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AYURVEDIC MANAGEMENT OF AUTOIMMUNE HYPOTHYROIDISM - A CASE REPORT

[1]Vd. Swetlana Upreti [2]Vd. Archana Zala [3]Vd. Rohini Salve
[1,2]Final Year PG Scholar, [3]Head of Department and Assistant Professor, Department of Panchakarma, Government Akhandanand Ayurveda College, Ahmedabad, Gujarat, India

ABSTRACT

Hypothyroidism is a common endocrine disorder in which autoimmune (Hashimoto's) thyroiditis is a leading cause. It has no direct nomenclature in classical Ayurvedic texts but can be correlated with Dhatvagnimandya (impaired tissue metabolism) involving Rasavaha and Medovaha Srotas (channels of nutrient fluid and fat tissue). This case report describes a 37-year-old married female homemaker with progressive weight gain (Sharira Bhara Vriddhi) for 2 years, bilateral knee and low back pain (Sandhi-Kati Shoola) and giddiness (Bhramanubhuti) for 6 months, and irregular menstruation for 1 year. Investigations revealed severe primary hypothyroidism (thyroid-stimulating hormone above 150 µIU/mL, thyroxine 0.1 µg/dL), anti-thyroid peroxidase antibodies (963 U/mL), sonographic thyroiditis and dyslipidemia. She had never received thyroid hormone replacement. She was managed with Virechana Karma (therapeutic purgation) with Abhayadi Modaka and Samsarjana Krama (graded diet), followed by Pippali Ksheera Paka and an Agni-restorative Shamana (palliative) regimen. At nine-month follow-up, thyroid-stimulating hormone had normalized to 4.740 µIU/mL and thyroxine to 5.4 µg/dL, cholesterol and triglycerides had improved, and body weight had reduced from 83.7 kg to 79.8 kg, with complete resolution of joint pain and menstrual irregularity. This case highlights the potential role of an Agni Mandya-directed Shodhana-Shamana approach in autoimmune hypothyroidism.

KEYWORDS: Hypothyroidism, Agni Mandya, Virechana Karma, Autoimmune thyroiditis

INTRODUCTION

Hypothyroidism is characterized by insufficient production of thyroid hormones, predominantly thyroxine (T4) and triiodothyronine (T3), which are crucial for regulating the body's metabolism, energy expenditure and cardiovascular function.[1] It is a common endocrine disorder with various causes, but autoimmune disease (Hashimoto's thyroiditis) and thyroid failure account for over 90% of cases, except where iodine deficiency is endemic.[2] Autoimmune (Hashimoto's) thyroiditis is increasingly recognized as a leading cause.[3]

It is classified into two types. Primary hypothyroidism results from intrinsic functional or structural destruction of the thyroid parenchyma and accounts for over 95% of all clinical cases.[4] Central (secondary or tertiary) hypothyroidism arises from inadequate thyrotropin (thyroid-stimulating hormone, TSH) stimulation by the anterior

pituitary gland or deficient thyrotropin-releasing hormone (TRH) secretion from the hypothalamus.[4,5]

Hashimoto's thyroiditis is an autoimmune disease in which antithyroid antibody formation and T-cell activation target thyroid tissue, leading to progressive fibrosis.[6] Both cellular (T cell-mediated) and humoral (B cell-mediated) immune responses play essential roles in its pathogenesis.[7] Individuals with Hashimoto's thyroiditis have polyclonal antibodies specific to thyroid antigens, namely thyroid peroxidase antibody (90% of individuals) and thyroglobulin antibody (50–80% of individuals).[8] CD8+ (cluster of differentiation 8) T cells are a central part of the immune dysfunction underlying its pathogenesis; these cytotoxic cells infiltrate thyroid tissue, causing inflammation and destruction of follicular cells.[9] CD4+ T cells activate other immune cells, particularly macrophages and B cells, thereby promoting the production of autoantibodies.[7] The condition was first described by Hakaru Hashimoto in 1912 as struma lymphomatosa.[10]

Individuals with Hashimoto's thyroiditis have varying presentations, including euthyroid state, subclinical hypothyroidism, overt hypothyroidism and thyroiditis. In the euthyroid state, thyroid hormone levels are normal. In subclinical hypothyroidism, TSH is elevated, usually below 10 mIU/L, with normal T4 and T3 levels. Individuals with overt hypothyroidism present with symptoms of hypothyroidism, TSH above 10 mIU/L, low T4 and, rarely, low T3 levels. Usual symptoms include fatigue, unintentional weight gain, cold intolerance, constipation, changes in menstrual cycles, hair loss, brittle nails, depression, confusion and brain fog. Individuals may notice periorbital puffiness, ankle swelling, voice changes, muscle aches or weakness, neuropathy and joint pain.[11,12,13] Conventional management rests on lifelong levothyroxine replacement.

