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DIRECTION-CHANGING GAZE-EVOKED NYSTAGMUS WITH PERSISTENT OSCILLOPSIA IN A PATIENT WITH HASHIMOTO'S THYROIDITIS: A CASE REPORT AND REVIEW OF VESTIBULO-OCULAR DYSFUNCTION IN AUTOIMMUNE THYROID DISEASE

Dr. Rahul[1*], Dr. Subha L[2], Dr. Balambighai V[3]

[1]Postgraduate Resident Department of General Medicine Sree Balaji Medical College & Hospital, BIHER Chennai, Tamil Nadu, India
Email: dravidrahul80@gmail.com
[2]Professor Department of OphthalmologySree Balaji Medical College & Hospital, BIHER Chennai, Tamil Nadu, India,
Email: drsubharaghu@gmail.com
[3]MBBS, DNB (Paediatrics), DrNB (Neurology), Associate Professor Department of Neurology Sree Balaji Medical College & Hospital, BIHER Chromepet, Chennai – 600075 Tamil Nadu, India, balapeds@gmail.com

ABSTRACT

Background: Direction-changing gaze-evoked nystagmus with persistent oscillopsia is an unusual neuro-otological manifestation and may indicate dysfunction of central or peripheral vestibulo-ocular pathways. Autoimmune thyroid disease, particularly Hashimoto's thyroiditis, has been increasingly recognised as a contributor to vestibular and ocular-motor abnormalities. The coexistence of disabling oscillopsia, mixed-pattern nystagmus, and markedly elevated thyroid autoantibody titres remains rarely documented.

Case Presentation: A 31-year-old woman presented with a 10-month history of oscillopsia and shaking of images on lateral gaze, preceded by blurring of lateral vision during the fifth month of pregnancy. Her antenatal period had been complicated by severe hyperemesis and neurological symptoms for which she was treated as Wernicke's encephalopathy earlier in the year. Despite partial improvement, oscillopsia and intermittent headaches persisted. She had a known history of hypothyroidism for four years on levothyroxine 100 mcg/ day On examination, she was conscious and afebrile, with stable vital signs. Neurological evaluation revealed bilateral gaze-evoked nystagmus with rotatory, direction-changing, upbeat, and downbeat components. Motor, sensory, and cerebellar functions were largely normal except for mild difficulty in tandem walking. MRI brain showed small bilateral frontoparietal T2/FLAIR hyperintensities of nonspecific etiology, and VEMP (**vestibular evoked myogenic potential**) testing was normal. Laboratory workup demonstrated markedly elevated thyroid peroxidase antibodies (>1000 IU/mL) and mildly raised alkaline phosphatase. Thyroid ultrasonography revealed a hypoechoic nodule in the left lobe.Endocrinologist opined to start intravenous pulse dose steroids.

The patient was initiated on intravenous methylprednisolone 1g for five days along with thiamine and supportive therapy. She tolerated treatment well, with mild subjective improvement in oscillopsia, and was discharged in stable condition with a tapering steroid regimen, continuation of levothyroxine, and structured follow-up.

Conclusion: This case highlights a rare presentation of direction-changing gaze-evoked nystagmus and persistent oscillopsia in the context of autoimmune thyroid disease. The findings emphasise the importance of evaluating thyroid autoimmunity in patients with atypical nystagmus patterns, even when vestibular testing is normal. Early neuro-vestibular assessment and timely immunomodulatory therapy may improve outcomes. This report contributes to the emerging evidence linking thyroid autoimmunity with vestibulo-ocular dysfunction and underscores the need for heightened diagnostic vigilance.

KEYWORDS: Hashimoto Thyroiditis , Oscillopsia. , Vestibulo-Ocular dysfunction .

INTRODUCTION

Oscillopsia accompanied by direction-changing gaze-evoked nystagmus represents an uncommon and diagnostically challenging neuro-otological presentation, particularly when it arises in association with autoimmune thyroid disease. Hashimoto's thyroiditis is one of the most prevalent autoimmune endocrinological disorders worldwide and exhibits considerable clinical variability across geographic regions, including India, where distinct phenotypic patterns and antibody trends have been described [1]. This heterogeneity underscores the importance of recognising unusual neurological manifestations in individuals with thyroid autoimmunity.

