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ORIGINAL ARTICLE

Clinical data analysis of 22 cases with hypoparathyroidism misdiagnosed as epilepsy

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Abstract

OBJECTIVE: Patients with hypoparathyroidism always present with recurrent tetany caused by hypocalcemia. These patients are usually misdiagnosed with epilepsy and incorrectly treated with anti-epileptic drugs. This research analyzed clinical data about 22 patients with hypoparathyroidism misdiagnosed as epilepsy and summarized the clinical experience for reducing misdiagnosis and incorrect therapy about hypoparathyroidism.

METHOD: Totally 160 patients with hypoparathyroidism, administrated to the First Medical Center of Chinese PLA General Hospital from January 1st, 2008, to July 1st, 2021, were enrolled in this report. Clinical data about 22 patients initially misdiagnosed with epilepsy were analyzed.

RESULTS: Of the 160 cases with hypoparathyroidism, 22 patients (12 males and 10 females) were misdiagnosed with epilepsy in local hospitals. The misdiagnosis rate was 13.75% and the median duration of misdiagnosis was 8.0 (2.0, 14.8) years. The clinical manifestations of the 22 patients misdiagnosed as epilepsy included tetany 81.8% (18/22), disturbance of consciousness 27.3% (6/22), limb numbness 13.6% (3/22), limb weakness 27.3% (6/22), mental and behavioral abnormality 9.1% (2/22), and memory impairment 13.6% (3/22), etc. Electroencephalogram (EEG) was performed in 9 cases, which presented as slow wave and spike-slow complex wave in 3 cases, slowing down of θ and δ band background in 2 cases and normal EEG in 4 cases. Out of the 15 cases that underwent head computed tomography (CT) scan, in which 13 cases had intracranial calcification. Anti-epileptic drugs were used to treat 22 patients, of which 17 patients were treated with two kinds of drugs. With calcium and calcitriol supplement in all these 22 patients, the anti-epileptic drugs were gradually reduced and withdrawn in 17 cases. In the other 5 cases with secondary epilepsy, the type of anti-epileptic drugs was reduced to one and the clinical condition improved obviously.

CONCLUSION: The clinical manifestations of hypoparathyroidism are complex and usually be misdiagnosed as primary epilepsy. Detection of serum calcium, phosphorus and parathyroid hormone is very important to avoid misdiagnosis and incorrect therapy about hypoparathyroidism.

INTRODUCTION

Hypoparathyroidism (HP) is an endocrine disorder of the parathyroid glands affecting the level of calcium and phosphorus in the body, caused by deficiency or insufficient effect of parathyroid hormone (PTH). The clinical manifestations of this disorder are mainly tetany and neuropsychiatric symptoms due to increased neuromuscular excitability induced by hypocalcemia (Marcucci *et al.* 2018; Abate EG & Clarke BL, 2016). Hypocalcemia combined with hypophosphatemia can lead to complications such as dilated cardiomyopathy, soft tissue ectopic calcification, cataract and intracranial calcification (Marcucci *et al.* 2018; Abate EG & Clarke BL, 2016). Intracranial calcification can cause secondary neurological symptoms, such as epilepsy, intellectual decline, agitation (Clarke *et al.* 2016). As a result of these secondary neurological manifestations, hypoparathyroidism has often been misdiagnosed as epilepsy or other psychiatric diseases and been given incorrect treatment with anti-epilepsy drugs (Tang *et al.* 2017; Kim *et al.* 2020). This study reviewed the clinical data of 22 patients initially misdiagnosed with epilepsy in local hospitals and summarized the causes of the misdiagnosis.

METHODS

Participants

From January 1st, 2008, to July 1st, 2021, the clinical data of 160 patients with hypoparathyroidism diagnosed in the First Medical Center of PLA General Hospital were collected. The study was approved by the Ethics Committee of PLA General Hospital. Informed consents were obtained from all patients prior to the use of their data and images for publication. We abstracted information from the medical records of these patients regarding their diagnosis and therapy of HP using a standardized data extraction form. Inclusion criteria: All patients met the diagnostic criteria of the 2018 China Clinical Diagnosis and Treatment Guidelines for hypoparathyroidism. Total blood calcium ≤ 2.13mmol/L (8.5mg/dL) with undetectable or inappropriate low levels of PTH, the serum PTH of pseudohypoparathyroidism (PHP) higher than the upper limit of the normal reference value. The Exclusion criteria: vitamin D deficiency and resistance, liver insufficiency, renal insufficiency, neoplastic osteomalacia, malnutrition, drug-induced hypocalcemia and (or) abnormal PTH levels.

