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Bullous Disorder - An Ayurvedic review study

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ABSTRACT

Vesicles and bullae are accumulation of fluid within or under the epidermis. Blisters, whether large bullae or small vesicles, can arise in a variety of conditions. Blisters may result from destruction of epidermal cells (a burn or a herpes virus infection). Loss of adhesion between the cells may occur within the epidermis (pemphigus) or at the basement membrane (pemphigoid). Sometimes, there are associated inflammatory changes in the dermis (erythema multiforme/vasculitis) or a metabolic defect (as in porphyria). In Ayurveda all skin disease have been described under the umbrella of *Kustha. Kushtha* is divided into two types *Maha Kushtha* and *Kshudra Kushtha. Mahakushtha* again divided in to seven types and *Kshudra Kushtha* into eleven types. Out of eleven types *Vishphot* is one of them. Vesiculobullous disorder resemble to *Vishphot.* The objective of this article is to analyze *Vishphot* and *Charmadal* its etiology, etiopathogenesis, management from different Ayurvedic literature. Though *Vishphot* and *Charmadal* is included under *Kushta*, hence etiology, etiopathogenesis, and management are same. *Sapta Dravya* i.e., Three *Dosha* and *Tvak, Rakta, Mamsa, Lasika* plays an important role in etiopathogenesis of *Kushtha* as well as *Vishphot* having predominance *Pitta Kapha Dosha.*

Key words: *Kustha, Vesiculobullous disorder, Vishphot, Shodhan, Shaman*

INTRODUCTION

The skin is an important and largest organ of our body which communicate with the external world. It is one of the five *Gyanendriya* described in *Ayurvedic* text which is responsible for *Sparsh Gyan* or touch sensation.[1] Most of the skin disorders have been describe under the umbrella of *Kustha.* Skin is a mirror that reflects internal & external pathology & thus helps in diagnosis of diseases. Large community prevalence studies have demonstrated that between 20-30% of the population have various skin problems requiring attention. In Dermatology, we can observe a wide array of skin manifestations with different names. In present day science, it is observed that there are over 2000 skin disorders. Depending upon the aetiology, they can be classified into various groups such as, Genetic, Infectious, Allergic, Autoimmune, Traumatic, Developmental, Occupational, Climatic etc. Vesicles and bullae are accumulations of fluid within or under the epidermis. They can have varied causes, but in most cases, clinical diagnosis is based on some salient clinical features, which have to be confirmed by investigations. The appearance of a blister is determined by the level at which it forms. Tense bullae are characteristic of blistering diseases with subepidermal split level such as pemphigoid, whereas flaccid bullae that break easily are seen in bullous diseases with intraepidermal split (such as pemphigus). Vesiculobullous disorder are closely resemble to *Vishphot*, *Charmadal* and *Kachhu* which is described in Ayurvedic classics.

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Materials and Methods

Material is collected from *Brihatrayi* and *Laghutrayi.* Various textbook of dermatology. From National and International journal research paper and review articles

Disease Review

Vesiculobullous Disorder in Modern Medicine

Subcorneal and intraepidermal disorder

Pemphigus group

Pemphigus refers to a group of disorders with loss of intraepidermal adhesion because of autoantibodies directed against proteins of the desmosomal complex that hold keratinocytes together. The desmosome is a complex structure, with many of its components targets for autoantibodies. In pemphigus, desmogleins 1 and 3 are important and they have a variable distribution in the epidermis. Desmoglein 3 is crucial for cell adhesion and is found in the oral mucosa and the lower layers of the epidermis, while desmoglein 1 is almost only present in the skin and most expressed in the upper layers. Thus, pemphigus foliaceus never involves the mucosa and has superficial erosions, while pemphigus vulgaris often presents with oral disease and may have full- thickness acantholysis.[2]

Pemphigus vulgaris

Severe, potentially fatal disease with interaepidermal blister formation on skin and mucosa caused by autoantibodies against desmogleins pemphigus vulgaris is particularly common in people of Mediterranean or Indian origin. Patient develop antibodies against desmoglein 3 and letter desmoglein 1. The bound antibodies activate protease that damage the desmosome, leading to acantholysis clinical features - Oral mucosa, scalp, face, mechanically stressed area, nail fold involvement. Flaccid blister are not stable, 70% of patient oral involvement start in the mouth with painful erosion.[3]