An eight-city Indian epidemiological study reported an overall hypothyroidism prevalence of 10.95%, significantly higher in women than in men,[14] while a global systematic review and meta-analysis have documented the rising epidemiological burden of Hashimoto's thyroiditis in adults.[15]

In *Ayurveda*, hypothyroidism lacks a direct classical counterpart but is functionally understood as a multidimensional state involving *Agnimandya* (deranged digestive and cellular fire), *Ama* (metabolic endotoxins) and *Kapha-Vata* predominance, culminating in *Rasa-Medovaha Srotorodha* (microvascular and tissue obstruction). The clinical presentation in this case can be interpreted primarily through *Kapha* and *Vata* involvement, with associated disturbance of *Agni* (digestive and metabolic fire) and tissue metabolism. The progressive weight gain and generalized heaviness indicate *Kapha* predominance and *Medo Dushti* (vitiation of fat tissue), while bilateral knee pain, low back pain, giddiness, abdominal distension, and irregular menstruation suggest associated *Vata*, particularly disturbance of *Apana Vata* (the downward-moving subtype of *Vata*). *Agnimandya* leads to impaired *Dhatvagni* (tissue-level metabolism) and subsequently disturbs the nourishment and metabolism of

Rasa and *Meda Dhatu* (nutrient fluid and fat tissue). The thyroiditis in the present case represents the inflammatory pathology identified by contemporary investigations, and *Pitta* may be considered in relation to these inflammatory processes.

This case report documents *Ayurvedic* management, based on an *Agni Mandya*-centered understanding, of a patient with biochemically and serologically confirmed autoimmune primary hypothyroidism and coexisting dyslipidemia, who received no allopathic treatment at any stage, evaluating outcomes through serial thyroid and lipid biochemistry.

MATERIALS AND METHODS

Case presentation

A 37-year-old married female homemaker, resident of Ahmedabad, presented to the *Panchakarma* outpatient department (OPD) of Government Akhandanand Ayurveda College, Ahmedabad.

Chief complaints (*Pradhana Vedana*)

TABLE NO. 1: CHIEF COMPLAINTS

Sr. no.	Complaint	Duration
1	*Sharira Bhara Vriddhi* (progressive weight gain)	2 years
2	*Ubhaya Janu Sandhi Shoola* (bilateral knee joint pain)	6 months
3	*Katishoola* (low back pain)	6 months
4	*Bhramanubhuti* (giddiness)	6 months
5	*Aniyamita Artava Pravritti* (irregular menstruation)	1 year
6	*Udara Gauravata* with *Adhmana* (abdominal heaviness and distension)	1 year
7	*Sharira Gauravata* (generalized body heaviness)	1 year

History of present illness

The patient was apparently well two years ago. She gradually noticed progressive weight gain, later associated with joint pain, low back pain and giddiness. She had no prior awareness of any thyroid abnormality. On biochemical work-up for the weight gain, severe primary hypothyroidism was detected (TSH >150 µIU/mL, T4 0.1 µg/dL), subsequently confirmed as autoimmune in origin (anti-thyroid peroxidase [anti-TPO] antibody 963 U/mL), with sonographic evidence of thyroiditis.

Past history

No known history of hypertension, diabetes mellitus, cardiac disease or similar prior episodes. No history of trauma or major surgery other than tubal ligation 6–7 years earlier. The patient confirmed that she had never taken any allopathic medication, including thyroid hormone replacement or lipid-lowering agents, at any point before or during *Ayurvedic* management.

Obstetric history

G3P3L2 (gravida 3, para 3, living 2): the first two pregnancies were full-term normal deliveries (a living male aged 16 years and a living female aged 13 years); the third (female) child died within two years of birth.

Personal history

Diet: vegetarian; appetite (*Kshudha*) and sleep (*Nidra*): normal (*Samyak*); daytime sleep (*Diva Swapna*): 1–2 hours; physical activity: nil (sedentary lifestyle); tea intake: 5–7 times/day; no known drug allergy.

TABLE NO. 2: General examination

Parameter	Finding
Blood pressure	130/80 mmHg
Temperature	98 °F
Respiratory rate	16/min
Height	180 cm
Weight / BMI	83.7 kg / 25.8 kg/m²
Pallor	Present
Icterus	Absent
Oedema	Non-pitting oedema present
Xanthoma	Absent

BMI: body mass index.

