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Case Study

AYURVEDIC MANAGEMENT OF PROGRESSIVE CEREBELLAR ATAXIA ASSOCIATED WITH AUTOIMMUNE THYROID DISEASE

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ABSTRACT

Cerebellar ataxia is characterized by impaired balance, coordination and motor control and may result from hereditary, degenerative, metabolic, endocrine or autoimmune disorders. Its association with autoimmune thyroid disease, particularly Hashimoto thyroiditis, is uncommon and may occur even after restoration of euthyroidism. Ayurveda considers such neurological manifestations within the broader concept of *Vatavyadhi*, with *Vata*, *Avarana* and *Dhatu-kshaya* being relevant to the clinical presentation. A 20-year-old male with a seven-year history of hypothyroidism presented with progressive gait imbalance, recurrent falls, impaired coordination, and slowness of speech and writing. MRI of the brain demonstrated diffuse bilateral cerebellar volume loss disproportionate to age, with prominence of cerebellar folia and ex-vacuo dilatation of the fourth ventricle. Thyroid evaluation showed markedly elevated anti-TPO and anti-thyroglobulin antibodies, while testing for common spinocerebellar ataxia repeat expansions was negative. Ayurvedic assessment indicated predominantly *Vata-vyadhi* with *Agnimandya, Ama, Srotodushti, Avarana* and *Dhatu-kshaya*. The patient received individualized multimodal Ayurvedic management comprising internal medicines, *Snehapana, Virechana, Abhyanga, Swedana, Nasya, Shiro Pichu, Talapothichil, Pizhichil* and *Mustadi Rajayapana Basti*. The Scale for the Assessment and Rating of Ataxia (SARA) score improved from 13 at baseline to 6 at the time of discharge, with improvement in gait, stance and limb coordination. Multimodal Ayurvedic management was associated with marked functional improvement in this case of cerebellar ataxia with autoimmune thyroid disease. However, being a single case, causality cannot be established and longer-term follow-up is required.

INTRODUCTION

Ataxia is a neurological disorder characterized by impaired balance, coordination and motor control, commonly resulting from dysfunction of the cerebellum or its associated pathways. Clinically, cerebellar ataxia may present with unsteady gait, impaired limb coordination, dysarthria and difficulty performing precise movements. The clinical pattern, tempo and symmetry of progression are important in determining the underlying cause. Ataxia may be acute, subacute or chronic.

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Acute causes include intoxication, drugs and post-infectious cerebellitis, whereas subacute ataxia may occur with metabolic, nutritional, autoimmune or paraneoplastic disorders. Chronic progressive ataxia raises consideration of hereditary or degenerative disorders, metabolic causes and potentially reversible endocrine disorders such as hypothyroidism.[1]

Hashimoto thyroiditis is a chronic autoimmune thyroid disorder characterized by immune-mediated destruction of thyroid follicular cells and the presence of circulating thyroid autoantibodies, particularly anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies. Neurological manifestations associated with autoimmune thyroid disease may occur in the setting of thyroid hormone deficiency as well as through mechanisms independent of thyroid hormone status. Thyroid hormone plays an important role in neuronal development and cerebellar function;

therefore, significant hypothyroidism may result in neurological dysfunction including cerebellar ataxia. In patients in whom hypothyroidism is responsible for the cerebellar syndrome, thyroid hormone replacement may result in partial or complete improvement of ataxia.[2,3]

However, cerebellar ataxia may occasionally persist or progress despite restoration of euthyroidism. Selim and Drachman described progressive non-familial cerebellar degeneration in patients with autoimmune thyroiditis and elevated antithyroid antibodies, in whom cerebellar atrophy was demonstrated on magnetic resonance imaging and the progression of ataxia was not modified by L-thyroxine treatment. These observations suggested that mechanisms other than thyroid hormone deficiency, including an autoimmune mechanism, may contribute to cerebellar dysfunction associated with autoimmune thyroid disease.[3]

From an Ayurvedic perspective, the clinical presentation of Ataxia can be classified under *Vatavyadhi*, particularly given the role of *Vata* in maintaining normal movement and neurological functions. Sushruta describes the progressive nature of *Vata* disorders, stating that *Vata* initially localised to a particular body part or *Dhatu*, when neglected, may progressively spread and become *Sarvangagata*.[4]

Case Report

Patient Information

Age– 20 years

Gender– Male

Marital status- Unmarried

Socioeconomic status- Lower middle class

A 20-year-old male college student, a known case of hypothyroidism, presented with a seven-year history of progressive impairment of balance while walking, difficulty in lifting and carrying heavy objects with a tendency to fall, and slowness in writing and speech.