Methods

The clinical data of patients with HP were analyzed retrospectively. The clinical information included: (1) General conditions: gender, age, age of disease, disease duration and time interval of diagnosis; (2) Clinical manifestations : numbness of hand and foot, lower limb weakness, limb tetany, state of consciousness and attack frequency, memory, facial nerve tapping, beam arm compression, appearance development; (3) Biochemical indicators: corrected blood calcium, blood phosphorus, blood PTH, alkaline phosphatase, blood creatinine, serum albumin, 24 hours urinary calcium, urinary phosphorus, 25 hydroxyvitamin D; The correction formula is as follows: the corrected serum total calcium (mmol/L) = Serum total calcium determination values (mmol/L) + 0.02 × [40-serum albumin level (g/L)] (Bove-Fenderson & Manstadt, 2018). (4) Complications screening: cranial electronic computed tomography (computed tomography, CT), cranial nuclear magnetic resonance (magnetic resonance imaging, MRI), ocular slit lamp examination, electrocardiogram, cardiac ultrasound, urological ultrasound, electroencephalography (electroencephalogram, EEG); (5) Treatment and follow-up. According to the etiology, the patients were classified into various groups, including PHP, idiopathic hypoparathyroidism (IHP), postsurgical hypoparathyroidism (more than 6 months after the surgery of cervical disease) pseudopseudohypoparathyroidism (PPHP), and autoimmune polyglandular syndrome(APS, include hypoparathyroidism).

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics Version 23.0 (USA, Chicago,III). Count data are shown as sample size (composition ratio), normal distribution measurement data are indicated by mean($\bar{x}$) ± standard deviation(s), non-normal distributions are indicated by M(Q1, Q3).

RESULTS

General clinical data

Totally 160 patients with HP (75 males, 85 females) were enrolled in this report. The median age was 44.2 (24.2, 54.5) years, and the median disease course was 3.5 (0.5, 13.0) years. 68 of the 160 cases (42.5%) had seizure-like symptoms, of which 22 cases (12 males and 10 females) had been initially misdiagnosed with epilepsy in local hospitals. The misdiagnosis rate was 13.75%. The median age of onset was 9.5 years old, and the longest misdiagnosis duration was 39 years. Since most of the patients were teenagers at the onset of the disease and their symptoms were mainly epileptic, they usually visit department of pediatrics (12 cases, 54.5%) for medical care. Clinical data of the patients were shown in Table 1.

Clinical manifestation

Most of the misdiagnosed patients had multiple symptoms, including tetany, consciousness disorder and limb weakness, and mental behavior abnormalities. Nine cases underwent electroencephalogram examination. Three cases had slow spine-slow complex wave and 2 cases had slow θ and δ band background. CT examination showed intracranial calcification in 13 cases,

Tab. 1. Clinical data of 22 patients with hypoparathyroidism misdiagnosed as epilepsy

	PHP (n=10)	IHP (n=11)	Postsurgical HP (n=1)
Gender (male/female)	6/4	6/5	0/1
Age at onset [yrs, M(Q1,Q3)]	8.0 (4.9,13.0)	18.0 (0.6,32.0)	29
Age of diagnosis [yrs, M(Q1,Q3)]	11.5 (8.58,26.0)	38 (24.0,52.0)	59
Diagnosis interval [yrs, M(Q1,Q3)]	4.2 (1.6,14.0)	11.0 (2.0,20.0)	30
Main clinical symptoms n (%)			
Tetany	9 (90)	8 (72.7)	1 (100)
Disturbance of consciousness	2 (20)	3 (27.3)	1 (100)
Limb numbness	1 (10)	1 (9.1)	1 (100)
Limb weakness	4 (40)	2 (18.2)	0
Memory impairment	2 (20)	1 (9.1)	0
Chvostek's sign n (%)	2 (20)	2 (18.2)	0
Trousseau's sign n (%)	3 (30)	3 (27.3)	0
Corrected Serum total calcium (mmol/L)	1.63±0.37	1.63±0.31	1.29
Urinary calcium (mmol/24h)	0.4 (0.2,1.6)	2.5 (0.3,2.9)	2.1
Serum phosphorus (mmol/L)	2.24±0.72	1.92±0.43	2.77
Urinary phosphorus [mmol/24h, M(Q1,Q3)]	8.6 (5.9,11.1)	12.7 (6.8,21.4)	14.25
Serum PTH [pg/mL, M(Q1,Q3)]	146.0 (77.9,293.0)	5.5 (3.8,13.1)	9.28
Complication (n)			
Intracranial calcification (CT scan)	8/8	4/6	1/1
Results of EEG	2/4	3/5	-
Cataract	4/4	2/7	1/1
Kidney stones	1/2	1/6	-

