Cureus
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Open Access Case Report

An Unusual Case of Optic Neuritis With Roth Spots in Hashimoto's Thyroiditis

Neha Singh [1], Shagun Gaur [1], Alka Tripathi [1], Amit Thangjam [1]

1. Ophthalmology, All India Institute of Medical Sciences Gorakhpur, Gorakhpur, IND

Corresponding author: Neha Singh, nhsngh.89@gmail.com

Review began 09/17/2026
Review ended 11/04/2026
Published 11/06/2026

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DOI: 10.7759/cureus.119251

Abstract

Thyroid-associated optic neuropathy (TAON) is an infrequent complication linked to autoimmune hypothyroidism. We present the clinical course of a 22-year-old female with a presumed case of TAON who presented with bilateral visual impairment and ocular pain. Clinical assessment revealed bilateral optic disc swelling, with the right eye exhibiting pronounced edema accompanied by peripapillary hemorrhages and Roth spots. Laboratory investigations confirmed elevated anti-thyroid peroxidase (anti-TPO) antibodies and deranged thyroid profiles, consistent with Hashimoto's thyroiditis. Magnetic resonance imaging (MRI) demonstrated subtle enhancement of the right optic nerve, without evidence of demyelinating brain lesions. Serological assessments for aquaporin-4 and myelin oligodendrocyte glycoprotein (MOG) antibodies were negative. Management involved a five-day course of intravenous methylprednisolone, followed by oral corticosteroids, alongside levothyroxine therapy. At one month, visual acuity in the left eye improved to 3/60, while the right eye remained without light perception. Disc edema and retinal hemorrhages resolved, with residual bilateral optic disc pallor. This case reports a rare entity of TAON along with the unusual occurrence of a Roth spot, presenting a diagnostic challenge.

Categories: Neurology, Ophthalmology, Endocrinology/Diabetes/Metabolism
Keywords: autoimmune hypothyroidism, bilateral optic neuritis, hypothyroidism, optic disc edema, roths spot

Introduction

Inflammatory optic neuropathies, such as optic neuritis, characteristically manifest in a single eye and affect approximately five out of every 100,000 individuals annually [1]. Bilateral involvement is less common, making up 19-50% of adult optic neuropathy presentations [2,3]. These bilateral cases frequently stem from atypical etiologies like myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease (MOGAD), neuromyelitis optica spectrum disorder (NMOSD), or various infectious and systemic autoimmune conditions. Optic neuritis has very rarely been described in association with autoimmune hypothyroidism, and the entity is termed thyroid-associated optic neuropathy (TAON) [4]. The mechanism underlying the development of TAON largely remains uncertain because of the very few cases reported to date [4,5]. Immune-mediated inflammation and the toxic and metabolic effects of long-standing hypothyroidism on the optic nerve are some of the proposed explanations [4]. Roth spots, defined as retinal hemorrhages with pale centers, are classically tied to bacterial endocarditis, though they may also arise from leukemia, anoxia, HIV retinopathy, or profound anemia [6]. Their presence in demyelinating diseases like MOGAD is exceptionally rare. In this report, we describe a young female patient who developed bilateral optic neuropathy and Roth spots in the setting of Hashimoto's thyroiditis. A similar case of optic neuritis associated with autoimmune hypothyroidism in a 22-year-old woman has been reported by Hidig et al. [4]. The co-occurrence of Roth spots is an observation from our case.

Case Presentation

A 22-year-old female presented to the clinic reporting acute, painful vision loss in her right eye that had progressed over four days, accompanied by a one-day history of blurred vision in her left eye. The patient lacked any recent history of trauma, febrile illness, or systemic infection. However, she was a known case of hypothyroidism for five years, for which she had discontinued her prescribed medication one year earlier. Systemic evaluation indicated a thin physique and normal blood pressure. Her recorded weight was 35 kg and height approximately 1.37 m, corresponding to an estimated BMI of 18.6 kg/m^2. Ophthalmic examination showed that her left eye vision was limited to counting fingers close to the face, while her right eye had no light perception. Both anterior segments appeared unremarkable, though a relative afferent pupillary defect was evident on the right side. Ocular movements were full in both eyes, with no diplopia or limitation of ocular motility. Dilated fundoscopy revealed blurred disc margins bilaterally, indicative of optic disc edema. The swelling was more prominent in the right eye (Frisen grade 3-4, Figure *1A*), which additionally exhibited Roth spots and peripapillary hemorrhages, compared with the milder swelling in the left eye (Frisen grade 2, Figure *1B*). Formal visual field assessment, color vision testing, and optical coherence tomography (OCT) could not be performed because of the patient's markedly reduced visual acuity.

