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Conceptual Study On *Samprapti* (Pathogenies) Of *Śvitra* w.s.r To Vitiligo

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ABSTRACT: -

Skin is the first organ of the body interacting with the environmental agents like physical, chemical & biological agents. Variations in the environmental stimuli & natural ability of body to deal with these factors result in spontaneous remissions & relapses. Interaction with these factors results in specific reaction pattern producing characteristic skin lesions in different parts of the body. Skin is a mirror that reflects internal & external pathology & thus helps in diagnosis of diseases. Skin ailments affects all ages from the neonates to the elderly & cause harm in a number of ways, such as discomfort, disfigurement, disability, etc. *Svitra* (Vitiligo) can be correlated with Vitiligo in contemporary medicine. Vitiligo is an autoimmune disease against melanocyte characterized by hypopigmented patches. In this review article describe the Ayurvedic *Samprapti* (Pathogenesis) in detail according to Ayurveda.

Key words-*Svitra* (Vitiligo), Vitiligo, *Kustha*

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INTRODUCTION

Skin is the largest and visible organ of the human body. Vitiligo affects the estimated 1% of world's population.[1] Vitiligo cause destruction of melanocyte in the skin mucous membrane, hair bulbs and inner ear. Melanocytes provide the pigment that gives skin its colour. Loss of pigment most commonly is noted first on the arms, hand, feet, or lips.[2] Acharya Sushruta also mentioned about *Svitra(Vitiligo)* in *Kustharog adhyaya. Kuṣṭha* (skin disease) is a disease where there is involvement of all three *Doṣa* like *Vāta*, *Pita* and *Kapha*. [3] The *Dushya* are *Tvaka* (skin), *Lasikā*(Lymph), *Rakta* (blood) and *Māṁsa* (muscles) and they are vitiated due to vitiated *Doṣa*. The causes for vitiation are to be prone against the *Nidāna* (causative factor). After *Nidāna Sevana* (causative factor) these morbid *Doṣa* circulate in the body by mean of blood vessels. Then they engorge in specific *Dushya* and vitiate them also.[4] The *Doṣa* which situated in the *Dushya* are generating different typed of *Kuṣṭha* (skin disease) depending upon the degree of *Doṣh* involvement, configuration of *Doṣa* and *Dushyas*, types of *Srotodushti*, *āvaraṇa* (covering) and other numerous direct and indirect factors.[5] So in same way they produced variety of symptoms. There may be also variation in colour, site, shape, border, pain, secretion and intensity of the lesion due to variety of Sammurchanā.[6]

AIMS AND OBJECTIVE

To evaluate, elaborate and discuss the pathogenesis of *Svitra* (Vitiligo).

MATERIAL AND METHOD

Material related to *Svitra* (Vitiligo) is collected from ayurvedic texts books, modern text books, index medical journals and website.

***Samprapti* (Pathogenesis)**

Entire process of manifestation of disease is called *Samprāpti* (Pathogenesis). The *Samprāpti* gives knowledge about provocation of *Doṣa*, route of the disease, involved dhātus (body tissues) and *Srotas (*channels) affected and their prognosis.

The process of understanding the development of disease by the vitiated *doṣa* which, are constantly circulating inside the body is called as *Samprāpti* (Pathogenesis).

