

Review Article

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HYPOTHYROIDISM (*DHATVAGNIMANDYA*): AN INTEGRATIVE REVIEW BASED ON MODERN ENDOCRINOLOGY AND AYURVEDIC PRINCIPLES

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Abstract

Hypothyroidism is a common endocrine disorder characterized by deficient secretion of thyroid hormones, resulting in a generalized slowing of metabolic processes. Clinically, it presents with fatigue, weight gain, cold intolerance, constipation, edema, and cognitive impairment. Although Ayurveda does not describe the thyroid gland anatomically, a similar pathological condition can be understood through the concept of *Dhatvagnimandya*, wherein impairment of tissue metabolism (*Dhatu Agni*) leads to *Ama* formation, *Kapha-Meda* accumulation, and systemic hypometabolism. Classical Ayurvedic texts emphasize *Agni* as the root determinant of health and *Mandagni* as the origin of disease. This review integrates modern biomedical evidence with Ayurvedic theory, correlates etiopathogenesis and symptomatology, updates relevant epidemiological data within the Indian subcontinent, and discusses contemporary and traditional management strategies. An integrative therapeutic approach may optimize patient outcomes.

Keywords: Hypothyroidism, *Dhatvagnimandya*, *Agni*, *Ama*, *Kapha*, Thyroid hormones, Ayurveda, Integrative medicine

Introduction:

Hypothyroidism is defined as a clinical syndrome caused by insufficient production of triiodothyronine (T-3) and thyroxine (T-4). These hormones regulate basal metabolic rate, thermogenesis, lipid metabolism, and cellular growth. Reduced hormone levels result in systemic metabolic decline and multisystem dysfunction. The condition is widely prevalent among women, elderly individuals, and iodine-deficient or iodine-excess populations. Ayurveda attributes all metabolic activities to *Agni*, which governs digestion, absorption, and tissue transformation. Among the thirteen types of *Agni*, *Dhatu Agni* regulates tissue-level transformation and cellular metabolism. When *Dhatu Agni* becomes weak (*Dhatvagnimandya*), improper tissue nourishment, *Ama* (toxic, undigested metabolic by-products) formation, and *Kapha-Meda* accumulation occur, producing clinical features analogous to hypothyroidism.

Prevalence and Risk Factors: The prevalence of overt hypothyroidism in the general population varies between 0.3% and 3.7% in the United States and between 0.2% and 5.3% in Europe, depending on the clinical definitions utilized (1–5). A meta-analysis of studies across nine European countries estimated the prevalence of undiagnosed hypothyroidism, including both overt and subclinical cases, at approximately 5% (6). Differences in iodine status markedly affect regional prevalence; thyroid dysfunction occurs frequently both in populations with a relatively high iodine intake and in severely iodine-deficient regions (7,8). Hypothyroidism occurs more frequently in women, in older individuals (>65 years), and in white populations, although comprehensive data on distinct ethnic variations remain sparse (2,9,10). Furthermore, thyroid insufficiency shows a strong clinical clustering with other autoimmune disorders, such as type 1 diabetes mellitus, autoimmune gastric atrophy, and celiac disease, frequently manifesting as part of multiple autoimmune endocrinopathies (10).

Indian Epidemiological Context: In India, hypothyroidism represents a significant public health burden with distinct epidemiological characteristics. Large-scale multicenter epidemiological studies reveal that the prevalence of hypothyroidism is notably higher in India than in Western cohorts. Estimates indicate that subclinical and overt hypothyroidism affect approximately 11% of the adult Indian population, with women demonstrating a significantly higher risk profile than men. Data from national reference frameworks highlight that urban populations experience elevated rates of thyroid dysfunction, driven by a

combination of dietary shifts, high psychological stress, and environmental triggers. Furthermore, autoimmune thyroiditis (Hashimoto's thyroiditis) has emerged as the primary cause of hypothyroidism in post-iodization India, overriding historical nutritional iodine deficiency.

Aim and Objectives

Aim: To determine and elucidate the pathophysiological relationship between *Dhatvagnimandya* and hypothyroidism.

