WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 9, 1354-1369. Review Article ISSN 2277–7105

UNDERSTANDING SWITRA (VITILIGO) THROUGH AYURVEDIC CLASSICS AND MODERN DERMATOLOGY: A SYSTEMATIC REVIEW

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Article Received on 05 April 2026,
Article Revised on 25 April 2026,
Article Published on 01 May 2026,

https://doi.org/10.5281/zenodo.20024404

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How to cite this Article: *[1]Dr. Aman Kumar, [2]Dr. Shyamanta Kalita, [3]Dr. Niten Barman. (2026). Understanding switra (vitiligo) through ayurvedic classics and modern dermatology: a systematic review. World Journal of Pharmaceutical Research, 15(9), 1354–1369.

1. ABSTRACT

Switra, a subtype of Kushta described in classical Ayurvedic literature, is a chronic pigmentary disorder characterized by hypopigmented or depigmented macules. It is conceptualized as a Tridoshaja Vyadhi with a predominance of Pitta Dosha, particularly involving Bhrajaka Pitta, which governs cutaneous pigmentation and metabolic transformation. The vitiation of Doshas leads to pathological involvement of Twak, Rakta, Mamsa, and Lasika, resulting in impaired melanogenesis and visible depigmentation. Ayurvedic texts emphasize multifactorial etiopathogenesis, including Viruddha Ahara (incompatible dietary regimens), Mithya Ahara-Vihara (improper diet and lifestyle), and Manasika Nidana (psychological stressors), indicating a complex psychosomatic interplay. In contemporary dermatology, Switra is closely correlated with Vitiligo, an acquired depigmentary disorder characterized by selective destruction of melanocytes, leading to well-demarcated achromic macules. The global prevalence ranges from 0.5% to 1%, with higher incidence reported in the Indian population. The pathogenesis of vitiligo is multifactorial, involving autoimmune-mediated melanocyte destruction, genetic susceptibility, oxidative stress-induced cellular damage, and neurochemical dysregulation. This systematic literary review critically evaluates classical Ayurvedic texts alongside modern biomedical literature to establish a conceptual

correlation between Switra and vitiligo. The analysis reveals significant parallels between Ayurvedic Samprapti and contemporary immunopathological and oxidative stress models. Furthermore, Ayurveda advocates a holistic therapeutic framework incorporating Shodhana (bio purification), Shamana (palliative therapy), and Nidana Parivarjan (Avoidance of causative factors) aiming at root-cause management. An integrative approach combining traditional Ayurvedic principles with modern dermatological insights may offer improved therapeutic outcomes and pave the way for evidence-based integrative research in vitiligo management. This review underscores the potential of integrative approaches in bridging traditional and modern perspectives for improved clinical management of vitiligo.

2. KEYWORDS: Switra; Vitiligo; Kushta; Bhrajaka Pitta; Tridosha; Autoimmunity; Oxidative Stress; Melanocyte Dysfunction.

3. INTRODUCTION

Switra is a well-described dermatological condition mentioned in Ayurvedic classics under the broader category of Kushta. It is considered a Tridoshaja Vyadhi, with predominant involvement of Pitta Dosha, which governs pigmentation and metabolic activities in the body. The vitiation of Pitta, along with Vata and Kapha, leads to derangement of Twak, Rakta, Mamsa and Lasika, ultimately manifesting as depigmented patches on the skin. The disturbance in Bhrajaka Pitta, a subtype of Pitta responsible for skin coloration, plays a crucial role in the pathogenesis of Switra.

Ayurveda emphasizes multiple etiological factors (Nidana) in the development of Switra, including *Viruddha Ahara* (incompatible dietary combinations), excessive intake of sour, salty, and heavy foods, improper lifestyle practices, and suppression of natural urges. Additionally, psychological factors such as stress, grief, and unethical conduct (*Papa Karma*) have also been implicated in classical texts, indicating a psychosomatic dimension of the disease.