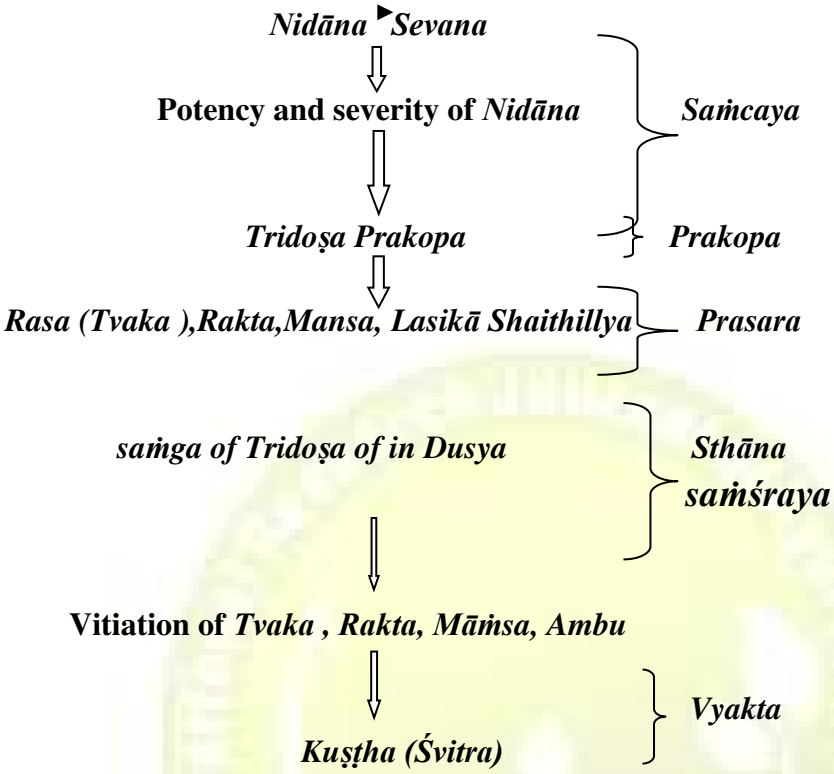
The specific action of vitiated *Vyādhijanya doṣa* responsible for the manifestation of *Vyādhi* (disease) is called as *Samprāpti* (Pathogenesis).[7] Vitiation of *Doṣa* takes place in various ways like *Prakrita* (nature), *Vikrita* (Imbalance), *Anubandhya* (Primary disease), *Ekdoṣaja*, *Dvidoṣaja* and *Tridoṣaja*. It all depends on various *Nidāna* (causative factor), *Vikrita doṣa* bring disturbance in *dhātus* (body tissues), *malas*(waste), and manifest diseases and understanding of such events is called *Samprāpti* (Pathogenesis).[8]

There are two types of *Samprāpti* of *Śvitra* (Vitiligo):

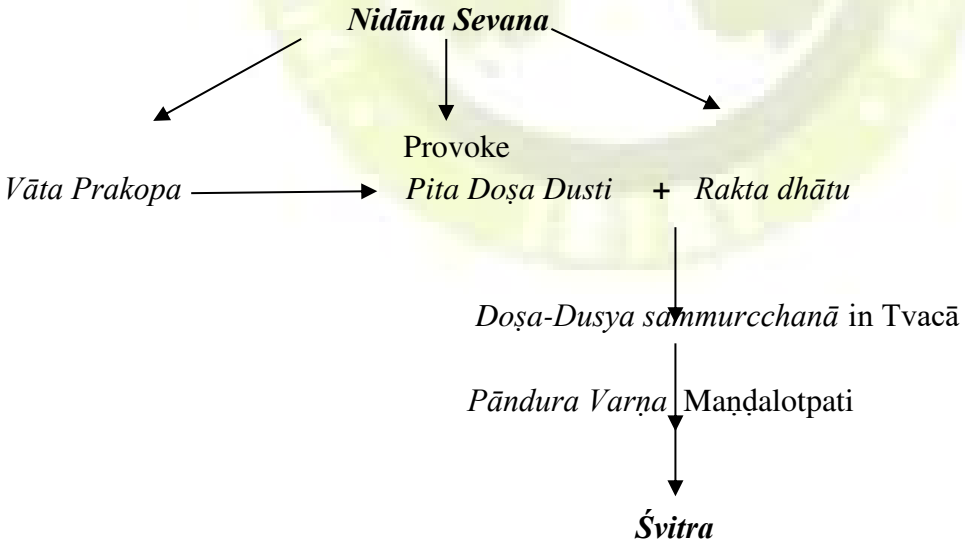
1. *Sāmanya* (General)
2. *Viśeṣa* (Special)

1. Śvitra (Vitiligo) described along with *Kuṣṭha* (skin disease) so, *Samprāpti* (Pathogenesis) of *Kuṣṭha* should be accepted as *Sāmanya Samprāpti* of *Śvitra* (Vitiligo).