Objectives

- To systematically study and analyze the clinical signs and symptoms of hypothyroidism in relation to *Dhatvagnimandya*.
- To evaluate and establish a comprehensive correlation between hypothyroidism and the Ayurvedic concept of *Dhatvagnimandya*.

Material & Methodology: A comprehensive literary review of hypothyroidism was conducted using standard modern medical textbooks and by exploring relevant clinical databases. The concept of *Dhatvagnimandya* was concurrently analyzed through an extensive review of classical Ayurvedic texts, including the *Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya*, alongside contemporary peer-reviewed integrative medical literature.

Physiological Effect of Thyroid Hormone: Thyroid hormones (T-3 and T-4) are the principal metabolic hormones influencing nearly every cell of the body throughout all stages of life (11). They play a crucial role in normal growth and development, particularly during the fetal period and early infancy (11). These hormones facilitate proper tissue development by enhancing amino acid uptake and stimulating enzymatic processes involved in protein synthesis, contributing significantly to systemic growth and maturation (11). In addition, thyroid hormones regulate a wide range of metabolic activities in various tissues, leading to multiple specific metabolic effects (11).

- **Action on Basal Metabolic Rate (BMR):** Thyroxine enhances metabolic activity in almost all body tissues, with the exception of the brain, retina, spleen, testes, and lungs (11). It elevates the BMR primarily by increasing oxygen consumption at the cellular level (11).

- **Action on Protein Metabolism:** Thyroid hormones stimulate protein synthesis within cells; however, alongside this anabolic effect, thyroxine also promotes protein breakdown (catabolism), especially when present in excess (11).
- **Action on Carbohydrate Metabolism:** Thyroid hormones enhance energy production by promoting glucose uptake into cells, as well as stimulating glycogenolysis and gluconeogenesis to increase the availability of free glucose in the body (11).
- **Action on Lipid Metabolism:** Thyroid hormones promote fat metabolism by mobilizing lipids from adipose tissue and enhancing their oxidation within mitochondria to generate energy, while increasing the number and size of mitochondria (11). Thyroxine reduces fat storage by releasing lipids from fat depots, converting them into free fatty acids that circulate in the bloodstream (11).
- **Action on Body Temperature:** Thyroid hormones raise body heat production by accelerating cellular metabolic activities and elevating the BMR (11).
- **Action on Growth:** Thyroid hormones exert both general and specific influences on growth; balanced thyroxine levels promote appropriate skeletal and soft tissue development during growth years (11).
- **Action on Body Weight:** Thyroxine plays a key role in regulating body weight: elevated levels reduce body weight and fat storage, while decreased levels lead to weight gain due to increased fat deposition (11).
- **Action on Blood:** Thyroxine enhances erythropoiesis by stimulating red blood cell production and increasing blood volume, making it an essential factor for normal erythropoietic activity (11).
- **Action on Cardiovascular System:** Thyroxine directly stimulates myocardial cells, increasing heart rate and the force of contraction, and promotes vasodilation by enhancing metabolic activity; together, these effects influence systemic cardiac output and blood pressure (11).
- **Action on Gastrointestinal Tract:** Thyroxine stimulates appetite and food intake while enhancing gastrointestinal secretions and motility; therefore, excess levels may lead to diarrhea, whereas deficiency can result in constipation (11).

- **Action on Central Nervous System:** Thyroxine is crucial for the normal development and functioning of the central nervous system, playing a vital role in brain growth and neuro-maturation during **fetal life** and the early postnatal years (11).
- **Action on Skeletal Muscle:** Thyroxine is necessary for proper skeletal muscle function; a mild increase enhances muscular performance, whereas excessive levels lead to muscle weakness due to accelerated protein breakdown (11).
- **Action on Sleep:** Adequate levels of thyroxine are essential for maintaining a normal sleep-wake cycle; excess secretion overstimulates muscles and the central nervous system, leading to fatigue with insomnia, whereas deficiency results in increased drowsiness (somnolence) (11).
- **Action on Sexual Function:** Normal levels of thyroxine are crucial for proper reproductive function (11). In men, hypothyroidism can reduce or abolish libido, while hyperthyroidism may lead to impotence (11). In women, hypothyroidism is associated with menorrhagia, polymenorrhagia, or irregular cycles, sometimes progressing to amenorrhea (11).
- **Action on Other Endocrine Glands:** Due to its widespread metabolic actions, thyroxine increases the functional demand on other endocrine glands, thereby stimulating their secretory activity (11).

