ISSN: 2322 - 0902 (P)
ISSN: 2322 - 0910 (O)

International Journal of Ayurveda and Pharma Research

Review Article

BRIDGING CLASSICAL AYURVEDA AND MODERN MEDICINE IN THE UNDERSTANDING OF *SHVITRA* (VITILIGO)

Chandraprabha Sharma[1]*, Harish Kumar Singhal[2], Manisha Goyal[3]

*[1]PG Scholar, [2]Professor & HOD, PG Department of Kaumarbhritya, [3]Associate Professor, PG Department of Rasa Shastra and Bhaishajya Kalpana, Postgraduate Institute of Ayurveda, Dr. S.R. Rajasthan Ayurved University, Jodhpur, Rajasthan, India.

Article info

Article History:
Received: 14-01-2026
Accepted: 20-02-2026
Published: 26-03-2026

KEYWORDS:
Ayurveda, *Kilasa, Rasayana, Shamana, Shodhana, Shvitra*, Vitiligo.

ABSTRACT

Vitiligo is a chronic dermatological disorder characterized by the loss of melanocytes, leading to depigmented skin patches. Ayurveda describes it as *Shvitra* or *Kilasa* and attributes its etiology to *Dosha* imbalances and impaired digestive function (*Agnimandya*), while contemporary medicine identifies autoimmunity, genetic susceptibility, and oxidative stress as the key etiological factors. This comparative analytical review was done by referring to classical Ayurvedic texts such as *Charaka Saṃhita*, *Sushruta Saṃhita, Ashtanga Hṛidaya*, and *Kashyapa Saṃhita* apart from the contemporary dermatology literature regarding etiology, classification, diagnostic tools, and therapeutic approaches, elucidating the integrative strategies. Ayurveda interprets *Shvitra* as a result of improper diet, impaired Agni, and *Dosha* vitiation, whereas in modern science, autoimmune mechanisms, especially CD8+ T-cell–mediated melanocyte destruction, with genetic predisposition as a major risk factor, are identified. Therapeutic approaches in Ayurveda consist of *Shodhana* (purificatory measures), *Shamana* (palliative therapies), and *Rasayana* (rejuvenative therapies), whereas modern management involves topical and systemic corticosteroids, calcineurin inhibitors, phototherapy, and surgical grafting. Integrating the two may provide for a holistic, patient-centered care wherein Ayurvedic *Rasayana* therapies support the modern immunomodulatory and phototherapeutic interventions, reducing recurrence and improving the long-term outcome. The bridging of the two approaches presents an opportunity to enhance therapeutic efficacy, minimize the relapse, and improve the overall quality of life in patients affected by vitiligo.

INTRODUCTION

Vitiligo is a long-term skin condition marked by the gradual loss of melanocytes, which results in distinct depigmented areas on the skin and occasionally on mucous membranes and hair (leukotrichia)[1]. It is believed that the word "vitiligo" comes from the Latin vitium, which means imperfection or blemish.

Early writers also connected it to vitellus (veal), noting the similarity in color between veal meat and vitiliginous patches[2].

Access this article online

Quick Response Code

https://doi.org/10.47070/ijapr.v14i3.4066

With advancing knowledge, vitiligo is now recognized as an acquired autoimmune condition in which melanocyte destruction underlies the loss of pigmentation[3].

The worldwide prevalence of vitiligo is roughly 1%, although there is significant geographical variation. A large meta-analysis estimated prevalence at 1.3%, with Europe having the highest rates (1.6%), the United States 1.4%, and Japan the lowest (0.5%) [4]. In India, prevalence varies widely: from 0.46% in Kolkata to 2.16% in Chandigarh, with some outpatient studies citing figures as high as 8.8%[5,6]. Nearly half of all cases develop before the age of 20, and up to 80% occur before the age of 30[7]. Both sexes are equally affected, though women more often seek care due to cosmetic and psychosocial concerns [8].

There is significant psychological morbidity associated with vitiligo. Rates of anxiety (28.8%) and depression (24.5%) are high in patients when compared with the general population. The conspicuous nature of the disorder often leads to stigma, social withdrawal, and lowered self-esteem, a fact that may indicate a need for a more holistic approach in management[9].

Descriptions of vitiligo also appear in ancient medical traditions. In Ayurveda, the disease is known as *Shvitra* or *Kilasa*. Classical texts like the *Charaka Saṃhita* and *Sushruta Saṃhita* attribute it to disturbances of the three *Doshas*, namely *Vata*, *Pitta*, and *Kapha*, along with impaired digestion (*Agnimandya*)[10,11]. The word *Shvitra* is derived from *Shweta*, which means white, manifesting the color of the lesions, while *Kilasa* suggests abnormal discoloration, *Vikṛta Varna*[12]. Sanskrit lexicons like *Sabdakalpadruma* and *Amarakosa* elaborate on the meaning of these terms, while *Kashyapa Saṃhita* states that any pathological change in skin color toward white may be referred to as *Shvitra*[13,14].

Ayurvedic management is comprehensive and staged. It begins with *Nidana Parivarjana* (removal of causative factors) and *Deepana-Pachana* (enhancing digestion and metabolism). Interventions include *Shodhana* (purificatory therapies such as *Vamana*, *Virechana*, and *Raktamokhshana*), *Shamana* (palliative measures with herbs like *Avalguja*, *Triphala*, and *Khadira*), along with external applications (*Lepa*), medicated oils (*Taila*), clarified butter preparations (*Ghṛita*), and *Rasayana* therapy aimed at tissue regeneration and immune strengthening[15,16].

AIMS AND OBJECTIVES

Aim

The aim is to systematically review and assess the effectiveness and potential of Ayurvedic treatments in the management of vitiligo, *Shvitra*, linking classical Ayurvedic concepts to contemporary clinical practices.

