nonoverlapping cases, and overlapping cases with the longest period and the largest case number were included. A total of 115 articles[1–115] were retrieved, which were about hypoparathyroidism and its misdiagnosis that were published in China between January 1994 and January 2013. All data were calculated with the actual reported case numbers.

4. Results

4.1. Literature reported case numbers and misdiagnosis rates

In the past 2 decades, the literature reported a total of 1020 hypoparathyroidism cases and 301 misdiagnosed cases, and the misdiagnosis rate was 29.51% (Table 1).

4.1.1 Misdiagnosed disease entities and their constitution

We reviewed 63[7–11,16–19,24–26,30–33,41–57,60–63,66–70,76–78,82,83,90–93,96–98,102–104,110–115] articles addressing the misdiagnosed disease entities explicitly (Table 2). Of these, epilepsy was most frequently misdiagnosed (60%), followed by hypocalcemia (12.95%), and neurosis and mental disorders (6.64%). Other less frequently misdiagnosed diseases include polymyositis, purulent

Table 2

Analysis of misdiagnosed disease entities.

Entity	Number of misdiagnosed cases	Percentage (vs total case number)	Percentage (vs misdiagnosed case number)
Epilepsy	179	17.55%	59.47%
Hypocalcemia	39	3.82%	12.95%
Neurosis and mental disorders	20	1.96%	6.64%
Cerebral palsy	14	1.37%	4.65%
Cerebrovascular disease	10	0.98%	3.32%
Convulsion	7	0.69%	2.33%
Viral encephalitis	4	0.39%	1.33%
Rickets	4	0.39%	1.33%
Parkinson disease	3	0.29%	0.99%
Vit D deficient carpopedal spasm	3	0.29%	0.99%
Encephalopathy and Vit D deficient rickets	2	0.20%	0.66%
Fahr disease	2	0.20%	0.66%
Toxic encephalopathy	2	0.20%	0.66%
Others	12	1.17%	3.99%

meningitis, tuberculous meningitis, pneumonia complicated with higher fever and convulsion, epilepsy with viral encephalitis, epilepsy with tuberculous cerebritis, parasitic disease, mesenteric lymph node inflammation, rheumatoid arthritis, delayed vitamin K deficiency, coronary artery disease, dilated cardiomyopathy, cervical spondylosis, Meniere disease, migraine, malnutrition, motor neuron disease, peripheral polyneuropathy disease, and viral myocarditis. Each of these entities was diagnosed in 1 case (19 cases in total).

5. Discussion

Hypoparathyroidism is a metabolic disorder, resulting from inadequate synthesis or secretion of PTH, or biologically inactive circulating PTH, or insusceptibility of target tissues and cells to PTH. The clinical manifestations are a carpopedal spasm, epilepsy-like seizures, hypocalcemia, and hyperphosphatemia. Hypoparathyroidism is characterized by many, highly variable symptoms and signs. The early manifestations are usually nonspecific; thereby, misdiagnosis is predisposed if a clinician or neurologist fail to recognize the condition. With respect to the current case report, some neurologists knew only little about this disease. Some nonspecific symptoms of the case were outstanding such that the clinician excluded or overlooked the abnormal auxiliary tests results, such as hypocalcemia, hyperphosphatemia, and symmetrical calcifications in bilateral basal ganglia, causing misdiagnosis for several years. Thus, PTH should be highlighted to avoid misdiagnosis.

We reviewed the literature on hypoparathyroidism and its misdiagnosis published in China in the past two decades, and summarized as follows.

IHP is a systemic disease, and its incidence is 0.55 to 0.88/100,000 (mean, 0.73/100,000) in China, similar to that reported in Japan.[116] The misdiagnosis rate of IHP was reported as 83.3%.[117] According to the present literature review, the misdiagnosis rate was 29.51% in the past 2 decades and 26.74% in the past 5 years, probably showing a tendency of decline, despite at a high level.

