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SHWITRA (VITILIGO): AN INTEGRATIVE REVIEW OF *AYURVEDIC* AND CONTEMPORARY PERSPECTIVES

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ABSTRACT

Shwitra is described in classical *Ayurvedic* literature as a type of *Kushta Roga* characterized by depigmented patches over the skin due to vitiation of *Doshas* and impairment of *Dhatus*, particularly *Rakta*, *Mamsa*, and *Meda*. Clinically, it resembles vitiligo, an acquired depigmenting disorder marked by progressive melanocyte destruction. Vitiligo affects approximately 0.5–2% of the global population and significantly impacts psychological well-being and quality of life. Contemporary medicine attributes its pathogenesis primarily to autoimmune mechanisms, oxidative stress, genetic predisposition, and neurohumoral factors. *Ayurveda* explains *Shwitra* through *Tridoshic* imbalance with predominance of *Pitta* and *Kapha*, along with involvement of *Bhrajaka Pitta* and *Rakta Dushti*. Classical texts such as the *Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya* elaborate its *Nidana*, *Samprapti*, *Lakshana*, and *Chikitsa* principles. This review critically analyzes the etiopathogenesis, clinical features, and therapeutic approaches of *Shwitra* from both

Ayurvedic and modern biomedical perspectives, highlighting possible integrative mechanisms and future research directions.

KEYWORDS: *Shwitra*, Vitiligo, *Kushta*, Autoimmune disorder, *Ayurveda*, Depigmentation.

INTRODUCTION

Skin disorders have profound psychosocial and cosmetic implications. Among them, vitiligo is a chronic acquired depigmenting disorder characterized by well-defined milky-white macules resulting from selective melanocyte destruction. Globally, vitiligo affects 0.5–2% of the population irrespective of gender or ethnicity. The disease often manifests before 30 years of age and is associated with autoimmune conditions such as thyroid disorders and diabetes mellitus.[1,2]

In *Ayurveda*, *Shwitra* is categorized under *Kushta Roga*. Although all *Kushtas* involve *Tridosha*, *Shwitra* is primarily associated with vitiation of *Pitta* and *Kapha* Dosha along with *Rakta Dushti*. The *Charaka Samhita* describes *Shwitra* as non-exudative, non-elevated white patches of skin occurring due to incompatible dietary habits (*Viruddha Ahara*), suppression of natural urges, and sinful acts (*Papa Karma*).[3] The *Sushruta Samhita* differentiates *Shwitra* based on involvement of *Dhatus—Rakta*, *Mamsa*, and *Meda*—resulting in varying discoloration patterns.[4] The *Ashtanga Hridaya* further elaborates therapeutic approaches including *Shodhana* (purificatory therapies) and *Shamana* (pacificatory measures).[5]

Modern biomedical understanding suggests multifactorial etiology including autoimmune cytotoxicity against melanocytes, oxidative stress-mediated apoptosis, neural mechanisms, and genetic susceptibility.[6,7] Histopathological studies confirm absence or reduction of functional melanocytes in affected areas.[8] Standard management includes topical corticosteroids, calcineurin inhibitors, phototherapy, and surgical grafting techniques; however, recurrence and incomplete repigmentation remain major challenges.[9]

Ayurveda emphasizes individualized management through *Nidana Parivarjana*, *Deepana-Pachana*, *Shodhana* procedures such as *Vamana* and *Virechana*, and administration of photosensitizing herbal drugs like *Bakuchi* (Psoralea corylifolia). Emerging studies suggest that certain *Ayurvedic* formulations may exert immunomodulatory and antioxidant effects, which align with contemporary pathogenic hypotheses.[10]

Given the chronic nature of vitiligo and limitations of current therapies, there is increasing interest in integrative approaches. This review aims to critically analyze classical descriptions of *Shwitra* and correlate them with contemporary biomedical insights to propose potential integrative therapeutic frameworks.