According to the patient, he was apparently healthy and able to perform his routine activities normally until the 9th standard. While studying in the 10th standard, he gradually developed weakness, difficulty in walking, and a tendency to fall, particularly while carrying heavy objects. In view of these complaints, he underwent detailed medical evaluation at Modern Hospital during which hypothyroidism was detected for the first time. Thyroid hormone replacement therapy was initiated, and he was also advised to continue physiotherapy. His symptoms showed some improvement with physiotherapy.

Initially, he was prescribed Thyronorm 100µg/day. The dose was subsequently reduced to 75µg/day approximately one year ago, and for the past eight months, he has been taking Thyronorm 50µg/day. There was no reported family history of hypothyroidism.

In 2021, the patient developed COVID-19 infection associated with severe vomiting, fever, and headache, following which there was a marked exacerbation of his pre-existing neurological symptoms. His balance deteriorated considerably, resulting in difficulty in maintaining an upright standing posture, for which he was hospitalised for one day.

An MRI brain with CVT study performed on 25 December 2021 revealed diffuse bilateral cerebellar volume loss, disproportionate to the patient's age, with resultant ex vacuo dilatation of the fourth ventricle and prominence of the cerebellar folia. The cerebrum was unremarkable, and the CVT study showed no significant abnormality.

Following this episode, the impairment of balance persisted. During his plus two studies, the complaints gradually worsened, with increasing difficulty in walking and maintaining balance, along with slowness in writing and speech. He was admitted to our hospital for further evaluation and management of the persisting neurological complaints.

Comorbidities

He was diagnosed with hypothyroidism since 7 years during adolescence and has been on thyroid hormone replacement therapy since then.

History of past illness

1. The patient had a single episode of seizure at the age of 5 years, with no reported recurrence thereafter.
2. H/o Covid-19: 3 yrs back.

Family History

Family history was largely non-contributory, except Father have DM. No other significant familial neurological illness was reported.

Drug History

The patient is currently receiving levothyroxine 50µg once daily for hypothyroidism. No other significant long-term medication history was reported.

Neuroimaging

MRI brain with CVT study dated 25 December 2021 demonstrated diffuse bilateral cerebellar volume loss disproportionate to the patient's age, with resultant ex vacuo dilatation of the fourth ventricle and prominence of the cerebellar folia. The cerebrum was unremarkable, and the CVT study revealed no significant abnormality. A subsequent CT brain reportedly demonstrated findings consistent with the previously documented cerebellar volume loss.

Personal History

- Bowel: Regular
- Appetite: Reduced

- Micturition: 4-5 times/day, 0-1 times/night
- Sleep: Sound
- Allergies: Nil
- Habits: Nil
- Addiction: Nil

Socioeconomic History: Lower middle class

Educational status: B com

Occupational status: Student

General Examination

On general examination at time of visit, vitals of patient were normal. BMI was 17.2 kg/m^2

Timeline

Year/period	Clinical events/investigations
Until 9th standard	Apparently healthy and able to perform routine activities normally.
10th standard	Gradual onset of weakness, difficulty walking, impaired balance, and tendency to fall, particularly while lifting/carrying heavy objects.
Initial medical evaluation	Evaluated at modern hospital. Hypothyroidism was diagnosed for the first time. Thyroid hormone replacement and physiotherapy were initiated.
Initial treatment period	Thyronorm 100µg/day was started. The dose was subsequently reduced to 75µg/day and later to 50µg/day.
2020	Thyroid investigations showed TSH 87.5, FT4 0.8 ng/dL and markedly elevated anti-TPO antibodies (>1000 IU/mL).
23/01/2021	Spinocerebellar Ataxia panel performed. SCA1, SCA2, SCA3, SCA6, SCA7, SCA12 and SCA17 repeat expansions were not detected.
December 2021	Developed COVID-19 infection with severe vomiting, fever and headache. Following the infection, his pre-existing neurological symptoms markedly worsened, with severe deterioration of balance and difficulty maintaining an upright posture. He required hospitalisation for one day.
2022–2025	Persistent impairment of balance and progressive difficulty in walking. During plus two studies, gait and postural instability gradually worsened, accompanied by increasing slowness of writing and speech.
20/02/2025	Thyroid profile showed improvement in thyroid function: FT4 1.05 ng/dL, TSH 1.227, while anti-TPO remained >1000 IU/mL and anti-TG was 806.24 IU/mL.
24/06/2026	Admitted to the hospital for detailed evaluation and Ayurvedic management of persistent neurological complaints.
24/08/2026	At discharge, clinical assessment showed improvement in ataxia, with the SARA score decreasing from 13 at baseline to 6 at discharge.