Physiological Functions of Agni: In Ayurveda, *Agni* is described as the fundamental bio-transformative force responsible for digestion and metabolism. It plays a vital role in breaking down, absorbing, and assimilating food, which are processes essential for sustaining life. *Agni* transforms food into energy, which supports all vital bodily functions and contributes to factors such as vitality, strength, complexion, immunity (*ojas*), and life force (*prana*).

According to Acharya Charaka, balanced *Agni* ensures good health and longevity, whereas impaired *Agni* disrupts metabolism and leads to disease. Thus, *Agni* is considered the root of life. Its activity is influenced by *Pitta*, *Vata* (especially *Samana Vata*), and *Kapha*. *Agni* performs both general and specific functions in the body and is classified into 13 types based on its location and role: one *Jatharagni*, five *Bhutagni*, and seven *Dhatvagni*.

Jatharagni: *Jatharagni* refers to the primary digestive fire located in the stomach and duodenum (*Jathara*). According to the *Ashtanga Hridaya*, its primary site is the *Grahani* (duodenum), which retains food in the stomach (*Amashaya*) for an appropriate duration to ensure proper digestion. *Jatharagni* plays a crucial role in sustaining life, health, strength, vitality, and overall metabolic efficiency.

The functioning of *Grahani* and *Agni* are interdependent; any imbalance in *Agni* leads to disturbance in *Grahani*, resulting in clinical disease (12). *Jatharagni* is considered the most important among all forms of *Agni* because all ingested food is first processed by it (13). Its primary function is the gross digestion of food, breaking down substances composed of the five basic elements and transforming them into a form suitable for tissue nourishment (*dhatu*) (13). It also separates digested food into the nutritive essence (*prasa*) and waste products (*kitta*) (13).

Furthermore, *Jatharagni* directly influences *Dhatvagni*, which governs cellular metabolism and tissue formation (*dhatu-paka*) (14). The proper functioning of all *Dhatvagni* depends on a balanced state of *Jatharagni* (14). If *Jatharagni* becomes hyperactive (*tikshna*) or hypoactive (*manda*), it disrupts tissue metabolism, leading to various systemic disorders (14). Thus, *Jatharagni* is regarded as the central regulator of all other *Agnis*, and the proper functioning of the remaining twelve *Agnis* depends entirely on its functional state (14).

Bhutagni: *Bhutagni* refers to the metabolic energy present within the five basic elements (*mahabhutas*)—*Parthiva* (earth), *Apya* (water), *Tejas* (fire), *Vayavya* (air), and *Nabhasa* (space). Each of these elements possesses its own specific *Bhutagni*. Since every cell in the body (*dhatu paramanu*) is composed of these five elements, *Bhutagni* is also present at the sub-cellular level.

The food we consume is similarly made up of the same five *mahabhutas* along with their respective *Bhutagni*, creating a fundamental structural similarity between the body and external nutrients. According to Acharya Charaka, each *Bhutagni* acts on its corresponding elemental component in food, digesting and transforming it (15). The processed nutrients, which retain qualities similar to their respective elements, then nourish the corresponding tissues in the body (15). *Bhutagni* functions after the action of *Jatharagni* (15). Once the initial macro-digestion is completed, *Bhutagni* carries out further subtle transformation and breakdown of nutrients, facilitating micro-digestion at the elemental and cellular levels.

Dhatvagni: *Dhatvagni* refers to the specific metabolic activity present in each of the seven body tissues (*dhatu*) (16). Every *dhatu* possesses its own *agni* responsible for processing and transforming nutrients delivered through its respective channels(16). The seven *Dhatvagni* are *Rasagni*, *Raktagni*, *Mamsagni*, *Medagni*, *Asthyagni*, *Majjagni*, and *Shukragni* (16).