***Ashtavidha Pariksha* (eight-fold examination)**

TABLE NO. 3: *ASHTAVIDHA PARIKSHA*

Pariksha	**Finding**
Nadi (pulse)	70/min; *Kapha-Pitta* predominant
Mutra (urine)	4–5 times/day, 0–1 time/night
Mala (stool)	Once daily, with straining (*Savibandha*)
Jihwa (tongue)	Coated (*Sama*)
Shabda (voice)	*Spashta* (clear)
Sparsha (touch/skin)	*Anushnasheeta* (neither hot nor cold)
Drik (eyes)	*Prakrita* (normal)
Akriti (appearance)	*Sthula* (obese)

Investigations

TABLE NO. 4: Before Treatment

Investigations

Investigation (date)	Finding	Significance
Haemoglobin (16/06/2025)	11 g%	Mild anaemia
Lipid profile (23/06/2025)	Cholesterol 355.43, TG 227.59, HDL 42.48 mg/dL (ref: cholesterol 140–250, TG 35–140, HDL 30–60)	Dyslipidemia
TSH / T4 (01/07/2025)	TSH >150 µIU/mL, T4 0.1 µg/dL	Severe primary hypothyroidism
Anti-TPO / Anti-TG (07/07/2025)	963.0 U/mL / 1.9 U/mL	Autoimmune (Hashimoto's) thyroiditis
USG neck (10/07/2025)	Normal size, altered echotexture, raised vascularity	Thyroiditis
Apolipoprotein B	161.00 mg/Dl	Within reference range

(12/07/2025)		(53–182)

TG: triglycerides; HDL: high-density lipoprotein; TSH: thyroid-stimulating hormone; T4: thyroxine; Anti-TPO: anti-thyroid peroxidase antibody; Anti-TG: anti-thyroglobulin antibody; USG: ultrasonography.

***Ayurvedic* diagnosis (*Nidana Panchaka*)**

TABLE NO. 5: *NIDANA PANCHAKA*

Component	Details
Nidana (causative factors)	*Guru-Snigdha Ahara* (heavy, unctuous food), *Paryushita Ahara* (stale food), *Adhyashana* (eating before digestion of the previous meal), *Divaswapna* (daytime sleep), *Avyayama* (lack of exercise), *Aniyamita Ahara* (irregular meals), excessive tea intake
Purvarupa (prodromal symptoms)	*Sharira Gauravata* (body heaviness), *Savibandha Mala Pravritti* (constipation), *Klama* (fatigue)
Rupa (clinical features)	*Sharira Bhara Vriddhi*, *Sandhi-Kati Shoola*, *Bhramanubhuti*, *Aniyamita Artava*
Upashaya / Anupashaya (relieving / aggravating factors)	*Upashaya* after *Pathya Palana* (adherence to wholesome diet and regimen)

***Samprapti* (pathogenesis)**

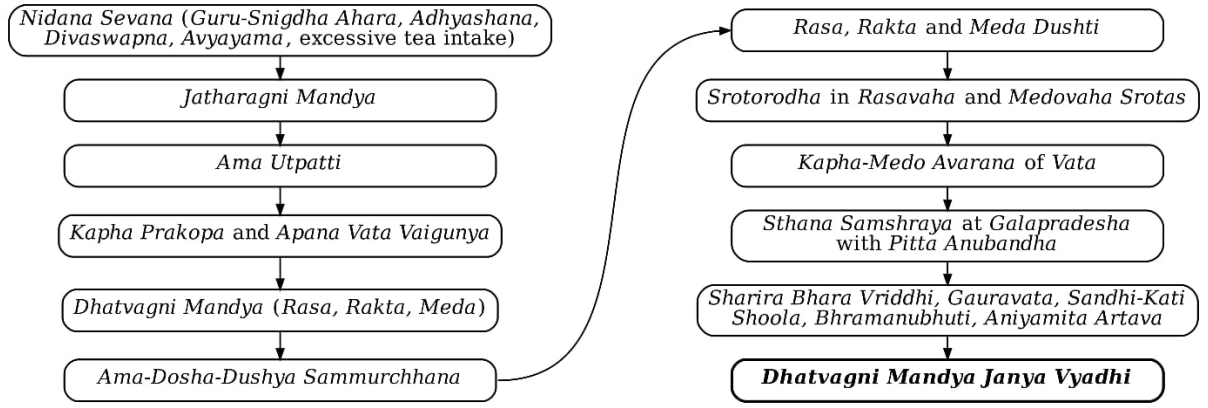


Figure 1: *Samprapti* (pathogenesis) of the present case

***Samprapti Ghataka* (components of pathogenesis)**

TABLE NO. 6: *SAMPRAPTI GHATAKA*

Sr. no.	Component	Details
1	*Dosha*	*Kapha-Vata Pradhana* with *Pittanubandha*
2	*Dushya*	*Rasa*, *Rakta*, *Meda*
3	*Srotas*	*Rasavaha*, *Raktavaha*, *Medovaha*
4	*Srotodushti*	*Sanga*, *Vimarga Gamana*
5	*Agni*	*Jatharagni*, *Dhatvagni*
6	*Adhishthana*	*Galapradesha* (thyroid gland)
7	*Udbhavasthana*	*Amashaya*
8	*Rogamarga*	*Madhyama*
9	*Vyaktisthana*	*Sarvanga Sharira* (whole body)

10	*Vyadhi Swabhava*	*Chirakari* (chronic)

Treatment plan

The treatment was planned in two phases: (I) *Shodhana Chikitsa* (purification therapy) and (II) *Shamana Chikitsa* (palliative therapy).

