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CASE REPORT

Thyroid-Associated Optic Neuropathy: A Case Report of Optic Neuritis Due to Autoimmune Hypothyroidism

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Introduction: Optic neuritis is an uncommon complication of autoimmune hypothyroidism, which is often referred to as thyroid-associated optic neuropathy (TAON).

Case Report: The case features a 22-year-old Somali woman who had no previous medical conditions. She had sudden vision loss, mainly in her right eye. This was accompanied by joint discomfort, muscular weakness, headaches, and weariness. The clinical examination showed complete absence of light perception in the right eye and there was a relative afferent pupillary deficit, and the thyroid function tests indicated severe hypothyroidism with significantly high levels of thyroid-stimulating hormone (TSH) and low levels of free Triiodothyronine (T3) and Thyroxine (T4). The diagnosis of autoimmune hypothyroidism was confirmed by the presence of positive anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin antibodies. Treatment was promptly initiated with high-dose corticosteroids (methylprednisolone) and levothyroxine replacement medication. Subsequently, her field of vision and visual acuity, when corrected with the best possible lenses, showed a considerable improvement. Further assessments demonstrated the restoration of normal thyroid function and the complete remission of symptoms related to ocular neuritis, with no further instances of recurrence.

Conclusion: The present case emphasizes the crucial need of promptly identifying and handling TAON in order to achieve positive visual results. This highlights the need of taking into account autoimmune hypothyroidism as a potential cause of ocular neuritis. Additional study is necessary to clarify the exact pathophysiological pathways that link autoimmune hypothyroidism to inflammation of the optic nerve, as well as to investigate the most effective therapy strategies.

Keywords: thyroid-associated optic neuropathy, optic neuritis, autoimmune hypothyroidism, Somalia

Introduction

Optic neuritis (ON) is an inflammatory disorder described by demyelination of the optic nerve, leading in acute or subacute vision loss, usually unilateral. While multiple sclerosis (MS) is the most frequently related condition, optic neuritis can develop from different factors, including autoimmune diseases.[1,2] One such autoimmune disease associated with optic neuritis is autoimmune hypothyroidism, also known as Hashimoto's thyroiditis.[3] Autoimmune hypothyroidism is described by the increased synthesis of autoantibodies, such as anti-thyroglobulin (anti-TG) and anti-thyroid peroxidase (anti-TPO) antibodies, which acts against thyroid antigens, resulting in thyroid gland inflammation and later developing hypothyroidism.[4] Despite being a known complication, optic neuritis secondary to autoimmune hypothyroidism (Thyroid-associated optic neuropathy) remains relatively rare and downplayed. This case report tends to describe a rare example of optic neuritis in the setting of autoimmune hypothyroidism, describing its clinical features, diagnostic approach, and management strategies.

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The relationship between autoimmune hypothyroidism and optic neuritis has been reported in several studies. Autoimmune diseases often have systemic manifestations affecting various organs, including the eyes. A study by Hennes and coworkers found an important association between autoimmune hypothyroidism and optic neuritis, suggesting a possible autoimmune-mediated mechanism underlying optic nerve inflammation.[5] İn addition, a retrospective study by Tellez and colleagues concluded an increased incidence of optic neuritis in patients with autoimmune hypothyroidism compared to the general population.[6] These results highlighting the significance of adding autoimmune hypothyroidism in the differential diagnosis of optic neuritis, specially in cases with atypical features or coexistence autoimmune manifestations.

TAON and dysthyroid optic neuropathy (DON) are two distinct complications, which differs significantly in etiology, clinical presentation, and management. TAON is rare and involves inflammatory demyelination of the optic nerve associated with thyroid dysfunction, resembling optic neuritis with painful vision loss, dyschromatopsia, while DON, a sight-threatening manifestation of Graves' orbitopathy, results from optic nerve compression at the orbital apex due to enlarged extraocular muscles and orbital fat.[7,8]

The pathophysiology of optic neuritis in autoimmune hypothyroidism remains mystery but is believed to be immune-mediated mechanisms. Thyroid hormone disparity in hypothyroidism can result in metabolic impairments and oxidative stress, possibly aggravating nerve injury in the optic nerve.[9] However, future studies are needed to explain the exact pathogenic mechanisms associating autoimmune hypothyroidism and optic neuritis.

In the presented case report, we describe a case of a 22-year-old female patient diagnosed with optic neuritis due to autoimmune hypothyroidism.

Case Report

A 22-year-old Somali woman patient who had previously been in good condition came to the emergency department with a complaint of an acute visual loss for 1 week. The visual loss was more severe in the right eye, and there was a complete no light perception, while the left eye had a 70% light perception. There was also right eye relative afferent pupillary defect. Intraocular pressure measured on left was 11 mmHg and right was 14 mmHg. Anterior segment examination was normal and both optic discs were seen to be normal in the fundus examination. Extraocular muscles were also examined as normal.