From a contemporary biomedical perspective, Switra can be correlated with vitiligo, a chronic depigmentary disorder characterized by the progressive loss of melanocytes, the cells responsible for skin pigmentation, resulting in the appearance of white patches on the skin. Clinically, vitiligo presents as well-demarcated, milky-white macules that lack functional melanocytes. It is a relatively common dermatological condition, with prevalence in India reported to be approximately 3–4%, which is higher compared to many other regions

globally. The term "vitiligo" is believed to originate from the Latin word vitium, meaning "blemish" or defect. Historical references to this condition are found in ancient texts; it has been described as Shwetakustha in the Atharva Veda and as Switra in the Manusmriti. In southern parts of India, particularly in regions where Tamil is spoken, the condition has traditionally been referred to as ven kushtam, signifying "white leprosy," reflecting its clinical resemblance to depigmented skin disorders.[1]

Despite advancements in modern medicine, complete cure of vitiligo remains challenging. Ayurveda offers a holistic approach targeting root causes, emphasizing detoxification, dietary regulation, and rejuvenation therapies. This study aims to systematically review classical Ayurvedic literature on Switra and correlate it with modern dermatological concepts.

4. MATERIALS AND METHODS

4.1 Study Design

The present study is a systematic literary review conducted to analyse and correlate the concept of *Switra* described in Ayurveda with the modern dermatological condition Vitiligo. The study involved a qualitative and comparative evaluation of classical Ayurvedic texts and contemporary scientific literature.

4.2 Data Sources

4.2.1 Ayurvedic Literature

Primary data were collected from classical Ayurvedic compendia including: Charaka Samhita, Sushruta Samhita and Ashtanga Hridaya. Relevant commentaries and translations were also reviewed to ensure accurate interpretation of concepts related to *Switra*, *Kushta*, *Kilasa*, and associated pathophysiological principles.

4.2.2 Modern Scientific Literature

Secondary data were retrieved from electronic databases including: PubMed, Google Scholar, Scopus and Standard Books Such as Essentials in Dermatology by DM Thappa, Harrison Etc. Search terms used included: "vitiligo," "autoimmune depigmentation," "melanocyte destruction," "oxidative stress," and "immunopathogenesis of vitiligo."

5. RESULTS AND DISCUSSION

The systematic review of classical Ayurvedic literature and modern dermatological studies revealed significant correlations between *Switra* described in Ayurveda and Vitiligo.

5.1 Diseases Review (Ayurveda Classic)

5.1.1 Paribhasa (Definition)

The name "Switra" comes from the Sanskrit word "Sweta," which means "white ". Thus, Switra is a condition in which the body develops white spots. As stated in Kashyapa Samhita “Shweta Bhava Michanti Switram”.[2]

5.1.2 Nidaan (Etiology)

5.1.2.1 Samanya Nidaan

The etiological factors are multifactorial and largely related to improper dietary habits, lifestyle practices, and behavioral conduct. Consumption of *Viruddha Ahara* (incompatible food combinations), along with excessive intake of *drava* (liquid), *snigdha* (unctuous), and *guru* (heavy-to-digest) substances, is considered a primary causative factor. Suppression of natural urges such as vomiting, and engaging in physical exertion or exposure to excessive heat immediately after consuming large quantities of food, further aggravate the Doshas. Irregular dietary patterns also play a significant role, including the intake of foods with opposing thermal properties (such as cold substances followed by hot ones) and alternating between prolonged fasting and heavy meals. Additional contributing factors include consumption of cold water immediately after exposure to intense sunlight, physical exertion, or emotional stress, as well as eating raw or improperly cooked food and taking meals before the previous food has been fully digested. Non-adherence to dietary restrictions prescribed during *Panchakarma* therapy is also considered detrimental.