How to cite this article
Singh N, Gaur S, Tripathi A, et al. (November 06, 2026) An Unusual Case of Optic Neuritis With Roth Spots in Hashimoto's Thyroiditis. Cureus 18(11): e119251. DOI 10.7759/cureus.119251

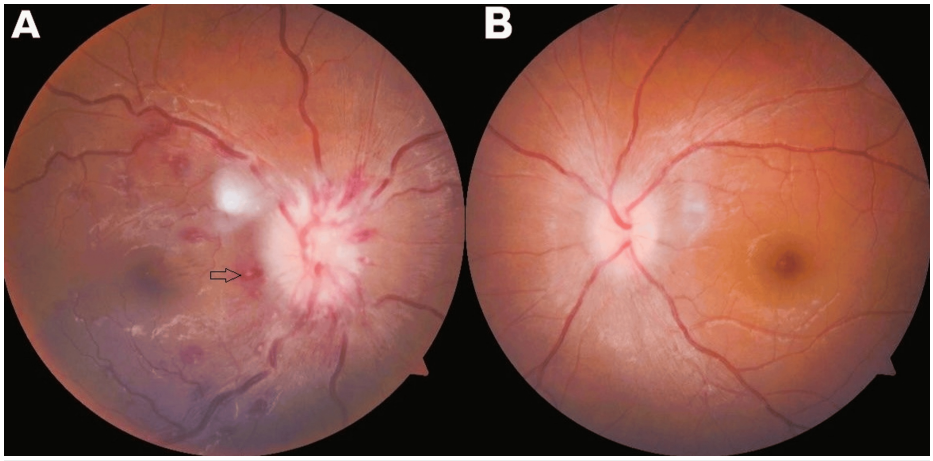


FIGURE 1: Fundus photographs at presentation

(A) The right eye with blurred disc margins, peripapillary hemorrhages, and Roth spots (arrow).

(B) The left eye with blurred disc margins.

Magnetic resonance imaging (MRI) of the brain and orbits with contrast showed mild swelling with subtle T2/fluid-attenuated inversion recovery (FLAIR) hyperintensity involving the right optic nerve (Figure *2*). The left optic nerve appeared normal. Brain parenchyma, the ventricular system, and cerebrospinal fluid (CSF) spaces were normal, with no evidence of raised intracranial pressure. No demyelinating lesions were identified. T1- and T2-weighted MRI images of the orbits showed no evidence of extraocular muscle enlargement or myopathy.