Moreover, the patient reported joint discomfort, muscle weakness, and headaches along with weariness. She appeared sick upon examination, but her consciousness level was intact, she was cooperative. No other relevant positive findings were detected in her examination.

The patient's past medical history was uneventful, and she had no family history of autoimmune diseases. Her vital signs indicate a temperature level of 35°C, her blood pressure was 115/80, her pulse was 70 pbm, and her respiratory rate was 18 breaths per minute. Brain MRI with contrast reported no brain parenchymal disease. Routine blood was normal except thyroid profile TSH was 115.6 mIU/L (range 0.35–5.1), T3 was 0.01 nmol/L (range 1.8–3.9), and T4 was 0.01 ng/dL (range 0.5–1.8). Further laboratory tests revealed a positive anti-tpo and anti- thyroglobulin tests, 74000 IU/mL (range 0–9) and 635.18 IU/mL (range 0–4), respectively. Thyroid ultrasound was reported enlarged thyroid gland with a hypoechoic, diffusely heterogeneous echotexture with no solid nodules or cystic features.

The patient was admitted to the neurology department under the diagnosis of retrobulbar neuritis optic neuritis possibly due to Hashimoto thyroiditis (Thyroid-associated optic neuropathy). A high-dose of corticosteroids (1 gram of methylprednisolone/day) was immediately started and with 100 mcg of levothyroxine treatment. The corticosteroid therapy was decreased after 3 days, and a 1mg/kg of daily dose of prednisolone was commenced. Subsequent to the corticosteroid treatment and thyroid replacement therapy, the visual field markedly recovered and the best corrected visual acuity were both measured at 0.5 (50%) in the right eye and 1 (100%) in the left eye.

These treatment regimens also diminished the general symptoms related to hypothyroidism and normalized TSH and free T4 levels after 2 months. No recurrence of optic neuritis occurred for follow-up after 3 months of treatment.

Discussion

The thyroid-associated optic neuropathy, also known as TAON, is a very uncommon but serious complication of thyroid ailments that can have an effect on both one's vision and their quality of life. Although the particular pathophysiology of

TAON is not well known, it is predicted to entail a complex link between immune-related inflammatory processes, vascular impairment, and the direct toxic implications of a reduced level of thyroid hormone on the optic nerve.[10,11] Other factors that may contribute to the development of TAON include vascular issues.

The significance of including TAON in the differential diagnosis of sudden vision loss is highlighted by our case, especially in young patients who also exhibit systemic signs of hypothyroidism. Initiating high-dose corticosteroid treatment promptly, as was done in this case, can effectively decrease inflammation and maintain visual function. Furthermore, it is crucial to treat the root cause of thyroid dysfunction with hormone replacement therapy in order to effectively manage optic neuritis in the long term and avoid its recurrence.

Research conducted globally has shown different occurrence rates of TAON, emphasizing the necessity for increased knowledge and attentiveness among medical professionals.[12,13] As an illustration, a case report that had been published by Domenico Bonifati and his coworkers provided a comprehensive account of the situation of a woman who was 26 years old and had experienced a rapid loss of eyesight as well as a swelling of the optic disc. Following further investigation, it was determined that the patient was suffering from Hashimoto's thyroiditis, which was successfully treated with corticosteroids and replacement thyroid hormones.[14] Additionally, in a case that was described by Chun et al, a woman who was 33 years old and suffered from optic neuritis and Hashimoto's thyroiditis showed significant improvement after receiving corticosteroid therapy and levothyroxine replacement.[15] These instances highlight the need to take into account autoimmune thyroid illness as a potential cause of optic neuritis and applying suitable techniques for its therapy.

In addition, Gorman et al conducted a study on six individuals with optic neuritis linked to autoimmune thyroid disease. The study highlighted the various ways in which the condition might manifest clinically and demonstrated the effectiveness of corticosteroid treatment in enhancing visual results.[16] This series emphasized the possibility of optic neuritis being a symptom of autoimmune thyroid disease, requiring a thorough assessment and coordinated care from other medical disciplines.

Case reports of thyroid association complications were reported previously in Somalia, but this was the first case report describing TAON in Somalia todate.[17,18] TAON was diagnosed based on the indications that were mentioned in our case report. These clues included elevated levels of thyroid-stimulating hormone (TSH), decreased levels of thyroxine (T4), and high titers of anti-thyroid antibodies. A considerable improvement was observed as a consequence of the rapid start of treatment for hypothyroidism, which lends credence to the findings of other studies that have demonstrated the beneficial effects of thyroid hormone replacement therapy on visual outcomes in patients who have TAON.[19,20] It is clear that ophthalmologists, endocrinologists, and neurologists have to collaborate together in order to deliver comprehensive care for TAON.