Each *Dhatvagni* acts selectively, converting the nutritive essence of food (*ahara rasa*) into the specific structural components required for its corresponding tissue (16). This transformation ensures proper nourishment, growth, and maintenance of each *dhatu* (16). As described by Acharya Charaka, these tissue-specific *agnis* digest and assimilate nutrients into forms similar to their own structure, supporting the body's metabolic integrity.

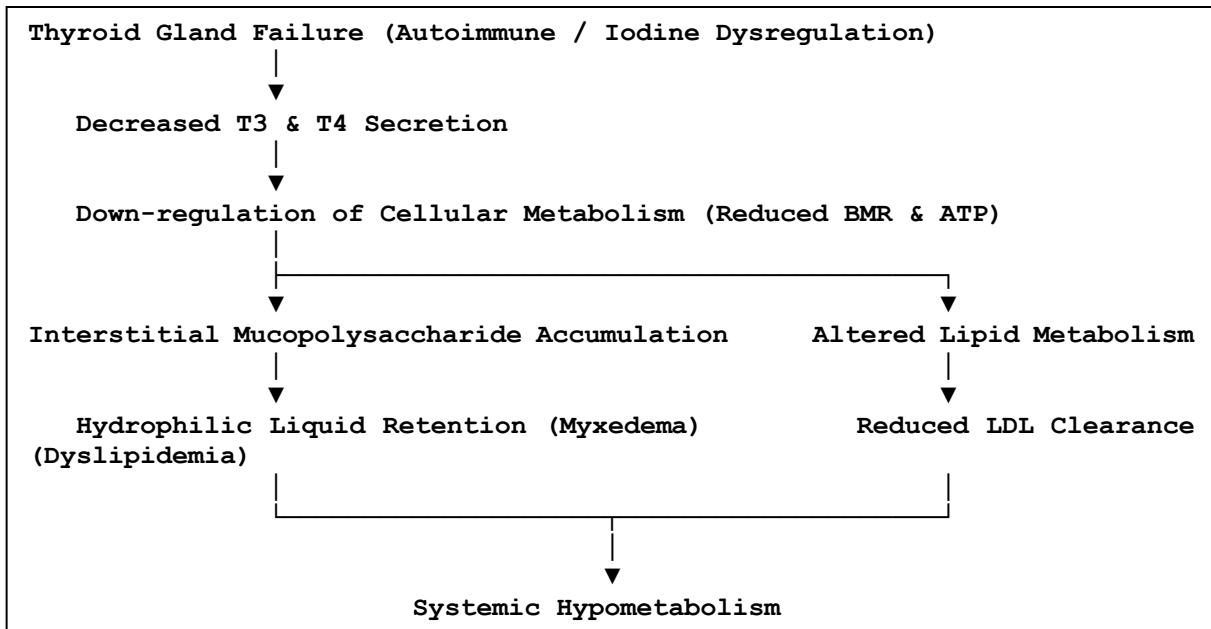
The overall digestive and metabolic process involves a sequential action of *Agni*: food is first digested by *Jatharagni*, followed by elemental transformation by *Bhutagni*, and finally refined at the tissue level by *Dhatvagni*. This coordinated process contributes to strength, complexion, vitality, and overall well-being. However, due to factors that weaken *Agni* (*agnimandyakara hetu*), the function of *Jatharagni* may become impaired, leading to improper digestion. As a result, the primary nutrient essence (*rasa dhatu*) remains incompletely processed and forms *Ama*. This *Ama* vitiates the *doshas*, causes obstruction in the *srotas* (*srotodushti*), and further weakens *Dhatvagni*, ultimately resulting in disturbed metabolism and systemic chronic disease.

Pathogenesis

Modern Pathophysiology: The pathogenesis of primary hypothyroidism begins with thyroid gland failure, most commonly triggered by autoimmune destruction (Hashimoto's thyroiditis) or severe iodine dysregulation. This results in a significant reduction in the synthesis and secretion of triiodothyronine (T-3) and thyroxine (T-4). The drop in circulating thyroid hormones removes the negative feedback inhibition on the anterior pituitary gland and hypothalamus, causing a compensatory increase in Thyroid-Stimulating Hormone (TSH).