Pemphigus Foliaceus

Pemphigus foliaceus (PF) is an acquired autoimmune blistering disease in which the body's immune system produces immunoglobulin G (IgG) autoantibodies that target the intercellular adhesion glycoprotein desmoglein-1 (dsg-1). The binding of these autoantibodies to dsg-1, which is principally expressed in the granular layer of the epidermis, results in the loss of intercellular connections between keratinocytes (acantholysis) and the formation of subcorneal blisters within the epidermis. The ultimate clinical manifestations of this process are fragile, superficial blisters and bullae of the cutaneous surface that easily rupture to yield erosive lesions.[4] Site of predelication include scalp, face, chest and back with diffuse scale and erosion

Subepidermal Immunobullous Disorder

Bullous Pemphigoid

Bullous pemphigoid (BP) is a relatively common autoimmune vesicobullous disease encountered in India. It is a subepidermal bullous disorder most commonly seen in the elderly and manifests as tense blisters on urticarial base, predominantly over flexures, and is associated with pruritus.[5] Autoantibodies are directed against two hemidesmosomal proteins. BP 230 or BP antigen 1 (BPAG1), a 230 kD component of the inner plaque of the hemidesmosome. BP 180 or BP antigen 2 (BPAG2), a 180 Kd transmembrane glycoprotein also known as type XVII collagen. BP 180 is more likely to be more involved in the initial immune response, since it is transmembrane. Before blisters develops pruritus, dermatitis and urticarial lesion may be seen. Tense bullae on erythematous base are characteristics.

Pemphigoid Gestationis

This is pemphogoid occurring in pregnancy or in the presence of a hydatidiform mole or a choriocarcinoma. Due to an HLA mismatch between the mother (HLA-B8, DR3 OR DR4) and father (HLA-DR2), the child is sensitized against placental antigen, BP 180 and less often BP 230. Grouped, periumblical, tense blister with pruritis develop in second or third trimester and persist until delivery and resolve within 3 month.

Cicatrical Pemphigoid

Cicatricial pemphigoid is a rare, chronic autoimmune blistering disorder which can produce scarring. It can

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affect the skin only, mucous membranes only, or both the skin and mucous membranes. When only mucous membranes are involved, the disease is often referred to as mucous membrane pemphigoid. When only the ocular membranes are involved, it may be referred to as ocular pemphigoid. Risk of scarring depends on the location of disease activity.

Initial diagnosis can be a challenge. Due to the risks of serious complications, such as blindness and airway compromise, early and aggressive treatment initiation may be warranted.[6] Several different target antigen BP180, BP230, Laminin 5, alpha 6 beta4 integrin, laminin 332 and type 7 collegen.

Epidermolysis Bullosa Acquisita

Epidermolysis bullosa acquisita (EBA) is an autoimmune subepidermal bullous disorder of the skin and mucous membranes. The disease results from the production of immunoglobulin G (IgG) antibodies against type-VII collagen, a major component of anchoring filaments in the dermal-epithelial junction. The disease has two major forms of presentation: the classical (non-inflammatory) type and the inflammatory type. Classical EBA is mainly characterized by the following features: development of non-inflammatory tense blisters on trauma-prone areas, multiple milia cysts, minimal or no inflammation findings on histopathology. Alternatively, inflammatory EBA is defined by widespread inflammatory blistering eruptions and a neutrophil-rich inflammatory infiltrate on standard histopathology.[7]

Linear IgA Disease

Chronic bullous disease of childhood. This is subepidermal blistering disease caused by deposits of IgA along BMZ. It is seen more commonly in female. Most common subepidermal blistering disorder in childhood. The lesion is distributed periorifacially in a rosette fashion. The classical arrangement of lesion is called the String of pearls or crown of jewels appearance

Dermatitis Herpetiformis

Pruritic vesicular disease caused by IgA autoantibodies directed against epidermal transglutaminase and presenting with granular pattern in papillary dermis. the classic clinical presenting of DH is very itchy polymorphous skin eruption comprising of erythema, urticarial plaque, papules, vesicles, excoriation and purpura sometimes in herpetiform configuration. the lesion typically on the extensor surfaces of the body such as knee, elbow and sacrum.

