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Case Report

Transient hypoparathyroidism masquerading as Guillain-Barré syndrome in a 7-year-old child

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ABSTRACT

Primary hypoparathyroidism may manifest in ill children or as a result of autoantibodies targeting the parathyroid glands. Guillain-Barré syndrome (GBS) is a prevalent worldwide condition that is a post-infectious, monophasic, immune-mediated polyradiculoneuropathy. Transient hypoparathyroidism, which necessitates short-term treatment, can occur in neuroinflammatory diseases like GBS. Here we report a seven-year-old girl experienced an ascending and symmetrical weakness, severe pain, muscle cramps, tingling sensations, and paresthesias in both lower limbs for one day. She struggled with micturition and defecation. Despite this, she had intact higher mental functions, hypotonia of both lower limbs, power (2/5), brisk lower limb reflexes, extensor plantar response, and tenderness in both calf muscles. A diagnosis of Guillain-Barré syndrome (GBS) was made due to ascending progressive weakness, and intravenous immunoglobulin was initiated at 2 g/kg. Due to severe muscle cramps, tingling, and distal paresthesias, further workup revealed hypocalcemia, hyperphosphatemia, low parathormone, and low 25(OH)D levels. After starting oral elemental calcium and vitamin D supplements, the child improved symptomatically in the second week. This case report emphasis the need for identification of transient primary hypoparathyroidism, sharing similar clinical characteristics with GBS, which could be overlooked and if not addressed, may result in seizures or cardiac arrhythmias.

Keywords: Immune-mediated polyradiculoneuropathy, Hypocalcemia, Hyperphosphatemia

INTRODUCTION

Guillain-Barré syndrome (GBS) is the leading cause of ascending neuromuscular paralysis globally, characterized by a devastating but manageable clinical progression. GBS is an inflammatory neurological condition that can become serious and potentially fatal without prompt intervention. The identification of GBS primarily relies on observing clinical symptoms, complemented by electrophysiological and laboratory tests as needed.[1] The clinical presentation can differ significantly, ranging from tingling sensations or loss of sensation in the extremities before the onset of weakness, which may or may not be accompanied by dysautonomia, as well as muscular, radicular, or neuropathic pain.[2]

Hypoparathyroidism is an uncommon endocrine condition in children marked by reduced levels of PTH, resulting in low calcium levels and elevated phosphate levels.[3] The clinical symptoms, including muscle cramps, tingling feelings, and paresthesias, closely resemble those seen in GBS. If not identified or addressed, it may result in severe hypocalcemia symptoms such as tetany, seizures, or heart rhythm disturbances.[4]

CASE REPORT

A seven-year-old girl presented with a history of sudden onset weakness of both lower limbs along with severe pain for one day. She had frequent falls the previous day, followed by an inability to walk, with an inability to void

urine and stools. She also had severe calf muscle cramps and tingling sensations in both feet. There was no history of fever, vomiting, loose stools, altered sensorium, seizures, difficulty in vision, or abnormal movements. There was no history of recent vaccination, dog bite, travel, tonsillectomy, or trauma. On examination, her vitals were unremarkable, with no neurocutaneous markers. She was conscious, oriented, with intact speech and memory. The single breath count test was normal, indicating no diaphragmatic involvement. Her cranial nerve examination was normal. Motor system examination revealed hypotonia of both lower limbs, with power of 2/5 (MRC Grading), brisk lower limb reflexes, and extensor plantar response. There was no sensory involvement, no cerebellar/meningeal signs. The child had severe calf muscle cramps, with muscle tenderness, tingling sensations, and distal paresthesias, which worsened on the third day of weakness.

GBS was clinically considered due to acute ascending symmetric flaccid paralysis, and immunotherapy was initiated with intravenous immunoglobulin at 2 g/kg over 2 days. Due to brisk reflexes, extensor plantar reflex, and bladder/bowel involvement, transverse myelitis was ruled out by MRI of the brain and spine. The child was simultaneously evaluated for other causes of acute flaccid paralysis. Complete hemogram showed leukocytosis (19.43×103 µ/l), neutrophilia (17.29×103 µ/l), and thrombocytosis (5.14×103 µ/l). Her serum potassium level was 4.8 mEq/l, and serum sodium was 142 mEq/l. Renal function tests and liver function tests were within normal limits. Investigations like fasting and postprandial blood glucose values were 75 mg/dl and 120 mg/dl, respectively, and serum vitamin B12 levels (408.52 pg/ml) were normal. Creatine kinase total (CK-Total) and creatine kinase–MB were within normal limits of 37 IU/l and 19.7 IU/l, respectively.

