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Review Article

CONCEPT OF EKAKUSHTA AND CHRONIC PLAQUE PSORIASIS

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Abstract

Skin diseases are afflicting the mankind since time immortal. Skin diseases have been a major concern of Physicians, Dermatologist and general practitioners since last few decades because of the rising trends of different skin disorders. Skin disorders occur about in 20%-30% of the general population among which Psoriasis and eczema are the major portion. In Ayurveda almost all the skin disorders are explained in the concept Kushta. Ekakushta is one among the Eleven Kshudra Kushta. The signs and symptoms of Ekakushta in Ayurveda are similar to that of chronic plaque psoriasis explained in western system of medicines. In this review article an effort has been made to understand the concept of Ekakushta explained in the classics in terms of Chronic Plaque Psoriasis.

Key words: Ekakushta; Chronic plaque psoriasis.

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INTRODUCTION

Skin is the largest organ in the human body which weighs 4 kg and covers 2 m square area in an adult individual. Patients with skin disease may experience physical, socio-economic and psychological embarrassment in the society.[1]

In Ayurveda almost all the skin diseases are explained under Kushta rogadhikara and classified as 7 Mahakushta and 11 kshudra kushta.[2] Apart from eighteen types of Kushta, another type of Kushta namely Kshwitra (Leucoderma) and its different types are explained in Ayurveda, on the contrary Ayurveda also considers skin diseases are innumerable.[3] Ekakustha is one among 11 varieties of Kshudra Kushta described in Ayurvedic classics.[2] It is characterized by Aswedana (anhydrotic / hypohydratic lesions), Mahavastu (covering of large surface area), Matsya shakalavat (scaly lesions) twacha which bears a greater resemblance with the symptoms of chronic plaque Psoriasis.[2]

Psoriasis is one among the most common skin disorder seen in day today clinical practice characterized by errythematous, circumscribed, silvery skin lesions.[4]

A survey conducted by the National Psoriasis Foundation reports that, almost 75% of Psoriasis patients believe that psoriasis had moderate to large negative impact on their quality of life (QoL) with alterations in their daily activities. This survey also opines that at least 10% of psoriasis patients had contemplated suicide. Furthermore, physical and emotional effects of psoriasis were found to have a significant negative impact at patient's workplace. Psoriasis is a serious condition strongly affecting the view in which a person sees himself and the way he is seen by others. It is linked with social stigmatization, pain, discomfort, physical disability and psychological distress.[5]

Hence in this review article an effort has been made to understand the concept of Ekakushta explained in the classics, in terms of Chronic Plaque Psoriasis.

REVIEW OF LITERATURE

Definition of Ekakushta

- Ekakushta is the prime among all varieties of Kshudra kushta.[6]
- Ekakushta is defined as one variety of Ashtadasha (18 types of skin diseases) kushta which is characterized by Aswedana, Mahavastu and Matsyashakalawat Twacha.[7]

Various definitions of psoriasis are as follows

Psoriasis is a common, chronic and non infective skin disease characterized by well defined slightly raised, dry errythematous macules with silvery scales and typical extensor distribution.[8]

Psoriasis is common, chronic, recurrent, inflammatory disease of the skin characterized by rounded, circumscribed, errythematous, dry scaling patches of various sizes, covered by greyish white or silvery white lamellar scales.[9]

Nidana (Etiology)

The etiological factors explained for all types of Kushta can be categorized as follows

Dosha hetu

Aharaja Nidana - Excessive intake of amla (sour), lavana (salt), kashaya (astringent) rasa, guru (food which are heavy to digest), snigdha (food made of ghee & fried substances) and drava ahara (food articles containing excess of oil and liquid contents), adhyashana (eating food before the digestion of previous meal), vishamashana (eating food irregularly and at

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improper time), atyashana (eating excessive food), asatmya ahara (eating food which is not suitable for an individual).