Objectives

1. To trace the various historical, traditional, and contemporary conceptualizations of vitiligo/ *Shvitra*, from its etymology through ancient Ayurveda to descriptions in biomedicine.
2. Assess the current global prevalence and epidemiology of vitiligo, especially in India, focusing on its psychosocial toll across different populations.
3. To study the etiopathogenesis of vitiligo through contemporary medicine (genetic factors, autoimmune responses, oxidative stress, neural theories) and Ayurveda (*Tridosha* imbalance, *Nidana*, *Samprapti*).
4. To classify and differentiate the clinical forms of vitiligo according to modern dermatological classifications and Ayurvedic literature, namely, *Charaka, Sushruta, Vagbhata, Kashyapa*.
5. To compare diagnostic approaches and therapeutic methods in both systems, including clinical, laboratory, dermoscopic, and scoring indices as used in current dermatology, and Ayurvedic *Shodhana, Shamana, Rasayana*, and external therapies

METHODOLOGY

The study was a qualitative narrative review that attempted to integrate traditional Ayurvedic literature with state-of-the-art biomedical research in psychodermatology. The approach was holistic, involving comparative analysis, conceptual mapping, and thematic classification.

Literature Sources

Ayurvedic Texts

Among the primary classical Ayurvedic sources reviewed are the following: *Charaka Samhita, Sushruta Samhita, Ashtanga Hridaya,* Commentaries such as *Chakrapani-on Charaka Samhita,* and *Dalhana- on Sushruta Samhita*. These texts were examined for mentions of *Shvitra*.

Modern Medical Literature

The following scientific databases were used: PubMed, Scopus, and Google Scholar. Relevant articles and systematic reviews from journals, as well as clinical studies published within the period from 2000 to 2024, have been chosen.

Modern Review

Etiopathogenesis

Vitiligo results from the complex interplay between genetic, environmental, immune, oxidative, endocrine, nutritional, and neurological factors[17]. The risk is higher in persons with a family history of the disease; genome-wide association studies have so far identified about 60 risk alleles predisposing individuals to the disease[18]. Various hypotheses have been advanced on the pathogenesis of melanocyte destruction, the most tenable being the autoimmune, neural, genetic, and oxidative stress hypotheses.

1. Autoimmune theory: Under cell stress, melanocytes release exosomes and extracellular vesicles that contain micro-RNAs, heat shock protein 70 (Hsp70), and damage-associated molecular patterns (DAMPs). These signals activate antigen-presenting cells through the stimulation of interferon-γ production, which subsequently upregulates chemokines CXCL9, CXCL10, and CXCL11. These chemokines then attract autoreactive CD8+T cells to the epidermis, where they contribute to melanocyte destruction[19,20]. Evidence also exists for deregulation of regulatory T cells (Tregs) and persistence of CD8+ tissue-resident memory T cells (Trm), which may explain the causes for recurrence after treatment[20].

2. Neural theory: Neurochemical mediators released from peripheral nerve endings have been implicated in melanocyte cytotoxicity. Elevated plasma norepinephrine, increased urinary catecholamine metabolites, and higher levels of neuropeptide Y (NPY) in lesional and perilesional skin suggest dysregulated neuroimmune signaling in vitiligo[21,22,23,24]. This supports the concept that neuronal dysfunction and local neuropeptide imbalance contribute to melanocyte destruction.

3. Genetic theory: Several genetic loci have been implicated for susceptibility to vitiligo, which include NLRP1, PDGFRA, PTPN22, FOXD3, XBP1, and CTLA-4. HLA-DRB1 and DQA1 gene polymorphisms further point toward genetic susceptibility. There is also a genetic sharing of vitiligo with other autoimmune diseases like type 1 diabetes mellitus and autoimmune thyroiditis, indicating systemic autoimmunity[25].

4. Oxidative stress theory: Oxidative stress is considered one of the major factors in the pathogenesis of vitiligo. The overproduction of reactive oxygen species along with reduced levels of antioxidant defenses produces a cytotoxic milieu for melanocytes. The unfolded protein response via XBP1 may also promote secretion of pro-inflammatory cytokines IL-6 and IL-8 that dampen Treg function and enhance the immune-mediated injury. Additional suppression of nuclear factor erythroid 2-related factor 2 and mammalian target of rapamycin complex 1 enhances oxidative injury, making melanocytes highly susceptible to apoptosis[26].

Classification of Vitiligo

According to British Association of Dermatology (BAD)

Table 1: Classification of Vitiligo

Main Type	Subtype	Definition
Nonsegmental Vitiligo (NSV)	Acrofacial	Generally limited to face and extremities (head, hands, feet)
	Generalized	Involves progression of NSV to other body sites
	Universal	Most extensive form; depigmentation of >80% of total body surface
	Mucosal	Oral and/or genital mucosae affected
	Mixed	Coexistence of NSV and segmental vitiligo
	Rare variants	Follicular; Vitiligo minor (incomplete loss of pigmentation with pale skin compared to surrounding areas); Vitiligo punctata (1–1.5 mm sharply demarcated macules)
Segmental Vitiligo (SV)	Uni-, bi-, or plurisegmental	≥1 depigmented macules distributed on one side of the body
Undetermined / Unclassified Vitiligo	Focal	Small, isolated patch(es) or macules, not progressed into NSV after ≥2 years and not following a segmental pattern
	Mucosal	Single mucosal site involvement in isolation

Table 2: Comparative Features of Non-Segmental Vitiligo (NSV) and Segmental Vitiligo (SV)

Feature	Non-Segmental Vitiligo (NSV)	Segmental Vitiligo (SV)
1. Prevalence	Most common (80–90% of cases)	Less common (10–20% of cases)
2. Distribution	Bilateral and symmetrical	Unilateral and localized
3. Age of Onset	Can occur at any age	Usually in childhood or adolescence
4. Progression	Often progressive with multiple episodes	Rapid early spread, then usually stabilizes
5. Association with Autoimmunity	Strongly associated with autoimmune diseases	Rarely associated with autoimmune conditions
6. Family History	Frequently positive	Less frequent
7. Response to Therapy	Better response to medical treatments (e.g., phototherapy)	Limited response; may require surgical intervention
8. Affected Areas	Commonly face, hands, feet, and body folds	Usually, a specific dermatome or segment
9. Mechanism of Melanocyte Destruction	Systemic immune-mediated attack	Local neural or segmental melanocyte destruction

10. Stability	Often unstable with cycles of activity and remission	More stable after initial rapid spread

Diagnosis

The diagnosis of vitiligo relies primarily on a clinical examination, comprehensive history from the patient, and, in selected cases, supplemental laboratory studies. Dermatologists usually conduct a physical examination to look for typical depigmented macules and patches. Wood's lamp examination enhances the visibility of hypopigmented areas, especially in early or subtle lesions, while dermoscopy may give further detail on pigmentary changes and borders of lesions. Advanced imaging techniques may help in mapping the affected regions.