Specific dietary items such as newly harvested grains, curd, fish, excessively salty and sour foods, black gram, radish, preparations made from flour paste, sesame, milk, and jaggery are identified as aggravating factors. Lifestyle-related causes include engaging in sexual activity immediately after meals when digestion is incomplete, sleeping during daytime, and indulging in inappropriate social or ethical behaviors such as disrespect toward teachers or elders and involvement in unethical or sinful actions. Collectively, these factors contribute to Dosha vitiation and are recognized as important etiological contributors to the development of Kushta, including Switra.[3]

5.1.2.2 Vishista Nidaan

वचांस्यतथ्यानि कृतघ्नभावो निन्दा सुराणां गुरुधर्षणं च|

पापक्रिया पूर्वकृतं च कर्म हेतुः किलासस्य विरोधि चान्नम्|| (charak chikitsa-7/177)

Unethical and inappropriate behavioral factors such as dishonesty, ingratitude, lack of reverence toward deities, disrespect toward teachers or elders, and engagement in immoral activities are described as important causative elements. Additionally, the effects of past actions (*Purva Karma*) and the intake of incompatible dietary combinations (*Viruddha Ahara*) are also considered significant contributors in the etiopathogenesis of the disease.[4]

5.1.3 Samprapti

Classical Ayurvedic texts such as Charaka Samhita and Sushruta Samhita do not provide a distinctly separate and elaborated Samprapti (pathogenesis) exclusively for Switra. Instead, Switra is described under the broader category of Kushta Roga, where a generalized Samprapti applicable to various skin disorders is explained.

The vitiated three doshas- vata, pitta, kapha along with tvak, rakta, mamsa and ambu together constitute seven essential entities which play role in pathogenesis of kushtha.[5]

Nidana Sevana

↓

Dosha Prakopa (Tridosha, Pitta predominance)

↓

Dushya involvement (Twak, Rakta, Mamsa, Ambu)

↓

Srotodushti

↓

Sthanasamsraya in twak

↓

Vyakti (Switra – depigmented patches)

5.1.4 Types

“According to Acharya Vagbhata it is caused by the three Dosha’s Vata, Pitta and Kapha. There are three different types of Switra stated in Dhatus, depending on where it is found and what the color and texture of skin. When it is in Rakta Dhatu, it is dry and red in colour, this type of Switra is called Vataja Switra; when it is in Mamsa Dhatu, it is coppery in colour and with burning sensation and loss of hairs, this type of Switra is called Pittaj Switra; when it is

in Meda Dhatu, it is white in colour and with itching sensation, this type of Switra is called Kaphaja Switra.[6] According to Acharya Charak, it has 3 types Darun, Charun and Switra in which rakta, mamsa and meda dhatu respectively gets vitiated leading to rakta, tamra and shukla colour of skin respectively.[7]

5.1.5 Chikitsa[8]

In **Switra**, *Shodhana* (purificatory therapy) is considered the primary line of management, with *Virechana* (therapeutic purgation), particularly using *Malapurasa* along with jaggery, described as especially beneficial. Prior to purification, the patient should undergo *Snehana* (oleation), followed by administration of *Kushtaghna* formulations according to individual strength (*bala*) and disease severity. Subsequent exposure to sunlight is advised.

During the purgation phase, if the patient experiences thirst, *Peya* (light liquid diet) should be administered for three days. Any vesicles or eruptions appearing over depigmented patches should be carefully punctured to facilitate drainage. Thereafter, for approximately 15 days, preparations such as *Malapu*, *Asana*, *Priyangu*, and *Shatapushpa* decoction may be administered, often in combination with *Palasha Kshara* or *Phanita*, depending on the patient's condition.

Additionally, all *Kushtaghna* therapies are considered beneficial in Switra, particularly *Khadira*-based decoctions. External applications containing *Manahshila*, *Vidanga*, *Kasisa*, *Gorochana*, *Kanakpushpi* (Svarnakshiri), and *Saindhava* are also indicated in the management of depigmented lesions.