(PHP) - pseudohypoparathyroidism
(IHP) - idiopathic hypoparathyroidism
(HP) - Hypoparathyroidism
(PTH) - parathyroid hormone

multiple calcifications in 11 cases and basal ganglia calcification in 10 cases (76.9%). Basal ganglia calcification involved globus pallidus (69.2%), caudate nucleus (53.8%) and putamen (61.5%); Other region calcification included cerebellar hemisphere 46.2%, cerebellar dentate nucleus 30.7% and gray matter junction 30.7%. Slit lamp examination was carried out on 12 patients and 7 (58.3%) were found to have cataract. (Table 1)

Comparison of clinical characteristics between misdiagnosed patients and accurately diagnosed patients

1) There was significant difference in age and course of disease between the two groups ($p<0.05$). The misdiagnosed group was mainly adolescents with a longer course of disease, whilst the accurately diagnosed group was mostly adults with a shorter course of disease. 2) These two groups had statistically significant differences in some presentations such as tetany, consciousness disorder and memory loss. And the prevalence of such symptoms was higher in the misdiagnosed group. 3) The misdiagnosed group had a higher blood phosphorus level. Accurately diagnosed patients were initially admitted in endocrinology department (55.1%), while misdiagnosed patients were initially admitted into another department (90.1%). (Table 2)

Treatment and Discharge follow-up

All these 22 cases misdiagnosed with epilepsy were administrated with one or two antiepileptic drugs: phenytoin sodium 10/22 (45.5%), sodium valproate 8/22 (36.4%), carbamazepine 6/22 (27.3%), topiramate, levetiracetam, etc. The longest duration with antiepileptic drugs treatment lasted for 39 years. In our department, they got the definite diagnosis of HP, and were administrated with oral calcium carbonate (calcium 480mg~1200mg) combined with calcitriol (0.5ug~1.0ug) daily. Meanwhile, the antiepileptic drugs were gradually reduced or withdrawn. As a result, 17 of these 22 patients (77.3%) had no seizure attacks later, only occasionally presented with some slight neuromuscular symptoms such as numbness of limbs. According to electroencephalogram examination and neurologist consultation, the other 5 cases (22.7%) were diagnosed with secondary epilepsy. In these 5 cases, the therapy with one antiepileptic drug continued and the frequency and symptoms of seizures relieved significantly.

Tab. 2. Comparison of clinical characteristics between misdiagnosed and correctly diagnosed group

	Misdiagnosed group (n=22)	Correctly diagnosed group (n=138)	*p*
Gender n (%)			0.438
male	12 (54.5)	63 (45.7)	
female	10 (45.5)	75 (54.3)	
Age at onset [yrs, M(Q1,Q3)]	9.5 (4.9,24.5)	39 (16.8,49.4)	0.00
Course of disease [yrs, M(Q1,Q3)]	15.5 (2.0,22.3)	2 (0.4,10.0)	0.00
Main clinical symptoms n (%)			
Tetany	18 (81.8)	65 (47.1)	0.003
Disturbance of consciousness	6 (27.3)	11 (7.9)	0.006
Limb numbness	3 (13.6)	24 (17.4)	0.896
Memory impairment	3 (13.6)	3 (2.2)	0.035
Limb weakness	6 (27.3)	48 (34.8)	0.489
Chvostek's sign n (%)	4 (18.2)	21 (15.2)	0.968
Trousseau's sign n (%)	6 (27.3)	33 (23.9)	0.733
AHO symptoms n (%)	3 (13.6)	6 (4.3)	0.208
Corrected Serum total calcium (mmol/L)	1.61±0.33	1.68±0.30	0.287
Serum phosphorus (mmol/L)	2.10±0.6	1.85±0.51	0.039
Serum PTH [pg/mL, M(Q1,Q3)]	14.2 (5.4,133.3)	10.4 (5.6,19.1)	0.069
Complication (n)			
Intracranial calcification (CT scan)	13/15	42/52	0.887
Results of EEG	5/9	10/13	0.376
Cataract	5/12	19/50	0.815
Kidney stones	0/8	3/54	1.0
First consultation department n (%)			0.00
Endocrinology Department	2 (9.1)	76 (55.1)	
Non-endocrine department	20 (90.1)	62 (44.9)	