When the diagnosis is in doubt, skin biopsy should be considered to confirm the absence of melanocytes and also to rule out other dermatological disorders that may mimic vitiligo.

The VASI is widely utilized for standardized assessment and monitoring. It quantifies the extent of pigment loss in a given body region and overall disease severity. In this way, VASI allows for objective monitoring of disease progression or therapeutic response over time.

Treatment

The main goals of vitiligo treatment are the arrest of melanocyte loss, the induction of repigmentation, and cosmetic and psychosocial improvement. Management is multimodal, with various pharmacological, phototherapeutic, and surgical treatments.

Topical Therapies: Topical corticosteroids, mainly clobetasol, remain the first choice of treatment in localized vitiligo. These drugs are effective in promoting repigmentation but can produce skin atrophy with long-term use[27]. Topical calcineurin inhibitors such as tacrolimus are safer alternatives, especially for sensitive sites such as the face and intertriginous areas[28]. Vitamin D analogues (e.g., calcipotriol) have immunomodulatory effects and are commonly used in conjunction with phototherapy [29]. Studies indicate that vitiligo is associated with VDR gene polymorphisms, significantly Apa-I variant, which can modulate melanocytes. Family history of autoimmune diseases like thyroidopathies, diabetes mellitus, and rheumatoid arthritis suggests that vitamin D may act as a disease-modulating factor in the pathogenesis of vitiligo[30]. Other topical agents include pseudocatalase, which deactivates hydrogen peroxide to cut down oxidative stress[31], 5-fluorouracil, that results in melanocyte activation through a wound-healing process[32], and prostaglandin analogs (e.g., bimatoprost) are known to stimulate melanogenesis locally[33].

Systemic and Novel Pharmacological Therapies

Systemic corticosteroids are mainly given as pulse therapy in rapidly progressive vitiligo to suppress the immune-mediated destruction of melanocytes with minimal adverse effects[34]. Janus kinase inhibitors such as tofacitinib and ruxolitinib constitute novel therapies that modulate autoimmune cytokine activity. Initial studies seem to indicate their potential efficacy for inducing repigmentation [35,36].

Phototherapy: Phototherapy remains a cornerstone of vitiligo management. PUVA, or psoralen plus UVA, was historically the first successful protocol but is limited by phototoxicity, a risk of skin cancer, and contraindications in children and pregnant women [37]. NB-UVB therapy has largely replaced PUVA due to its higher efficacy, broader safety, and fewer adverse effects [38]. Other options include excimer lasers and lamps targeting localized lesions [39].

Surgical Interventions: Surgical options are considered for stable vitiligo unresponsive to medical therapy. [40]

- Punch grafting: works well for small lesions but may leave resultant cobble stoning.
- Suction blister epidermal grafting: ideal for small areas with less scarring.
- Split-thickness grafting: for larger lesions but may cause color mismatch.
- Non-cultured epidermal cell suspension (NCECS): involves the use of melanocyte-keratinocyte mixtures from donor skin. It has succeeded in achieving extensive repigmentation.

Depigmentation Therapy: MBEH has been used in resistant, diffuse vitiligo to render the skin uniformly depigmented and thereby improve cosmetic appearance [41]. In summary, the management of vitiligo is best accomplished by using an individualized, often multimodal approach based on medical, phototherapeutic, and surgical modalities to maximize repigmentation and minimize recurrence.

Table 3: Summary of Modern Treatment Modalities for Vitiligo

Treatment	Mechanism of Action	Indications	Efficacy	Limitations /Side Effects	References
Topical Corticosteroids (Clobetasol)	Anti-inflammatory, immunosuppressive	Localized lesions	Good for small/limited patches	Skin atrophy, telangiectasia, striae	27
Topical Calcineurin Inhibitors (Tacrolimus)	Inhibits T-cell activation	Sensitive areas	Moderate; safer long-term	Mild irritation, burning	28
Vitamin D Analogues (Calcipotriol)	Immunomodulatory, melanocyte support	Adjunct with phototherapy	Moderate, enhances repigmentation	Irritation, limited monotherapy	29, 30
Pseudocatalase	Reduces oxidative stress	Areas with oxidative damage	Limited; adjunct	Limited evidence	31
5-Fluorouracil	Stimulates melanocyte activation	Small localized patches	Variable	Irritation, erythema	32
Prostaglandin Analogs (Bimatoprost)	Stimulates melanogenesis	Eyelashes, small patches	Variable; cosmetic areas	Local irritation	33
Systemic Corticosteroids (Pulse)	Immunosuppressive	Rapidly progressive vitiligo	Effective in controlling spread	Weight gain, glucose intolerance	34
JAK Inhibitors (Tofacitinib, Ruxolitinib)	Cytokine modulation	Resistant or widespread vitiligo	Promising	Cost, long-term safety under study	35,36
PUVA	Photosensitization, melanocyte proliferation	Generalized vitiligo	Moderate	Phototoxicity, nausea, skin cancer risk	37
NB-UVB Phototherapy	Stimulates melanocyte proliferation	Widespread lesions	High efficacy, safe	Multiple sessions needed	38
Excimer Laser / Lamp	Targeted NB-UVB	Localized or stubborn lesions	Moderate; faster repigmentation	Cost, limited area	39
Surgical Grafting (Punch, SBEG, Split-thickness, NCECS)	Transplant melanocytes	Stable, refractory vitiligo	High for localized areas	Scarring, color mismatch	40
Monobenzyl Ether of Hydroquinone (MBEH)	Depigmentation	Recalcitrant diffuse vitiligo	Achieves uniform appearance	Permanent, irreversible	41

Ayurvedic Review

Nidana (Etiology)