5.1.6 Prognosis of Switra[9]

If the lesions are confluent, numerous in distribution, associated with reddish discoloration of hair over the patches, and of long duration (more than one year), such Switra is considered *asadhya* (incurable).

Conversely, if the hair over the lesions retains its normal color, the skin is thin and pale, the disease is of recent onset, and the intervening skin between patches is slightly elevated, such Switra is regarded as sadhya (curable).

5.2 Diseases Review (Modern Classic)

5.2.1 Definition[10,11]

Vitiligo is an acquired pigmentary disorder characterized by well-defined depigmented macules and patches, frequently accompanied by leukotrichia. It typically follows a progressive course and results from the selective destruction of melanocytes, affecting the skin, mucous membranes, inner ear, and leptomeninges. The Vitiligo European Task Force (VETF) defines vitiligo vulgaris/ nonsegmental vitiligo as an acquired chronic pigmentation disorder characterized by white patches, often symmetrical, which usually increase in size with time, corresponding to a substantial loss of functioning epidermal and sometimes hair follicle melanocytes.

5.2.2 Prevalence[12,13,14,15,16]

Vitiligo is the most prevalent pigmentary disorder, affecting approximately 0.5–1% of the global population. A comparatively higher occurrence has been documented in India, followed by countries such as Mexico and Japan. Within India, the reported prevalence shows considerable variation, ranging from 0.46% to 8.8%, with an average between 0.25% and 2.5%. Higher rates have been observed in regions such as Gujarat and Rajasthan. Epidemiological studies indicate variability across populations; for instance, a study from Surat reported prevalence rates of 0.47% in rural areas and 1.78% in urban populations. Similarly, prevalence rates of 2.64% in Uttarakhand and 0.46% in Kolkata have been documented. A retrospective analysis reported a prevalence of 2.5%, with nearly equal distribution among males and females. Such regional differences are likely attributable to variations in genetic background, environmental influences, and sociocultural factors.

5.2.3 Pathogenesis[17,18,19,20,21]

The pathogenesis of vitiligo is complex and multifactorial, primarily characterized by the progressive loss of functional melanocytes, resulting in depigmented skin lesions. Current evidence suggests that this process arises from the interplay of autoimmune, genetic, oxidative, neural, and biochemical mechanisms rather than a single causative pathway. Among these, the autoimmune hypothesis is the most widely accepted. It proposes that melanocytes are selectively destroyed by immune-mediated mechanisms, particularly through cytotoxic T lymphocytes and autoantibodies directed against melanocyte-specific antigens. This is supported by the frequent association of vitiligo with other autoimmune disorders, including thyroid diseases such as Hashimoto’s thyroiditis and Graves’ disease, Addison’s Disease, Diabetes Mellitus, Alopecia Areata, Systemic Lupus Erythematosus, Rheumatoid

Arthritis, Psoriasis, and Pernicious Anemia, indicating an underlying systemic autoimmune predisposition. Genetic susceptibility also plays a significant role, as evidenced by familial clustering and increased prevalence among first-degree relatives. However, incomplete concordance in monozygotic twins highlights the contribution of environmental and epigenetic factors. Vitiligo is considered a polygenic disorder involving multiple genes related to immune regulation and melanocyte function, including major histocompatibility complex (MHC), cytotoxic T-lymphocyte antigen-4 (CTLA-4), angiotensin-converting enzyme (ACE), catalase (CAT), and interleukin-2 receptor A (IL2RA).

Oxidative stress is regarded as a key initiating factor in melanocyte injury. Accumulation of reactive oxygen species, particularly hydrogen peroxide (H_2O_2), in the epidermis leads to mitochondrial dysfunction and apoptosis of melanocytes. This is further compounded by impaired antioxidant defense mechanisms, including reduced activity of catalase and glutathione peroxidase, along with decreased levels of vitamins C and E. Elevated oxidative markers such as malondialdehyde further support the role of oxidative damage in disease progression.