Growing scientific evidence indicates that thyroid autoantibodies may adversely influence both peripheral and central vestibular pathways. Studies conducted on euthyroid individuals with autoimmune thyroiditis have demonstrated vestibular abnormalities independent of hormonal status, suggesting that immune-mediated mechanisms contribute significantly to balance and ocular-motor disturbances [2]. Additional clinical

observations have confirmed that vestibular dysfunction can occur across a spectrum of patients with autoimmune thyroid disease, with manifestations ranging from episodic vertigo to persistent gait unsteadiness and abnormal ocular-motor responses [3].

More recent work has provided objective evidence of vestibular organ involvement in autoimmune thyroid disease, including measurable abnormalities in semicircular canal and otolithic function [4]. Systematic evaluation has also identified an association between benign paroxysmal positional vertigo and thyroid disorders, further supporting the concept that thyroid autoimmunity may predispose individuals to recurrent vestibular disturbances through metabolic, microvascular, or neuroepithelial mechanisms [5]. Beyond the peripheral vestibular system, autoimmune processes are increasingly recognised as contributors to central vertigo and ocular-motor dysfunction, with documented effects on cerebellar and brainstem pathways essential for gaze stability [6].

Despite these insights, reports detailing the coexistence of persistent oscillopsia, mixed-pattern nystagmus, and serological evidence of autoimmune thyroiditis remain limited. Oscillopsia, particularly when severe, reflects a disruption of neural integrators responsible for maintaining steady visual fixation and is therefore an important clinical clue toward deeper vestibulo-ocular or cerebellar involvement. When accompanied by direction-changing nystagmus, the diagnostic complexity increases, requiring careful correlation of neuroimaging, autoimmune markers, and metabolic history. This becomes especially relevant in patients who have undergone prior treatment for conditions such as Wernicke's encephalopathy, where overlapping clinical features may obscure the underlying pathology.

The present case gains significance in this context by illustrating a constellation of disabling oscillopsia, direction-changing gaze-evoked nystagmus, non-specific white-matter changes on MRI, and markedly elevated thyroid autoantibodies. The case highlights the importance of considering thyroid autoimmunity in the differential diagnosis of unexplained nystagmus and underscores the value of early recognition, comprehensive neuro-vestibular evaluation, and timely initiation of immunomodulatory therapy. Through this observation, the report contributes to the growing understanding of vestibulo-ocular dysfunction in autoimmune thyroid disease and reinforces the need for heightened clinical vigilance.

CASE REPORT

A 31-year-old woman presented with a 10-month history of persistent oscillopsia and shaking of images on both lateral gazes. The symptoms began in January 2025, following a preceding phase of blurring of vision which started during the fifth month of pregnancy in late December 2024. Her pregnancy had been complicated by severe hyperemesis until the fourth month of gestation, associated with progressive imbalance, vertigo, headache, and difficulty in walking. She had been treated earlier elsewhere for suspected Wernicke's encephalopathy, after which partial improvement in gait and vision occurred; however, oscillopsia and intermittent headaches persisted.

There was no history of trauma, seizures, or loss of consciousness. She was a known case of hypothyroidism for four years and was on levothyroxine 100 mcg daily. There was no history of diabetes mellitus, hypertension, coronary artery disease, or chronic kidney disease. Her dietary and sleep patterns were normal, and no relevant family history was noted.

On admission, she was conscious, oriented, and afebrile. Her blood pressure was 130/80 mmHg, heart rate 100 beats per minute, and oxygen saturation 99% on room air. Higher mental functions were normal. Cranial nerve examination showed normal primary gaze but revealed bilateral gaze-evoked nystagmus with a rotatory component. Direction-changing nystagmus was present on horizontal gaze, and both upbeat and downbeat components were elicited on respective gaze positions. Motor, sensory, autonomic, and extrapyramidal examinations were normal. Cerebellar assessment showed mild difficulty during tandem walking, although limb coordination remained intact. Systemic examinations were unremarkable.

MRI brain with contrast demonstrated small bilateral frontoparietal white-matter hyperintensities on T2/FLAIR sequences, reported as non-specific. Vestibular evoked myogenic potential (VEMP) was normal. Laboratory evaluation revealed markedly elevated thyroid peroxidase antibody titres (>1000 IU/mL) and mildly raised alkaline phosphatase, while other routine parameters remained within normal limits. Thyroid ultrasonography showed a hypoechoic nodule in the left lobe of the thyroid gland.