The exact etiology (*Nidana*) of *Shvitra* has not been explained; however, the classical Ayurvedic texts acknowledge that its etiological factors greatly resemble those of *Kushtha*, and thus all etiological factors for *Kushtha* may directly or indirectly contribute to the development of *Shvitra*. Etiological factors include moral, behavioral, and dietary components. Moral and behavioral factors are described as *Vachansi Atathyani* (telling lies), *Kritaghna Bhava* (ingratitude), *Sadhunam Ninda* (disrespect toward divine entities), *Guru-Darshanam* (disobedience to teachers), *Papa Kriya* (sinful acts in the present life), and *Purva Kritam Karma* (sins from previous births). [42]

The dietary causes (*Aaharaja Nidana*) include intake of incompatible (*Viruddha*) or unwholesome (*Mithya*) food, excessive consumption of heavy foods, intake of unctuous (*Snigdha*) or liquid (*Drava*) foods beyond normal limits, overeating (*Atibhuktva*), and ingestion of raw or half-cooked food (*Ajirnadhyashana*) as well as specific dietary items like recently harvested cereals (*Navanna*), curd (*Dadhi*), fish (*Matsya*), salt (*Lavana*), sour fruits (*Amla*), black gram (*Masha*), radish (*Mulaka*), starchy foods (*Pishtanna*), sesame (*Tila*), milk (*Kshira*), jaggery (*Guda*), and meat of *Gramya, Aanupa*, and *Jaliya* animals taken with milk (*Payasa*) [43].

Lifestyle factors (*Viharaja Nidana*) include suppression of natural urges *Vega Dharana,* disrespectful behavior toward saints, teachers, Brahmins, or God, sinful actions in present or past life (*Papa Karma*), excessive physical activity in heat (*Ati Santapam*) or after heavy meals (*Ati Bhuktva*), consumption of cold water immediately after exposure to heat (*Shitambhupana*), stress due to fear (*Bhaya*), grief, or exhaustion (*Shrama*), improper administration or complications of *Panchakarma* therapies, excessive sexual activity (*Vyavaya*) during indigestion (*Ajeerne Anna*), and excessive daytime sleep (*Divaswapana*) [44].

Purvarupa (Pre-clinical Symptoms)

Shvitra often presents suddenly without characteristic premonitory features. It sometimes follows general premonitory signs described for *Kushtha*. According to the texts, the preclinical features include evidence of altered or loss of tactile sensation (*Sparsha-Agyatvam*), excessive or absent perspiration with raised papules (*Sweda Asweda*), changes in skin colour (*Vaivarnya*), rash (*Kotha*), horripilation (*Lomharsha*), itching (*Kandu*), physical fatigue (*Shrama*), mental fatigue (*Klama*), pain around lesions (*Vranana Amdhikam Shoolam*), numbness of limbs (*Suptaangata*), and a sensation of burning (*Daah*) [45].

Rupa (Clinical Symptoms)

According to *Charaka*, *Shwitra* is considered a subtype of *Kilasa*. Subclassification is mainly based on predominance of *Doshas* and involvement of specific *Dhatus*. The different subtypes represent various presentations or progressive stages in the evolution of the disease, with later forms being more serious and having poorer prognoses. Most are considered *Asadhya* (incurable), especially when deeper Dhatus are involved. Three types of *Kilasa* are described by *Charaka*: *Daruna* (red, involving *Rakta Dhatu*), *Aruna* (copper-colored, involving *Mamsa Dhatu*), and *Shwitra* (white, involving *Meda Dhatu*). [46]

Sushruta also classified *Kilasa* depending upon predominance of *Doshas* and mentioned that it is located mainly in the skin (*Twakgata*) and is *Aparisravi* (non-secreting) in nature. He preferred usage of *Kilasa* while describing its etiology and pathogenesis (*Nidana* and *Samprapti*) and kept *Shwitra* for describing the treatment. *Sushruta* documented three varieties of *Kilasa* which are - *Vataja* (*Arunam, Mandala, Parusham, Paridhwansi*), *Pittaja* (*Padmapatra-pratika, Daha*) and *Kaphaja* – (*Shweta, Snigdha, Bahalam, Kandu*) [47].

According to *Vagbhata*, *Shwitra* originates from the same causative factors as *Kushtha* but with no discharge (*Aparisravi*). *Shwitra* has been classified into three forms by him: *Vataja* type, which is *Aruna*, affecting *Rakta Dhatu*, characterized by *Rukshata*; *Pittaja* type, which is *Tamra*, affecting *Mamsa Dhatu*, presenting as *Kamala Patravat*, *Daha*, and *Roma Vidhwansi*; and *Kaphaja* type, which is *Shweta*, affecting *Meda Dhatu*, presenting as *Ghana*, *Guru*, and *Kandu* [48].

Kashyapa says that all discoloration of the skin in the direction of white is called *Shwitra*, highlighting that it is simply a discoloration of the skin without secretion [49].

Samprapti (Pathogenesis)

The classical Ayurvedic texts provide limited description of the exact pathogenesis (*Samprapti*) of *Shvitra Roga*, with the exception of the *Hareeta Samhita*. [50]. Based on various Samhitas, the pathogenesis involves disturbances in the Doshas, Dushyas, *Agni*, *Srotas*, and other bodily factors, ultimately manifesting as depigmentation of the skin (*Twak*). The *Samprapti Ghatak* of *Shvitra* can be delineated as a multifactorial pathogenic framework involving several interrelated components. The disorder is characterized by vitiation of *Tridosha*, with a predominance of *Kapha*. The affected *Dushya* include *Rasa*, *Rakta*, *Mamsa*, and *Meda*, reflecting involvement of multiple tissue systems. The pathogenesis is initiated by *Ama*, arising from impaired *Jatharagni* and *Dhatwagni*, resulting in metabolic and tissue-level dysfunction (*Jatharagni Mandya* and *Dhatwagni Mandya*). The disease process involves the channels (*Srotas*)- *Rasavaha*, *Raktavaha*, *Mamsavaha*, and *Medovaha*- where obstruction (*Srotodushti: Sanga*) contributes to the progression of pathology. The primary site of manifestation (*Adhishthana*) is the *Twak*, involving *Rakta*, *Mamsa*, and *Meda*, while the origin of the disease (*Udbhava Sthana*) is localized to the *Amashaya*, affecting the *Twak*. Clinically, the disorder follows an external trajectory (*Roga Marga: Bahya*) and manifests visibly on the body (*Vyaktasthana: Sharira*) as depigmented patches. This integrated understanding of *Dosha, Dushya*, and *Srotas* forms the conceptual basis for therapeutic interventions in Ayurveda. This conceptual framework highlights that the disease originates from impaired *Agni*, obstruction (*Srotodushti*), and imbalance of the

three *Doshas*, leading to disruption of *Dushyas* and culminating in clinical manifestations on the skin.