(AHO) - Albright hereditary osteodystrophy

DISCUSSION

Hypoparathyroidism is a relatively rare endocrinological disorder, with a prevalence of 22-37/100,000 (Cipriani C *et al.* 2017; Cianferotti L *et al.* 2018). Its pathophysiological changes are caused by decreased secretion or insufficient effect of PTH, and its biochemical characteristics are hypocalcemia and hyperphosphatemia (Bilezikian *et al.* 2020). The main clinical manifestations are tetany, paresthesia, followed by convulsion or epileptic seizures (Seedat *et al.* 2018; Quan *et al.* 2017). The accurately diagnosed is usually based on typical presentations, physical examinations and biochemical characteristics (Bilezikian *et al.* 2020; Seedat *et al.* 2018). Surgery is the most common cause of adult hypoparathyroidism (Kim *et al.* 2020), followed by IHP, APS syndrome and other syndrome involving paraplegia. The severity of hypocalcemia caused by different etiology is variable. For example, the serum calcium of PHP and IHP are lower than that of postoperative HP (Tang *et al.* 2017).

Convulsion and epilepsy-like seizures are the common symptoms of HP, with an incidence of 60%~70% (Seedat *et al.* 2018; Quan *et al.* 2017; Modi *et al.* 2014). Some younger patients may present with persistent epilepsy (Kamate *et al.* 2018). In this study, 68 cases (42.5%) of 160 patients had epileptic seizure symptoms, most of which only showed symptoms of tetany, paresthesia, muscle clonus. Among them, 18 cases (26.5%) had conscious disorder, and 6 cases were misdiagnosed as epilepsy. Compared with the confirmed group, consciousness disorder was more common in the misdiagnosed group, hence more likely to be misdiagnosed as epilepsy (Tang *et al.* 2017; Quan *et al.* 2017).

Hypocalcemia can increase neuromuscular excitability, hippocampal neuron discharge, intracranial pressure and metabolic disorders, which may lead to epileptic seizures (Bilezikian *et al.* 2020; Liu *et al.* 2016). Seizures may be the only symptom of hypocalcemia and is related to the severity of hypocalcemia (Seedat *et al.* 2018). Most of the neurological symptoms caused by hypocalcemia are functional changes, which can be quickly relieved after rectifying the hypocalcaemia (Liu *et al.* 2018). In these twenty-two patients with misdiagnosed epilepsy, the treatment

with calcium and calcitriol got good response when the hypocalcemia was rectified. When antiepileptic drugs were gradually withdrawn in 17 patients, no epileptic seizures occurred again. However, recurrent epileptic seizures can also lead to intracranial structural changes, especially calcium salt deposition (Seedat *et al.* 2018; Liu *et al.* 2016; Donzuso *et al.* 2019). The intracranial calcification rate of HP was 59.1%-69.0% (Tang *et al.* 2017; Liu *et al.* 2016), characterized by the symmetrical distribution of calcification foci in the thalamus and cortex of bilateral basal ganglia region (Qu *et al.* 2016).

Among all the patients undergoing craniocerebral CT examination, 82.1% had intracranial calcification and 91% had basal ganglia calcification. The calcification rate in the PHP group was 93.1%, significantly higher than that in the other groups (Qu *et al.* 2016). Intracranial calcification increases the risk of epileptic seizures and therefore it is more likely to be misdiagnosed (Bilezikian *et al.* 2020; Liu *et al.* 2016). The degree of calcification is associated with the severity of hypocalcemia (Abate *et al.* 2016; Seedat *et al.* 2018). In this group, 15 cases of 55 patients (27.3%) with intracranial calcification did not have epileptic seizures, which was considered to be related to the calcium deposition site. EEG in Hypocalcemia patients were present with extensive θδ range background rhythm slowing down and focal or extensive spike discharge, which is indistinguishable from epileptic EEG (Nardone *et al.* 2016; Shellhaas *et al.* 2019). EEG examinations were performed in 9 patients with misdiagnosed epilepsy. Slow wave and spike-slow complex wave present in 3 cases, and slow θ and δ band background in another 2 cases, which had no difference compared with the confirmed group.