The flow chart of the *Samprāpti* of *Kuṣṭha (Śvitra*) according to *ṣaṭakriyākāla* is as follows.[9]



***Viśeṣa Samprāpti* of *Śvitra*:** [10]



***Samprāpti Ghaṭaka* (Pathogenic factor) of *Śvitra* (Vitiligo):**

Doṣa	*Pita*	*Bhrāṁjaka, Raṁjaka,Pācaka*
	Vāta	*Udāna, Vyāna*
	Kapha	*Avalambaka*
Dusya	*Rasa* (plasma), *Rakta*(blood), *Māṁsa* (muscle),*Meda (fats) and Lasikā(lymph)*	
Mala (Waste)	*Loma* (hair follicle)	
Srotasa (Channels)	*Rasavaha, Raktavaha*	
Srotodusṭi	*saṁga* (obstruction)	
Mārga (Route)	*Bāhya Rogamārga* (external disease pathway)	
Udabhavasthāna (Origin)	*Āmashaya* (stomach)	
Svabhāva (Nature)	*Cirakaāri* (chronic)	
Sādhyasādhyatā(Prognosis)	*Asādhya* (not curable) and *Kriccasādhya* (difficult to cure)	

The exact etiopathologies of Vitiligo is not fully understood. There are a few major hypotheses for the pathogenesis of Vitiligo.[11] Autoimmune pathogenesis is a long-standing and popular hypothesis; [12]the neural hypothesis suggests that nerve endings release neurochemical substances that can decrease melanin production or damage melanocytes,[13] the biochemical hypothesis implicates the accumulation of toxic intermediate metabolites of melanin synthesis and defective free radical defense, and the build-up of excessive quantities of hydrogen peroxide (H_2O_2) as a cause for destruction of melanocytes.[14]

Approximately one in four vitiligo patients present with additional autoimmune disorders including pernicious anaemia, autoimmune thyroid disease, psoriasis, rheumatoid arthritis, adult-onset autoimmune diabetes mellitus, Addison's disease, and systemic lupus erythematosus

Genetic influence-

Vitiligo is a polygenic disease, several candidate genes including major histocompatibility complex (MHC), angiotensin-converting enzyme (ACE), catalase (CAT), cytotoxic T lymphocyte antigen-4 (CTLA-4), catechol-O-methyltransferase (COMT), estrogen receptor (ESR), mannan-binding lectin (MBL2), protein tyrosine phosphatase, non-receptor type 22 (PTPN22), human leukocyte antigen (HLA), NACHT leucine-rich repeat protein 1 (NALP1), X-box binding protein 1 (XBP1), forkhead box P1 (FOXP1) and interleukin-2 receptor A (IL-2RA), that are involved in the regulation of immunity have been tested for genetic association with generalized Vitiligo.[15]

Neurohumoral hypothesis: psycho-neuro-endocrine-immune connection in Vitiligo:-

There is enough scientific evidence that supports the view that psychological stressors and nervous pathways have an impact on the release of neuropeptides (NPs), various cell behaviours and expression of innate and adaptive immunity in the skin. Evidence in support of the neurohumoral pathogenesis of vitiligo includes common origin of both the melanocytes and nerves from the neural crest cells, the usual presence of SV in a dermatomal fashion, alterations in perspiration and nerve structure in vitiliginous skin and expression of specific neuropeptides in patients with vitiligo. Stress is known to induce the production of various NPs in the skin such as neuropeptide Y (NPY). Immunohistochemical stains have also

shown an increase in NPY, both within and around a vitiliginous patch. The release of this neuropeptide from nerve fibres is suspected to initiate a cascade of reactions leading to the destruction of melanocytes, probably, mediated by mast cells and granulocytes. This theory is of major interest when applied to SV. Vitiligo lesions also exhibit increased levels of norepinephrine and low levels of acetylcholine esterase activity.[16] An increased level of neurotransmitters may be directly cytotoxic to cells or may have an indirect effect by causing constriction of supplying blood vessels, thereby causing hypoxia and subsequently cell death. Increased levels of homovanillic and vanillylmandelic acids in the 24-hour urine samples of patients with recent onset or progressive disease support the role of stress and the resultant increase in levels of catecholamines in initiating and perpetuating vitiligo.[17]