Viharaja nidana – Atapasevana (excessive exposure of the body to the sun rays), Anila sevana (exposure of the body to the cold wind), Atishrama (excessive physical work), divaswapna (indulgence in day time sleep).

Vyadhi hetu

Mithya Ahara / Viruddha ahara - Incompatible food, Dushi visha (artificial poisoning), polluted water, shitoshna vyatyasa sevana (sudden diving into cold water or drinking cold water after fear, exhaustion and coming from sunlight)

Practice of Physical exercise and sunbath after heavy meals.

Mitya vihara - Suppression of chardi, mutra vegas (suppression of vomiting and urine urges), sexual indulgence after snehana karma (oleation therapy).

Mithya achara - Papa Karma, Guru Tiraskara, Sadhu Ninda (Idiopathic causes like doing sin, not respecting teachers, etc.)

Ubhaya hetu

Aharaja Nidana – Excessive intake of ksheera (milk), dadhi (curds), navanna (food prepared with fresh grains), pishtanna (food containing excess of oil), navadhanya (fresh grains), masha (urad dal), kulatha (horse gram), matsya (fish), varaha (excess of meat), mulaka (raddish), guda (jaggery), madhu (honey)**.**

Mitya ahara - Vidahi vidagdha ahara (food which increases pitta / burning sensation), intake of food in ajirna avastha (indigestion phase).

Virudha (incompatible food), ahara-gunataha viruddha. For example intake of mulaka (radish), lashuna (garlic) with Milk; gramya anupa audaka mamsa with milk (intake of marshy animal's meat with milk), intake of fish with Milk.

Acharaja Nidana - Behavioural misconduct, antisocial activities, sinful activities are considered as acharaja nidanas. These acharajanya nidanas bring about psychogenic stress which is of prime importance in aggravation of psoriasis.

Etiology of psoriasis

Psoriasis has been considered as an Idiopathic disease. It is now believed that Psoriasis is essentially a genetic disorder that is triggered by some form of stress.[9]

The factors that appear to contribute to its appearance are listed below:

Genetic factor

Solomons (2005), stated that the familial incidence of all cases is about 30%.[10] Hunter (1995) described a 15% chance that an off spring would develop psoriasis if one parent is affected.[11] This would rise to 50% if both parents were sufferers. The psoriasis association of Australia (1996) suggested that the appearance of psoriasis is solely a genetic disposition that is expressed due to a form of stress. There is a higher expected frequency of certain white cell antigens (Class 1 human leukocyte antigen or HLAs) on cells of people with psoriasis and their close relatives.

This finding also supports inheritability and also suggests that the gene(s) involved in the psoriasis may be on the same chromosome that holds the gene for HLA. There are many types of HLA in HLA complex and studies have shown that HLA type may be associated in some degree with timing of disease onset, type of psoriasis and severity of the disease.

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Bio chemical factors

Hunter (1995) stated that the increased epidermal proliferation was due to increased levels of prostaglandins, leukotrienes, hydroxyeicosatetraenoic acids in the epidermis. Hunter stated that altered Arachidonic acid metabolism resulted in decreased cyclic Nucleotides, raised polyamines and raised proteases when Psoriasis is seen.[12]

Role of immune system

When the immune system is triggered it sends a false alarm to the skin that they have been damaged the skin cells reaction by attempting to repair the damage. They being reproduced in accelerated rate (the process that takes roughly 26 days in normal skin now occurs in about 5days), rising in the skin surface, dying and building up there. As blood vessels expand, and more blood flows to the skin it become red. It results in reddish erythematous patches.[13]

Role of T cells and Cytokines play

The process is driven by T cells & by the attack force of the immune system. T cells start off as innate, unable to recognize antigens and with no instinct to attack them. However once T cells are exposed to an antigen, they bind together with the antigen and become active. They will be able to recognize the signal given off by the antigen and target it for the destruction whenever the signal is packed up.