Epileptiform seizures are common presentations in HP patients. As a result, such patients have been usually misdiagnosed with primary epilepsy, with a misdiagnosis rate of 16.5%-34.3% (Tang *et al.* 2017; Kim *et al.* 2020). Based on the analysis of clinical data in these misdiagnosed group and confirmed group, we summarized the causes of misdiagnosis as follows: 1) HP is a rare disease with complex etiology. The clinical symptoms are mainly increased neuromuscular excitability and epileptic seizures (Marcucci *et al.* 2018; Abate *et al.* 2016). 2). In terms of etiology, IHP and PHP are more common in patients with epilepsy misdiagnosed and HP after surgery are rare, (Kim *et al.* 2020; Modi *et al.* 2014; Liu *et al.* 2016). The reason is that physicians have been alert to secondary HP after thyroid operation and have paid more attention to monitoring and rectifying hypocalcemia (Tang *et al.* 2017; Bilezikian *et al.* 2020; Marcinkowska *et al.* 2017). However, PHP usually develops in childhood and the onset age is about 8.5 years old (Qu *et al.* 2016). Hypocalcemia in children usually manifests as convulsive epileptic convulsions (Liu *et al.* 2016; Modi *et al.* 2014), so there is higher probability to be misdiagnosed. In this study, the median age of the misdiagnosed epilepsy PHP group was 8.0 years, and 90% of them had seizure-like convulsions. IHP often has insidious onset and progress slowly, causing delay in diagnosis (Modi *et al.* 2014). In this report, the median misdiagnosis duration of IHP patients was about 11 years. 3). For most epileptic patients, the monitoring on serum calcium, phosphorus and PTH has always been ignored, increasing the probability of misdiagnosis (Tang *et al.* 2017). 4). Hypocalcemia can lead to intracranial calcification which is also a potential risk factor of epilepsy (Quan *et al.* 2017; Liu *et al.* 2016). 4). When HP was misdiagnosed as primary epilepsy, antiepileptic drug is not always effective. If physicians did not further investigate the true cause of epileptiform seizures and are satisfied with the slight effect of antiepileptics, such misdiagnosis might continue. (Seedat *et al.* 2018; Donzuso *et al.* 2019). For example, all these 22 patients misdiagnosed in this study were treated with antiepileptic drugs, and 17 cases were even treated with two antiepileptic drugs when monotherapy was deemed ineffective.

When the diagnosis of HP is confirmed in patients presenting with epileptiform seizures, calcium and active vitamin D are needed to maintain blood calcium levels within the normal limit or slightly below normal level. Maintaining the product of calcium and phosphorus below $55mg^2/dL^2$ can effectively alleviate epileptic seizures (Liu *et al.* 2016; Qu *et al.* 2016). Antiepileptic drugs can be replaced in 71% of IHP epileptic patients with calcium and vitamin D supplements. In 70% patients diagnosed with secondary epilepsy caused by long term hypocalcemia, also the usage of antiepileptic drugs can be reduced (Seedat *et al.* 2018; Modi *et al.* 2014). In this report, all these 22 patients misdiagnosed were administrated with calcium and active vitamin D supplement. As a result, 17 cases of them did not suffer any more seizures , even after antiepileptic drugs were gradually withdrawn.

In conclusion, we analyzed the clinical data of patients with hypoparathyroidism admitted in our hospital in recent 13 years and summarized the experience and lesson in different diagnosis with epilepsy. We hope it can offer some valuable references for physicians in dealing with such disorder. In clinical practice, for patients with recurrent epileptic convulsions and/or disturbance of consciousness, especially those with poor reaction to anti-epileptic treatment and/or symmetrical calcification foci in the brain, routine detection of serum calcium, phosphorus, PTH and craniocerebral CT should be performed to investigate hypoparathyroidism. As this will be an efficient way to avoid misdiagnosis and consequently administer correct therapy.

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DECLARATION OF COMPETING INTEREST

The authors declared that they have no competing interests.

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