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Extensive Intracranial Calcification in a Case of Hypoparathyroidism Which Presented With Generalized Tonic-Clonic Seizure: A Case Report

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ABSTRACT

Hypoparathyroidism is an endocrine disorder that can be congenital or acquired. Generally, hypoparathyroidism is characterized by hypocalcemia, hyperphosphatemia, and low or abnormal levels of Parathyroid Hormone (PTH). It can be asymptomatic or symptomatic. The symptoms include seizures, paresthesia, depression, psychosis, extrapyramidal manifestations, and increased intracranial pressure. In this case study, we reported a 40-year-old male patient who was admitted to the emergency department with generalized tonic-clonic movements and urine incontinency. Laboratory investigations revealed hypocalcemia, hyperphosphatemia, and low parathyroid hormone levels and in paraclinical studies, including Electroencephalography (EEG) and brain CT-scan, despite normal EEG, extensive intracranial calcification involving the basal ganglia, thalamus, white matter of the cerebral hemispheres, and subcortical area of the frontal and parietal lobes were observed on CT-scan.

Introduction

Hypoparathyroidism is an endocrine disorder that can be congenital or acquired and the most common cause of its acquired type is iatrogenic damage due to anterior cervical surgery [1]. It can also be caused by radiotherapy to the cervical region, drugs, infiltration of the parathyroid glands, such as Wilson's disease, suppression of glandular function, or idiopathic [2, 3]. Generally, hypoparathyroidism is characterized by hypocalcemia, hyperphosphatemia, and low or abnormal levels of Parathyroid Hormone (PTH) and can be asymptomatic or with various manifestations, including neurological symptoms, such as seizures, paresthesia, de-

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pression, psychosis, extrapyramidal manifestations, and increased intracranial symptoms [1]. Hypoparathyroidism and pseudohypoparathyroidism can cause calcification of basal ganglia, cerebellum, subcortical white matter of cerebral lobes, including frontal and parietal, and generally cause extensive intracranial calcification [2, 4].

Case Presentation

A 40-year-old man from Rafsanjan city entered the emergency department of Aliebne Abitaleb General Hospital with generalized tonic-clonic movements followed by 40 minutes of urine incontinency. His movements were abnormal and periodic, and each time lasted for 3-4 minutes. Each attack consisted of consciousness loss with an interval of 5-10 minutes. The first episode had occurred in adolescence around 15 years of age and he had been treated by anticonvulsants for a short time and had stopped since then. Although the patient did not receive medication, he has not had another seizure for ten years. Also, according to the patient's companion, the patient has experienced jumping movements in his hands in the last 2 months and has been experiencing gastrointestinal symptoms since last week, including diarrhea and vomiting, which have not been treated specifically. The patient was married at the age of 25 and had two children. He had no family history of seizures but had a history of cataract surgery for both eyes at the age of 20 and 32, and a history of heart disorders over the past five years that have not been treated specifically.

On admission, only some parts of physical examinations were possible due to seizures (obvious tonic-clonic generalized movements) and lack of consciousness. The pupils were mid-sized and light-responsive, and his eye movements were normal, but papilledema was observed on fundoscopy. On the other hand, there were no abnormal findings in the examination of some cranial nerves, and the corneal reflex was normal, but the deep tendon reflexes were reduced and the plantar reflex was positive. Also, sensory examinations, including surface and deep touch, vibration, heat and pain, cortical sensations, and sensory level could not be examined. The patient's skin had no scarring indicating previous surgery, and there was no evidence of candidiasis or vitiligo in the mucosa and skin, and the nails growing were normal. The patient's height was also 153 cm.

In routine laboratory tests at the time of admission, the patient's hemoglobin was 13 mg/dl (Nl range: 14-16) and his White Blood Cell (WBC) count was 7,600 (neutrophil 86% and lymphocyte 7%. Also, the Blood Sugar (BS) was 126 mg/dl, blood urea was 39 mg/dl, and serum creatinine was 1.5 mg/dl (Nl range: 0.5 - 1.5). Moreover, the hepatic enzymes, including alkaline phosphatase, aspartate aminotransferase, and alanine aminotransferase, and serum bilirubin, and albumin were in the normal range. Serum electrolytes analysis showed following results: sodium: 140 mEq/l (Nl range: 135-145), potassium: 3.9 mEq/l (normal: 3.5 - 5.3), calcium: 4.1 mg/dl (Nl range: 8.5 to 10.4), serum phosphate: 5.6 mg/dl (Nl range: 2.5 to 5), and serum magnesium: 1.5 mg/dl (Nl range: 1.3 - 2.5). Vitamin D level was lower than normal range and Parathyroid Hormone (PTH) was 4 PG/ML (Nl range: 15-65), but thyroid hormones were in the normal range.

In paraclinical studies, the patient's EEG (Electroencephalography) was not abnormal, but in CT scans of the brain, bilateral symmetrical hyper-dependence lesions, and bilateral diffuse calcification were observed in the area of the basal ganglia, thalamus, white matter of the cerebral hemispheres, and subcortical area of the frontal and parietal lobes (Figures 1 and 2).