In psoriasis activated T cells moves to the dermis. This triggers the release of protein called cytokines that serves as chemical messenger in the immune system. These cytokines send out false alarm to the skin cells, activating their accelerated reproduction cycle. Cytokines also make the process 'snow ball' which triggers the inflammation. They cause the activation of even more T cells and call T Cells in other part of the body to come to the skin. They even set off the release of cytokines by the skin cells by themselves.

One of the cytokines released by the T cell is called "tumor necrosing factor" (TNF) which play a role in almost all psoriasis symptoms ie- Inflammation, redness, pain and itching in the plaques. It may also lead to the multiplication of capillaries so as to lead the Auspitz sign in psoriasis.[13]

Dermal factors

Hunter wrote that the increased epidermal cell proliferation of Psoriasis is related to increased replication and metabolism of dermal fibroblasts.[12]

Hence the nidanas explained in Ayurveda, emphasizes more on external causes like improper food intake and improper lifestyle. The concept of Viruddha bhojana as kushta hetu mentioned in Ayurveda having research scope, is the important contribution of Ayurveda to present the Kushta like Ekakushta.

Whereas etiology in western science explains on internal causes like genetic, biochemical and autoimmune factors. Therefore both the sciences are having their valuable contribution in understanding the nidana or etiology of Ekakushta/plaque psoriasis.

Samprapti (Pathogenesis)

Nidanas of kushta aggravates the doshas, causes agnimandya (indigestion) and in other hand produces dhatu shaitilyata (cause weakness of the muscles etc). Among all the doshas, vata and kapha get aggravated predominantly and causes the shithilyata of dushyas like twak (skin), rakta (blood), mamsa (muscles), lasika (channels) and obstruct the lomakupa (sweat glsnds) leading to the sangatmaka vikriti (vitiation) in swedavaha srotas (channels of sweat glands).

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This prakupita (vitiated) doshas enters into rasaraktadi paribhramana (systemic circulation) especially sanchara (movement) in tiryaka siras (vein) and lodges in bahya roga marga viz. twak and resulting in mandalotpatti (formation of skin lesions).

Pathogenesis

Psoriasis appears to be largely a disorder of keratinization.[14] The process comprising the pathophysiology of Psoriasis include

- Abnormalities in the kinetics of epidermal proliferation (hyperproliferation).
- Dilatation and proliferation of dermal blood vessels
- Activation of immune pathways (accumulation of inflammatory cells particularly neutrophils and T – lymphocytes)

The basic defect is rapid replacement of epidermis in psoriatic lesion (3-4 days instead of 28 days in normal skin)

Abnormalities in the kinetics of epidermal proliferation (hyperproliferation)

Accelerated epidermopoiesis has been considered to be the fundamental pathogenic event in the initiation of Psoriasis.

There is increase in the number of proliferating keratinocytes in the basal layer of the epidermis. The mitotic rate in the germinative layer of the epidermis shows a substantial increase in skin affected with psoriasis as compared with normal epidermis. The growth rate in psoriasis is up to 10 times that of normal epidermis.

The transit time of an epidermal basal cell to the stratum corneum can be markedly reduced from about 28 days to 3-4 days when the disease is in an active phase.

Abnormal cellular differentiation of keratinocytes is associated with the hyperproliferative state, which is evident by greatly increased production of keratin. This together is responsible for the thick, silvery scales. A variety of technique have demonstrated that the increased keratinocyte proliferation in psoriasis is a consequence of an increase in the proliferating cell compartment in the basal and suprabasal levels of the epidermis and not because of shortened cell cycle time.[14]

Dilatation and proliferation of dermal blood vessels

In the early stage, capillary in superficial part of dermis are dilated, elongated and twisted. There is a fourfold increase in endothelium of superficial microvessels but not in deep microvasculature, indicating that vascular growth or angiogenesis is an important component of this process. Epidermal keratinocytes are the primary source of angiogenic activity.

