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**Review Article** 

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# LITERARY RESEARCH ON *EKA* KUSTHA WITH SPECIAL REFERANCE TO PSORIASIS

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#### **ABSTRACT**

The term *Eka Kustha* is found in various *Ayurvedic* texts and its characteristic features gives a clear picture of what the disease is, like *Aswedanam*, *Mahavastu* and *Matsyashakolopama*. It is a *vata-kapha* predominant *tridoshaja vikar*. It can be easily treated by both *shodhana* and *shamana chikitsa*. *Raktamokshan*, *Vaman* and *Virechan* are the mainstay. *Amlaki*, *Rasanjan*, *Manjistha*, *Lelitaka* are important among internal medications. The disease is quite similar to Psoriasis in modern medicine. According to them, it is a hereditary, autoimmune chronic inflammatory skin disease of unknown etiology. The five main types of psoriasis are: 1. Plaque 2. Guttate 3. Inverse , 4. Pustule and

5. Erythrodermic. It typically manifests as red and white scaly patches on the top layer of the skin. Skin biopsy is confirmatory.

**KEYWORDS**: Aswedanam, Eka Kustha, Mahavastu, Matsyashakolopama, Raktamokshan, Vaman, Virechan etc.

# INTRODUCTION

The term 'Kustha' is originated from the word "Nikushi", defined as 'Kusnati rogam and kusnati sharirastha shonitam vikruti' denotes that kustha is a disease causing disgraceful situation in which vitiated rakta becomes destructed in the body <sup>1</sup>. The term Eka Kustha comprises of two parts: 'Eka' & 'Kustha'. Here the term eka signifies some unique features which will be described below.

Eka Kustha is characterized as follows - Absence of perspiration (Aswedanam), Extensive localization (Mahavastu) and resembles the scales of fish (Matsyashakolopamam)<sup>2, 3</sup>. In

Ayurvedic texts eighteen clinical varieties of Kusthas are described. Ekakustha is one of them. Clinically it is considered as 'Khudra Kustha'. Morphologically it co-relates with Psoriasis of modern medicine.

#### Doshik In Volvement

Eka Kustha is a vata-kapha predominant tridoshaja vikar. View of Caraka denotes that, among vata and kapha, if either dosha is predominant then the disease is not so difficult to cure. One can determine the nature of the predominant dosha from specific variety of Kustha and vice versa. The causative factors are determined on the basis of specific manifestation and from the manifestations one can also determine the cause<sup>4</sup>. All varieties of Kustha are caused by the simultaneous vitiation of all the three doshas<sup>5</sup>. However some doshas are predominant and others are not. Keeping this in view and after ascertaining this from manifested sign and symptoms, the physician should decide the time of treatment. In the beginning the predominantly vitiated dosha(s) should be alleviated. Thereafter, the remaining secondarily vitiated doshas should be alleviated<sup>6</sup>.

# Nidana (Etiological Factors)

*Madhava* has clearly mentioned the *nidana* or etiological factors of *Kustha*. Indulgence in incompatible food and drinks, foods which are very watery, fatty and hard to digest; suppressing the urges of vomiting and others, heavy physical exercises and too much exposure to heat immediately after taking food, immersing in cold water soon after exposure to sunlight, hard work causing fatigue and incidents of fear, use of uncooked foods and over eating, improper methods of administering the five purificatory therapies, use of fresh grains, fish, foods which are very salty and sour, black gram, raddish, dry or powdery foods, sesame milk and jiggery, over indulging in sexual intercourse, sleeping during day though suffering from indigestion, showing disrespect to God, teachers and many other kinds of sinful acts, etc make for increase of all the three *doshas* and derangement in the *twak*, *rakta*, *mamsa*, *ambu*, and produce *Kustha*. This group of seven causes *Kustha* of seven and eleven different types<sup>7</sup>.