Although the autoimmune cause of TAON is well acknowledged, it is important to consider other aspects that may contribute to the condition, such as microvascular ischemia and the direct toxic effects of thyroid dysfunction on the optic nerve.[12,21] Research conducted in several parts of the world has demonstrated a connection between thyroid dysfunction and damage to the optic nerve, underscoring the worldwide importance of this issue.[22,23] It is crucial to conduct regular and extended monitoring of thyroid function in order to avoid the reoccurence of optic neuropathy and enhance visual results in individuals with TAON.

Optic neuritis in multiple sclerosis (MS) and neuromyelitis optica spectrum disorder (NMOSD) differ from TAON in pathophysiology, presentation, and management. MS-related (ON) involves autoimmune demyelination, while NMOSD is linked to anti-AQP4 antibodies causing astrocyte injury. Clinically, MS/NMOSD ON presents with acute, painful vision loss, often severe in NMOSD, whereas TAON features resembling optic neuritis with painful vision loss, dyschromatopsia, and optic nerve enhancement on imaging. MS/NMOSD ON is treated with immunomodulatory therapies, while TAON requires corticosteroids. Recognizing these differences is essential for targeted treatment and optimal patient outcomes[7,8] Table 1.

The likelihood of developing MS in a patient who has an episode of acute optic neuritis is around 75% in women and 34% in men over the next 15–20 years. The highest risk occurs within the first 5 years following the incident, so the patient has to be followed up routinely.[8]

Table 1 The Differential Diagnosis of Acute Optic Nerve Pathology

Diseases	Key Features	Imaging Findings	Laboratory Tests
Neuromyelitis Optica (NMOSD)	Severe vision loss, often bilateral. Poor recovery. Associated with anti-AQP4 antibodies.	Optic nerve enhancement, often long segments.	Serum AQP4-IgG antibodies.
Ischemic Optic Neuropathy	Sudden, painless vision loss. Associated with arteritic (eg, GCA) or non-arteritic causes.	No enhancement on imaging. Swollen optic disc.	ESR, CRP, temporal artery biopsy (GCA).
Dysthyroid Optic Neuropathy	Gradual vision loss, proptosis, restricted eye movements. Related to Graves' orbitopathy.	Orbital muscle enlargement, no tendon involvement.	Thyroid function tests, TSH receptor antibodies.
Thyroid-Associated Optic Neuritis (TAON)	Painful vision loss, dyschromatopsia. Resembles inflammatory optic neuritis. Associated with thyroid dysfunction.	Optic nerve enhancement without compression.	Thyroid function tests, TSH receptor antibodies.
Ocular Vasculitis	Pain, ischemic optic neuropathy, systemic vasculitis symptoms (eg, rash, joint pain).	May show ischemia; optic nerve infarction.	ANCA, ESR, CRP.
Compressive Optic Neuropathy	Progressive vision loss, often painless. Can be caused by tumors or trauma.	Mass effect or optic nerve compression on MRI.	Specific tumor related tests.
Toxic/Metabolic Neuropathy	Gradual vision loss, bilateral. Associated with toxins (eg, methanol) or vitamin deficiencies.	Normal imaging.	Serum B12, folate, toxicology screen.
Infectious Optic Neuropathy	Associated with syphilis, tuberculosis, or viral infections. May have systemic symptoms.	May show optic nerve inflammation.	VDRL, FTA-ABS, TB tests, viral serologies.
Leber's Hereditary Optic Neuropathy (LHON)	Gradual, painless vision loss. Typically affects young males.	Normal or atrophic optic nerves.	Mitochondrial DNA testing.

Conclusion

In short, the findings of our case study highlight the critical role that thyroid hormone replacement treatment plays in attaining good visual recovery in individuals who have TAON. Increased awareness, quick diagnosis, and care that involves several disciplines are essential components in order to achieve optimal results in this infrequent but potentially life-threatening ailment. It is necessary to conduct additional studies in order to acquire a better understanding of the mechanisms that underlie TAON and to investigate innovative treatment strategies that are aimed at maintaining the integrity of the optic nerve and enhancing visual results.

Abbreviation

Anti-TG, anti-thyroglobulin; Anti-TPO, Anti-thyroid peroxidase; MS, Multiple sclerosis; TAON, Thyroid-associated optic neuropathy; TSH, thyroid-stimulating hormone.

Ethical Approval and Consent for Publication

Mogadishu Somali Turkish Training and Research Hospital waived approval for this case report. A written and Oral informed consent was obtained from the patient. İn addition, a consent for publishing this case report anonymously was granted by the patient.

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Disclosure

The authors declare no competing interests relating to this case report.

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