On the third day of weakness, power became 0/5, with absent deep tendon reflexes and mute plantar reflex and worsening of muscle aches. Due to severe muscle cramps and calf muscle tenderness along with tingling sensations in the feet, serum total calcium was done, which was less (7.0 mg/dl), and inorganic phosphorous was elevated (6.9 mg/dl) with normal serum magnesium levels (2.2 mg/dl). Ionized calcium levels were low (3.6 mg/dl). Due to hypocalcemia and hyperphosphatemia, parathormone (PTH) levels and 25 (OD) levels were done and found to be low at 1 pg/ml and 9 ng/ml, respectively. QTc was 0.49 msec with no ST changes in the 12-lead ECG.

Thyroid stimulating hormone (TSH) was 1.73 µIU/ml with normal FT3 and FT4 levels. Ultrasonogram of the neck was normal. 8 AM serum cortisol levels were normal (5 mcg/dl). There were no recent neck surgeries, radiation therapy, or features suggestive of other autoimmune or endocrine disorders. The possibility of hypoparathyroidism, probably of autoimmune etiology, was initially considered and due to ECG changes, child was initiated on intravenous calcium gluconate 10% 10 ml every 6th hourly till 48 hours followed by oral elemental calcium at 65 mg/kg/day in 4 divided doses, along with vitamin D3 at 800 IU/day. Anti-nuclear antibody (ANA) was negative. A nerve conduction study (NCS) done on the seventh day of weakness turned out to be normal. A CSF analysis on the fourteenth day of weakness revealed lymphocytes and monocytes in a proteinaceous background with normal protein levels (45 mg/dl). There was symptomatic improvement in muscle cramps, tingling sensations, and distal paresthesias by the third week of illness. Muscle weakness improved in tone and power, with reappearance of deep tendon reflexes and bladder/bowel functions. Serum calcium, phosphorous, and parathormone levels normalized after 4 weeks, and 25 (OD) levels became sufficient after 8 weeks.

DISCUSSION

GBS is an autoimmune polyneuropathy that can be triggered by several infectious organisms and is characterized by rapidly progressive ascending symmetric paralysis, which can involve cranial nerves and respiratory muscles. The disease can be of varying severity, from mild weakness to complete paralysis. GBS onset may be acute or subacute, so maximum disability is usually reached within 2 weeks of onset of illness.[5] Hyporeflexia or areflexia in the form of absent deep tendon reflexes, especially ankle jerk and patellar jerk, serves as a diagnostic criterion of GBS.[6] However, 10% of cases of GBS have shown to have normal or exaggerated deep tendon reflexes, as in our case.[5,7]

All children with GBS should undergo laboratory testing to exclude other causes of acute flaccid paralysis, like a complete hemogram, serum electrolytes, and renal and liver function tests. Other investigations to rule out common GBS mimics like myelopathies, myopathies, and peripheral neuropathies should be done.[5] Immunotherapy with intravenous immunoglobulin (IVIG) or plasma exchange can effectively prevent irreversible axonal damage and improve neurological outcomes in GBS. An IVIG regimen of 2 g/kg over 2 days is known to improve clinical outcomes by 2 weeks and independent ambulation by 6 months.[8]

Electrophysiological studies like NCS form an important aspect in the diagnosis of GBS; however, NCS might be normal in the early days of the disease, like in our case. CSF analysis in GBS is usually performed to rule out GBS mimics like infections. The classic finding of albumin cytological dissociation is characterized by elevated protein levels with normal CSF cells; however, 30-50% of patients and 10-30% of patients have normal CSF protein levels in the first and second weeks, respectively, as seen in our case. Hence, GBS cannot be excluded with lumbar puncture findings.[5] So all the investigations regarding GBS does not clearly rule out the disease which lkead to increase the suspicion. Moreover, contrast MRI was taken and was found to be normal.