#### Purvarupa

Ayurveda has described different *Purvarupa* (premonitory symptoms) of various types of *Kustha*. Modern science is lacking in this regard. They are- the skin will be either very smooth or rough, presence or absence of perspiration, discoloration, burning sensation, itching, loss of tactile sensation, pricking pain, appearance of elevated patches, giddiness, severe pain on injury, quick formation of ulcer and remaining for long periods without

healing; though healed with difficulty, roughness of skin persisting and re-appearance of ulcer even with trivial causes, frequent horripulations and blackish discoloration of the blood are premonitory symptoms <sup>8</sup>.

#### **Clinical Features**

The basic clinical manifestations of *Kustha* according to *Ayurveda* are based on *doshik* involvement. Like roughness, blue or bluish red colour, dryness and pain are due to *vata* in all the kinds of *Kustha*; putrefaction, burning sensation, redness and exudation are due to *pitta*; unctuousness, thickness, greasiness, itching, cold and feeling of heaviness are due to *kapha*; presence of symptoms of two *doshas* and of all the three will also manifest respectively <sup>9</sup>.

# **Prognosis**

Now the Prognosis of *Kustha* is also mentioned in *Ayurvedic* texts. Those which invades *twak*, *rakta* and *mamsha* and those varieties which are caused by predominance of *vata* and *kapha* are easy to cure, that invades *medas* and caused by combination of two *doshas* to either become chronic and that which invades *majja* and *asthi* is to be refused treatment <sup>10</sup>. Also presence of worms in the ulcers, thirst, feeling of burning sensation, poor digestive capacity, involvement of all the three *doshas* together, mutilation and loss of body parts, redness of the eyes, loss of voice and that affecting patients who are unsuitable for the five purificatory treatments are going to kill the patients <sup>11</sup>.

# **Treatment Principles (Ayurvedic Concept)**

The sage (Lord Punarvasu) has explained various details on the treatment of Kustha with a view of sharpening the memory and intellect of his disciple (Agnivesha)<sup>12</sup>. To expel out the vitiated doshas, bloodletting ,external and internal administration of alleviation therapies and administration of medicated ghee in appropriate time, the curable types of Kustha gets <sup>13</sup>.Chardana (Vaman) should be done every fortnight, purification(Virechan) every third day and Raktamokshana every six months intervals <sup>14</sup>. Caraka has instituted lots of mineral compound to treat Kustha. Administration of Lelitak (sulphar) with the juice of *Jati (Amlaki)* together with honey is the remedy per excellence for the cure of seventeen types of Kustha. Similarly it is the therapeutic efficacy of Makshika (Copper pyrite) that goes very well if taken together with cow's urine in the disease<sup>15</sup>. Intake of Rasanjana (Solid extract) prepared of decoction of Daruharidra along with cow's urine cures *Kustha*. Similarly intake of *Abhaya* along with Trikatu (*Sunthi, Pippali, Marich*), *Guda* (jiggery) and sesame oil for one month cures *Kustha* <sup>16</sup>.

# Dietary Modification In Kustha In Ayurveda

The patient suffering from kustha should take the following diet  $^{17}$ :

- 1. Light and wholesome food.
- 2. Leafy vegetables having bitter taste.
- 3. Food and medicated Ghee prepared by boiling with *Bhallataka*, *Trifala* and *Nimba*.
- 4. Old cereals and
- 5. Meat of animals inhabiting arid land and preparations of *mudga* mixed with *patola*.

Intake of heavy and sour food, milk, curd, meat of animals inhabiting marshy lands, fish, *guda* and *taila* (sesame) is prohibited for patients of *Kustha* <sup>18</sup>.

#### **Modern View**

In contrary to the *Ayurvedic* theory, modern view is quite different. It is considered to be a hereditary, autoimmune chronic inflammatory skin disease of unknown etiology which can affect people of any age<sup>19</sup>.

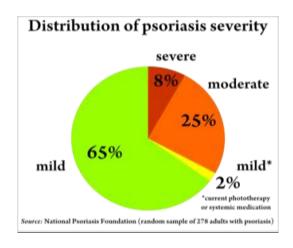
# **Epidemiology Of Psoriasis And Psoriatic Arthritis**

Psoriasis is prevalent world-wide though more common in northern part than tropical part. Among the races, Caucasians are more affected than any other races<sup>20</sup>. The world-wide prevalence of Psoriasis ranges from 0 - 11.8  $\%^{21}$ . The prevalence of Psoriasis is low among African-American than Caucasians <sup>22</sup>. The prevalence of Psoriasis is 0.5 – 1.5 % in India. In dry rainless countries of eastern Africa, the prevalence of Psoriasis is higher in comparison to humid and rainy countries of Western Africa.