At the cellular level, deficiency of T-3 and T-4 leads to a generalized down-regulation of metabolic enzymes, causing a reduction in the basal metabolic rate, decreased cellular oxygen consumption, and diminished ATP production. A hallmark clinical feature is the systemic accumulation of hydrophilic mucopolysaccharides—primarily hyaluronic acid and chondroitin sulfate—in the interstitial spaces of subcutaneous and mucosal tissues. This

interstitial deposition binds water, leading to the non-pitting, generalized interstitial edema known as myxedema. Simultaneously, down-regulated lipid metabolism decreases LDL receptor expression, precipitating secondary dyslipidemia, marked by elevated serum cholesterol and triglycerides, which drives global systemic hypometabolism.

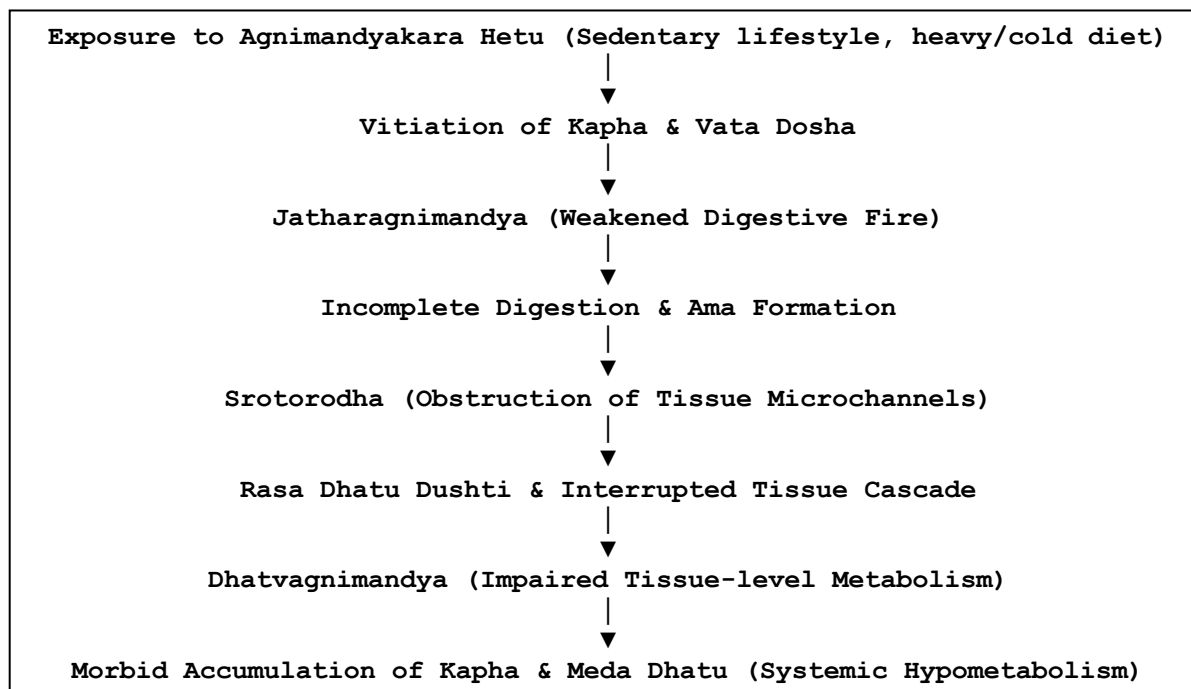


Ayurvedic Samprapti (Etiopathogenesis): The *samprapti* of hypothyroidism is initiated by exposure to predisposing factors (*agnimandyakara hetu*), such as a sedentary lifestyle (*avyayama*), daytime sleep (*divasvapna*), and excessive intake of heavy, cold, and unctuous food (*snigda, guru, sheeta ahara*). These factors primarily vitiate *Kapha* and *Vata*, which directly compromises the central digestive fire, causing *Jatharagnimandya*. This compromised state leads to the incomplete digestion of food, resulting in the generation of *Ama* (highly toxic, undigested metabolic debris).