Hypoparathyroidism in children is a rare entity characterized by low PTH levels with hypocalcemia and hyperphosphatemia.[3]

Clinical features like muscle cramps, tingling sensations, and paresthesias are similar to those of GBS. If left undiagnosed or untreated, it can lead to life-threatening complications of severe hypocalcemia.[4] Case reports have shown that severe hypocalcemia can cause quadriplegia mimicking GBS in adults. The reason for the nerve involvement is explained as reversibly increasing inward sodium currents, moderate depolarization, and increased nerve excitability due to a decrease in calcium concentration prevent muscle fibers from being excited by supramaximal stimulation of peripheral nerves.[9] Etiology of hypoparathyroidism can be primary, secondary, or tertiary. Low parathormone levels are a hallmark of primary hypoparathyroidism, either due to absent/dysfunctional parathyroid glands or autoantibodies against parathyroid cells.

It is possible that numerthatous patients with early-onset or congenital hypoparathyroidism may have genetic causes. Conversely, those with late-onset hypoparathyroidism might have an autoimmune cause underlying their condition. When a clear cause for hypoparathyroidism is not identified, these individuals should be closely monitored for the emergence of other disorders.[3] Though there is no proven association between GBS and hypoparathyroidism, a few case reports in adults show a potential link, as both of them have similar clinical features and share autoimmune pathogenesis.[9] In research conducted by Gay and Grimes et al on a 68-year-old man suffering from hypoparathyroidism, it was found that he had impaired position sense in his feet, a lack of vibration sensation, and diminished deep tendon reflexes. Nerve conduction studies revealed extended latency and reduced conduction velocity in both the median and peroneal nerves, which showed improvement six weeks after initiating treatment with vitamin D and calcium carbonate.[10]

Gomez et al documented a 25-year-old male patient with a known history of hypoparathyroidism. When he was 20 years old, the patient experienced weakness in his limbs along with reduced tendon reflexes. Nerve conduction studies indicated that there were lower nerve conduction velocities in both the median motor and ulnar nerves. A nerve biopsy revealed signs of axonal degeneration.[11] Dionisi et al reported that a 15-month-old child with hypoparathyroidism and distal sensorimotor peripheral neuropathy recovered after receiving vitamin D medication for five months.[12]

Axonal sensorimotor neuropathy was discovered during nerve conduction investigations in another case of idiopathic hypoparathyroidism that Goswami et al reported.[13] For two years, he was given calcium and vitamin D supplementation. Neuropathy improved gradually on both a clinical and electrophysiological level. Peripheral neuropathy's occurrence in hypocalcemia-related disorders like osteomalacia and hypoparathyroidism, as well as its reversibility following calcium and vitamin D normalization, indicate that it plays a critical role in peripheral axon function.[13] In patients with hypoparathyroidism, ataxia, paraplegia, dysphagia, and dysarthria have all been reported. Fortunately, several symptoms, ranging from papilledema to Chvostek and Trousseau indications, have been shown to return when the hypocalcemia is corrected.[14] Even worse, any concurrent hypokalemia, hypomagnesemia, or alkalosis should be ruled out when treating a patient with hypoparathyroidism since they would exacerbate the signs and symptoms of hypocalcemia.[15]

Autoimmune hypoparathyroidism can occur as a part of autoimmune polyglandular syndrome (APS-1) comprising APECED (autoimmune polyendocrinopathy candidiasis ectodermal dystrophy), or as an isolated disorder. APS-1 is characterized by autoantibodies against parathyroid, thyroid, and adrenal glands. Children with isolated autoimmune hypoparathyroidism have circulating antibodies targeted against the extracellular domain of the calcium-sensing receptor (CaSR).[3]

However, authors couldn't screen the child for CaSR antibodies. Thyroid profile done initially was normal. As GBS is a hyperinflammatory state, the possibility of sick euthyroid syndrome and elevated serum cortisol levels due to acute stress were anticipated; hence, 8 AM serum cortisol levels (5 mcg/dl) and repeat thyroid profile were done after 4 weeks. As the child recovered soon by the third week, with normalization of serum calcium, inorganic phosphorus, and parathormone levels after four weeks, the possibility of transient hypoparathyroidism was considered in our case.