***Chikitsa* (Management)**

***Nidana Parivarjana*:** The first measure in the treatment of *Shvitra* or *Kilasa* is *Nidana Parivarjana*, or the avoidance of the causative factors. Many classical texts refer to prevention from exposure to etiological factors, which greatly enhances therapeutic results. The basic principle, "Prevention of disease is better than cure", is that the avoidance of dietary and behavioural factors or environmental provocations decreases the chance of recurrence and progression of the disease.

***Deepana and Pachana*:** Therapies form an important part of the management of *Shvitra*. According to Ayurveda, *Ama* or metabolic toxins due to *Mandagni*, which is hypo-functioning of digestive fire, is the primary cause of this disease. Stimulation of *Agni* and elimination of *Ama* are involved in restoring metabolic balance through *Deepana* and *Pachana*. The commonly recommended formulations are *Trikatu Churna, Triphala Churna*, *Hingwashtaka Churna*, *Panchakola Churna*, *Ajamoda Churna*, *Aampachana Vati,* and *Chitrakadi Vati*.

***Shodhana*:** *Shvitra* is best treated with *Shodhana* therapies like *Snehan* (oleation), *Swedana* (fomentation), *Vaman* (therapeutic emesis), and *Virechan* (therapeutic purgation), followed by *Sansran Karma*. *Charaka* recommends *Virechan* as a part of *Sansran Karma* with *Malapee Rasa* with *Guda* (jaggery), followed by *Snehan* and sun exposure according to patient tolerance. *Peya* administration is advised for three consecutive days and local application of *Kwath* prepared from *Kathgullar* bark, *Asana*, *Priyangu*, and *Sauf*, or *Palash Kshar Phanita* for 15 days, is recommended. [51]

Sushruta outlines a comprehensive approach for *Shodhana* according to the *Dhatu* involved. In skin-localized *Kushtha*, *Shodhana* and *Aalepana* are warranted. *Rakta Dhatu* involvement warrants *Shodhana*, *Aalepana*, *Kashaya Pana*, and *Raktamokshana*. *Mamsa Dhatu* is indicated by *Shodhana*, *Lepa*, *Kashaya Pana*, *Raktamokshana*, *Asava - Arishta*, *Mantha*, and *Prash/Avaleh*. In *Meda Dhatu, initial Shodhana* and *Raktamokshana* are followed by *Bhallataka, Shilajeeta, Svarnamakshika, Guggulu, Agaru, Twak, Khadir, Asana,* and *Ayaskruti* therapies. When *Asthi Dhatu* is involved, the disease becomes *Asadhya* or incurable [52].

The additional management principles for *Shvitra* mentioned by *Vagbhata* in the *Shvitra Krimi Rogadhikar* bring forth the role of structured therapeutic interventions. The *Ashtanga Hridaya* advocates the use of *Vaman* fortnightly, *Virechan* every month, *Shiro Virechan* every three days, and *Raktamoikshanas* every six months. [53]

In addition, therapeutic sun exposure is also one of the uniform recommendations in most classical texts as an adjunctive management. The *Brihattriayi* consolidates these principles of management to a more comprehensive approach, including *Langhan Chikitsa* (comprising seven types of *Shamana* and five types of *Shodhana*) Samshodhan, with special emphasis on *Virechan*, *Raktamokshana*, *Kashaya Pana*, which is the internal administration of medicaments, *Aalepana*, the topical application of therapeutic formulation; and regular sun therapy. Thus, this systematic framework underlines the holistic approach with multiple modes adopted in Ayurvedic management of *Shvitra*, targeting both the root cause and symptomatic manifestations of the disease.

***Shamana*:** *Shamana Chikitsa* consists of internal and external therapies, often supplemented with controlled sun exposure in an effort to balance the *Doshas* and encourage repigmentation. The classical texts contain many single and compound formulations that can be used internally as well as externally for *Shvitra* management.

Table 4: Comparative Treatment Approaches for *Shvitra*/Vitiligo

Treatment Category	Ayurvedic Approach	Modern Medical Approach	Notes / Mechanism
Etiological Control	*Nidana Parivarjana* (avoidance of causative factors: *Viruddha Ahara,* excessive Sun exposure, sinful acts)	Lifestyle counseling, trigger avoidance	Both aim to prevent disease progression
Digestive / Metabolic Correction	*Deepana* and *Pachana* (e.g., *Trikatu Churna*, *Triphala Churna*, *Aampachana Vati*)	Nutritional support, vitamin D supplementation	Targets *Ama* and metabolic dysfunction in Ayurveda; oxidative stress and vitamin D pathways in modern medicine
Detoxification	*Shodhana (Vaman, Virechan,*	Systemic corticosteroid	Both aim to modulate