These cells produce an array of soluble mediators with angiogenic activity including interleukin -8(IL-8), tumour necrosis factor– (TNF-a) thymidine phosphorylase and endothelial cell- stimulating angiogenesis factor and perhaps most importantly, vascular endothelial growth factor (VEGF), VEGF is over expressed in psoriatic epidermis.

In addition to vascular growth, dermal capillaries contribute to the inflammatory process actively through surface expression of molecules involved in leukocyte homing, induced by inflammatory mediators such as histamine, neuropeptides, IL-1 and TNF – a, importantly E-selection is induced and intercellular adhesion molecule – 1(ICAM – 1) up regulated on dermal vessels in lesional tissue thus providing a mechanism for skin homing T – Lymphocytes to accumulate within dermis and epidermis.[14]

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Activation of immune pathways (Accumulation of inflammatory cells particularly neutrophils and T-lymphocytes)

In Psoriasis Parakeratosis is seen which signifies oedema of prickle cell layer, usually due to an inflammatory process. This inflammatory reaction may be part of an immunological response to yet unknown antigens. Immunocomponent cells that process antigens appear to initiate the activation of T-cells in the skin, resulting in the production of cytokines that potentiate the continuation of the mechanisms phenotypically expressed as psoriasis

There is considerable evidence that T – lymphocytes have an important role in the development of psoriasis plaques.

Cellular basis of psoriasis

Most current models of disease pathogenesis are focused on actions or secreted cytokines of activated leucocytes that in turn, produces reactive changes in skin cells according to preprogrammed pathways. Neutrophils are actually quite variably expressed in psoriasis lesions from different patients. In contrast, increased numbers of T lymphocytes are a highly consistent finding in psoriasis biopsies. Dendritic cells form another major class of leucocytes that is found in increased abundance in psoriatic skin lesions

Genetic basis of psoriasis

In recent years, genetic analysis of multiple affected families or cases has identified some susceptibility variants for psoriasis and PsA. Genome-wide linkage scans suggest many additional susceptibility loci for which genes still need to be identified. One of the most compelling susceptibility factors for psoriasis is the presence of human leucocyte antigen (HLA)

Biochemical basis of psoriasis

The anomalies in protein expression can be divided into three areas: abnormal keratinocyte differentiation, hyperproliferation of the keratinocyte, and infiltration of inflammatory elements (Duvic et al. 1998). At least six markers of abnormal keratinocyte differentiation have been found, and all have implications in the pathogenesis of the disease. These include aberrations of keratinocyte transglutaminase type I (TGase K), skin-derived antileukoproteinase (SKALP), migration inhibitory factor-related protein-8 (MRP-8), Involucrin, Filaggrin and keratin expression.

Hyperproliferation

Several possible biochemical causes for the overproduction of the keratinocytes have been found in psoriatic skin: epidermal growth factor (EGF), bone morphogenetic protein-6 (BMP-6), transforming growth factor-alpha (TGF-a), ornithine decarboxylase, activating protein (AP1) and mitogen-activated protein kinase (MAPK).

Inflammatory elements

The inflammatory aspect of psoriasis is physically evident by the redness of psoriatic plaques. The biochemical basis for this inflammation stems from several immune modulators including various cytokines released from keratinocytes and other proteins involved in the inflammatory response, which are increased in psoriasis at both local and systemic level. These inflammatory mediators are most likely to play a crucial role in the pathogenesis of psoriasis.

Hence one can say that twak dushti, Rakta dushti, Ambu dushti (vitiation of skin, blood and serum) mentioned in Ayurvedic classics can be considered in terms of

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- Epidermal proliferation, dilation & proliferation of dermal blood vessels
- Accumulation of inflammatory cells particularly neutrophils and T-lymphocytes
- Intra epidermal occlusion of sweat ducts explained in the allopathic science respectively.[15]

For understanding Mamsa dhatu dushti (vitiation of muscles) there are no explanations available in the allopathic medicines. Therefore this component of samprapti of kushta (pathogenesis of skin disease) is the contribution of Ayurveda.