Humoral Immunity: Various subsets of antibodies are seen in patients with vitiligo and are categorized as those against cell surface pigment cell antigens, intracellular pigment cell antigens and non-pigment cell antigens.[18] Certain antigens namely VIT 40/75/90, named after their respective weights, have been identified in around 83% patients with vitiligo. Although VIT 90 is found exclusively on pigment cells, VIT 40 and VIT 75 are considered common to both pigment and non-pigment cells. Non-specific antibodies against these antigens have been found in patients with vitiligo.[19] As melanocytes are much more sensitive to immune-mediated injury, it is probable that minimal injury from non-specific antibodies may induce lethal harm to melanocytes, but not to the surrounding cells. Antibodies against tyrosinase and tyrosinase-related proteins 1 and 2 (TRP-1 and TRP-2), SOX9 and SOX 10 (transcription factors involved in the differentiation of cells derived from the neural crest) have also been detected in patients with autoimmune polyendocrine syndrome type 1 (APS1) and in patients with vitiligo without any concomitant disease.[20]

Cellular Immunity: - As far as cellular immunity is concerned, the main culprits are the $CD8^+$ cytotoxic T-cells. Perilesional skin biopsies have shown epidermotropic cutaneous lymphocyte antigen positive lymphocytes with an increased $CD8^+/CD4^+$ ratio, substantiating the role of cytotoxic T-cells in the pathogenesis of vitiligo. These T-cells have been shown to bring about degenerative changes in melanocytes and vacuolization of basal cells in the normal-appearing perilesional skin in patients with actively spreading lesions. An increased expression of CD25 and MHC II (specifically HLA-DR) and ability to secrete interferon gamma (IFN γ) has been noted in these T-cells which lead to increased expression of intercellular adhesion molecule-1 and, consequently, increased T-cell migration to the skin leading to a vicious cycle. Also, high frequencies of Melan-A-specific $CD8^+$ T-cells have been found in patients with vitiligo, and their number may correlate with disease extent[i].Another subset of T-cells, called the follicular T helper (Tfh) cells, has been described lately in the pathogenesis of vitiligo[21]

Oxidative stress: -

The oxidative stress theory of vitiligo suggests that the main culprit in the pathogenesis of vitiligo is the intra-epidermal accumulation of reactive oxygen species (ROS), the most notorious of which is H2O2 whose concentration may reach upto one milimole. At this concentration, H2O2 leads to changes in the mitochondria and, consequently, apoptosis/death of the melanocytes.[22]

Melanocytorrhagy: -

Theory of melanocytorrhagy proposes that NSV is a primary melanocytorrhagic disorder with altered melanocyte responses to friction, which induces their detachment, apoptosis and subsequent trans epidermal loss. This theory adequately explains the Koebner's phenomenon because it proposes that weakly anchored melanocytes upon facing minor friction and/or other stress undergo separation from the basement membrane, migrate upward across the

epidermis and are eventually lost to the environment resulting in vitiligo at the sites of trauma [23]

Vitamin D deficiency: -

Vitamin D exerts a significant effect on the melanocytes and keratinocytes via various mechanisms. In vitro studies have shown that vitamin D3 increases melanogenesis and tyrosinase content of cultured human melanocytes and protects the melanocytes from UVB-induced apoptosis, thus, contributing to re-pigmentation in vitiligo macules.[24]

DISCUSSION

Svitra (Vitiligo) is a *Pitta pradhana tridoshaja Vyadhi***.** The pathogenesis of *Śvitra* (Vitiligo) not described separately in texts except *Hārīta Saṁhitā*. *Sushruta, Vagabhatta* and other *Samgraha Kalina* workers, except *Harita, Harita* endeavours to mention the *Samprapti* of *Svitra(Vitiligo)* separately and says that *Vata* provokes the *Pitta,* which is situated in *Twak* (skin). This vitiated Pitta along with *Rakta* (blood) produces Pandura Varna, which is known as *Svitra* (Vitiligo).[25] According to *Hārīta Saṁhitā* the vitiation of *Vāta* along with the *Pita Doṣa* spoil the *Rakta dhātu* (blood tissue) and create the spot of *Pāndura Varṇa* (paleness) that is called *Śvitra* and has mentioned *Pāndura* (paleness) as *Śvitra* (Vitiligo). In this way, *Acārya Hārīta* alone gives the specific line of treatment .

CONCLUSION

Attraction of individual depends upon skin health including physical and mental health. Colour of skin plays important role in society. Vitiligo has major impact on quality of life of patients. Ayurveda can play an important role in the understood pathogenesis of *Svitra* (Vitiligo).

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