PATHOGENESIS

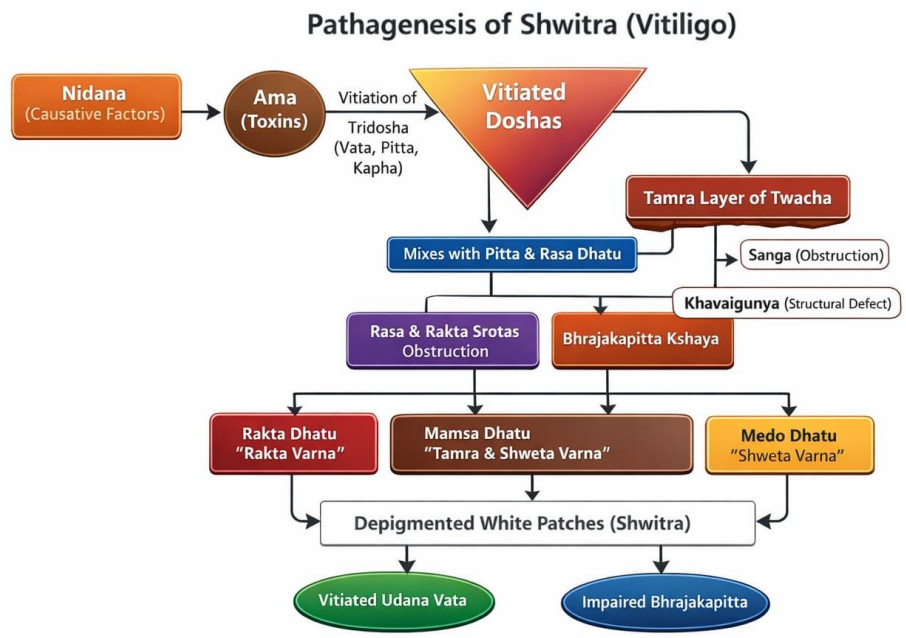
Vitiligo is an acquired depigmentary disorder in which functional melanocytes are progressively lost from the epidermis, leading to well-demarcated white macules. Its pathogenesis is multifactorial and involves a complex interplay between genetic predisposition, oxidative imbalance, and immune dysregulation. Excess production of reactive oxygen species within melanocytes creates cellular stress, impairing mitochondrial function and triggering the release of inflammatory mediators. These signals activate the innate immune system and subsequently promote a melanocyte-specific adaptive immune response, predominantly mediated by CD8+ cytotoxic T lymphocytes. Key cytokines, particularly interferon-gamma, enhance immune activation through pathways such as JAK-STAT signaling, resulting in targeted melanocyte destruction. In addition, defective melanocyte adhesion and neural influences may contribute to their detachment and loss. Together, these mechanisms culminate in selective melanocyte depletion and the clinical appearance of depigmented patches seen in vitiligo.[11-13]

Table: Probable Pathogenetic Hypotheses in Vitiligo (with Type Correlation).[11,18]

Hypothesis/ Mechanism	Description	Types of Vitiligo Most Linked	Key Evidence / Notes
Autoimmune Hypothesis	Autoimmune targeting of melanocytes via CD8+ T cells and autoantibodies.	Non-Segmental Vitiligo (NSV) predominantly	Strong association with autoimmune diseases; IFN-gamma mediated cytotoxicity.
Oxidative Stress Hypothesis	Accumulation of reactive oxygen species leading to melanocyte apoptosis.	NSV; possible trigger in SV	Increased H2O2 levels; decreased antioxidant enzymes in lesional skin.
Neural Hypothesis	Neurochemical mediators from nerve endings disrupt melanocyte function.	Segmental Vitiligo (SV)	Explains unilateral distribution; limited direct evidence.
Genetic Susceptibility	Polygenic predisposition	Mainly NSV	GWAS studies show immune-related

Theory	involving immune-regulatory genes.		susceptibility loci.
Melano-cytorrhagy (Adhesion Defect)	Defective melanocyte adhesion leading to mechanical detachment.	Acral / friction-prone NSV	Reduced E-cadherin expression; Koebner phenomenon correlation.
Intrinsic Melanocyte Defect	Melanocytes have inherent vulnerability to stress and apoptosis.	Both NSV and SV	Altered mitochondrial function observed in vitro.
Somatic Mosaicism	Localized genetic mosaicism causing selective melanocyte targeting.	Segmental Vitiligo (SV)	Explains stable unilateral pattern.
Microvascular Theory	Altered local blood flow facilitates immune infiltration.	Proposed in SV	Increased lesional blood flow reported in some studies.
Biochemical / Metabolic Toxicity	Toxic melanin intermediates accumulate and damage melanocytes.	NSV	Impaired detoxification pathways contribute to oxidative stress.
Environmental & Stress Triggers	Physical trauma, chemicals, sunburn, psychological stress initiate lesions.	All types	Koebner phenomenon; catecholamine-mediated immune modulation.