Given the symptom profile, examination findings, significantly elevated thyroid autoantibody levels, and non-specific white-matter abnormalities, a working diagnosis of direction-changing gaze-evoked nystagmus with persistent oscillopsia in the setting of possible autoimmune thyroiditis was made. After multidisciplinary discussion, the patient was initiated on intravenous methylprednisolone for five days along with thiamine supplementation and supportive therapy. She tolerated treatment well and remained hemodynamically stable throughout hospitalization. Mild symptomatic improvement was observed, with slight reduction in oscillopsia, though nystagmus persisted.

No new neurological deficits developed during the hospital stay. She was discharged in stable condition with a tapering schedule of oral corticosteroids, continuation of levothyroxine, nutritional supplementation, and advice for assisted ambulation initially. Follow-up with neurology and endocrinology was arranged for further evaluation of autoimmune thyroid disease and monitoring of visual-vestibular symptoms.

This case demonstrates a rare presentation of persistent oscillopsia with direction-changing nystagmus in a patient with strong autoimmune thyroid markers, emphasizing the need for thorough neuro-vestibular assessment in individuals with thyroid autoimmunity presenting with unexplained ocular-motor disturbances.

Table 1. Vital signs and laboratory parameters of the patient on admission

S. No	Parameter	Result
1	Blood Pressure	130/80 mmHg
2	Pulse Rate	100 bpm
3	Temperature	Afebrile
4	SpO_2	99% (Room Air)
5	Thyroid Peroxidase Antibodies (TPO Ab)	>1000 IU/mL (Markedly elevated)
6	Alkaline Phosphatase (ALP)	Mildly elevated
7	MRI Brain (T2/FLAIR)	Small bilateral frontoparietal hyperintensities (Non-specific)
8	VEMP	Normal

Table 2. Clinical presentation, neurological examination, and hospital course

S. No	Parameter	Result
1	Primary Complaint	Oscillopsia and shaking of images on lateral gaze
2	Duration of Symptoms	Since January 2025
3	Visual Complaint	Blurring of lateral vision since December 2024
4	Obstetric History	Severe hyperemesis till 4th month of gestation
5	Past Neurological History	Treated as Wernicke's encephalopathy in April 2025
6	Associated Symptoms	Headache, vertigo, gait difficulty
7	Past Medical History	Hypothyroidism × 4 years (Thyronorm 100 mcg)
8	Negative History	No trauma / No seizures / No LOC / No T2DM / No HTN / No CAD / No CKD
9	General Condition	Conscious, oriented, afebrile
10	Higher Mental Functions	Normal
11	Cranial Nerve Examination	Primary gaze normal
12	Nystagmus Findings	Bilateral gaze-evoked; rotatory; direction-changing; upbeat and downbeat components
13	Motor System	Normal
14	Sensory System	Normal
15	Cerebellar System	Mild difficulty in tandem walking
16	Autonomic System	Normal
17	Extrapyramidal System	Normal
18	Thyroid Ultrasound	Hypoechoic nodule in left thyroid lobe
19	In-hospital Treatment	IV methylprednisolone × 5 days + Thiamine + supportive care
20	Treatment Response	Mild improvement; oscillopsia persists; no new deficits
21	Condition at Discharge	Stable
22	Discharge Medications	Thyronorm, Folvite, Wysolone taper, PAN, Shelcal-Neu, Benfomet Forte

DISCUSSION

Direction-changing gaze-evoked nystagmus with persistent oscillopsia is an uncommon manifestation in autoimmune thyroid disease, making this case noteworthy both diagnostically and pathophysiologically. The markedly elevated thyroid peroxidase antibody titre in the present patient (>1000 IU/mL) and the constellation of vestibulo-ocular abnormalities suggest a strong immune-mediated mechanism affecting central and/or peripheral vestibular pathways. Several Indian and international studies support this association, strengthening the biological plausibility of the patient's clinical presentation.

Thyroid-related vestibular dysfunction has been increasingly recognised in Indian settings. Rai et al. reported that abnormal thyroid metabolism can influence vestibular symptoms even in patients presenting primarily with vertigo, emphasizing a potential endocrine–vestibular interface that may modify ocular motor control [7]. In the present case, despite the absence of overt hypothyroid symptoms during admission, profoundly elevated TPO antibodies indicate an underlying autoimmune process capable of altering vestibulo-ocular stability.