Clinical Findings

Nervous System Examination

The patient was conscious and oriented to place and time. Immediate, recent and remote memory were normal. Speech was dysfluent. All cranial nerves were intact.

Co-ordination		
	Right	**Left**
Diadochokinesis	Possible	Possible
Finger nose test	Slightly impaired	Slightly impaired
Heel shin test	Slightly impaired	Slightly impaired
Romberg test	Negative	
Tandem walking	Not possible	

Investigation

	28/10/2020	20/2/2025
FT4	0.8 ng/dl	1.05 ng/dl
T4	5.43	
T3	108.2	
TSH	87.5	1.227
Anti TPOAb	>1000 IU/ml	>1000 IU/ml
ATG	-	806.24 IU/ml

Spinocerebellar Ataxia (SCA) Panel-23/01/2021

Ataxia	CAG Repeats	Interpretation
SCA1	28 on Allele 1; 30 on Allele 2	Not detected – Diagnosis of SCA1 excluded
SCA2	22 on both alleles	Not detected – Diagnosis of SCA2 excluded
MJD/SCA3	19 on Allele 1; 23 on Allele 2	Not detected – Diagnosis of SCA3 excluded
SCA6	11 on both alleles	Not detected – Diagnosis of SCA6 excluded
SCA7	9 on both alleles	Not detected – Diagnosis of SCA7 excluded
SCA12	10 on both alleles	Not detected – Diagnosis of SCA12 excluded
SCA17	36 on Allele 1; 38 on Allele 2	Not detected – Diagnosis of SCA17 excluded

Ayurvedic Clinical Assessment

Dasavidha pareeksha	
Dooshyam	*Dhatu-Rasa, Mamsa, Majja* *Upadhatu- Snayu*
Desham	*Bhoomi-Sadharanam* *Deha- Sarva deha*
Balam	*Rogi- Avara* *Rogam- Madhyama*
Kalam	*Kshanadi- Sarvakala* *Vyadhyavastha- Purana*
Analam	*Madhyamam*
Prakriti	*Vatapitta*
Vaya	*Youvana*
Satwam	*Madura*
Sathyam	*Madhyamam*
Ahara	*Jaranasakthi- Madhyama* *Abhyaavaaharana sakthi- Madhyama*

Samprapti Ghataka	
Dosha	*Tridosha*
Dhatu	*Rasa, Mamsa, Majja*
Upadhatu	*Snayu*
Agni	*Vishamagni*
Srotas	*Rasa, Mamsa, Majja*
Srotodushti	*Sanga*
Rogamargam	*Madhyama*

Udbhavasthanam	*Siras*
Vyakthasthanam	*Sarvadeha*

Treatment Protocol

Internal medicines	External therapy
1. *Punarnavasavam* 20 ml Bd after food 2*. Panchakola choornam* 5g bd with rice gruel before food 3. *Rasnadasamoola ghritam* 5g in morning on empty stomach. 4. *Maharasnadi kashayam* 90 ml bd before food	1. *Snehapana* with *Indukantham ghritam* for 6 days 2. *Abhyanga* and *Ushma sweda* with *Sahacharadi Tailam* 3 days 3.*Virechana* with *Gandharvaeranda tailam* 20 ml with milk. 4. *Pratimarsha nasya* with *Ksheerabala 21 Avartti* 4 drops at 4 pm 5. *Abhyanga* and *Ushma sweda* with *Sahacharadi tailam* 7 days 6. *Talapothichil* with *Musta,Vacha, Amala*ki in *Takra* 3 days 7. *Siropichu* with *Vatasini tailam* 3 days 8*. Pizhichil* with *Balatailam* and *Satahwadi tailam* 3 days 9. *Mustadi Rajayapana vasthi* 3 days

Advice on Discharge

1. *Mashatmaguptadi kashayam* 90ml at 11am b/f for 2 weeks
2. *Vidaryadi Kashayam* 90 ml at 11 am b/f from 3rd week
3. *Badradarvadi kashayam* 90 ml at 11 am b/f from 5th week
4. *Rasnadasamoola ghritam* 5ml early morning on empty stomach
5. *Pratimarsha nasya with dhanvantaram* 7 A 4 drops at 4 pm

RESULTS

At baseline, the patient had progressive gait imbalance, recurrent falls, dysfluent speech and impaired coordination. MRI brain showed diffuse bilateral cerebellar volume loss disproportionate to age, with prominence of the cerebellar folia and ex-vacuo dilatation of the fourth ventricle. The cerebrum was unremarkable. Anti-TPO and anti-thyroglobulin antibodies were markedly elevated, and no pathogenic repeat expansion was detected in the evaluated spinocerebellar ataxia panel.