AYURVEDA PATHOGENESIS OF SHWITRA



Classification of Vitiligo

Vitiligo is a chronic acquired depigmenting disorder characterized by the selective destruction of melanocytes, resulting in well-demarcated depigmented macules and patches. Over time, several classification systems have been proposed based on clinical morphology, distribution, and disease behaviour. The currently accepted classification is based on consensus recommendations of the Vitiligo Global Issues Consensus Conference (VGICC), which categorizes vitiligo into segmental, non-segmental, and unclassified forms.[19]

Segmental Vitiligo (SV)

Segmental vitiligo is characterized by unilateral depigmented macules distributed in a dermatomal or quasi-dermatomal pattern. It typically has an early onset, often in childhood, and demonstrates rapid progression followed by early stabilization within one to two years.[20] Unlike non-segmental vitiligo, SV shows limited association with systemic autoimmune diseases. Leukotrichia is more frequently observed in this type, and repigmentation with medical therapy is often less satisfactory compared to non-segmental forms.[21]

Recent pathogenetic theories suggest that SV may be associated with somatic mosaicism, where genetically altered melanocyte populations are selectively targeted by immune responses.[22] Although autoimmune mechanisms may contribute, the localized nature and clinical behavior distinguish it from non-segmental variants.

Non-Segmental Vitiligo (NSV)

Non-segmental vitiligo is the most common clinical type, accounting for approximately 80–90% of cases.[23] It is characterized by bilateral, symmetrical depigmented patches and typically follows a chronic, progressive course with periods of activity and remission.

NSV includes several subtypes

- **Generalized vitiligo** – the most prevalent subtype, involving widespread macules on trunk and extremities.
- **Acrofacial vitiligo** – primarily affecting distal extremities and facial areas.
- **Mucosal vitiligo** – limited to mucous membranes.
- **Universal vitiligo** – involving nearly complete depigmentation (>80–90% body surface area).
- **Mixed vitiligo** – coexistence of segmental and non-segmental patterns.[24]

NSV is strongly associated with autoimmune disorders such as autoimmune thyroid disease, type 1 diabetes mellitus, and alopecia areata.[25] Immunologically, it is characterized by CD8+ cytotoxic T-cell infiltration targeting melanocyte-specific antigens, elevated interferon-gamma levels, and dysregulation of immune tolerance mechanisms.[26] Genetic studies have identified multiple susceptibility loci related to immune regulation and melanocyte biology.[27]

Unclassified or Undetermined Vitiligo

Certain cases do not clearly fit into segmental or non-segmental categories. These include focal vitiligo (localized, non-progressive lesions without clear segmental distribution) and isolated mucosal vitiligo. Early-stage lesions may also remain indeterminate until progression clarifies their pattern.[19]

Evolution of Classification Systems

Earlier classification systems categorized vitiligo into localized and generalized types. However, inconsistencies in terminology and overlap between patterns led to the need for a standardized system. The VGICC classification improved clinical uniformity, particularly for research purposes and therapeutic trials.[19-23]

Clinical Importance of Classification

Accurate classification is clinically significant because disease behavior, prognosis, therapeutic response, and autoimmune associations differ between types. Segmental vitiligo tends to stabilize early and may respond better to surgical grafting procedures, whereas non-segmental vitiligo often requires long-term immunomodulatory therapies such as topical corticosteroids, calcineurin inhibitors, and phototherapy.[24-26]

Thus, the contemporary classification system reflects both clinical morphology and underlying pathogenetic differences, aiding in better disease stratification and management.

Comparative Chart: Differential Diagnosis of White Patches.[28-39]