Phase I: *Shodhana Chikitsa* (purification therapy)

(a) *Deepana-Pachana* (appetiser and digestive therapy)

TABLE NO. 7: *DEEPANA-PACHANA*

Drug	**Dose and mode (11–19 Aug 2025)**
Trikatu Churna	3 g twice daily before food with *Ghrita* (ghee)
Haritaki Churna	5 g at night with warm water

(b) *Vardhamana Snehapana* (ascending-dose internal oleation) with *Tila Taila*

TABLE NO. 8: *SNEHAPANA*

Day	**Date**	**Dose (mL)**	**Time of intake**	***Kshudha Pravritti* (onset of hunger)**
1	20/08/2025	30	7:30 am	12:00 pm
2	21/08/2025	50	7:00 am	1:15 pm
3	22/08/2025	75	7:00 am	3:25 pm
4	23/08/2025	95	6:30 am	2:00 pm
5	24/08/2025	120	7:00 am	3:44 pm
6	25/08/2025	150	6:45 am	6:30 pm
7	26/08/2025	170	7:00 am	8:00 pm

Samyak Snigdha Lakshana (signs of adequate oleation) such as *Vatanulomana* (normal downward movement of *Vata*), *Diptagni* (enhanced appetite), *Snigdha* and *Asamhata Varcha* (unctuous, unformed stools) progressed through the seven days and were fully observed on day 7.

(c) *Sarvanga Abhyanga-Swedana* (whole-body massage and sudation)

Whole-body oleation massage and sudation were given for 3 days (27–29 August 2025) as pre-purificatory measures.

(d) *Virechana Karma* (therapeutic purgation)

TABLE NO. 9: *VIRECHANA KARMA*

Drug	**Dose and mode (29 August 2025)**	***Anupana***	**Time**	***Vega / Upavega***
Abhayadi Modaka	10 tablets orally (1 tablet = 250 mg)	*Sheeta Jala* (cold water)	10:30 am	*Vega* 8 / *Upavega* 13

Vega: number of purgative bowel movements; *Upavega*: number of minor (subsidiary) bowel movements.

Antiki (end-point of purgation): *Kaphaja*; *Shuddhi Lakshana* (signs of purification): *Laghavata* (lightness), *Agni Vriddhi* (improved appetite) and mild *Daurbalya* (weakness); grade of *Shuddhi*: *Alpa Shuddhi* (mild purification). Baseline blood pressure was 130/80 mmHg and pulse 84/min; vitals remained stable throughout.

(e) *Samsarjana Krama* (graded diet)

Based on the *Alpa Shuddhi* assessment, a 3-day graded *Samsarjana Krama* diet was given.

Phase II: *Shamana Chikitsa* (palliative therapy)

TABLE NO. 10: *SHAMANA CHIKITSA*

Sr. no.	Duration	Drug	Dose	Frequency	Timing	*Anupana*
1	4/09/25–4/11/25 5/11/25–30/01/26	*Guduchi* + *Dhatrinisha* + *Punarnava Churna* (0.8 g each) + *Trikatu Churna* (0.5 g)	3 g	TDS	Before food	*Ushna Jala*
2	4/09/25–30/09/25 16/10/25–10/11/25	*Arogyavardhini Vati*[16]	125 mg	2 tablets TDS	After food	*Ushna Jala*
3	4/09/25–10/11/25 1/01/26–25/02/26	*Kanchnara Guggulu*[17]	375 mg	2 tablets TDS	After food	*Ushna Jala*
4	4/09/25–30/10/25 15/11/25–18/12/25 1/01/26–25/02/26	*Guduchyadi* [18]+ *Pathyadi Kwatha*[19]	15 mL	OD	Empty stomach, evening	–
5	13/09/2025–20/11/2025	*Pippali*[20] *Ksheera Paka* (4 *Pippali*)	40 mL	OD	Morning	–

TDS: three times a day; OD: once a day; *Ushna Jala*: warm water.

Management of hair loss and brittle nails: Approximately 2 months after treatment initiation, the patient reported excessive hair loss (*Khalitya*) and brittle, breaking nails (*Nakha Bhanjana*). These were considered manifestations of persisting hypothyroidism (TSH 8.525 µIU/mL at that time) rather than an adverse drug reaction. The prescription was revised and a new combination of *Guduchi* 125 mg + *Amalaki* 125 mg + *Mustaka* 125 mg (three times a day) was added. Complete resolution of hair loss and nail fragility (100% recovery per patient report) was achieved within one month.

