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Optic neuritis, as unusual manifestations of hypoparathyroidism: a rare case report

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Abstract

Background Optic neuritis, commonly associated with multiple sclerosis (MS), can also occur in the context of hypocalcemia as a rare intriguing clinical scenario. Most recent articles have reported the association of papilledema and hypoparathyroidism, However, very rarely, case reports revealed hypocalcemia-induced optic neuritis as well as our case.

Case presentation We report a 43-year-old woman with a history of hypocalcemia presenting with optic neuritis, characterized by sudden vision loss, painful eye movements, and pulsatile headache. Despite the normalization of serum calcium level and pulses of steroid treatment for the most probable underlying cause (demyelination); visual acuity improvement was not regained. Lumbar puncture revealed normal intracranial pressure, and MRI showed unspecific periventricular signal changes with T1 hyperintensity in bilateral basal ganglia and thalamic mostly in favor of calcifications. Additional investigations ruled out alternative conditions like multiple sclerosis and other inflammatory-based disorders.

Discussion Optic neuritis could have an association with hypocalcemia, with or without elevated intracranial pressure, which highlights the necessity for comprehensive evaluations in patients with visual symptoms despite normal intracranial pressure. While serum calcium correction effectively treats papilledema, its benefit for optic neuritis remains unclear. Further evaluations are needed to understand the exact pathology and optimal management of hypocalcemia-related optic neuritis.

Keywords Hypocalcemia, Optic neuritis, Papilledema

Background

Optic neuritis, characterized by inflammation of the optic nerve, has been recognized as a clinical entity associated with various systemic and neurological conditions [1]. While it is often linked to multiple sclerosis (MS) [1], the coincidence of optic neuritis within the context of hypocalcemia introduces a distinctive and unusual clinical scenario [2]. Hypocalcemia manifests with a diverse range of symptoms, including both neurological and ocular abnormalities [2, 3]. Reports have shown associations between hypocalcemia and optic disc involvement, especially papilledema or optic neuropathy. Notably, some cases have presented symptoms of optic neuritis in the setting of hypoparathyroidism [3], emphasizing the critical necessity to investigate the relationship between hypocalcemia and optic nerve involvement.

Herein, we report a 43-year-old female patient presented with optic neuritis manifestations and a history of hypocalcemia, so we aim to discuss the connection