# **Clinical Picture Of Psoriasis And Psoriatic Arthritis**

Modern medicine categorized the types of Psoriasis according to the clinical manifestations. There are five main types of psoriasis: 1. Plaque 2.Guttate 3. Inverse 4. Pustule and 5. Erythrodermic <sup>23</sup>. Plaque Psoriasis, which is also known as psoriasis vulgaris typically manifests as red and white scaly patches on the top layer of the skin. Skin cells rapidly accumulate at these plaque sites and create a silvery-white appearance. Plaques frequently occur on the skin of the elbows and knees, but can affect any area, including the scalp, palms of hands and soles of feet, and genitals. In contrast to eczema, Psoriasis is more likely to be

found on the outer side of the joint. Fingernails and toe nails are frequently affected (Psoriatic nail dystrophy) and can be seen as an isolated sign. Inflammation of the joints in the context of psoriatic disease, known as psoriatic arthritis, affects up to 30% of individuals with psoriasis<sup>24</sup>. These above features of Psoriasis can be co-related with other varieties of *Kustha* and can be categorized and treated accordingly.



The disease does not have any sex predilection. The average age of onset of Psoriasis is 15 to 25 years of age, but can develop at any age. Active Psoriatic lesions are characterized by *Koebner phenomenon* in which new lesions appear at the site of trauma. It has been found that in children Guttate Psoriasis can develop after streptococcal throat infections or viral infection. In most of the guttate Psoriasis is self limiting solved within 3-4 months from the date of onset. About 5-40% of Psoriatic patients may develop psoriatic arthritis (PsA) several years after the onset of Psoriasis. Usually psoriatic arthritis is characterized by involvement of small joints of hands including interphalangeal joints of fingers, toes, calcaneal joints and sometimes involvement of spine. As like other chronic diseases, patients with Psoriasis and Psoriatic arthritis suffer from psychological and financial burdens which interfere with their quality of life. Apart from psychological and financial burden these patients also have increased chance of other co-morbidities like CVD, Obesity and metabolic disorder<sup>25</sup>.

#### **Genetics Of Psoriatic Disease**

Although the exact cause of Psoriasis and its associated arthritis has remained unidentified, but the role of genetic, immunological and environmental factors in the pathogenesis is clearly evident. In Psoriasis and psoriatic arthritis (PsA) genetic component plays an important role. The genetic basis of psoriasis is supported from evidence from family and twin studies, linkage studies and population based association studies. Regarding the

association between psoriasis and different loci of immune system, such as the Th17 pathway (IL12B,IL23A,IL23R,TRAF3IP2,TYK2), innate immunity [NF-Kb and IFN], signaling pathway (TNFAIP3,TNIP1,NFKBIA,REL,TYK2,IFIH,IL23RA) and the Th2 pathway (IL4,IL13) and adaptive immunity involving CD8 T cells (ERAP1,ZAP70). Elder and his colleagues have reviewed the SNP analyses of several major studies in this field to provide collective information <sup>26</sup>. Cornified envelope LCE3B and LCE3C, which are important for skin barrier function have also been found to have an association with psoriasis phenotype. From these studies, importance of both keratinocytes and the immune ststem in the pathophysiology of psoriasis has been well established. In PsA,HLA-B22 is protective for disease progresion whereas HLA-B27 is associated with spinal involvement and HLA-B38 & HLA-B39 are associated with peripheral polyarthritis. Patients with both HLACw6 and HLA-DRB1 are reported to have a less severe course of arthritis than patients with HLA-Cw6 or HLA-DRB1alone.Susceptibility of PsA is also determined by interactions between certain HLA-class I alleles and killer inhibitory receptors (kirS), located on chromosome 19. It has been found that presence of KIR2DS1 and/or KIR2DS2 plus HLA-Cw ligand group homozygosity is strongly associated with the susceptibility to PsA.