Roopa (Symptomatology)[16]

Aswedana

Aswedanam means no perspiration / no sweating on the skin. Aswedana symptom is mainly because of increased rooksha guna of vata. Aswedana is one of the swedavaha srotodushti lakshana caused due to sanga (obstruction) in swedavaha srotas. In Ekakushta, both vata and kapha are predominant doshas. Aggravated vata makes shosha (emaciation) in malayana (waste) by its ruksha guna (dry property) which lead to aswedana. Kleda soukumaryakrith and kledavidrathi (moistness) are the sweda karma. They get disturbed by asweda or swedakshaya leading to the manifestation of signs like twacha shosha (emaciation of skin), sputana (breaking of the skin), kharatha, parushatha (dryness). These above mentioned symptoms suggest the predominance of Vatadosha.

Mahavastu

The symptom mahavastu in Ekakushta may be due to the vaikrita kapha dosha. Mahavastu means a large foundation or its site is abundant. In this context, mahavastu refers that the lesions occupy large area, either as a single big or as multiple small lesions.

Psoriasis characteristically involves area like scalp, the extensor surface of the extremities, umblicus or anogenital region. Occasionally the disease is generalized.

Matsyashakalopamam

This can be considered as a cardinal feature of Ekakushta. Shakala shabda is related to the skin which refers that the affected twacha in Ekakushta resembles the scaly skin of a fish.

This may also suggest that, the lesions in Ekakushta resemble mica sheets.

Psoriasis is a scaling popular disease in which the lesions of various sizes are seen which are characterized by dry silvery scales. Typical lesions are covered with overlapping thick silvery Micaceous or slightly opalescent shiny scales.

Krishnarunata or Krishna or Arunata

In Ekakushta, these varnas are manifested due to vitiated vata. Rakta is said to be main dhatu for varnaprasadana. When rakta gets vitiated by vata, these varnas are manifested.

In Psoriasis, the lesions are red or dull red often referred to as salmon pink in colour. In erythrodermic Psoriasis, entire cutaneous surface is red. Rupoid lesions are thick and black in colour.

Vikunam

Vikunam means shrinking, contraction and wrinkles. Vitiated vata makes shoshana of twakgata sneha by its rukshaguna which leads to vikunam.

In Psoriasis, the lesions are well defined with a sharply delineated edge. When they merge, annular and gyrate pattern of figures are produced.

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Clinical features of psoriasis[17]

Onset and Course

Onset is usually gradual, but occasionally an acute attack may occur as in Guttate Psoriasis. Course is inconstant or unpredictable and the variations are numerous. The typical course is one of the chronic remissions and recurrences or occasionally of acute exacerbations that vary in frequency and duration.

The disease usually starts in the scalp with small patches followed by increase in size of lesions. After onset it may remain localized or become generalized over the body's surface.

Distribution

Usually bilaterally symmetrical distribution is noticed occasionally unilaterally arranged. Typical distribution is extensor areas. The size of the lesion varies from a pinpoint to plaques that cover large areas of the body.

Area

Psoriasis characteristically involves more area like scalp, extensor surface or the extremities, the sacral areas and buttocks. The nails, eyebrows, axillae, umbilicus or anogenital area may also be affected.

Lesions

Lesions are rounded, circumscribed, erythematous, dry, variable sized, covered by greyish white or silvery white, imbricated and lamellar scales. Lesions are sharply demarcated with clear borders, consists of non-coherent silvery scales. Under the scale the skin has a glossy homogenous Erythema.

Nature

Condition is gradually progressive; some time spread rapidly within few months and sometime remains constant for long period of time.

Chronic plaque psoriasis (Psoriasis vulgaris)[17]

This is the most typical and common form of the disease. Four out of five people with psoriasis have this type. The technical name for plaque psoriasis is Psoriasis Vulgaris (vulgaris means common). A Plaque is the name used to describe the lesion i.e, well defined patches of red, raised skin.

Characters of lesion – Sharply defined erythematosquamous Plaques or Raised inflamed red lesion covered by a silvery white scales with fairly symmetrical distribution.