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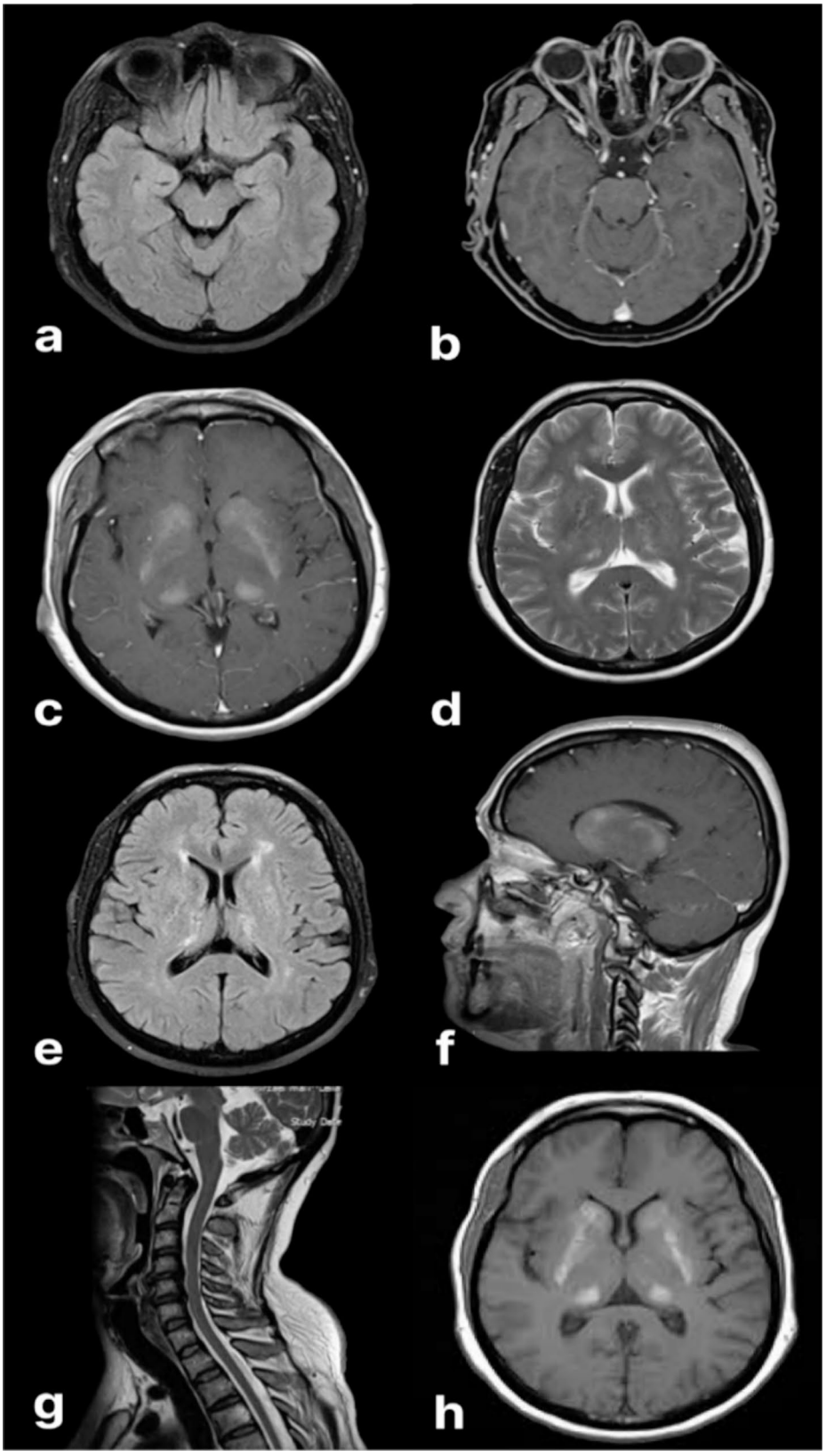

Fig. 1 (See legend on next page.)

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Fig. 1 Brain and cervical MRI. Brain MRI: **a**. No significant abnormalities were identified in the orbital and parenchymal regions. **b**. The T1-weighted images with gadolinium contrast showed no evidence of enhancement in the basilar region, optic nerve, or parenchymal areas. **c**. Unspecific hyperintense lesions of basal ganglia and thalamus were noted on the T1 sequence contrasted with GAD. **d**. The T2-weighted image showed Bilateral high-intense lesions in the thalamus and BG likely indicative of a mineralization process. **e**. Non-specific hyperintensities in the periventricular region observed on the FLAIR sequence (do not align with those typical of demyelinating lesions). **f**. The T1-weighted sagittal view demonstrates hyperintensity in the thalamus. **g**. T2-weighted (sagittal view) cervical MRI reveals disc bulging at C4-C5 and C5-C6, causing mild cord compression, with no inflammatory plaques present.

between systemic calcium imbalances and ocular pathology. This investigation offers a broader perspective on the potential consequences of hypocalcemia on the optic nerve and contributes to the understanding of rare clinical manifestations.

Case report

A 43-year-old female with a history of hypocalcemia and seizures since the age of 10 presented to the Bu Ali Sina Medical Center in Qazvin on October 3, 2022, with sudden left visual acuity loss, painful eye movements, and pulsatile left temporal headache that had started a week before. The patient's headache did not respond well to painkillers. In the initial examination, the visual field was limited in the left inferior and nasal area, and the visual acuity of the left eye was reduced to 6 of 10, while the right eye was 9 of 10. The left eye Marcus Gunn sign was positive, and fundoscopy showed blurred optic disc margins consistent with left eye papillitis. Other neurological examinations were normal. On October 5th, 2022, a brain and cervical MRI was conducted to rule out multiple sclerosis. The results showed bilateral T1 hyperintense signal within the basal ganglia and thalamus compatible with calcification and some unspecific preventricular hyperintensity changes. (see Fig. 1b and a).