Regarding seizures, history of bilateral cataracts, hypocalcemia, hyperphosphatemia, and low levels of parathyroid hormone, the diagnosis of hypoparathyroidism and the resulting hypocalcemia were discussed. In this regard, the patient was treated with 10% intravenous calcium gluconate and calcium retainer, and cardiac monitoring was started for him. In spite of receiving intravenous calcium and magnesium, the patient's seizures were continued. Therefore, the patient was treated for status epilepsy. Unfortunately, despite the correction of calcium and magnesium disorders and vitamin D, the patient's consciousness did not return. At last, after a few days, the patient was died, which might be due to prolonged hypoxia before reaching the hospital.

Discussion

Hypoparathyroidism, regardless of its cause, can have a wide range of neurological, psychological, skin, and ocular symptoms [5, 6]. Psycho-neurologic symptoms include extrapyramidal manifestations, such as rigidity and tremor, seizures, decreased level of consciousness, cerebral symptoms, psychosis, depression, and cognitive impairment [5]. Moreover, skin and eye manifestations include skin changes, alopecia, changes in the nail bed, and cataracts [6]. Also, if the disease is left untreated, it can cause papilledema, dental diseases, and mental retardation [7]. Generally, the most common symptoms of the disease, which are also associated with hypocalcemia, are numbness, paraesthesia,

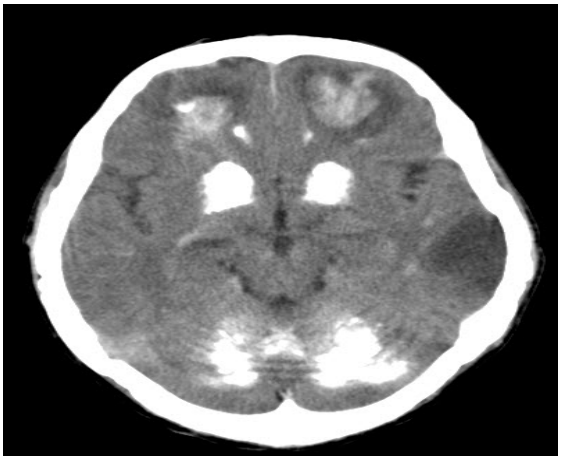

Figure 1. Non-contrast enhanced CT-scan at the level of mid-brain showing multiple bilaterally symmetrical hyperdense lesions with CT value of calcification in basal ganglia, thalami, and white matter of cerebellar hemispheres.

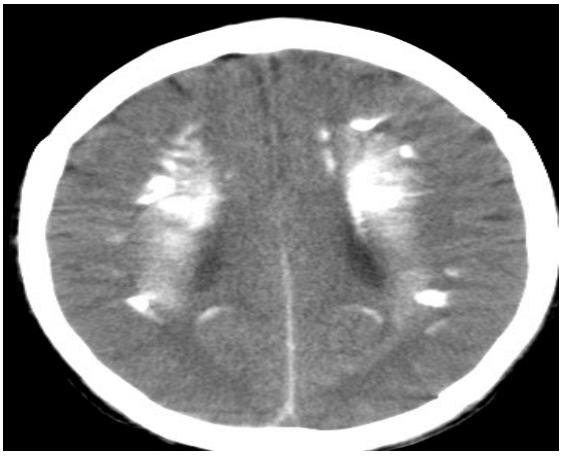

Figure 2. Non-contrast enhanced CT-scan at the level of the roof of lateral ventricles showing bilaterally symmetrical hyperdense lesions with CT value of calcification in the subcortical white matter of bilateral frontal and parietal.

muscle spasms, laryngeal spasms, tetany, and seizures [8]. However, seizures may even be the only sign of disease [9]. The case reported in this article, like the case reported by Moushumi et al. in 2014, involved abnormal body movements, rigidity, and tremor due to hypocalcemia. Also, despite the normal EEG, there were abnormal findings on brain CT scans that indicated brain calcification [10]. Although intracranial calcification is seen physiologically in 0.3-1.5% of individuals [11], the most common causes of basal ganglia calcification are hypoparathyroidism and pseudohypoparathyroidism [9]. Various studies have reported patients with obvious findings on brain CT scans, including basal ganglia calcification due to hypoparathyroidism and with different clinical manifestations [4, 9, 12].

Movement disorders, chorea, or parkinsonism are also seen in 20 to 30% of patients with basal ganglia calcification, while some patients may be asymptomatic [13]. In addition to basal ganglia calcification, in rare cases, as reported in this patient, some patients may develop calcification of the cerebral hemispheres, subcortical white matter of the cerebral lobes, including the frontal and parietal, and, more generally, extensive intracranial calcification [4, 14]. Calcification of basal ganglia can be also caused by other factors, such as carbon monoxide poisoning, Fahr's disease, encephalitis, idiopathic calcification of the basal ganglia, tuberculous, Cocayne syndrome, cerebellar vascular disease, brain parasites, neurofibromatosis, but decreased PTH level and hypocalcemia negate these causes [15]. It is noteworthy that due to the lack of facilities, it was not possible to perform genetic tests. Despite the correction of calcium and other electrolytes, the patient's seizures continued, which might be due to extensive cerebral calcification.

