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. Śvitra (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-Śvitra (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. 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. 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. The Dushya are Tvaka (skin), Lasīkā (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. 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. 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 Sammunchanā.

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.

Samprāpti (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). Vitiation of Doṣa takes place in various ways like Prakriti (nature), Vikriti (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).

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 āṣṭakriyākāla is as follows.\(^9\)

\[\text{Nidāna} \rightarrow \text{Sevana} \]

\[\text{Potency and severity of Nidāna} \rightarrow \text{Saṁcaya} \]

\[\text{Tridoṣa Prakopa} \rightarrow \text{Prakopa} \]

\[\text{Rasa (Tvaka), Rakta, Mansa, Lasikā Shaithilīya} \rightarrow \text{Prasara} \]

\[\text{saṁga of Tridoṣa of in Dusya} \rightarrow \text{Sīhāna saṁśraya} \]

\[\text{Vitiation of Tvaka, Rakta, Māṁsa, Ambu} \rightarrow \text{Vyakta} \]

\[\text{V)išeṣa Samprāpti of Śvitra:} \(^{10}\)

\[\text{Nidāna Sevana} \]

\[\text{Provoke} \rightarrow \text{Vāta Prakopa} \rightarrow \text{Pita Doṣa Dusti} + \text{Rakta dhātu} \]

\[\text{Doṣa-Dusya sammurcchanā in Tvacā} \]

\[\text{Pāndura Varṇa} \rightarrow \text{Maṇḍalotpati} \rightarrow \text{Śvitra} \]
The exact etiopathologies of Vitiligo is not fully understood. There are a few major hypotheses for the pathogenesis of Vitiligo. Autoimmune pathogenesis is a long-standing and popular hypothesis; the neural hypothesis suggests that nerve endings release neurochemical substances that can decrease melanin production or damage melanocytes, 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$_2$O$_2$) as a cause for destruction of melanocytes. 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.

**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. 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.

**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. 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. 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.

**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.

Another subset of T-cells, called the follicular T helper (Tfh) cells, has been described lately in the pathogenesis of vitiligo.

**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 up to one millimole. At this concentration, H2O2 leads to changes in the mitochondria and, consequently, apoptosis/death of the melanocytes.

**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 Svitra (Vitiligo) not described separately in texts except Hārīta Sanhitā. 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 Sanhitā 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, Ācārya 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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