RESULT

Thyroid and lipid parameters

TABLE NO. 11: OUTCOME ASSESSMENT – THYROID AND LIPID PARAMETERS

Parameter [reference range]	Baseline (01/07/2025)	2-month (04/09/2025)	4-month (15/11/2025)	9-month / final (11/03/2026)
TSH (µIU/mL) [0.35–5.50]	>150 (H)	8.525 (H)	4.889 (N)	4.740 (N)
T3 (ng/mL) [0.6–1.81]	–	–	0.93 (N)	0.93 (N)
FT3 (pg/mL) [2.3–4.2]	–	3.42 (N)	–	–

T4 (µg/dL) [4.5–12.6]	0.1 (L)	–	6.3 (N)	5.4 (N)
FT4 (ng/dL) [0.89–1.76]	–	0.83 (L)	–	–
Anti-TPO (U/mL) [<60]	963.0 (07 Jul) (H)	>1000 (H)	–	–
Cholesterol (mg/dL) [140–250]	355.43 (23 Jun)	243.07	–	247
Triglycerides (mg/dL) [35–140]	227.59 (23 Jun)	114.03	–	118
HDL (mg/dL) [30–60]	42.48 (23 Jun)	66.85	–	46.0
Haemoglobin (g/dL)	11	11.9	–	11.5
Body weight (kg)	83.7	76.6	78	79.8

TSH: thyroid-stimulating hormone; T3: triiodothyronine; FT3: free triiodothyronine; T4: thyroxine; FT4: free thyroxine; Anti-TPO: anti-thyroid peroxidase antibody; HDL: high-density lipoprotein; N: normal; H: high (above reference range); L: low (below reference range); –: not tested at this visit.

TSH normalized from >150 to 4.740 µIU/mL over approximately 9 months (baseline to final follow-up), a shift from severe overt hypothyroidism to euthyroid status. TSH and T4 had already reached the normal range by the 4-month (November 2025) assessment (TSH 4.889 µIU/mL, T4 6.3 µg/dL) and remained there through the 9-month follow-up without any allopathic thyroid hormone replacement (Figures 2–4).