Conclusion

Although seizures are not a common finding in hypoparathyroidism, and extensive cerebral calcification is rare in hypoparathyroidism, it seems that clinicians should consider this disease and its brain calcification in patients with progressive neurological symptoms and treatment-resistant seizures, and by taking appropriate and early diagnostic and therapeutic measures, prevent the progression of symptoms and death of the patient. It also highlights the importance of checking calcium in patients with seizures.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Conflict of interest

The authors declared no conflict of interest.

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References

[1] Al-Azem H, Khan AA. Hypoparathyroidism. Best Practice & Research Clinical Endocrinology & Metabolism. 2012; 26(4):517-22. [DOI:10.1016/j.beem.2012.01.004] [PMID]

[2] Zisimopoulou V, Siatouni A, Tsoukalos G, Tavernarakis A, Gatzonis S. Extensive bilateral intracranial calcifications: A case of iatrogenic hypoparathyroidism. Case Reports in Medicine. 2013; 2013:932184 [DOI:10.1155/2013/932184] [PMID] [PMCID]

[3] Venugopal A, Acharya P, Kishore MS. Extensive intracranial calcification in idiopathic hypoparathyroidism: Rare presentation in an adolescent. Indian Journal of Radiology and Imaging. 2006; 16(2):181. [DOI:10.4103/0971-3026.29083]

[4] Rizvi I, Ansari NA, Beg M, Shamim D. Widespread intracranial calcification, seizures and extrapyramidal manifestations in a case of hypoparathyroidism. North American Journal of Medical Sciences. 2012; 4(8):369-72. [DOI:10.4103/1947-2714.99523] [PMID] [PMCID]

[5] Aggarwal S, Kailash S, Sagar R, Tripathi M, Sreenivas V, Sharma R, et al. Neuropsychological dysfunction in idiopathic hypoparathyroidism and its relationship with intracranial calcification and serum total calcium. European Journal of Endocrinology. 2013; 168(6):895-903. [DOI:10.1530/EJE-12-0946] [PMID]

[6] Stelmasiak Z, Tarach JS, Nowicka-Tarach BM, Mitosek-Szewczyk K, Drop A. Idiopathic hypoparathyroidism with intracranial calcifications and dominant skin manifestations. Medical Science Monitor. 2000; 6(1):145-50. [PMID]

[7] Fowler WM Jr, Ling S. Convulsions and papilledema in a child with idiopathic hypoparathyroidism. California Medicine. 1961; 95(3):180-2. [PMID] [PMCID]

[8] Rubin MR, Bilezikian JP. Hypoparathyroidism: Clinical features, skeletal microstructure and parathyroid hormone replacement. Arquivos Brasileiros de Endocrinologia & Metabologia. 2010; 54(2):220-6. [DOI:10.1590/S0004-27302010000200019] [PMID] [PMCID]

[9] Verma I, Gupta R, Singh S, Warkade D, Subhash N, Bose CH. Bilateral symmetrical basal ganglia calcification with atypical presentation: A case series. 2016. https://www.semanticscholar.org/paper/Bilateral-Symmetrical-Basal-Ganglia-Calcification-A-Verma-Gupta/0b1d8f05221aeff42c470f42a0148fbd8e5c5db5

[10] Moushumi L, Rajarshi M. Primary hypoparathyroidism misdiagnosed as epilepsy - a case report: Seizures, hypocalcemia and cerebral calcification. The Journal of the International Federation of Clinical Chemistry and Laboratory Medicine. 2014; 25(2):195-8. [PMID] [PMCID]

[11] Verulashvili IV, Glonti LSh, Miminoshvili DK, Maniia MN, Mdivani KS. Basal ganglia calcification: Clinical manifestations and diagnostic evaluation. Georgian Medical News. 2006; 140:39-43. [PMID]

[12] Basak RC. A case report of basal ganglia calcification: A rare finding of hypoparathyroidism. Oman Medical Journal. 2009; 24(3):220-2. [DOI:10.5001/omj.2009.44] [PMID] [PMCID]

[13] Koller WC, Cochran JW, Klawans HL. Calcification of the basal ganglia: Computerized tomography and clinical correlation. Neurology. 1979; 29(3):328-33. [DOI:10.1212/WNL.29.3.328] [PMID]

[14] Ehsaei MR, Abouei Mehrizi MA, Firouz F. Extensive intracranial calcification in a case of hypoparathyroidism: Case report. Iranian Journal of Neurosurgery. 2017; 2(4):22-4. [DOI:10.29252/irjns.2.4.22]

[15] Sabau M, Comanescu A, Maghiar T, Dinulescu D. Hypoparathyroidism diagnosed by neurological signs and widespread intracerebral calcifications. Romanian Journal of Neurology. 2010; 9(1):44-50. [DOI:10.37897/RJN.2010.1.8]