#### Mechanism

Psoriasis is characterized by an abnormally excessive and rapid growth of the epidermal layer of the skin  $^{27}$ . Abnormal production of skin cells (especially during wound repair) and an overabundance of skin cells result from the sequence of pathological events in psoriasis. Skin cells are replaced every 3-5 days in psoriasis rather than the usual 28-30 days<sup>28, 29</sup>. These changes are believed to stem from the premature maturation of keratinocytes induced by an inflammatory cascade in the dermis involving dendrite cells, macrophages, and T cells (three subtypes of white blood cells). These immune cells move from the dermis to the epidermis and secrete inflammatory chemical signals (cytokines) such as tumor necrosis factor- $\alpha$ , interleukin-1 $\beta$ , interleukin-6, and interleukin-22 a defect in regulatory T cells, and in the regulatory cytokine interleukin-10  $^{30}$ .

Gene mutations of proteins involved in the skin's ability to function as a barrier have been identified as markers of susceptibility for the development of psoriasis. DNA is an inflammatory stimulus in Psoriasis and stimulates the receptors on certain dendrite cells, which in turn produce the cytokine interferon- $\alpha$ . In response to these chemical messages from dendrite cells and T cells, keratinocytes also secrete cytokines such as interleukin-1,

interleukin-6, and tumor necrosis factor- $\alpha$ , which signal downstream inflammatory cells to arrive and stimulate additional inflammation.

# **Diagnosis**

A diagnosis of Psoriasis is usually based on the appearance of the skin. Skin characteristics typical for psoriasis are scaly, red, plaques, papules, or patches of skin that may be painful and itch. There are no special blood tests or diagnostic procedures needed to make the diagnosis.

The differential diagnosis of Psoriasis includes dermatological conditions similar in appearance such as discoid eczema, seborrhoeic eczema, pityriasis rosea (may be confused with guttate psoriasis), nail fungus (may be confused with nail psoriasis) or cutaneous T cell lymphoma (50% of individuals with this cancer are initially misdiagnosed with psoriasis). Dermatologic manifestations of systemic illnesses such as the rash of secondary syphilis may also be confused with psoriasis.

If the clinical diagnosis is uncertain, a skin biopsy or scraping may be performed to rule out other disorders and to confirm the diagnosis. Skin from a biopsy will show clubbed epidermal projections that interdigitate with dermis on microscopy. Epidermal thickening is another characteristic histological finding of psoriasis lesions. The stratum granulosum layer of the epidermis is often missing or significantly decreased in psoriatic lesions; the skin cells from the most superficial layer of skin are also abnormally as they never fully mature. Unlike their mature counterparts, these superficial cells keep their nucleus. Inflammatory infiltrates can typically be visualized on microscopy when examining skin tissue or joint tissue affected by psoriasis. Epidermal skin tissue affected by psoriatic inflammation often has many CD8+ T cells while a predominance of CD4+ T the inflammatory infiltrates of the dermal layer of skin and the joints.

#### **CONCLUSION**

To conclude, *Eka Kustha* has a very important clinical significance as the disease mentioned by the ancient *Ayurvedic* scholars has a very close similarity to the common presentation the disease. The features: Absence of perspiration (*Aswedanam*), Extensive localization (*Mahavastu*) and the scales resembles the fish (*Matsyashakolopamam*) are worth mentioning. Morphologically *Eka Kustha* correlates Psoriasis. Also the *nidanas* mentioned here have a distinct value in appearance of the disease. The treatment module is worth mentioning. The

shodhana and the shamana chikitsa mentioned earlier gives a promising result as compared to modern medicine. Scales have been seen to be eradicated within few days of treatment. Further randomized studies need to be done to clarify the role of the therapeutic regimens, shodhana and shamana for the betterment of the society. Many of the discussed topics may not stand the test of time and can make a path in forming a newer concept. On the other hand, many points that were not discussed in this article may prove noteworthy in the future. We just need to keep our eyes open.

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