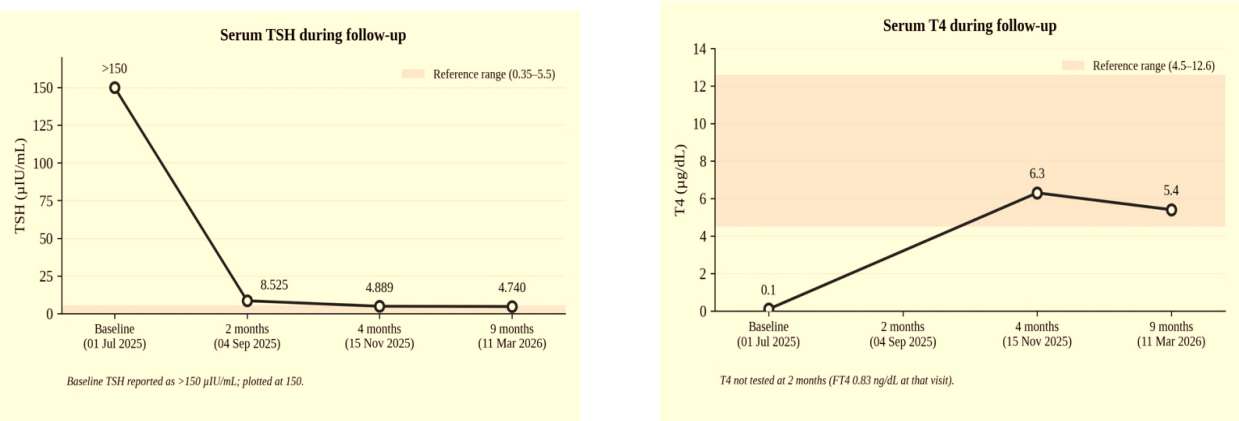


Figure 2: Serum TSH (left) and serum T4 (right) during follow-up

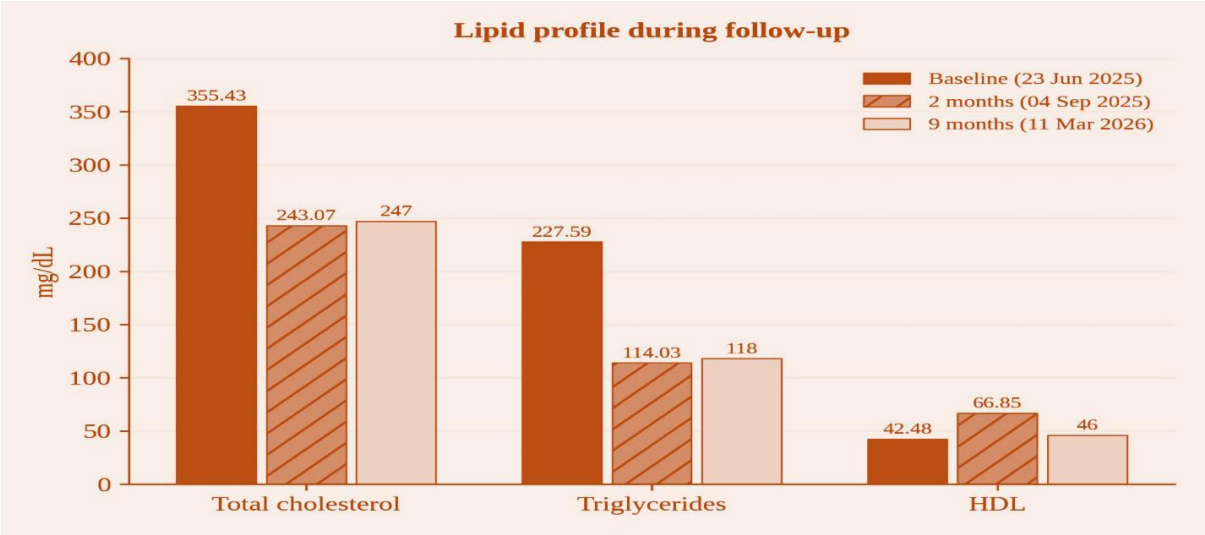


Figure 3: Lipid profile during follow-up

Subjective parameters

By March 2026, knee pain, low back pain, giddiness, menstrual irregularity, distension and body heaviness had resolved completely, and abdominal heaviness was mildly persistent.

DISCUSSION

***Deepana-Pachana* (*Trikatu Churna* and *Haritaki Churna*)**

Trikatu Churna (*Shunthi, Maricha, Pippali*) was selected for its *Deepana-Pachana* and *Kapha-Vatahara* action, directly targeting the *Jatharagni Mandya* (impaired digestive fire) identified as the root of this patient's *Samprapti* and correcting the underlying *Ama* before oleation therapy. Administering *Sneha* to a patient with active *Ama* risks further *Srotorodha* rather than absorption. Its *Katu Rasa*, *Katu Vipaka* and *Ushna Virya* (pungent taste, pungent post-digestive effect and hot potency) directly counteract the *Guru*, *Snigdha* and *Picchila* (heavy, unctuous and slimy) qualities of vitiated *Ama*[21] and *Kapha*. *Trikatu* is *Deepana* and *Kaphamedoghna*,[22] acting to kindle *Jatharagni* and prevent further *Ama-Meda* accumulation. Network pharmacology and *in vivo* work have shown *Trikatu* to support hepatic and lipid-metabolic function, consistent with this *Agni*-correcting role.[23]

Haritaki Churna was added at night for its *Anulomana* (carminative, downward moving) action, addressing the patient's chronic tendency toward *Savibandha Mala Pravritti* (constipation with straining) and easing the downward movement of *Apana Vata*. Experimental data support a prokinetic effect of *Terminalia chebula* on gastric emptying, lending pharmacological plausibility to its classical *Anulomana* indication.[24]

Vardhamana Snehapana* with *Tila Taila

Tila Taila (sesame oil) was chosen for *Snehapana* because it has *Ushna*, *Tikshna* and *Lekhana* (hot, penetrating and scraping) properties, as described by *Acharya Sushruta*,[25] making it inherently opposed to the *Kapha*-predominant, *Guru-Snigdha* qualities of vitiated *Meda*. The *Vardhamana* (ascending-dose) schedule over seven days

allowed gradual saturation of the *Dhatus* while monitoring for *Samyak Snigdha Lakshana*, which appeared appropriately at each stage and was fully established by day 7, confirming adequate *Dosha* mobilization in preparation for *Virechana*. A recent controlled study in experimentally induced hypothyroidism found that sesame oil supplementation improved the lipid profile and hepatic and cardiac markers to a degree comparable with, and in some parameters exceeding, L-thyroxine treatment.[26]

Sarvanga Abhyanga-Swedana

Three days of whole-body oleation massage and sudation were interposed between *Snehapana* and *Virechana* as classical *Purvakarma* (preparatory procedure), intended to further loosen *Dosha* adherent to peripheral *Srotas* and bring it toward the *Koshtha* (gut), improving the efficiency and completeness of the subsequent purgation.

Virechana Karma* with *Abhayadi Modaka

To address systemic hypometabolism and *Kapha-Medo Dushti*, *Virechana* was done using *Abhayadi Modaka* to eliminate the *Doshas*. The formulation's effectiveness stems from its dual action: potent purgative herbs such as *Trivrit*, *Danti* and *Jaipala* induce *Rechana* and *Bhedana* (purgation and breaking of hard stools) owing to their *Ushna* and *Tikshna* qualities, removing *Ama* and reducing vascular and tissue resistance, while *Haritaki*, *Amalaki*, *Trikatu* and *Vidanga* promote gentle *Anulomana*, calming bowel movement and reducing mucosal irritation. The resulting *Kaphaja Antiki Shuddhi* and the appearance of *Laghavata* indicated successful channel cleansing (*Srotoshodhana*), thus revitalizing *Agni*.