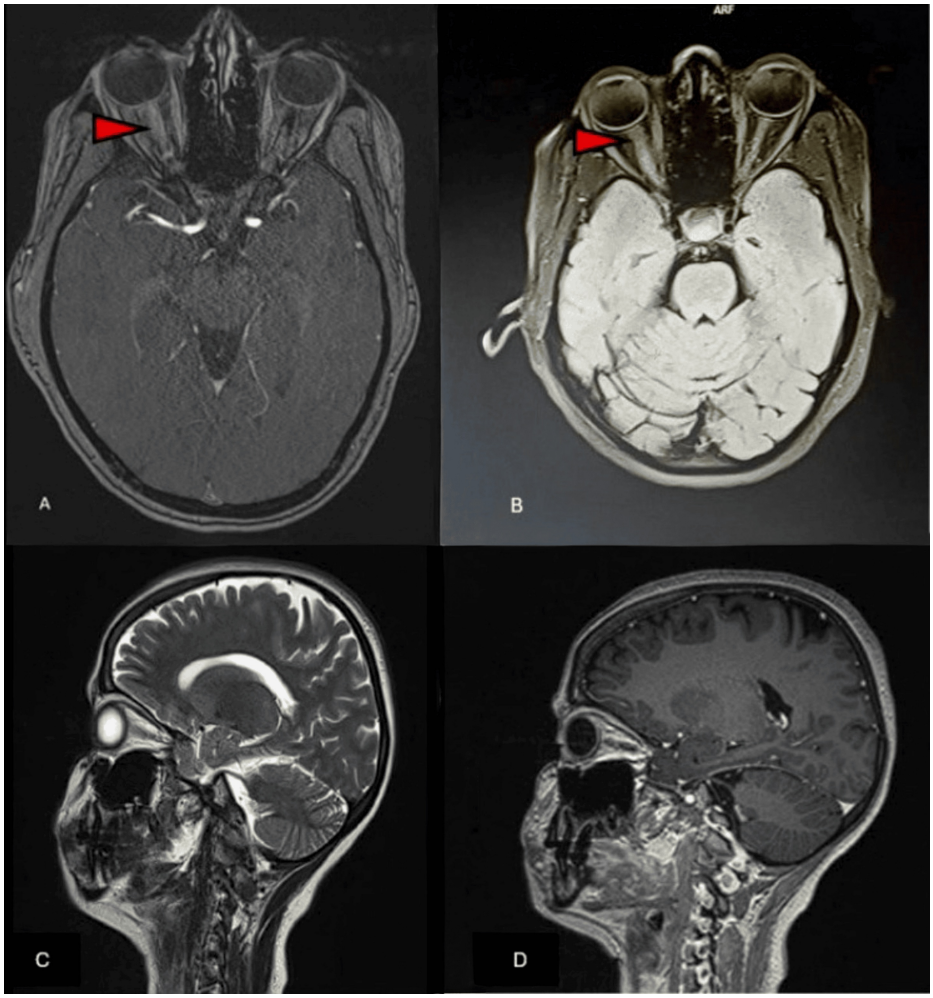


FIGURE 2: MRI of the brain and orbits

(A) Axial post-contrast T1-weighted image demonstrating enhancement of the right optic nerve (red arrowhead).

(B) Axial T2-weighted FLAIR image demonstrating signal abnormality involving the right optic nerve (red arrowhead).

(C) Sagittal T2-weighted image showing normal ventricular size and preserved brain architecture.

(D) Sagittal post-contrast T1-weighted image showing no abnormal parenchymal or optic nerve enhancement.

FLAIR, fluid-attenuated inversion recovery; MRI, magnetic resonance imaging

Endocrine workup revealed a deranged thyroid state: thyroid-stimulating hormone (TSH) exceeded 150 µIU/mL (normal range: 0.4-4.9 µIU/mL), free thyroxine (T4) was reduced at 3.28 µg/dL (normal range: 5-14 µg/dL), and triiodothyronine (T3) was 10.5 ng/dL (normal range: 80-200 ng/dL). Autoimmune screening showed positive anti-thyroid peroxidase (anti-TPO) antibodies at 77.79 IU/mL (normal range: 0-34 IU/mL), while anti-thyroglobulin titers remained normal (range: <1.30 IU/mL). In view of the presence of Roth spots, systemic investigations were performed to evaluate potential hematological, inflammatory, or infectious causes. Complete blood counts, peripheral blood smear, erythrocyte sedimentation rate, C-reactive protein, and liver and kidney function tests were within normal limits. Furthermore, serology for MOG and aquaporin-4 (NMO) antibodies returned negative. A lumbar puncture performed in the lateral decubitus position showed normal CSF constituents and an opening pressure of 18 cm of water. Ultrasound imaging of the neck revealed an atrophic thyroid gland, pointing toward chronic thyroiditis.

Therapeutic intervention commenced with admission for pulse corticosteroid therapy, consisting of intravenous methylprednisolone (1 g/day) for five days. Concurrently, she resumed oral levothyroxine (50 µg/day) to address the underlying hypothyroidism. Following the intravenous regimen, she was transitioned to oral prednisolone (40 mg/day) with a weekly tapering schedule.