Following the multimodal Ayurvedic treatment protocol, the total Scale for the Assessment and Rating of Ataxia (SARA) score decreased from 13 at baseline to 6 at discharge. Improvements were recorded in gait, stance, finger chase, nose–finger testing, rapid alternating hand movements and heel–shin slide. These findings document an improvement in measured ataxia severity during the treatment period.

SARA Scale for Ataxia

	24/06/26	24/08/2026
Gait	3	2
Stance	1	0
Sitting	0	0
Speech disturbance	1	1
Finger chase test	2	1
Nose finger test	2	1
Fast alternating hand movements	1	0
Heel shin slide	2	1

DISCUSSION

The present case describes a young male with progressive cerebellar dysfunction manifested by gait imbalance, recurrent falls, dysfluent speech and impaired coordination, in association with autoimmune hypothyroidism. MRI brain demonstrated diffuse bilateral cerebellar volume loss disproportionate to age, with prominence of the cerebellar folia and ex-vacuo dilatation of the fourth ventricle, while the cerebrum remained unremarkable. The absence of pathogenic repeat expansions in the evaluated Spinocerebellar Ataxia panel made the tested common hereditary SCA subtypes less likely.

The patient had markedly elevated anti-TPO and anti-thyroglobulin antibodies, supporting an autoimmune thyroid disease background. Hashimoto's thyroiditis is a chronic autoimmune disorder in which both cellular and humoral immune mechanisms contribute to thyroid injury. Neurological manifestations associated with autoimmune thyroid disease have been described, and autoimmune mechanisms have been proposed in cerebellar dysfunction. These include antibody-mediated neuronal injury, immune-mediated inflammation and possible involvement of cerebellar Purkinje cells. However, in the present case, the association between Hashimoto's thyroiditis and cerebellar atrophy should be considered a possible relationship rather than a proven causal mechanism.

From an Ayurvedic perspective, the clinical presentation can be interpreted predominantly through *Agnimandya, Ama, Kapha pitha dusti,*

Margavarana of *Vatha* and *Dhatu-kshaya*. The proposed *Samprapti* can be represented as:

Due to *Agni mandhya* at *Kostha* level *Ama* originates and localized at *Kapha pitha stana* (thyroid gland) and results in *Dhathu paka* and *Dhathwagnimandhya* (T3 and T4) further leads to *Dhathu nasha* at the level of *Mastulunga* (cerebellum) due to *Beejabhaga avayava dusti*. Resultant *Khavaigunya* at *Mastulunga* leads to *Margavarana* of *Vyanavatha* and produces the symptoms like *Gathi vaishamya, Vak graha.*

Agnimandya can be considered the initial pathological factor leading to impaired digestion and tissue metabolism, followed by accumulation of *Ama* and subsequent *Srotodushti*. *Sthana-samsraya* at *Shiras* can be correlated with the predominant neurological manifestations. *Avarana* of *Vyana Vata* is considered relevant due to the disturbances of gait, coordination, and speech, while *Dhatu-kshaya* can be correlated with the chronicity and structural changes demonstrated on neuroimaging.

The treatment protocol was therefore planned to address *Agnimandya*, *Ama*, *Avarana*, and *Vata* predominance through a combination of internal medicines, *Shodhana*, and external therapies. *Panchakola Churna*, *Maharasnadi Kashaya*, and *Rasnadashamoola Ghrita* were used with the objective of improving *Agni*, reducing *Ama*, and supporting *Vata* management.

Indukantham Ghrita was administered as part of the therapeutic protocol, considering its role within the Ayurvedic management of disorders associated with impaired *Agni* and systemic involvement. In the context of the present case, it was considered as supportive therapy for the underlying pathological process associated with autoimmune hypothyroidism. This interpretation represents an Ayurvedic therapeutic rationale and should not be taken as evidence of a direct immunomodulatory effect on Hashimoto's thyroiditis.

The external therapies were selected according to the proposed site and mechanism of disease. *Abhyanga* and *Ushma Sweda* with *Sahacharadi Taila* were administered for *Vata-shamana* and functional support. *Shirolepa (Talapothichil)* using *Musta*, *Vacha*, and *Amalaki* in *Takra* was specifically considered in view of the proposed *Sthana-samsraya* at *Shiras*. *Shiro Pichu* and *Pratimarsha Nasya* were also incorporated as *Shiro*-oriented therapies, with the intention of addressing *Vata* involvement and *Dhatu-kshaya* at the site of manifestation.