The neural hypothesis suggests that neurochemical mediators released from peripheral nerve endings may exert cytotoxic effects on melanocytes or disrupt melanin synthesis. Additionally, decreased catalase activity in both lesional and non-lesional skin contributes to oxidative imbalance and further melanocyte damage. From a biochemical perspective, the accumulation of toxic intermediates during melanin synthesis, combined with defective antioxidant systems, leads to melanocyte dysfunction and destruction. Structural abnormalities in melanocytes, impaired cellular signaling, and reduced melanocyte growth factors further contribute to depigmentation. In summary, vitiligo is best understood as a multifactorial disorder resulting from a complex interaction between genetic predisposition, environmental triggers, immune dysregulation, and oxidative stress. These interrelated mechanisms collectively lead to melanocyte destruction and the clinical manifestation of depigmented patches.

Table 1: Comparative Pathogenesis of Switra (Ayurveda) and Vitiligo (Modern Medicine).

Aspect	Ayurvedic Perspective (Switra)	Modern Dermatology (Vitiligo)
Basic Concept	Tridoṣaja Vyadhi with predominance of Pitta (especially Bhrajaka Pitta)	Acquired depigmentary disorder due to melanocyte loss
Primary Cause	Doṣha vitiation (Vata, Pitta, Kapha) due to Nidāna Sevana	Multifactorial: autoimmune, genetic, oxidative stress
Key Pathological Factors	Vitiation of Doṣha + involvement of Dhatus (Twak, Rakta, Mamsa, Lasika)	Immune-mediated melanocyte destruction
Initiating Factors	Viruddha Aahara, Mithya Aahara-Vihara, suppression of urges, psychological factors (Manasika Nidāna)	Genetic predisposition + environmental triggers (stress, chemicals, UV exposure)
Core Mechanism	Doṣha-Dhatu Dushti → Srotodushti → Twak Vaivarnya (discoloration)	Autoimmune cytotoxic T-cell attack on melanocytes
Role of Pitta / Metabolism	Bhrajaka Pitta dysfunction → impaired त्वक वर्ण (skin pigmentation)	Defective melanogenesis and melanocyte apoptosis
Tissue Involvement	Twak, Rakta, Mamsa, Lasika	Epidermal melanocytes, hair follicle melanocyte reservoir
Role of Immunity	Not explicitly described but implied via Doṣha imbalance and Ojas disturbance	Strong autoimmune basis (T-cells, autoantibodies)
Oxidative Stress Correlation	Can be interpreted as Agni imbalance and Dhatu Kṣaya	ROS accumulation (H_2O_2), decreased catalase → melanocyte damage
Genetic Component	Not directly mentioned (conceptual via Beeja Doṣha)	Polygenic inheritance (MHC, CTLA-4, IL2RA, etc.)
Neural Factors	Indirect (Manasika Nidana, stress involvement)	Neurochemical mediators toxic to melanocytes
Disease Progression	Sthanasamsraya in Dhatus → Vyakti (clinical manifestation)	Progressive melanocyte destruction → depigmented patches
Clinical Outcome	Twak Vaivarnya (white patches)	Well-defined depigmented macules/patches
Holistic Interpretation	Systemic imbalance involving body, mind, and lifestyle	Localized + systemic autoimmune and biochemical disorder

"The comparative analysis highlights that while Ayurveda explains Switra through Dosha–Dhatu imbalance and Srotodushti, modern dermatology attributes vitiligo primarily to immune-mediated melanocyte destruction and oxidative stress. Interestingly, the Ayurvedic concept of Bhrajaka Pitta dysfunction can be functionally correlated with melanocyte activity and melanin synthesis. Similarly, Agni imbalance and Dhatu Kshaya may reflect oxidative stress and cellular damage described in modern science. These parallels suggest that both systems describe the same disease process using different conceptual frameworks."