	Raktamokshana, Sansran Karma)	pulse therapy	immune response and reduce inflammation
Immunomodulation & Tissue Rejuvenation	*Shamana* (internal formulations: *Shashilesha Vati*, *Aarogyavardhini Vati*, *Swayambhu Guggulu*; external: *Lepa*, *Taila*, *Kwath*, *Ghrita*, *Rasa*), Sun therapy (*Surya Bhedana*)	Topical corticosteroids, calcineurin inhibitors, JAK inhibitors, phototherapy (NB-UVB, PUVA)	Ayurveda uses herbal/mineral compounds for immune balance and tissue regeneration; modern therapy targets cytokines and melanocyte stimulation
Topical Therapy	*Lepa* (e.g., *Manahshiladi Lepa*, *Shvitrakushthahar Lepa*), *Taila* (*Marichyadi Taila*, *Bhallatakaadi Taila*)	Topical corticosteroids, calcineurin inhibitors, prostaglandin analogs, pseudocatalase	Both aim to induce repigmentation locally
Oral/ Internal Therapy	*Kwath* (*Dhatri Khadir Kwath*), *Vati/Gutika*, *Ghrita*, *Rasa* preparations	Oral corticosteroids, vitamin D analogs, JAK inhibitors	Systemic approach to modulate autoimmunity and melanocyte survival
Phototherapy/Sun Exposure	Sunrays therapy (*Surya Bhedana*)	Narrow-band UVB (NB-UVB), PUVA, excimer laser	Stimulates melanocyte proliferation and repigmentation
Surgical/Procedural Therapy	*Raktamokshana* (bloodletting), *Aalepana* (medicated pastes) on depigmented areas	Punch grafting, suction blister epidermal grafting, split-thickness grafting, non-cultured epidermal cell suspension (NCECS), MBEH depigmentation	Ayurveda uses minimally invasive external procedures; modern therapy uses precise melanocyte transplantation or depigmentation technique

DISCUSSION

Vitiligo is a chronic depigmentation disorder caused by the interplay of genetic, environmental, immunological, and oxidative stress factors. Modern medicine views vitiligo as an autoimmune disease, where epidermal melanocytes are targeted and destroyed by CD8+ T-cells due to structural similarities with other antigens; this results in depigmented white patches. In contrast, Ayurveda describes *Shvitra* as a *Tridosha* imbalance, predominantly *Kapha*-related, giving rise to systemic derangements that affect the skin, blood, muscles, and fat through the *Nadis* or bodily channels. The prevalence of vitiligo is about 0.5-2% worldwide and is considered to be even higher in India. Environmental triggers, trauma, and micronutrient deficiencies-especially vitamin D, B12, and copper-exacerbate the condition. Classifications such as Segmental and Non-Segmental Vitiligo are useful to note the spread and progression of the disease. Nonsegmental vitiligo is symmetrical, bilateral, and progressive, whereas segmental vitiligo is localized and usually stable without any further progression. The Vitiligo Area Scoring Index is now in common use to quantify the extent of the disease and monitor response to treatment.

Ayurvedic texts describe *Shvitra* according to *Dosha* dominance: *Vataja*, which presents as dry and reddish; *Pittaja*, which presents with a burning sensation and has a coppery hue; and *Kaphaja*, which is white, thick, and itchy. According to the Ayurvedic concept of disease progression, temporal features related to the involvement of *Dhatu* are featured. The emphasis of pathogenesis in Ayurveda is on *Mandagni* (weak digestion) and *Ama* (toxins), which correspond with the oxidative stress and immune dysfunction that are recognized in modern practice. Diagnosis in contemporary practice is based on Wood's lamp examination, dermoscopy, and investigations in the laboratory, while Ayurveda relies on detailed clinical observation and assessment of the *Doshas*.

***Virechana*:** *Virechana* (therapeutic purgation) evacuates morbid humors through the inferior channels (*Adhobhaga*), mainly pacifying *Pitta* in the *Pakvasaya*. Formulations used in *Virechana* are those possessing properties like *Ushna* (hot), *Tikshna* (sharp), *Sukshma* (subtle), *Vyavayi* (rapid absorption), and Vikasi (expansive action). The procedure also provides liquefaction of *Doshas* and *Mala*, invasion to microchannels, and resurfacing of *Bhrajaka Pitta* to the

skin, thereby improving the complexion and repigmentation [54].

***Raktamokshana*:** *Raktamokshana*, especially *Siravedha* (venesection), drains the local *Pitta Dosha*, purifies *Rakta Dhatu*, and brings about *Varnaprasadana* (normalization of skin color). The treatment activates melanocyte secretion from the pituitary which helps in melanin production and skin pigmentation restoration.

***Bakuchi* (*Psoralea corylifolia* Linn.):** The ingredient psoralen present in *Bakuchi Beeja Churna* acts by promoting melanogenesis. Local action of *Bakuchi* is described in ancient Ayurvedic texts leading to vasodilation of subcapillary arterioles and stimulation of melanoblasts. Psoralens intercalate with DNA forming adducts in the presence of UVA, which promote repigmentation in hypopigmented lesions. Clinical improvement in pigmentation has been reported with oral psoralen in a dose of 4 mg/g combined with sunlight exposure [55].

***Rasayana*:** The therapy improves tissue regeneration, immunity, and vitality. Longevity, strength, acumen of mind, good utterance of voice, youthfulness, fine complexion, fertility, and good social respect are the accompanying benefits. *Rasayana* herbs act as immunomodulatory and antioxidants by modulating macrophage activity, promoting phagocytosis, natural killer cell activity, and cytotoxic T-lymphocytes. They nullify ROS generation; epitope expression of oxidative damage is repaired, and hence cellular mutations are not allowed to take place. This helps in repigmentation of lesions and overall systemic balance and prevents relapse also [56–58].

An integration of Ayurvedic and modern approaches thus presents a comprehensive, personalized strategy for the management of disease, fusing root-cause elimination, immunomodulation, mitigation of oxidative stress, and local repigmentation therapies. This integrative perspective may improve outcomes, reduce recurrence, and enhance patient quality of life.

CONCLUSION

Vitiligo is a chronic skin disorder with significant medical and psychosocial implications. In modern medicine, vitiligo is conceptualized as a multivariate disease due to genetic predisposition, autoimmune mechanisms, and oxidative stress leading to the destruction of melanocytes. Ayurveda interprets it as a manifestation of the imbalance of *Tridosha* and impaired digestion (*Mandagni*), and its diagnostic and therapeutic approaches are tailored to the constitution and disease stage of the individual. The integration of modern interventions comprising corticosteroids and phototherapy along with Ayurvedic treatments such as *Shodhana*, *Rasayana* therapy, and herbal agents like *Bakuchi*, *Kalajaji*, *Haridra*, *Khadira*, and *Nimba* presents a holistic, personalized approach. These Ayurvedic therapies exert melanogenic, antioxidant, and immunomodulatory actions that facilitate repigmentation and restore systemic balance and emotional well-being, making this an integrative approach that will facilitate long-term management of the disease, reduce recurrence, and address the physical and psychosocial aspects of vitiligo.