Hence, from the above factors it is clear that, the lakshanas of ekakushta explained in the context of Charaka samhita are similar to the signs and symptoms of chronic plaque psoriasis explained in the western system of medicine.

Comparison of symptoms of Ekakushta with that of chronic plaque psoriasis is explained in Table 1.

DISCUSSION

Viruddha ahara

All substances which act as antagonist or incompatible to normal dhatus of the body are regarded as viruddha ahara (incompatible foods).[18]

Viruddha ahara can be considered as nidana only when a patient has history of regular consumption viruddha ahara vidhis for a longer duration and it causes the ninditha vyadhis (diseases) like Kushta (skin diseases) and Kshwitra (leucoderma).[19] Viruddha ahara causes Jatharagnimandya (poor digestive poor) leading to the production of ama (undigested materials).

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Table 1: Comparison of symptoms of Ekakushta with that of chronic plaque psoriasis

Ekakusta	Chronic plaque Psorisis
Aswedana	Anhidrotic / Hypohidrotic skin due to intra-epidermal occlusion of the sweat duct
Mahavastu	Involves Scalp, extensor surface of the extremities, sacral area, buttocks, penis. Occasionally the disease is generalized
Matsyashakhalopamam	Lesions having silvery scales
Chakrakara abhrakapatravat	Overlapping thick silvery micaceous scales
Arunata	Erythematous, salmon pink or cutaneous surface is red
Krishnata	Rupoid lesions are black in colour

This ama turns sour and stays in amashaya (small intestine) for longer duration and causes the production of Amavisha (food poison). Amavisha causes the dhatu shithilata particularly Twak, rakta, Mamsa and Ambu in the body and vitiates tridoshas.

Fish with Milk

It is an example of Samyoga (combination) and Veerya (potency) Viruddha ahara.[19]

Both milk and fish are Madhura rasa (sweet taste), Madhura Vipaka (sweet taste in post digestion) and Maha abhishyandikaraka (moistness) in nature. However, milk is Shita veerya (cold potency) while fish is Ushna Veerya (hot potency).[19]

Due to incompatibility at the level of veerya, when these are taken together, it causes dosha dushti (probably vata dosha) and due to Maha Abhishyandikara property it may produce Kapha dushti and Amavisha at the level of jatharagni (digestive fire) and dhatus. This ultimately leads to the obstruction of different srotas of the body (channels in the body).

Viruddha ahara causes the production of Amavisha. Amavisha due to the combination of gunas (properties) of Ama and visha i.e. picchila guna (sliminess), Ushna (hot), Tikshna Sukshma gunas (strong and penetrating) of Ama and ushna, teekshna, sukshma (dry), vyvayi (moves fastly), vikasi (enters into minute pores) gunas of visha, it renders the dhatu shithilata particularly twak, rakta, mamsa and ambu. Here amavisha can be considered as Garavisha (artificial poison).

The Dhatushithilata i.e. khavaigunya and vyadhi kshamatva abhava (reduced resistance against disease process), favors the settlement of vikrita doshas at the level of different dhatus enhancing the pathogenesis of different disorders of the Skin.

Madhya (alcohol drinks) with milk

Madhya is Amla katu (sour & pungent) rasa, katu vipaka and ushna veerya in nature while milk is madhura rasa, madhura vipaka and Sheeta veerya. Due to incompatibility at the level of veerya (potency) it causes tridosha prakopa which inturn causes Rasa, Rakta, ambu and mamsa dusthi.

Anupa Audaka Mamsa & Matsya Sevana

Anupa and Audaka mamsa (meat of animals living near the marshy land) is kaphakaraka and matsya mamsa (fish meat) is pittakara and abhishyandi. This causes obstruction in srotas (channels of the body) of the body especially in the annavaha srotas (gastrointestinal tract) leading to the production of Ama. Ama causes the Prakopa of sarvadoshas.