Condition	Etiology	Nature of Pigment Loss	Borders	Scaling	Distribution	Wood's Lamp	Histopathology	Distinguishing Features
Vitiligo	Autoimmune melanocyte destruction	Complete depigmentation	Well-defined	Absent	Symmetrical (NSV) / unilateral (SV)	Bright chalky white fluorescence	Absence of melanocytes	Associated with autoimmune diseases, Koebner phenomenon
Pityriasis Versicolor	Malassezia fungal infection	Hypopigmented or hyperpigmented	Ill-defined	Fine scaling present	Trunk, shoulders	Yellowish-green fluorescence	Fungal hyphae ('spaghetti & meatballs')	Positive KOH test
Pityriasis Alba	Atopic dermatitis related	Mild hypopigmentation	Ill-defined	Mild scaling	Face (children)	Minimal accentuation	Mild spongiosis	Common in children; history of atopy
Tinea Corporis	Dermatophyte infection	Hypopigmented center	Raised erythematous border	Prominent scaling	Exposed areas	Variable	Fungal elements	Annular lesion with active border
Nevus Depigmentosus	Congenital melanocyte defect	Stable hypopigmentation	Irregular but stable	Absent	Localized	Off-white accentuation	Reduced melanin, normal melanocyte number	Present since birth, non-progressive
Leprosy (Indeterminate/Tuberculoid)	Mycobacterium leprae	Hypopigmented	Ill-defined	Absent	Localized	No enhancement	Granulomatous inflammation	Sensory loss over lesion
Post-inflammatory Hypopigmentation	Secondary to dermatitis/trauma	Partial pigment loss	Irregular	May be present	Site of previous inflammation	Mild accentuation	Decreased melanin	History of prior inflammation
Idiopathic Guttate Hypomelanosis	Age-related melanocyte loss	Small depigmented macules	Well-defined	Absent	Sun-exposed areas	Bright white	Reduced melanocytes	Elderly patients
Albinism	Genetic tyrosinase deficiency	Generalized depigmentation	Diffuse	Absent	Generalized	Diffuse enhance	Reduced/absent melanin	Congenital, ocular involvement

						ment		
Chemical Leukoderma	Contact chemical melanocyte toxicity	Depigmented	Confined to contact areas	Absent	Exposure sites	Similar to vitiligo	Melanocyte destruction	History of phenolic exposure
Lichen Sclerosus	Autoimmune/inflammatory	Porcelain-white plaque	Well-defined	Atrophic	Genital > extragenital	No fluorescence	Epidermal thinning, sclerosis	Atrophic shiny surface

Comparative Diagnosis: Vitiligo and Other White Patch Disorders with *Ayurvedic* Correlation

In modern dermatology, vitiligo is an acquired depigmenting disorder characterized by melanocyte destruction. In *Ayurveda*, depigmented and hypopigmented lesions are primarily described under *Kushta Roga*, especially *Shwitra* and *Kilasa*.[40-41] Proper differential diagnosis is essential because *Ayurvedic* entities are classified based on *Dosha* predominance, *Dhatu* involvement, color, texture, and associated symptoms.

Modern and *Ayurvedic* Diagnostic Correlation

Modern Diagnosis	***Ayurvedic Correlation***	***Dosha/Dhatu Involvement***	**Distinguishing Ayurvedic Feature**
Vitiligo	*Shwitra*	*Tridosha (Pitta-Kapha), Rakta Dushti*	Non-exudative white patches
Pityriasis Versicolor	*Kaphaja Kushta*	*Kapha* predominance	Scaling, mild itching
Tinea Corporis	*Dadru Kushta*	*Kapha-Pitta*	Circular itching lesions
Leprosy	*Mahakushta*	*Tridosha* with *Supti*	Loss of sensation
Nevus Depigmentosus	*Janmabala Pravrutta Vyadhi*	Congenital defect	Present since birth
Post-inflammatory Hypopigmentation	*Pittaja Twak Vikara*	*Pitta* involvement	After inflammation
Idiopathic Guttate Hypomelanosis	*Vataja Kushta*	*Vata* predominance	Dry small lesions in elderly
Chemical Leukoderma	*Agantuja* leading *to Shwitra*	External *Dosha* vitiation	Chemical exposure history

Theoretical Correlation

1. *Shwitra* vs Vitiligo

Shwitra is described in the *Charaka Samhita* under *Kushta Chikitsa Adhyaya* as a non-exudative white patch disorder caused by consumption of incompatible foods (*Viruddha Ahara*), suppression of natural urges, and moral transgressions. The *Sushruta Samhita* further categorizes *Shwitra* based on Dhatu involvement

Rakta (reddish), *Mamsa* (coppery), *Meda* (white).

This closely resembles progressive melanocyte destruction seen in vitiligo.

2. Kilasa

Kilasa is sometimes described interchangeably with *Shwitra* but often refers to more superficial discoloration without deeper *Dhatu* involvement.[42] Some scholars interpret *Kilasa* as milder hypopigmentation compared to *Shwitra*.

3. *Kushta* Spectrum

Ayurveda broadly categorizes skin disorders into *Mahakushta* and *Kshudra Kushta*. Conditions like *Dadru* (fungal infections) and other hypopigmented lesions are differentiated based on itching, discharge, pain, scaling, and Dosha predominance.