Guduchi + Dhatrinisha + Punarnava + Trikatu

Tinospora cordifolia (*Guduchi*) serves a dual metabolic and immunomodulatory role, targeting both systemic metabolic hypofunction and the underlying autoimmune pathology. *Bhavaprakasha Nighantu* describes *Guduchi* as *Tridoshahara*, with *Tikta*, *Kashaya* and *Katu Rasa*, *Ushna Virya* and *Madhura Vipaka*.[27] Its *Agni Deepana* and *Balya* (strength-promoting) actions counteract the core pathogenesis of *Jatharagni* and *Dhatvagni Mandya* without aggravating *Pitta*. *T. cordifolia* has significant immunomodulatory, free-radical scavenging and anti-inflammatory properties.[28]

Dhatrinisha is a combination of *Amalaki* (*Dhatri*) and *Haridra* (*Nisha*). *Amalaki* has a *Rasayana* (rejuvenative) property that supports *Rasa Dhatu* nourishment after *Shodhana*, and *Haridra* has a *Lekhana* property that scrapes excess *Meda* and clears channel obstruction (*Srotorodha*). *Punarnava* was selected for its *Shothahara* (anti-oedematous) and *Kaphahara* action to address residual *Kapha* accumulation.

Arogyavardhini Vati

It contains mineral *Bhasmas* (*Lauha*, *Abhraka*, *Tamra*), *Triphala*, *Shilajatu*, *Guggulu*, *Chitraka* and *Katuki* (*Picrorhiza kurroa*), levigated with *Nimba Patra Swarasa* (fresh juice of neem leaves). It is *Medovinashini*, *Pachani* and *Deepani* (fat-reducing, digestive and appetiser). *Tamra Bhasma* and *Shuddha Guggulu* exert potent *Lekhana* properties, clearing excess *Meda*.

Picrorhiza kurroa is a premier *Yakrit-Uttejaka* (hepatostimulant) and *Bhedana* agent; its iridoid glycosides (picrosides) have been documented to provide membrane-stabilizing and hepatoprotective actions, optimizing hepatic function relevant to systemic thyroid hormone homeostasis.[29]

Kanchnara Guggulu

It is classically indicated in *Granthi* and *Gandamala* (glandular swellings, including goitre), making it directly relevant to thyroid pathology. *Kanchnara* is *Grahi* (absorbent), of *Kashaya Rasa*, *Shleshmapittanut* (pacifies *Kapha* and *Pitta*) and *Gandamalapaha* (removes glandular swellings).[30] The *Kashaya* and *Grahi* properties of *Kanchnara* act by reducing oedema and scraping away *Medo-Kaphaja* channel occlusion (*Srotorodha*). Ethanolic extracts of *Bauhinia* have been shown to stimulate thyroid follicular activity, significantly increasing circulating T3 and T4 levels while protecting the glandular parenchyma from lipid peroxidation.[31]

Guduchyadi Kwatha + Pathyadi Kwatha

Guduchyadi Kwatha contains *Guduchi*, *Dhanyaka*, *Nimba*, *Padmaka* and *Raktachandana*. It modulates *Pitta-Kapha* morbidity and clears systemic *Ama* (endotoxins) without escalating metabolic heat. *Pathyadi Kwatha* contains *Triphala*, *Kiratatikta* and *Nimba*; it provides systemic *Anulomana* (downward regulation of *Vata*, relieving stubborn constipation) and *Lekhana* action.

Pippali Ksheera Paka

Pippali (*Piper longum*) boiled in milk was introduced as a gentle, sustained *Agni-Deepana* and *Rasayana* measure, providing ongoing correction of *Jatharagni Mandya*. *Ksheera* (milk) as the medium was specifically chosen to offset the *Ushna-Tikshna* qualities of *Pippali*, allowing sustained *Agni* stimulation without provoking *Pitta* or causing *Dhatu Kshaya* (tissue depletion).

***Pathya-Apathya*:** The patient was advised a *Laghu* (light), *Kapha-Medohara* diet: *Mudga* (green gram), *Masura* (red lentil), *Purana Shali* (rice aged one year), *Yava* (barley) , *Jowar* (sorghum) and *Khichdi*; vegetables such as *Alabu* (bottle gourd), *sponge gourd*, *Koshataki* (ridge gourd), *Karavellaka* (bitter gourd) and *Patola* (pointed gourd); *Dadima* (pomegranate) and *Amalaki*; *Ushnodaka* (warm water) for drinking; and a daily morning walk.

As *Apathya*, patient was asked to avoid *Divaswapna* (daytime sleep), *Ratrijagarana* (night waking), excessive tea, raw and cold food, *Sheeta Jala* (cold water), *Adhyashana*, *Atyashana* (overeating) and *Paryushita Ahara* (stale food).