At the one-month follow-up assessment, visual acuity in her left eye had improved to 3/60, whereas the right eye remained without light perception. Fundoscopic evaluation demonstrated resolution of retinal hemorrhages and disc edema in both eyes, leaving behind bilateral optic disc pallor (Figure 3).

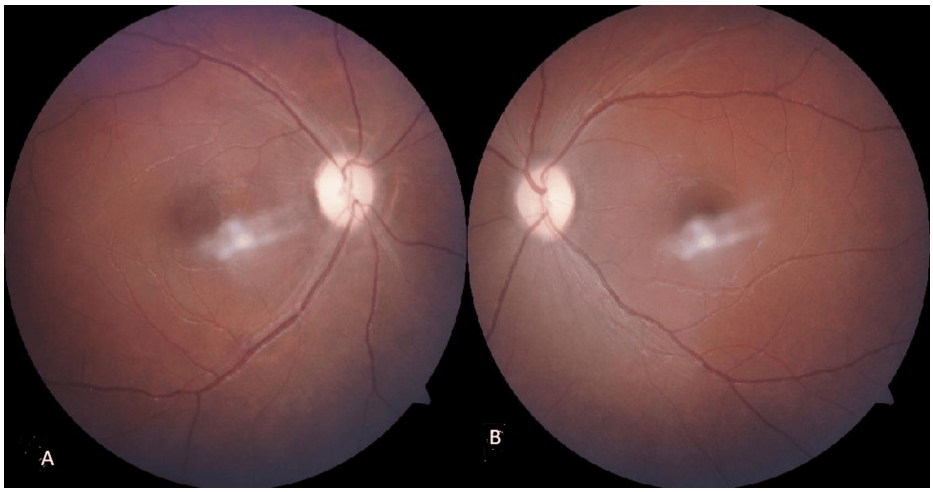


FIGURE 3: Fundus photographs at one-month follow-up

(A,B) Optic disc pallor in both eyes with resolved hemorrhages and edema.

Discussion

Bilateral optic disc edema in a young patient presents a broad differential diagnosis, including inflammatory optic neuropathy, demyelinating disorders, vascular causes, and raised intracranial pressure. The differential diagnosis process is summarized in Table 1, formulated by the authors to reflect stepwise clinical reasoning based on available evidence.

Differential Diagnosis	Considered Because	Rationale for Lower Diagnostic Likelihood
MS	A common cause of optic neuritis in young adults; typically, unilateral	No demyelinating lesions on MRI; no other neurological signs; bilateral onset is atypical for MS.
NMOSD	Presents with bilateral optic neuritis	Negative serum AQP4-IgG antibodies. Seronegative patients require at least two core clinical characteristics, including at least one of optic neuritis, acute myelitis, or area postrema syndrome, together with additional specified clinical and MRI criteria [7], which were absent.
MOGAD	Can cause bilateral optic neuritis with a good steroid response	MOG antibodies negative; poor visual recovery despite treatment; no typical imaging signs. However, these findings do not independently exclude the diagnosis.
Dysthyroid optic neuropathy	Deranged thyroid profile	There were no orbital signs or muscle enlargement on imaging.
Ischemic optic neuropathy	May occur in severe systemic hypotension or anemia	No hypotension or risk factors; young age makes this unlikely.
Autoimmune optic neuropathy (Hashimoto-related)	Rarely reported with Hashimoto's thyroiditis	Diagnosis of exclusion; compatible with prior reports of optic neuropathy linked to autoimmune hypothyroidism.
IIH	Bilateral disc edema; association with hypothyroidism	Low body mass index; normal CSF opening pressure, asymmetric painful vision loss; optic nerve signal changes.