Mustadi Rajayapana Basti was selected in the present case with the therapeutic aim of providing *Brimhana* and *Rasayana* effects, along with the classical therapeutic benefits of *Vasti* in *Vata* disorders. *Yapana Vasti* is traditionally described as nourishing, strengthening, and restorative, making it particularly relevant in conditions characterised by *Dhatu-kshaya* and *Vata* aggravation. In the management of *Dhatu-asrita Vyadhi*, the therapeutic approach includes *Snehapana*, *Shodhana*, and *Rasayana*, with particular emphasis on *Brimhana* and *Rasayana Chikitsa* when tissue depletion and weakness are predominant. Accordingly, *Mustadi Rajayapana Basti* was incorporated into the treatment protocol to address the presumed *Vata* predominance and *Dhatu-kshaya* while providing nourishment and restorative support.

The therapeutic response was assessed using the Scale for the Assessment and Rating of Ataxia (SARA). The total SARA score decreased from 13 at baseline to 6 at discharge, indicating an improvement in the overall severity of ataxia. Improvements were observed across several components, including gait, stance, finger chase, nose-finger testing, rapid alternating hand movements and heel-shin test. The reduction in SARA score suggests improvement in cerebellar motor performance following the treatment protocol. However, as this is a single case, the observed improvement cannot be attributed definitively to the intervention, and longer-term follow-up with repeated standardized assessments would be required to establish sustained benefit.

CONCLUSION

The case is noteworthy because of the early-onset progressive cerebellar manifestations, marked cerebellar atrophy on neuroimaging, autoimmune thyroid disease with markedly elevated thyroid autoantibodies, and negative testing for the evaluated common spinocerebellar repeat-expansion disorders. The improvement in SARA score following the multimodal Ayurvedic intervention provides a clinically relevant observation and supports the need for further systematic investigation of Ayurvedic approaches in neurological manifestations associated with autoimmune thyroid disorders.

Nevertheless, the limitations of a single-case report, including the absence of a control group and the possibility of natural fluctuation or other contributing factors, should be considered. Further longitudinal clinical, neurological, radiological and immunological evaluation would be necessary to clarify the relationship between autoimmune thyroid disease and cerebellar dysfunction and to determine the reproducibility and sustained effectiveness of the therapeutic approach.

Primary Takeaway Lessons from this Case Report

This case highlights the potential clinical value of an individualized, Ayurveda-based approach in the management of progressive cerebellar ataxia associated with autoimmune thyroid disease. The treatment incorporated internal medicines along with *Snehapana, Virechana, Abhyanga, Swedana, Nasya,*

Shiro Pichu, Talapothichil, Pizhichil and *Mustadi Rajayapana Basti*, based on the assessment of *Vata*, *Agnimandya, Ama, Avarana* and *Dhatu-kshaya*. Following treatment, improvement was observed in gait, stance, limb coordination and overall motor performance, with the SARA score improving from 13 at baseline to 6 at discharge. This case suggests that a personalized, multimodal Ayurvedic approach may have a role in the supportive management of chronic cerebellar ataxia. However, as this is a single case, the observed improvement cannot establish a causal relationship between the treatment and the outcome.

Patient Perspective

The patient and his family observed improvement in balance, walking ability, limb coordination and overall functional performance following Ayurvedic treatment. He experienced better stability while walking and improved ability to perform daily activities. The patient considered the improvement meaningful, particularly in relation to his long-standing difficulties with balance and coordination. He expressed satisfaction with the progress achieved during the treatment period and willingness to continue follow-up and further management.

Informed Consent

The patient provided informed consent for publication of the clinical details, investigations, treatment and clinical outcomes presented in this case report.

REFERENCES

1. Hauser SL, Josephson SA. Harrison`s Neurology in Clinical Medicine. 3rd edition.
2. Ercoli T, Defazio G, Muroni A. Cerebellar syndrome associated with thyroid disorders. Cerebellum. 2019; 18(5): 932-940. doi:10.1007/s12311-019-01059-9.
3. Selim M, Drachman DA. Ataxia associated with Hashimoto's disease: progressive non-familial adult-onset cerebellar degeneration with autoimmune thyroiditis. J Neurol Neurosurg Psychiatry. 2001; 71(1): 81-87. doi:10.1136/jnnp.71.1.81.
4. Sharma P. Sushruta Samhita. Volume 2. Nidana Sthana, Chapter 1 verse 30, Year 2010. Chaukhambha Visvabharati. P 8

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