This circulating *Ama* enters the vascular system and infiltrates the macro- and micro-channels (*srotas*), causing mechanical and functional obstruction (*srotorodha*). The initial tissue channel affected is the *Rasavaha Srotas*, leading to *Rasa Dhatu Dushti*. Because subsequent tissue nourishment depends on a sequential cascade (*dhatu-poshana krama*), the block in the *srotas* restricts the supply of nutrition to deeper tissues.

Consequently, *Rasagni* and *Medagni* become severely hypoactive, establishing a state of chronic *Dhatvagnimandya*. This tissue-level metabolic arrest prevents the proper transformation of *Rasa* into *Rakta* (blood) and *Meda* (fat). Instead of functional tissue

development, it causes a morbid, un-metabolized over-accumulation of heavy *Kapha* and *Meda* (*Kapha-Meda Vriddhi*), resulting in the hypometabolic state clinically identified as hypothyroidism.



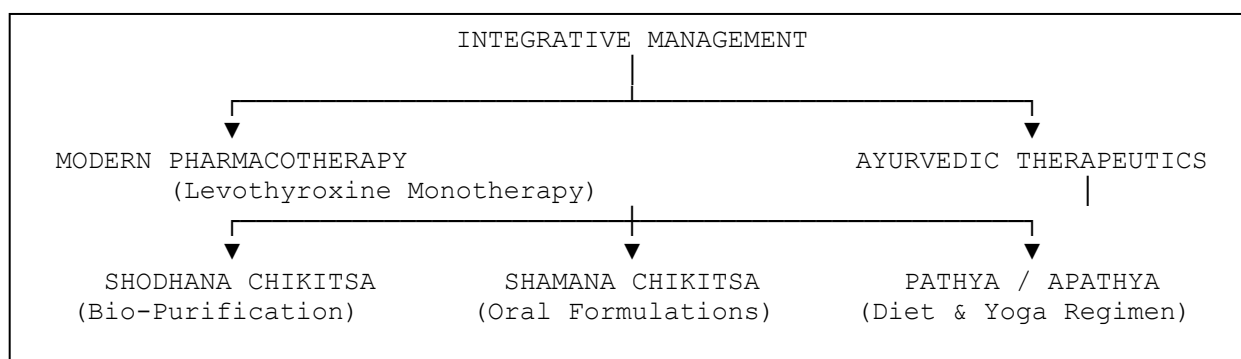
Symptomatology and Ayurvedic Correlations (17): To optimize clinical assessment in integrative settings, the conventional presentation of hypothyroidism is cross-referenced with traditional Ayurvedic tissue-level equivalents below (17):

Comparative Analysis of Symptoms

Modern Biomedical Symptom	Ayurvedic Pathophysiological Equivalent	Primary Involved Dosha / Dhatu / Srotas
Abnormal Weight Gain (17)	<i>Medovruddhi</i> (Excessive fat accumulation) (17)	<i>Meda Dhatu, Medovaha Srotas</i> (17)
Puffy Appearance / Edema (17)	<i>Shotha / Shopha</i> (Kapha-driven swelling) (17)	<i>Kapha Dosha, Rasa Dhatu</i> (17)
Loss of Appetite (17)	<i>Agnimandya / Aruchi</i> (Diminished appetite) (17)	<i>Jatharagni, Annavaaha Srotas</i> (17)
Dry, Coarse Skin & Hair (17)	<i>Twak Rukshata / Khara Kesa</i> (17)	<i>Vata Dosha, Rasa-Rakta Dhatu</i> (17)