TABLE 1: Differential diagnosis approach

MS, multiple sclerosis; NMOSD, neuromyelitis optica spectrum disorder; MOG, myelin oligodendrocyte glycoprotein; MOGAD, MOG antibody-associated disease; IIH, idiopathic intracranial hypertension; AQP4-IgG, aquaporin-4 immunoglobulin G; MRI, magnetic resonance imaging; CSF, cerebrospinal fluid

Given the patient's age and clinical presentation, the initial differential diagnosis included demyelinating disorders such as multiple sclerosis (MS), NMOSD, and MOGAD [8,9]. MS was considered less likely because of the absence of characteristic demyelinating lesions on brain MRI and other neurological manifestations, although these findings do not definitively exclude the diagnosis. NMOSD was also considered less likely in view of negative serum aquaporin-4 immunoglobulin G (AQP4-IgG) antibody testing, the absence of other systemic features, and the supportive MRI findings. Similarly, MOGAD was considered less likely because of negative serum MOG-IgG testing, the absence of characteristic supportive MRI findings, and poor visual recovery following corticosteroid therapy. Nevertheless, negative antibody results and the absence of characteristic MRI abnormalities reduce the likelihood of these disorders but do not independently exclude them.

Idiopathic intracranial hypertension (IIH) was also considered because of the bilateral optic disc edema. However, brain MRI revealed no mass lesion or hydrocephalus, and lumbar puncture performed in the lateral decubitus position demonstrated normal CSF constituents and an opening pressure of 18 cm H_2O. These findings did not support IIH. Ischemic optic neuropathy was considered less likely given the patient's young age and the absence of documented hypotension or other recognized vascular risk factors. In the absence of a more compelling alternative diagnosis, autoimmune optic neuropathy associated with Hashimoto's thyroiditis was considered the most plausible explanation for the clinical presentation.

TAON and dysthyroid optic neuropathy (DON) represent distinct potential mechanisms of optic nerve dysfunction in the setting of thyroid disease. DON typically occurs in association with thyroid eye disease and results from optic nerve compression at the orbital apex, commonly due to extraocular muscle enlargement and apical crowding [10]. In the present case, the absence of orbital signs and imaging evidence of extraocular muscle enlargement or orbital apical crowding made DON less likely. In contrast, TAON has been described as a rare optic neuropathy associated with autoimmune thyroid dysfunction that may clinically resemble inflammatory optic neuritis, with features such as painful visual loss and dyschromatopsia [11]. However, its clinical characteristics and pathophysiology remain incompletely defined, and the diagnosis is largely one of exclusion.

The pathophysiology of TAON is not fully understood but may involve immune-mediated inflammation, microvascular dysfunction, and metabolic alterations associated with thyroid hormone deficiency [10,11]. Case reports by Hidig et al. [4] and Singhai et al. [5] have described optic neuropathy in patients with autoimmune hypothyroidism, suggesting a possible association between thyroid autoimmunity and optic nerve dysfunction.

Plasma exchange may be considered in severe inflammatory optic neuritis with an inadequate response to high-dose intravenous corticosteroids, particularly when an antibody-mediated demyelinating disorder is suspected. However, its role in TAON remains uncertain, and there is insufficient evidence to recommend it routinely for this condition.

A particularly unique feature in this case was the manifestation of Roth spots. While these lesions are classically observed in oncologic, inflammatory, or infectious scenarios, our patient's comprehensive negative systemic workup made these secondary causes less likely. Though Roth spots have been documented alongside MOG-related optic neuritis [12,13], their presence here likely reflects severe ischemic and oxidative injury to the right optic nerve, explaining the profound and irreversible visual deficit in that eye. However, this explanation is speculative, and the causal association cannot be established by a single case report.

This case serves to emphasize an atypical manifestation of bilateral optic neuropathy driven by long-standing, unmanaged Hashimoto's thyroiditis.

Conclusions

This case highlights the diagnostic challenges of bilateral optic neuropathy in a patient with long-standing, inadequately treated Hashimoto's thyroiditis. It reinforces the importance of a systematic evaluation to identify alternative inflammatory, demyelinating, compressive, vascular, and intracranial causes before considering TAON. The coexistence of Roth spots represents an additional unusual finding whose clinical significance requires further investigation.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Neha Singh, Shagun Gaur

Critical review of the manuscript for important intellectual content: Neha Singh, Shagun Gaur, Alka Tripathi, Amit Thangjam

Acquisition, analysis, or interpretation of data: Shagun Gaur, Alka Tripathi, Amit Thangjam

Drafting of the manuscript: Shagun Gaur, Amit Thangjam

Supervision: Alka Tripathi

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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