According to our literature review, 301 cases of hypoparathyroidism were misdiagnosed in China in the past 2 decades. Of these, 179 cases were misdiagnosed as epilepsy (59.79%). Carpopedal spasm is the most common symptom of hypoparathyroidism. The severe cases manifesting systemic skeletal and smooth muscle spasticity may be diagnosed as various types of epilepsy-like seizures,[118–120] and the literature reported incidence was 40% to 80%.[121] As the electroencephalogram varied between hypoparathyroidism and epilepsy patients, although insignificantly, and also as anti-epileptics can control the seizures temporarily, hypoparathyroidism is liable to be misdiagnosed as epilepsy. In hypoparathyroidism patients, the incidence of mental symptoms is high. For instance, Bronsky et al[122] reported an incidence of 25%. The primary symptoms of the mental disorder include restlessness, depression, insomnia, and excessive dreams, and the symptoms may worsen or improve with the season, mood, tiredness, and menstrual cycle. Severe cases may also present hallucination and delirium, and they could be misdiagnosed as mental diseases. According to our review, in the past 2 decades, 20 cases were misdiagnosed as mental disorders with a misdiagnosis rate of 6.64%. Hypoparathyroidism may also cause cognitive impairment[123] and parapyramidal system symptoms.[124–126] Thus, hypoparathyroidism may be misdiagnosed as any of the above 30 disease entities, which can be categorized as neurology, cardiology, pediatrics, gastroenterology, orthopedics, and otorhinolaryngology.

According to the current review, we summarized the causes of misdiagnosis of hypoparathyroidism as follows. First, hypoparathyroidism is characterized by complex manifestations and atypical clinical symptoms. Lack of understanding of this disease is the main cause of misdiagnosis. Hypoparathyroidism is not a common endocrine disease, and primary practitioners usually know little about this disease in China, leading to ignorance regarding the disease in clinical practice. Some nonspecific symptoms can conceal the patient's underlying condition. For instance, in the literature, one 5-year-old patient underwent blood electrolyte test and head CT during the initial assessment of the new onset of symptoms. In addition, hypocalcemia, hyperphosphatemia, and symmetrical calcifications in bilateral basal ganglia were identified. Despite these findings, the patient was diagnosed with primary epilepsy and administered antiepileptic treatment. The patient was not diagnosed definitely for 8 years and 3 months, although he visited multiple hospitals (outpatient visits to 6 hospitals and diagnosed and treated by 12 clinicians). This case suggests that several Chinese clinicians may lack the understanding of this disease. Therefore, it should be brought to the attention of clinicians, especially neurologists at various tiers of hospitals. The neurologists should pay attention to the disease in clinical diagnosis because the new-onset symptoms of IHP are commonly neurological or brain-related, concealing the endocrine disease and leading to misdiagnosis. In addition, omitting or overlooking some critical auxiliary examinations/tests may also cause misdiagnosis. For instance, in the literature, 2 patients had cognitive impairment, carpopedal spasm, and brain calcifications, but were misdiagnosed with Fahr disease.[53]

Our literature review suggests a severe misdiagnosis of hypoparathyroidism, which should be cautioned against by clinicians, especially neurologists. Nevertheless, most Chinese medical care institutions are equipped to decrease the misdiagnosis rate of IHP based on the currently available medical parameters. Thus, a clinician should enrich the understanding of this disease, and emphasize the screening of high-risk population. In order to achieve a definite diagnosis of this disease, a clinician should discriminate between specific neuromuscular hyperexcitability symptoms, neuropsychiatric symptoms, and ectodermal dystrophic symptoms, and screen the high-risk populations, such as chronic carpopedal spasm patients with unknown causes, patients with hypocalcemia and neurological or mental symptoms, refractory epilepsy patients, and young patients with symmetrical cranial calcifications. Moreover, auxiliary examinations/tests should be performed in suspected patients to verify the levels of calcium, phosphate, and PTH in blood, renal tubular reuptake, as well as head CT scan. The diagnosis of the disease is not difficult if the clinician has mastered its clinical characteristics, and analyzed the patient's history, signs, and results of auxiliary examinations/tests thoroughly.

6. Conclusion

The misdiagnosis rate of IHP in China increased in the past 2 decades, which were predisposed to be misdiagnosed as epilepsy or mental and other diseases. It is essential that a clinician understand this disease better and emphasizes the screening of high-risk population, especially in those patients with hypocalcemia and hyperphosphatemia, increased blood CK with unknown causes, as well as typical clinical symptoms.

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