4. Importance of Sensory Examination

Modern leprosy differentiation parallels *Ayurvedic* emphasis on *Supti* (loss of sensation), which is absent in *Shwitra* but present in certain *Kushta* types.[43]

Comparative Diagnostic Principles of *Shwitra* (Vitiligo): Modern Dermatology vs Ayurveda.[40-45]

Aspect	Modern Dermatology	Ayurveda
Etiology	Autoimmune melanocyte destruction; oxidative stress; genetic susceptibility	*Tridosha* imbalance *(*predominantly *Pitta–Vata); Rakta Dushti; Agnimandya; Nidana Sevana*
Pathogenesis	Loss or dysfunction of melanocytes leading to depigmentation	*Dosha–Dushya Sammurchana; Bhrajakapitta Kshaya; Rakta, Mamsa* and *Meda Dhatu Dushti; Srotorodha*
Clinical Examination	Wood's lamp examination; Skin biopsy; KOH (differential diagnosis)	*Darshana (*inspection*); Sparshana (*palpation*); Prashna (*history*); Dosha–Dhatu* assessment
Classification	Segmental Vitiligo (SV); Non-segmental Vitiligo (NSV)	*Mahakushta; Kshudra Kushta; Dosha*-based variants
Investigations	Autoimmune markers; Thyroid profile; Histopathology	Primarily clinical*;* systemic assessment *of Agni*, *Dosha*, and *Dhatu* involvement
Disease Nature	Chronic, autoimmune, relapsing	*Krichra Sadhya; Twak-gata* with systemic *Doshic* origin
Prognostic Factors	Early onset, rapid spread, mucosal involvement	*Chronicity; Dosha* predominance*; Dhatu* depth*;* response *to Shodhana*

DISCUSSION

Shwitra, described under *Kushta* in classical *Ayurvedic* literature, is characterized by *Twak Vaivarnya* without *Srava* and comparatively limited systemic manifestations. Although classified under *Kushta*, classical *Acharyas* distinctly recognize its localized cutaneous presentation and *Krichra Sadhyata*. Clinically, *Shwitra* corresponds to Vitiligo, a chronic depigmenting disorder characterized by selective melanocyte destruction. While modern dermatology attributes vitiligo primarily to autoimmune mechanisms, oxidative stress, and

genetic predisposition, *Ayurveda* conceptualizes *Shwitra* as a consequence of *Dosha* imbalance, *Rakta Dushti*, and *Dhatu*-level pathology.

In ***Charaka Samhita (Chikitsa Sthana** 7), Shwitra* is managed within the broader framework of *Kushta Chikitsa*. *Charaka* advocates *Nidana Parivarjana* as the foundational step, followed by *Deepana–Pachana* to eliminate *Ama* and restore *Agni*. This is succeeded by *Snehana* and *Swedana* as *Purva Karma* and *Virechana* as the principal *Shodhana* therapy, particularly in *Pitta*-dominant conditions. *Charaka's* repeated emphasis on *Shodhana* in chronic *Kushta* reflects an understanding that systemic *Dosha* elimination is essential before attempting *Dhatu* restoration. The subsequent administration of *Rasayana* suggests a strategy aimed at tissue regeneration and immune stabilization. From a contemporary perspective, this sequence parallels systemic immunomodulatory intervention prior to localized therapeutic stimulation.[46]

Sushruta Samhita (Chikitsa Sthana 9) presents a more procedure-oriented therapeutic strategy. *Sushruta* recommends *Vamana* and *Virechana* according to *Dosha* predominance and specifically includes *Raktamokshana* in cases involving *Rakta Dushti*. Importantly, *Sushruta* restricts *Shwitra* pathology to *Twak* level and differentiates it from other *Kushta* types by the absence of discharge, indicating careful clinical observation. The inclusion of *Raktamokshana* signifies recognition of vascular and *Rakta* involvement, which may be interpreted in the context of inflammatory and immune-mediated processes implicated in vitiligo pathogenesis. Thus, *Sushruta's* model appears to address both systemic and microcirculatory components of the disorder.[47]

In ***Ashtanga Hridaya (Chikitsa Sthana*** 19), *Acharya Vagbhata* consolidates earlier doctrines and emphasizes correction of *Agni* prior to any *Shamana* therapy. *Vagbhata* reiterates the importance of *Shodhana* but presents a more concise therapeutic algorithm integrating internal purification, external applications, and strict *Pathya* adherence. His stress on metabolic correction underscores the concept that *Dhatu* nourishment and pigmentation restoration are dependent on optimal digestive and metabolic function. This aligns with modern insights linking oxidative stress and metabolic dysregulation with melanocyte vulnerability.[48]