Minimal / Absent Sweating (17)	<i>Anabhishtyanda / Asveda</i> (17)	<i>Pitta Dosha, Meda Dhatu (Sweda)</i> (17)
Anemia (17)	<i>Pandu Roga</i> (Pale, low-nourished blood) (17)	<i>Rakta Dhatu, Raktavaha Srotas</i> (17)
Constipation (17)	<i>Malavashtambha</i> (Sluggish bowel evacuation) (17)	<i>Apana Vayu, Purishavaha Srotas</i> (17)
Hoarseness of Voice (17)	<i>Svara Bhedha</i> (Obstructed vocal cords) (17)	<i>Kapha Dosha, Pranavaha Srotas</i> (17)
Generalized Aches & Pains (17)	<i>Angamarda / Ruja</i> (Widespread body pain) (17)	<i>Vata Dosha, Rasa Dhatu</i> (17)
Sluggishness & Lethargy (17)	<i>Alasya / Shaithilya</i> (Physical inertia) (17)	<i>Kapha Dosha, Ama</i> (17)
Tiredness & Weakness (17)	<i>Daurbalya / Klama</i> (Fatigue without exertion) (17)	<i>Vata Dosha, Ojas</i> (17)
Menstrual Disturbances (17)	<i>Artava Dushti / Anartava</i> (Irregular flow) (17)	<i>Rasa Dhatu, Artavavaha Srotas</i> (17)
Cold Intolerance (17)	<i>Sheetasatmya</i> (Hypersensitivity to cold) (17)	<i>Agni Mandya, Rakta Dhatu</i> (17)
Forgetfulness / Brain Fog (17)	<i>Smrutialpata / Tamas Vriddhi</i> (17)	<i>Tamo Guna, Manovaha Srotas</i> (17)
Excessive Sleepiness (17)	<i>Tandra / Atinidra</i> (Somnolence) (17)	<i>Kapha Dosha, Rasa Dhatu</i> (17)

Management and Treatment Strategies: An effective therapeutic framework requires a dual approach, balancing modern hormonal optimization with classic Ayurvedic metabolic re-ignition.



Modern Biomedical Management: Standard contemporary treatment centers exclusively on hormone replacement therapy to restore a euthyroid state.

- **Pharmacotherapy:** Levothyroxine sodium (T-4) monotherapy remains the gold standard. It is administered orally on an empty stomach at least 30–60 minutes before breakfast to guarantee optimal intestinal absorption. The dose is calculated based on body weight (typically $1.6 \mu\text{g/kg/day}$ in young adults) and modified based on clinical response.
- **Clinical Monitoring:** Serum TSH levels are the primary indicator for monitoring dose adequacy. Serum TSH is evaluated 6–8 weeks following treatment initiation or any dosage adjustments. Long-term monitoring is scheduled every 6–12 months once therapeutic stability is reached.

Ayurvedic Management Principles: Ayurvedic treatment strategies do not focus on external hormone supplementation; instead, they target the correction of *Dhatvagnimandya*, the digestion of *Ama*, and clearing channel blockages (*Srotoshodhana*).

1. Shodhana Chikitsa (Bio-Purification Procedures): For patients displaying robust physical stamina (*Bahu-Dosha Avastha*), metabolic detoxification is introduced to root out deep-seated morbidities.

- **Deepana-Pachana:** Pre-procedural optimization is conducted using carminative and digestive stimulants like *Chitrakadi Vati*, *Shunthi*, or *Panchakola Churna* to process circulating *Ama*.
- **Snehapana (Internal Oleation):** Administering medicated lipophilic carriers in escalating daily doses, utilizing *Panchatikta Ghrita* or *Guggulu Tiktaka Ghrita*, to penetrate cellular lipid bilayers and mobilize fat-soluble metabolic wastes.
- **Vamana Karma (Therapeutic Emesis):** Specifically indicated to eliminate excess *Kapha* from its primary locus (*Amashaya*). This clearance stimulates central metabolic activity and enhances systemic *Jatharagni* efficiency.
- **Virechana Karma (Therapeutic Purgation):** Administered to clear *Pitta* and *Kapha* debris via the gastrointestinal tract, which cleanses systemic *srotas* and regulates thyroid-axis feedback pathways.

2. Shamana Chikitsa (Conservative Oral Formulations): Oral herbs are chosen for their hot potency (*Ushna Veerya*), scraping properties (*Lekhana Guna*), and ability to stimulate tissue metabolism.