Audiovestibular involvement in hypothyroidism has been described more than two decades ago. Malik et al. demonstrated significant auditory and vestibular abnormalities in hypothyroid patients, indicating that thyroid

dysfunction can exert subclinical yet measurable effects on neural pathways involved in balance and gaze fixation [8]. The direction-changing nature of nystagmus observed in the current case parallels these findings, suggesting central integrator or cerebellar involvement.

Objective vestibular testing findings in Indian cohorts further corroborate this relationship. Moideen et al. found that videonystagmography frequently detects gaze-evoked and positional nystagmus patterns in patients with balance disorders, many of whom had underlying metabolic or endocrine abnormalities, including thyroid dysfunction [9]. This aligns with the patient's bilateral gaze-evoked nystagmus and instability in tandem gait, despite normal VEMP responses.

Audiological studies reinforce a neurophysiological basis for thyroid-related vestibular abnormalities. Singh et al. demonstrated that hypothyroid patients often present with combined auditory and vestibular deficits, which improve with thyroxine therapy, underscoring hormonal and immunological contributions to vestibular impairment [10]. Although the present patient was already on appropriate levothyroxine replacement, the persistent elevation of TPO antibodies suggests that the autoimmune rather than hormonal component may be the dominant driver in her case.

Autoimmune thyroid disease can also impact central auditory pathways. Sharma et al. reported abnormal brainstem auditory evoked potentials in hypothyroid individuals, pointing to central conduction delays attributable to metabolic or immune-mediated myelination defects [11]. The nonspecific white-matter hyperintensities seen on MRI in the current case correspond well with these observations, signifying possible subclinical demyelination or inflammation.

Additional Indian evidence from Trivedi et al. confirms that thyroid hormone imbalance contributes to audiological dysfunction, with measurable derangements even in mild or subclinical disease [12]. This highlights the need for vigilance regarding vestibulo-ocular manifestations in patients with thyroid autoimmunity, as seen in this case where symptoms persisted despite stable thyroid hormone replacement.

The role of thyroxine therapy in reversing neuro-otological deficits has also been documented. Singh et al. described significant improvement in auditory and vestibular parameters following replacement therapy in hypothyroid patients [13]. However, persistent symptoms in this patient despite ongoing therapy suggest a predominantly immune-driven rather than solely metabolic pathology.

Thyroid-related comorbidities can predispose to neuro-sensory dysfunction. Hsu et al. identified hypothyroidism as a risk factor for tinnitus through alterations in neurovascular and neuroinflammatory pathways, supporting the broader concept that thyroid dysfunction can affect sensory integration across multiple systems [14,15]. Although tinnitus was not reported by the patient, the study reinforces the multisystemic nature of thyroid autoimmunity and its capacity to disrupt sensory pathways, including vestibulo-ocular circuits.

Taken together, the referenced literature substantiates the association between autoimmune thyroid disease and vestibulo-ocular disturbances. The patient's presentation—direction-changing nystagmus, oscillopsia, subtle cerebellar signs, and strongly positive thyroid antibodies—fits within the expanding spectrum of thyroid-related neuro-otological dysfunction. The partial improvement following corticosteroid therapy further supports an inflammatory or autoimmune mechanism. This case highlights the importance of incorporating thyroid autoimmunity into the differential diagnosis of atypical nystagmus presentations and underscores the need for early immunomodulatory intervention in selected patients.

CONCLUSION

This case highlights an uncommon but clinically significant association between autoimmune thyroid disease and complex vestibulo-ocular disturbances. The presence of direction-changing gaze-evoked nystagmus, persistent oscillopsia, and subtle cerebellar signs in a patient with markedly elevated thyroid peroxidase antibodies underscores the importance of recognising thyroid autoimmunity as a potential contributor to unexplained ocular-motor abnormalities. The findings reinforce the growing evidence that immune-mediated mechanisms, rather than hormonal imbalance alone, may disrupt central and peripheral vestibular pathways.

The partial clinical improvement following corticosteroid therapy further supports an underlying autoimmune inflammatory process. Early identification, comprehensive neuro-vestibular evaluation, and prompt initiation of immunomodulatory treatment may help prevent progression and improve functional outcomes in similar cases. This report adds to the limited but evolving literature on vestibulo-ocular dysfunction in autoimmune thyroid disease and highlights the need for heightened diagnostic vigilance in patients presenting with atypical nystagmus patterns.

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