Chakrapani Datta, through his *Ayurveda Dipika* commentary on *Charaka*, provides interpretative clarity on *Dosha–Dushya Sammurchana* in *Kushta* and rationalizes the

necessity of *Shodhana* preceding *Rasayana* therapy. He indicates that inadequate elimination of vitiated *Doshas* predisposes to recurrence, indirectly acknowledging the chronic and relapsing nature of *Shwitra*. This commentary reflects a sophisticated appreciation of disease persistence and the need for comprehensive systemic correction.[49]

A more pharmacologically detailed exposition is observed in ***Bhavaprakasha***, wherein specific *Ekal Dravyas* such as *Bakuchi, Bhallataka, and Khadira* are described with *Kushtaghna, Raktashodhaka,* and *Varnya* properties. These drugs are intended to restore normal *Varna* following systemic purification. *Bakuchi*, traditionally indicated in *Shwitra*, contains psoralen compounds that are known to stimulate melanogenesis when activated by ultraviolet exposure. This offers a plausible bridge between classical *Varnya karma* and contemporary photochemotherapy principles. *Bhavaprakasha* thus represents a shift toward pharmacotherapeutic specificity while preserving the foundational *Shodhana* approach.[50]

Yogaratnakara further synthesizes earlier teachings and provides compound formulations and Lepa preparations for *Kushta* including *Shwitra*. The integrative approach combining systemic detoxification, internal medication, external application, and dietary regulation reflect a multidimensional therapeutic model. Such multimodal management corresponds with current integrative strategies that combine systemic immune modulation with local pigment-inducing therapies.[51]

Collectively, classical *Ayurvedic* management of *Shwitra* demonstrates a structured and hierarchical therapeutic model beginning with systemic purification, followed by metabolic correction, tissue-specific pharmacotherapy, and *Rasayana* support. Early *Samhitas* emphasize *Dosha* elimination and *Rakta* correction, whereas later texts elaborate drug-specific pigmentation strategies. When viewed through a modern lens, the concepts of *Bhrajakapitta Kshaya, Rakta Dushti,* and *Srotorodha* may be interpreted in relation to melanocyte dysfunction, immune dysregulation, and oxidative stress. Therefore, classical *Ayurvedic* therapeutics offer a coherent immunometabolic framework that may provide complementary insights into the management of vitiligo.

CONCLUSION

Shwitra, described under *Kushta* in classical *Ayurvedic* literature, represents a distinct pigmentary disorder characterized by *Twak Vaivarnya* without *Srava* and comparatively localized pathology. Although grouped under *Kushta*, classical *Acharyas* consistently

emphasized its unique clinical behavior and therapeutic considerations. The systematic approach outlined in the *Samhitas* demonstrates a hierarchical management strategy beginning with *Nidana Parivarjana*, followed by *Shodhana* for systemic *Dosha* elimination, subsequent *Shamana* with *Kushtaghna* and *Varnya* drugs, and long-term *Rasayana* support for *Dhatu* restoration.

The comparative evaluation of classical texts reveals conceptual continuity across *Acharyas*, with early *Samhitas* focusing primarily on systemic purification and *Rakta* correction, while later treatises elaborated drug-specific pharmacotherapeutic interventions such as *Bakuchi*-based formulations. The consistent emphasis on *Agni* correction, *Dosha–Dushya Sammurchana*, and *Bhrajakapitta Kshaya* indicates that *Shwitra* was understood as a disorder of systemic origin manifesting at the cutaneous level.

When interpreted in light of contemporary understanding of Vitiligo, classical concepts such as *Rakta Dushti*, *Srotorodha*, and *Dhatu Kshaya* may be correlated with immune dysregulation, oxidative stress, and melanocyte dysfunction. Thus, *Ayurvedic* therapeutics present a coherent immunometabolic framework integrating systemic detoxification, metabolic correction, tissue-specific pharmacology, and rejuvenation therapy.

In conclusion, the classical *Ayurvedic* approach to *Shwitra* offers a multidimensional and logically structured management paradigm that remains conceptually relevant in the era of integrative dermatology. Future well-designed clinical trials and translational research are warranted to validate and refine these traditional therapeutic strategies within evidence-based frameworks.

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