CONCLUSION

Transient hypoparathyroidism in children is temporary and triggered by an illness, infection, or stress, which can present with muscle cramps, tingling sensations, and fatigue, overlapping with the clinical features of GBS. These symptoms resolve once the underlying disease is treated and mostly require symptomatic short-term treatment like calcium and vitamin D3 supplementation. Hence, we must consider on treating hypoparathyroidism in suspected case of GBS as primary hypoparathyroidism can be transient in case of other causes like fever and stress.

The child responded well to calcium and vitamin D therapy; neuromuscular weakness started improving by the end of the third week by regaining power, tone, and reflexes, and she was able to get up and sit on her own. After 4 weeks, total leukocyte counts, platelet counts, and inflammatory markers became normal, coinciding with resolution of neuroinflammation. This leads to the conclusion that the reported case is a transient hypoparathyroidism overlapping with symptoms of GBS.

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REFERENCES

1. Yuki N, Kokubun N, Kuwabara S, Sekiguchi Y, Ito M, Odaka M, et al. Guillain-Barré syndrome associated with normal or exaggerated tendon reflexes. J Neurol. 2012;259(6):1181–90.
2. Papri N, Islam Z, Leonhard SE, Mohammad QD, Endtz HP, Jacobs BC. Guillain–Barré syndrome in low-income and middle-income countries: challenges and prospects. Nature Rev Neurol. 2021;17(5):285-96.
3. Pasieka JL, Wentworth K, Yeo CT, Cremers S, Dempster D, Fukumoto S, et al. Etiology and Pathophysiology of Hypoparathyroidism: A Narrative Review. J Bone Miner Res Off J Am Soc Bone Miner Res. 2022;37(12):2586–601.
4. Korkmaz HA, Ozkan B. Hypoparathyroidism in children and adolescents. Ann Pediatr Endocrinol Metab. 2023;28(3):159–67.
5. Leonhard SE, Mandarakas MR, Gondim FAA, Bateman K, Ferreira MLB, Cornblath DR, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671–83.
6. Elendu C, Osamuyi EI, Afolayan IA, Opara NC, Chinedu-Anunaso NA, Okoro CB, et al. Clinical presentation and symptomatology of Guillain-Barré syndrome: A literature review. Med (Baltimore). 2024;103(30):38890.
7. Yuki N, Kokubun N, Kuwabara S, Sekiguchi Y, Ito M, Odaka M, et al. Guillain-Barré syndrome associated with normal or exaggerated tendon reflexes. J Neurol. 2012;259(6):1181–90.
8. Liu S, Dong C, Ubogu EE. Immunotherapy of guillain-barre syndrome. Human vacc Immunotherap. 2018;14(11):2568-79.
9. Zaidi SS, Siddiqui T, Siddiqui SJ. Acute Onset of Quadriplegia Secondary to Hypoparathyroidism: Mimicker of AMAN Variant of GBS. J Coll Physicians Surg--Pak. 2018;28(3):23–5.
10. Gay JD, Grimes JD. Idiopathic hypoparathyroidism with impaired vitamin B12 absorption and neuropathy. Can Med Assoc J. 1972;107:54-6.
11. Gomez MR, Engel AG, Dyck PJ. Progressive ataxia, retinaldegeneration, neuromyopathy, and mental subnormality in apatient with true hypoparathyroidism, dwarfism, mal absorption and cholelithiasis. Neurol. 1972;22:849-55.
12. Dionisi-Vici C, Garavaglia B, Burlina AB, Bertini E, Saponara I, Sabetta G, et al. Hypoparathyroidism in mitochondrial trifunctional protein deficiency. J Pediatr. 1996;129:159-62.
13. Goswami R, Bhatia M, Goyal R, Kochupillai N. Reversible peripheral neuropathy in idiopathic hypoparathyroidism. Acta Neurol Scand 2002;105:128-31.
14. Shield KD, Parry C, Rehm J. Chronic diseases and conditions related to alcohol use. Alcohol Res Curr Rev. 2014;35(2):155.
15. Ghosh K, Bhattacharya A, Sengupta N. Pandora's box: Neurological manifestations of hypoparathyroidism. Indian J Endocrinol Metabol. 2017;21(4):638.

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