As neuroretinitis was a possible diagnosis, an ophthalmology consultation was conducted, which ruled out this possibility.

Due to the patient's low serum calcium level of 6.1 mg/dL, a serum PTH level test and thyroid ultrasound imaging were performed. The serum PTH level was found to be lower than the normal range (2 mg./dl, normal: 14–65 mg/dl). The thyroid ultrasound imaging showed a 7*6 mm isoechoic nodule with small internal cystic areas and a brief lobulated margin in the middle posterior lobe of the right thyroid (TIRADS III). In addition, there was a slight increase in serum phosphorus level (4.8 MG/DL, normal: 304.5 mg/dl) and a decrease in serum magnesium level (1.5 mg/dl, normal: 1.7–2.2 mg/dl). However, other serum parameters, such as albumin level (3.7 mg/dl) and thyroid hormone levels (T4: 73nmol/l and TSH: 2.6 U/L), MOG (cut-off for the presence of MOG-Ab: 1:3200) were found to be normal.

Methylprednisolone therapy was initiated during the patient's first hospitalization for a suspected demyelinating process, administered for three days at a dose of 500 mg twice daily (total: 7 doses). At the time of discharge on October 9, 2022, the patient's symptoms, slightly improved after the serum calcium level was normalized by oral calcium replacement (CA: 9.0 MG/DL) and recommended to oral intake of prednisolone 50 mg which tapered and discontinue within 10 days and oral calcium replacement therapy for calcium depletion.

Following an initial improvement, the patient experienced a recrudescence of her presenting symptoms, including painful eye movements, pulsatile headache, and blurred vision, prompting her return to the medical center on November 7, 2022. A cerebrospinal fluid (CSF) sample was obtained through a lumbar puncture (LP). The results showed a normal opening pressure of 17 cmH2O, a glucose level within the normal range at 48 mg/dl, and a slightly elevated lactate dehydrogenase (LDH) level of 35 units/L. The protein level was also normal at 38 mg/dl, along with other parameters remaining normal. Despite there was no improvement in the patient's symptoms after LP. No pathological lesions were found in the patient's orbital MRI conducted after the recurrence of symptoms. The orbital MRI conducted after the recurrence of symptoms revealed no pathological lesions. (Fig. 1a). The Visual Evoked Potentials (Table 1) showed an abnormality of more than 8ms interpeak latency of p100 latency on the left side compared to the right side, as well as low amplitude on the left side. Other laboratory findings included negative NMO AB (IgG) (AQP4) and C ANCA, anti-phospholipid antibody (IgG) within the normal range, normal C3 and CH50 levels, negative anti-double-stranded DNA, negative anti-HCV, HIV (Ab & Ag), and HBS (Ag & Ab), negative HLA B5, and negative CSF elect-OCB. The patient followed up for one year. Although the patient's initial headache resolved completely, her visual acuity declined progressively to 4/10 without any response to treatment.

Discussion

Hypocalcemia can cause numerous health issues affecting various bodily systems such as the neuromuscular, cardiovascular, and ocular systems [4]. Although muscle cramps, tetany, and seizures are commonly associated with hypocalcemia; ocular symptoms such as cataracts, papilledema, and optic neuritis may also occur [4, 5]. Analyzing past cases uncovers some interesting similarities around the ocular manifestations. While papilledema is the most common sign [6–8], there have been a few reports where optic neuritis has occurred in the context

Table 1 VEP

	wave	Latency		
		N75	**P100**	**N145**
Right	**A1**	66.9ms	96.9ms	124.5ms
Left	**A1**	76.5ms	110.1ms	125.1ms