Masha and pishtanna sevana

Masha (urad dal) and pishtanna (food containing excessive oil) is guru in nature. Its guru (heavy) and snigdha (unctousness) guna causes the Kapaha prakopa. When a person of

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mridu koshta or mandagni (weak digestive fire) consumes this food, it causes the production of Ama leading to the prakopa of Tridoshas in particular kapha dosha.

Excessive Guru, Snigdha and Drava ahara

Excess of guru, snigdha and drava (heavy and unctuous food) ahara causes the prakopa of Kapha dosha. Guru and snigdha ahara in excess produces rasavaha sroto dushti and Guru Ahara also causes mamsavaha sroto dusti[20]and excess drava ahara also causes raktavaha sroto dusti[20]

Vegadharana (Suppression of natural urges)

Chardi Vegadharana causes the Vata prakopa. According to Charaka samhita, suppression of chardi Vega (suppression of vomiting urge) leads to Kushta.[21] Chardi Vega is present only when the doshas are dislodged from their seat and are ready to be expelled out from the nearest outlet, but when the vega is suppressed, the outlet is not open and the dislodged doshas cannot be expelled out. These dislodged doshas start the etiopathogenesis.

Divaswapna (indulgence in day time sleep)

It is Snigdha, abhishyandikara and vitiates KaphaPitta leading to the manifestation of Kandu, Kotha, (itching and burning sensation and popular rashes) Pidaka. It is also Srotorodhaka (obstruction of the channels) and Agnimandhyakara (weakens the digestive fire) which ultimately produces Kushta.[22] It is also said that day sleep after abhishyandi and guru(heavy and unctuous food) ahara causes dushti of Mamsavaha and medovaha (connective tissue and adipose tissues) srotas[23] and here diwaswapna can be considered as kapha pradhana tridosha prakopaka hetu.

Acharaja Nidana

Behavioral misconduct, antisocial activities, sinful activities are considered as Acharaja Nidana (Hetu). These acharajanya nidana bring about psychogenic stress which is of prime importance in the pathogenesis and aggravation of Psoriasis. Chinta, Bhaya, Krodha (tension, fear, grief and anger) are Vata Prakopaka hetus, Bhaya and Krodha (fear and grief) are vatapitta prakopaka Nidana and Bhaya, Krodha and Shoka causes Dushti of Swedavaha Srotas.[24]

Ati Vyayama & Atapa sevana (excess physical work and exposing the body to cold wind)

Ati Vyayama causes vata prakopa (vyanavata in particular) and atapa sevana causes vata pitta prakopa (vyanavata & twakastha bhrajaka pitta in particular). Aggravated vata dosha increases rukshata and kharata (dryness and roughness in body) in shareera and twak in particular, which is the ashrayasthana and vyakta sthana of kushta roga.

CONCLUSION

There is no separate explanation mentioned in classics regarding Nidana, Poorvarupa, Samprapti and chikitsa of Ekakushta. Hence, the Nidana panchakas of Kushta vyadhi can be considered for Ekakushta.

Genetic factors and the Autoimmunity are the main etiological factors responsible for the manifestation of Psoriasis where as in Ayurveda Viruddha ahara vihara is the Vyadhi utpadaka Hetu (main etiological factor responsible for the production of disease) and Papakarma (poorvajanma kruta papa) and Beejadosha is explained as another etiological factor which can be merely considered as the Genetic Factor

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Even though the Exact pathology of Psoriasis is not yet understood in the contemporary system of medicines', Accelerated Epidermopoiesis is Explained as the Basic Pathological event , where as in Ayurveda Vikrita Vata kapha dosha along with Twak, Rakta, Mamsa and Ambu is explained as the factors which plays a main role in production of the disease Ekakushta.

Symptoms like Matsya shakalopamam, Krishna aruna mandala, Mahavastu and Abhrakapatra sadrusha lesions are the cardinal features of Ekakushta and they are similar to that of Chronic Plaque Psoriasis.

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