Acknowledgement

The authors express their sincere gratitude to the faculty and staff of the Department of Kaumarbhritya and Kayachiktasa for their guidance and support during the preparation of this review. We acknowledge the invaluable contributions of classical Ayurvedic texts and contemporary literature, which have enriched the understanding of *Shvitra* (vitiligo) from both traditional and modern perspectives. Special thanks are extended to colleagues and peers who provided critical insights and suggestions that enhanced the quality of this work.

REFERENCES

1. Alikhan A, Felsten LM, Daly M, Petronic-Rosic V. Vitiligo: a comprehensive overview Part I. Introduction, epidemiology, quality of life, diagnosis, differential diagnosis, associations, histopathology, etiology, and work-up. J Am Acad Dermatol. 2011; 65(3): 473–491.
2. Lerner AB. Vitiligo. J Invest Dermatol. 1959; 32(2 Pt 2): 285–310.
3. Ezzedine K, Lim HW, Suzuki T, Katayama I, Hamzavi I, Lan CC, et al. Revised classification/nomenclature of vitiligo and related issues: Vitiligo Global Issues Consensus Conference. Pigment Cell Melanoma Res. 2012; 25(3): E1–E13.
4. Krüger C, Schallreuter KU. A review of the worldwide prevalence of vitiligo in children, adolescents and adults. Int J Dermatol. 2012; 51(10): 1206–1212.
5. Singh M, Singh G, Kanwar AJ, Belhaj MS. Clinical pattern of vitiligo in Libya. Int J Dermatol. 1985; 24(4): 233–235.
6. Gopal K, Rama Rao GR, Kumar YH, Appa Rao MV, Vasudev P. Vitiligo: a part of a systemic autoimmune process. Indian J Dermatol Venereol Leprol. 2007; 73: 162–165.
7. Howitz J, Brodthagen H, Schwartz M, Thomsen K. Prevalence of vitiligo: epidemiological survey on the Isle of Bornholm, Denmark. Arch Dermatol. 1977; 113(1): 47–52.
8. Boisseau-Garsaud AM, Garsaud P, Calès-Quist D, Hélénon R, Quénéhervé C, Claire RC. Epidemiology of vitiligo in the French West Indies (Isle of Martinique). Int J Dermatol. 2000; 39(1): 18–20.

9. Mattoo SK, Handa S, Kaur I, Gupta N, Malhotra R. Psychiatric morbidity in vitiligo: prevalence and correlates in India. J Eur Acad Dermatol Venereol. 2002; 16(6): 573–578.
10. Sharma PV, editor. Charaka Saṃhitā: Text with English Translation. Vol I–IV. Varanasi: Chaukhambha Orientalia; 2014.
11. Murthy KRS, Sushruta Saṃhita: Text with English Translation. Vol I–III. Varanasi: Chaukhambha Orientalia; 2012.
12. Raghunathacharya T, Śabdakalpadruma. Vol II. Varanasi: Chowkhamba Sanskrit Series Office; 1967.
13. Apte VS, Amarakośa with English Translation. Delhi: Motilal Banarsidass; 2000.
14. Sharma PV, Kaśyapa Saṃhita (Vṛddha Jīvakīya Tantra). Varanasi: Chaukhambha Visvabharati; 2008.
15. Dash B, editor. Materia Medica of Ayurveda. Varanasi: Chowkhamba Sanskrit Series Office; 2015.
16. Tripathi B, editor. Ashtaṅga Hṛidaya of Vāgbhaṭa with English Translation. Varanasi: Chaukhambha Sanskrit Pratishthan; 2013.
17. Spritz RA. The genetics of vitiligo. J Invest Dermatol. 2011; 131(E1): E18–E20.
18. Jin Y, Birlea SA, Fain PR, Ferrara TM, Ben S, Riccardi SL, et al. Genome-wide association analyses identify 13 new susceptibility loci for generalized vitiligo. Nat Genet. 2012; 44(6): 676–680.
19. Rashighi M, Harris JE. Interfering with the IFN-γ/CXCL10 pathway to develop new targeted treatments for vitiligo. Ann Transl Med. 2015; 3(21): 343.
20. Richmond JM, Strassner JP, Zapata L Jr, Garg M, Riding RL, Refat MA, et al. Antibody blockade of IL-15 signaling has the potential to durably reverse vitiligo. Sci Transl Med. 2018; 10(450): eaam7710.
21. Laddha NC, Dwivedi M, Mansuri MS, Gani AR, Ansarullah M, Ramachandran AV, et al. Vitiligo: interplay between oxidative stress and immune system. Exp Dermatol. 2013; 22(4): 245–250.
22. Yu R, Broady R, Huang Y, Wang Y, Yu J, Gao M, et al. Transcriptome analysis reveals markers of aberrantly activated innate immunity in vitiligo lesional and non-lesional skin. PLoS One. 2012; 7(12): e51040.
23. Tanwar S, Thakur V, Bhatia A, et al. Catecholamines' accumulation and disturbed metabolism at perilesional site: a possible cause of vitiligo progression. Arch Dermatol Res. 2023; 315: 173–180.
24. Laddha NC, Dwivedi M, Mansuri MS, Singh M, Patel HH, Agarwal N, et al. Association of NPY and IL1B genetic variants with vitiligo susceptibility. PLoS One. 2014; 9(9): e107020.
25. Alkhateeb A, Fain PR, Thody A, Bennett DC, Spritz RA. Epidemiology of vitiligo and associated autoimmune diseases. Pigment Cell Res. 2003; 16(3): 208–214.
26. Xuan Y, Yang Y, Xiang L, Zhang C. The role of oxidative stress in the pathogenesis of vitiligo: a culprit for melanocyte death. Oxid Med Cell Longev. 2022: 8498472.
27. Whitton ME, Pinart M, Batchelor J, Leonardi-Bee J, González U, Vander H, et al. Interventions for vitiligo. Cochrane Database Syst Rev. 2015; 2: CD003263.
28. Sarkar R, Dogra S, Vinay K, Sinha S, Narayan VR, Kumaran MS, et al. Topical tacrolimus in vitiligo: consensus paper. Clin Cosmet Investig Dermatol. 2024; 17: 2875–2886.
29. Khullar G, Kanwar AJ, Singh S, Parsad D. Calcipotriol with NB-UVB vs NB-UVB alone in vitiligo. J Eur Acad Dermatol Venereol. 2015; 29(5): 925–932.
30. Zhang JZ, Wang M, Ding Y, Gao F, Feng YY, Yakeya B, et al. Vitamin D receptor polymorphism and vitiligo: meta-analysis. Medicine (Baltimore). 2018; 97(29): e11506.
31. Schallreuter KU, Wood JM, Lemke KR, Levenig C. Pseudocatalase and UVB therapy in vitiligo. Dermatology. 1995; 190(3): 223–229.
32. Abdelwahab M, Salah M, Samy N, Rabie A, Farrag A. Topical 5-fluorouracil with Er:YAG laser in vitiligo. Clin Cosmet Investig Dermatol. 2020; 13: 77–85.
33. Biswas K, Kaliyadan F. Treatment of vitiligo-associated leucotrichia. Indian Dermatol Online J. 2025; 16: 220–226.
34. Banerjee K, Barbhuiya JN, Ghosh AP, Dey SK, Karmakar PR. Low-dose oral corticosteroids in vitiligo. Indian J Dermatol Venereol Leprol. 2003; 69(2): 135–137.
35. Rosmarin D, Pandya AG, Lebwohl M, Grimes P, Hamzavi I, Gottlieb AB, et al. Ruxolitinib cream for vitiligo. Lancet. 2020; 396(10244): 110–120.
36. Liu LY, Strassner JP, Refat MA, Harris JE, King BA. Tofacitinib and light exposure in vitiligo. J Am Acad Dermatol. 2017; 77(4): 675–682.e1.
37. Westerhof W, Schallreuter KU. PUVA for vitiligo and skin cancer. Clin Exp Dermatol. 1997; 22(1): 54.
38. Khanna U, Khandpur S. Advances in NB-UVB therapy for vitiligo. Indian Dermatol Online J. 2019; 10(3): 234–243.