Limitations of the study

This is a single case report; findings cannot be generalized. The anti-TPO titer was not followed to resolution. No control or comparator group was available.

CONCLUSION

This single case report demonstrates that a systematic protocol directed at correcting *Agni Mandya* (*Deepana-Pachana*, *Vardhamana Snehapana* and *Virechana Karma*), followed by a targeted *Agni*-restorative *Shamana* regimen, was associated with normalization of thyroid function and lipid parameters in a patient with autoimmune primary hypothyroidism

understood as an *Agni Mandya Janita Vikara* (disorder arising from impaired *Agni*), sustained over nine months without any allopathic intervention. Further multi-centre studies with larger sample sizes are recommended to validate *Agni Mandya*-based *Ayurvedic* protocols for autoimmune hypothyroidism.

DECLARATION OF PATIENT CONSENT

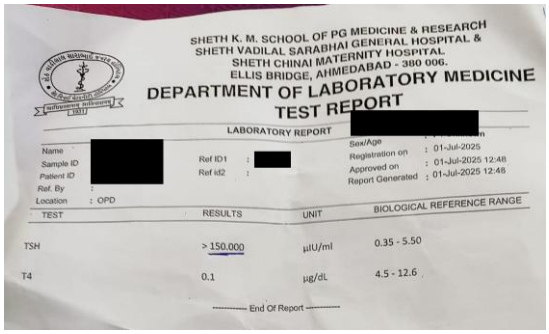
The authors certify that they have obtained all appropriate patient consent for the use of clinical information in this report. The patient understands that her name and initials will not be published and that due effort will be made to conceal her identity.

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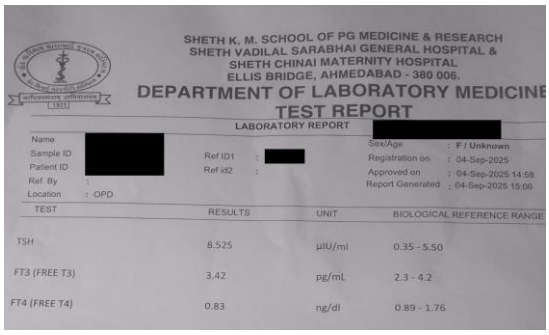
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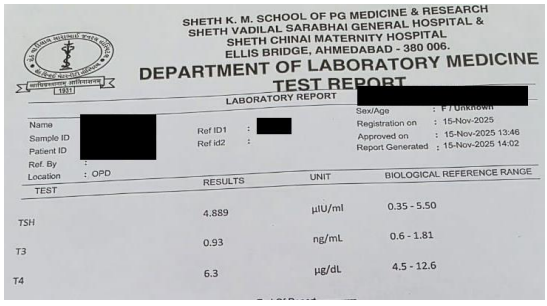

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TEST	RESULTS	UNIT	BIOLOGICAL REFERENCE RANGE
TSH	> 150.000	µIU/ml	0.35 - 5.50
T4	0.1	µg/dL	4.5 - 12.6

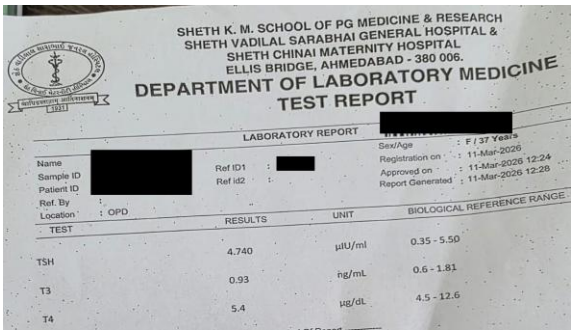
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TSH	8.525	µIU/ml	0.35 - 5.50
FT3 (FREE T3)	3.42	pg/mL	2.3 - 4.2
FT4 (FREE T4)	0.83	ng/dl	0.89 - 1.76


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TSH	4.889	µIU/ml	0.35 - 5.50
T3	0.93	ng/mL	0.6 - 1.81
T4	6.3	µg/dL	4.5 - 12.6



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DEPARTMENT OF LABORATORY MEDICINE
TEST REPORT

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TEST	RESULTS	UNIT	BIOLOGICAL REFERENCE RANGE
TSH	4.740	µIU/ml	0.35 - 5.50
T3	0.93	ng/mL	0.6 - 1.81
T4	5.4	µg/dL	4.5 - 12.6

Figure 4: Thyroid function laboratory reports at baseline (01/07/2025), 2 months (04/09/2025), 4 months (15/11/2025) and 9 months (11/03/2026)

CORRESPONDING AUTHOR
Vd. Swetlana Upreti
PG Scholar, Department of Panchakarma, Government Akhandanand Ayurveda College, Ahmedabad, Gujarat, India
E-mail: swatiupreti7@gmail.com

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