5.2.4 Clinical Features[22,23]

The hallmark lesion of vitiligo is a well-demarcated depigmented macule or patch, typically milky white in appearance, with round or oval configuration and often irregular or scalloped borders. Lesion size can range from small, localized spots to extensive areas of depigmentation.

Vitiligo may present at any age, although peak onset is commonly observed between 5 and 30 years. It affects males and females almost equally. The disease generally follows a gradual and progressive course, with an increase in the number and size of lesions over time; however, in certain cases, rapid progression may occur, referred to as vitiligo fulminans or galloping vitiligo. Mucosal involvement is common, particularly affecting the lips, oral cavity, and genital mucosa, and is often more prominent in individuals with darker skin types. Leukotrichia, defined as depigmentation of hair within the lesions, is an important clinical feature and serves as a marker of disease activity and prognosis.

5.2.5 Classification of Vitiligo[24,25]

Vitiligo can be broadly classified based on the distribution and extent of lesions into the following categories.

5.2.5.1 Localized Vitiligo

- **Focal Vitiligo:** One or more depigmented macules confined to a limited area, without a segmental or dermatomal pattern.
- **Segmental Vitiligo:** Unilateral lesions distributed along a dermatome or segment, typically not crossing the midline and often showing early stabilization.
- **Mucosal Vitiligo:** Depigmentation restricted to mucous membranes, such as the lips, oral cavity, or genitalia.

5.2.5.2 Generalized Vitiligo

- **Vitiligo Vulgaris:** The most common form, characterized by widespread, bilaterally symmetrical depigmented lesions.
- **Acrofacial Vitiligo:** Involvement of distal extremities (hands and feet) and facial regions, particularly around body orifices.
- **Mixed Vitiligo:** Coexistence of segmental and non-segmental patterns or a combination of different clinical types.

5.2.5.3 Special Types

- **Lip-tip Vitiligo:** Involvement of the lips and distal digits.
- **Universal Vitiligo:** Extensive depigmentation affecting nearly the entire body surface.

5.2.6 Management of Vitiligo[18,26,27]

The management of vitiligo remains challenging due to its chronic course, unpredictable progression, and variable response to therapy. It is important to recognize that vitiligo is not merely a cosmetic disorder but is associated with significant psychosocial burden. Therefore, a comprehensive and individualized treatment strategy is essential. The primary goals of therapy are to halt disease progression, induce repigmentation, and improve cosmetic outcomes. Treatment selection depends on several factors, including disease type and extent, rate of progression, patient age, skin phototype, and impact on quality of life. Anatomical site also influences response to therapy; lesions on the face, neck, and trunk generally respond better, whereas acral and mucosal areas are relatively resistant.

Repigmentation typically occurs either from the margins of lesions or in a perifollicular pattern. Most therapeutic modalities require a minimum duration of 2–3 months to assess efficacy, and long-term follow-up is often necessary. Combination therapy has been shown to provide superior outcomes compared to monotherapy.

5.2.6.1 Pharmacological Management

a. Topical Corticosteroids

Topical corticosteroids are considered first-line therapy, particularly in localized vitiligo. They exert anti-inflammatory and immunosuppressive effects, thereby reducing melanocyte destruction. Potent agents such as betamethasone valerate and clobetasol propionate are commonly used. Better responses are observed in sun-exposed areas, while acral regions tend to show limited improvement.

b. Topical Calcineurin Inhibitors

Calcineurin inhibitors, including tacrolimus (0.03% or 0.1%) and pimecrolimus (1%), are particularly effective for lesions on the face and neck. They inhibit T-cell activation and cytokine release, with minimal adverse effects such as skin atrophy. These agents are typically applied twice daily over several months.

c. Vitamin D Analogues

Topical vitamin D analogues, such as calcipotriol, exhibit immunomodulatory effects and may promote melanocyte proliferation and melanogenesis. Although less effective as monotherapy, they are often used in combination with corticosteroids or phototherapy to enhance outcomes.

d. Other Pharmacological Agents

Several adjunctive and emerging therapies include:

- 5-fluorouracil (5-FU)
- Methotrexate
- Janus kinase (JAK) inhibitors
- Prostaglandin analogues
- Apremilast
- Minocycline

These agents primarily act through immunomodulatory, anti-inflammatory, or melanocyte-stimulating mechanisms.