- **Kanchanar Guggulu:** The definitive formulation utilized in traditional glandular enlargements and metabolic growths (*Granthi/Arbudha*). *Kanchanar* (*Bauhinia variegata*) provides notable scraping actions, reducing glandular sluggishness.
- **Vyoshadi Guggulu & Shadushana Churna:** Rich in pungent compounds (*Trikatu*), these formulations directly re-ignite *Rasagni* and *Medagni*, lowering elevated *Kapha* and secondary *Meda* accumulation.
- **Varunadi Kwatha:** Promotes optimal fluid kinetics, reduces peripheral *Shotha* (myxedematous edema), and eliminates fluid retention.
- **Rasayana Herbs:** Single herbs such as *Ashwagandha* (*Withania somnifera*) and *Guduchi* (*Tinospora cordifolia*) provide adaptogenic support, stabilizing the neuro-endocrine axis and mitigating chronic fatigue.

3. Pathya (Dietary and Lifestyle Regimen)

- **Dietary Adjustments:** Patients should emphasize *Kaphahara Ahara*, incorporating barley (*Yava*), green gram (*Mudga*), bitter melon (*Karavellaka*), garlic (*Lasuna*), and warm water (*Ushna Jala*). Heavy, cold, processed dairy, white flour, and sweet, unctuous foods (*Apathya*) must be strictly avoided.
- **Yogic Interventions:** Daily execution of specific postures that stimulate neck-region circulation, such as *Sarvangasana* (Shoulder Stand), *Matsyasana* (Fish Pose), and *Halasana* (Plow Pose), coupled with *Ujjayi Pranayama* and *Kapalbhati*, provides mechanical and bio-energetic stimulation to the thyroid area.

Discussion

In classical Ayurvedic literature, hypothyroidism is not documented as a distinct nosological disease entity; however, its clinical presentation can be closely mapped to *Dhatvagnimandya* (diminished metabolic activity at the tissue level). According to Ayurvedic philosophy, the human body is sustained by a delicate equilibrium of *Dosha* (functional principles), *Dhatu* (structural tissues), and *Mala* (waste products) (18). The appropriate nourishment and chronological development of these structural elements depend on the functional capacity of *Agni* at various physiological levels.

In modern physiology, global metabolic activity is regulated by circulating thyroid hormones. In contrast, Ayurveda explains systemic metabolism through the coordinated

action of *Jatharagni* (digestive fire), *Bhutagni* (elemental metabolism), and *Dhatvagni* (tissue-specific metabolism). When these *Agnis* become impaired due to improper dietary and behavioral habits (*agnimandyakara hetu sevana*), it prompts a state of *Agnimandya*. This causes incomplete macro-digestion, yielding *Ama*.

The accumulation of *Ama* leads to *Srotorodha* (obstruction of micro-channels), disrupting the downstream delivery of essential nutrients. Initially, *Rasa Dhatu* becomes improperly formed, failing to satisfy the downstream *Dhatu-Poshana* cascade. This tissue-level metabolic arrest prevents the proper transformation of nutrients, manifesting as lethargy, weight gain, cold intolerance, and a sluggish basal metabolic rate.

Thus, hypothyroidism is best understood through an integrative lens as a systemic metabolic disorder rooted in *Agnimandya* and *Ama* formation, culminating in chronic *Dhatvagnimandya*.

Conclusion

Ayurvedic diagnostics prioritize underlying pathophysiology and individual constitutional imbalances over nomenclature. When a condition is not explicitly defined in classical texts, the physician should analyze the signs, symptoms, and structural changes based on the core principles of *Dosha*, *Dhatu*, *Agni*, and *Srotas*.

Hypothyroidism can be effectively interpreted through this foundational framework. Based on its metabolic derangements, it correlates directly with *Dhatvagnimandya*, where impaired tissue-level metabolism plays a central role. Adopting an integrative therapeutic approach—combining modern levothyroxine replacement with Ayurvedic *Deepana-Pachana*, *Srotoshodhana*, and *Guggulu* formulations—may address both hormone deficiency and systemic metabolic slowing, potentially improving clinical outcomes.

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