Abnormal pattern study due to left-side interpeak latency of P100 being more than 8 ms compared to the related right side, along with low amplitude on the left side

of hypocalcemia as well [9]. It is important to note that increased intracranial pressure is a prevalent factor for such cases, and the link between optic nerve involvement and raised intracranial pressure is well-established [6, 8]. However, it is worth noting that there are some rare cases where the elevation of ICP is absent [9], as observed in our case. One of the most similar cases to ours, presented as a 54-year-old man who suffered from optic neuritis due to primary hypoparathyroidism. The patient's lumbar puncture revealed an opening pressure of 21 cm, and their serum calcium level was below the normal range at 1.32 mmol/L [9]. Due to lacking evidence of increased intracranial pressure in our case, which is the most recognized pathology for ocular nerve involvement, underscores the importance of investigating other etiological factors and pathways. Therefore, it is crucial to conduct a comprehensive evaluation of patients presenting with visual symptoms, particularly those with normal ICP levels, to identify other possible causes of optic nerve inflammation. On the other hand, there was a significant possibility that we were dealing with demyelinating processes, particularly multiple sclerosis and neuromyelitis optica, which were ruled out due to not meeting the McDonald and NMOSD criteria, furthermore not responding to prednisolone pulses. Upon analyzing patient outcomes, it was revealed that cases of optic neuritis did not exhibit any improvement in visual field enhancement, as well as our case.

However, it can be noted that papilledema can be effectively treated by correcting the levels of serum calcium. These findings suggest that serum calcium correction may be an optional treatment for papilledema in the context of hypocalcemia, however, in the case of optic neuritis, no ocular improvement should be expected for calcium replacement. Thereby, it is worth mentioning that idiopathic optic neuritis may potentially arise as a consequence of hypocalcemia.

Another hypothesis is that there was a coincidence between hypocalcemia and optic neuritis.

So, in the future, further investigations are crucial for well knowing the underlying mechanisms and management of patients with demyelination conditions that are associated with hypocalcemia.

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Author contributions
This article was written by Dr. Yousef Mirmazloomi, who contributed the main text, images, and tables. Dr. Forough Ebrahimtabar assisted with scientific editing and data collection. Dr. Hossein Mozhdehipanah performed the final editing.

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Data availability
The sequence data that support the findings of this study have been deposited in the Qazvin University of Medical Science under primary accession code ir.qums.rec.1403.063.

Declarations

Consent for publication
The patient has provided **written informed consent** to publish their personal and clinical details, including any identifying images, as part of this study.

Competing interests
The authors declare no competing interests.

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References

1. Group* ONS. The 5-year risk of MS after optic neuritis: experience of the optic neuritis treatment trial. Neurology. 1997;49(5):1404–13.
2. McLean C, Lobo R, Brazier D. Clinical & laboratory research-optic disc involvement in hypocalcaemia with hypoparathyroidism: papilloedema or optic neuropathy? Neuro Ophthalmol. 1998;20(3):117–24.
3. BAJANDAS FJ, SMITH JL. Optic neuritis in hypoparathyroidism. Neurology. 1976;26(5):451- Goltzman D, Rosen C, Mulder J. Clinical manifestations of hypocalcemia. UpToDate Rose BD, ed Wellesley MA: UpToDate. 2010.
4. Rao MN, Shoback DM. Hypocalcemia. 2016.
5. Goyal J, Kang J, Gupta R, Anand A, Arora R, Jain P. Bilateral papilledema in hypocalcemia. Delhi J Ophthalmol. 2012;23(2):127–30.
6. Palmer RF, Searles HH, Boldrey EB. Papilloedema and hypoparathyroidism simulating brain tumor. J Neurosurg. 1959;16(4):378–84.
7. Sheldon RS, Becker WJ, Hanley DA, Culver RL. Hypoparathyroidism and pseudotumor cerebri: an infrequent clinical association. Can J Neurol Sci. 1987;14(4):622–5.
8. Vukovic D, Pajic S. Hypocalcemia induced severe heart insufficiency and visual acuity Impairment–Will calcium supplementation improve heart and eye function. J Blood Lymph. 2015;4(130):2.
9. Arshad MF, Karim R, Paling D, Jayamanne G. Optic neuritis as presenting primary hypoparathyroidism. BMJ Case Rep CP. 2018;11(1):e227032.

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