5.2.6.2 Phototherapy

a. Narrow-Band Ultraviolet B (NB-UVB)

NB-UVB (311 nm) is currently the most widely used and effective phototherapeutic modality. It promotes repigmentation by stimulating melanocyte proliferation, migration, and melanogenesis, along with immunosuppressive effects.

b. Psoralen plus Ultraviolet A (PUVA)

PUVA therapy involves administration of psoralen followed by exposure to UVA radiation (320–400 nm). It enhances melanocyte activity and melanin synthesis. Although effective, it is generally considered a second-line therapy due to potential adverse effects.

5.2.6.3 Surgical Management

Surgical interventions are reserved for stable vitiligo cases unresponsive to medical therapy. Common techniques include

- Skin grafting
- Melanocyte transplantation
- Cellular grafting methods

5.2.6.4 Cosmetic and Supportive Therapy

Cosmetic camouflage plays a significant role, particularly for lesions in exposed areas. Available options include

- Dermocosmetic camouflage
- Self-tanning agents (e.g., dihydroxyacetone)
- Corrective cosmetic formulations

In addition, psychological counselling and patient education are essential components of holistic management.

5.2.6.5 Treatment Guidelines

According to the European Dermatology Forum guidelines

- **First-line:** Topical corticosteroids, calcineurin inhibitors
- **Second-line:** Phototherapy (NB-UVB, PUVA), systemic corticosteroids
- **Third-line:** Surgical interventions
- **Fourth-line:** Depigmentation therapy (for extensive disease)

6. CONCLUSION

This systematic review establishes a clear conceptual and pathophysiological correlation between the Ayurvedic entity Switra and the modern dermatological condition vitiligo. Classical Ayurvedic texts describe Switra as a Tridoshaja Vyadhi with predominant involvement of Pitta—particularly Bhrajaka Pitta—along with vitiation of Rakta, Mamsa, and Meda Dhatus. These descriptions closely parallel contemporary biomedical mechanisms, including melanocyte dysfunction, autoimmune-mediated cytotoxicity, and oxidative stress-induced cellular damage. The Ayurvedic framework of Samprapti, encompassing Dosha–Dhatu imbalance and Srotodushti, can be interpreted in modern terms as a complex interaction of immune dysregulation, genetic susceptibility, oxidative stress, and neuroendocrine influences. The autoimmune hypothesis of vitiligo, supported by its association with systemic autoimmune disorders, aligns conceptually with Ayurvedic etiological factors such as Viruddha Ahara, Mithya Vihara, and Manasika Nidana, reflecting a shared understanding of disease as a multifactorial and systemic process. From a therapeutic perspective, Ayurveda offers a holistic and individualized approach through Shodhana, Shamana, and Rasayana, emphasizing root-cause management, lifestyle modification, and psychosomatic balance. These principles may complement modern modalities such as phototherapy and immunomodulatory treatments, supporting the development of integrative treatment strategies.

However, despite these conceptual correlations, there remains a need for robust scientific validation through well-designed clinical trials, molecular studies, and standardized protocols. Future research should focus on evidence-based integration of Ayurvedic interventions with modern dermatological practices to enhance therapeutic outcomes. In conclusion, an integrative approach combining Ayurvedic wisdom with contemporary biomedical science holds significant promise for advancing the understanding and management of vitiligo, paving the way for more holistic, patient-centered, and effective care.

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