39. Bibi K. Le vitiligo: physiopathologies associées et traitements. Thesis. France; 2008.
40. Falabella R. Surgical therapies for vitiligo. Clin Dermatol. 1997; 15(6): 927–939.
41. Grimes PE, Nashawati R. Depigmentation therapies for vitiligo. Dermatol Clin. 2017; 35(2): 219–227.
42. Shastri K. Charaka Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 7/177. Varanasi: Chaukhambha Sanskrit Sansthan; 2018
43. Shastri K. Charaka Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 7/4. Varanasi: Chaukhambha Sanskrit Sansthan; 2018.
44. Shastri K. Charaka Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 7/6. Varanasi: Chaukhambha Sanskrit Sansthan; 2018.
45. Shastri K. Charaka Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 7/12. Varanasi: Chaukhambha Sanskrit Sansthan; 2018.
46. Shastri K. Charaka Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 7/173. Varanasi: Chaukhambha Sanskrit Sansthan; 2018.
47. Shastri AD. Sushruta Samhita, Nidana Sthana, Kushtha Nidana Adhyaya 5/17. Varanasi: Chaukhambha Sanskrit Sansthan; 2019.
48. Tripathi B. Vagbhata Samhita, Nidana Sthana, Kushtha Shvitra Krimi Adhyaya 14/37–38. Varanasi: Chaukhambha Sanskrit Sansthan; 2022.
49. Vriddha Jivaka Acharya. Kasyapa Samhita, Chikitsa Sthana, Kushtha Adhyaya 9/2. Varanasi: Chaukhambha Visvabharati; 2002.
50. Pandey J. Hareeta Samhita, Tritiya Sthana 39/50–51. Varanasi: Chaukhambha Bharti Academy; 2016.
51. Shastri K. Charaka Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 7/162–165. Varanasi: Chaukhambha Sanskrit Sansthan; 2018.
52. Shastri AD. Sushruta Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 9/6. Varanasi: Chaukhambha Sanskrit Sansthan; 2019.
53. Tripathi B. Vagbhata Samhita, Chikitsa Sthana, Kushtha Chikitsa Adhyaya 19/96. Varanasi: Chaukhambha Sanskrit Sansthan; 2022.
54. Shastri K. Charaka Samhita, Kalpa Sthana, Madana Kalpa Adhyaya 1/5. Varanasi: Chaukhambha Sanskrit Sansthan; 2018.
55. Pathak MA, Fitzpatrick TB. Evolution of photochemotherapy with psoralens and UVA. J Photochem Photobiol B. 1992; 14(1–2): 3–22.
56. An overview of immunomodulatory activity of Rasayana. Int J Ayurveda Pharma Res. 2024; 12(8): 38–42.
57. Tiwari N. Globalization of Rasayana as antioxidants in ageing. J Ayurveda Holist Med. 2021; 5(4).
58. Shalini D. Effect and action of Rasayana in autoimmune disorders. J ReAttach Ther Dev Divers. 2023; 6(1): 1703–1709

Cite this article as:
Chandraprabha Sharma, Harish Kumar Singhal, Manisha Goyal. Bridging Classical Ayurveda and Modern Medicine in the Understanding of Shvitra (Vitiligo). International Journal of Ayurveda and Pharma Research. 2026;14(3):183-193.
https://doi.org/10.47070/ijapr.v14i3.4066
Source of support: Nil, Conflict of interest: None Declared

***Address for correspondence**
Dr. Chandraprabha Sharma
PG Scholar,
PG Department of Kaumarbhritya,
Postgraduate Institute of Ayurveda,
Dr. S.R. Rajasthan Ayurved University,
Jodhpur, Rajasthan.
Email: chandraprabha938@gmail.com