Epidermolysis Bullosa

This is group of disorder with mechanical defects leading to easy blistering caused by defective structural protein.

EB Simlex

Least disturbing form of EB patient tend to easily develop blister from minor mechanical trauma such as crawling on knee and elbow or walking. The most common mutation is in keratine 5 and 14 which is paired and expressed low in the epidermis, either in basal layer or just above.[8]

Junctional EB

In junctional epidermolysis bullosa simplex (JEB), the site of blister formation within skin is the lamina lucida within the basement membrane zone. It causes generalized blistering of the skin and internal mucous membranes of varying severity.[9] The separation occur in the lamina lucida of the basement membrane, usually following mutation in the gene responsible laminin -332 formation.

Dystrophic EB

Most severe form mutation in the type VII collagen, the main component of the anchoring fibril in the papillary dermis invariable scarring often mutilating. In the autosomal dominant type blister appear in the late infancy and are localized to the friction site healing with scarring and milia formation.

Recessive dystrophic epidermolysis bullosa is a fetal form of epidermolysis bullosa where in extensive, sometime hemorrhagic subepidermal blister start in infancy and heal with scarring the teeth mouth and upper part of the esophagus are all affected.

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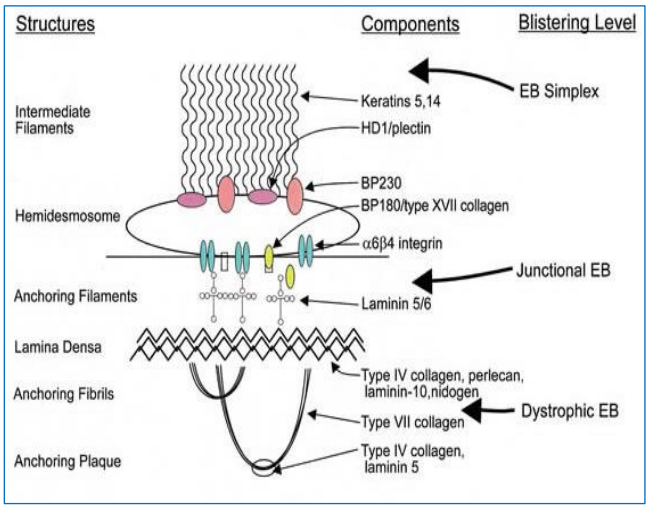

Treatment

Classic standard of care treatments are reviewed, such as dapsone for dermatitis herpetiformis. The advent of corticosteroids has improved mortality rates for many dermatoses, and it remains a crucial aspect of care for diseases such as pemphigoid and pemphigus. However, clinicians should plan for a transition to nonsteroidal therapy to avoid side effects from chronic corticosteroid use. Common alternatives include mycophenolate mofetil, methotrexate, azathioprine, intravenous immunoglobulin, and cyclosporine. Rituximab, a monoclonal antibody against CD20+ B-cells, has been approved as a first-line treatment for moderate-to-severe pemphigus foliaceus and pemphigus vulgaris and offers an excellent steroid-sparing alternative for these disease. Treatments currently revolve around immunosuppressive therapies, including systemic corticosteroids, intravenous immunoglobulin (IVIg), cyclosporine, and TNF-alpha inhibitors. Controversy remains regarding which therapies provide a mortality benefit.[10]

Ayurvedic Review of Bullous Disorder

Visphot (Pemphigus Foliaceus)

स्फोटाः श्वेतारुणाभासो विस्फोटाः स्युस्तनुत्वचः[11]

तनुत्वग्भिश्चितं स्फोटैः सितारुणैःविस्फोटम्[12]

स्फोट - Subcorneal blisters within the epidermis

स्युस्तनुत्वचः - binding of autoantibodies to dsg-1, which is principally expressed in the granular layer of the epidermis (superficial layer).

Blisters are fragile, superficial blisters and bullae of the cutaneous surface that easily rupture to yield erosive lesions.

Charmadal (Bullous Pemphigoid)

सकण्डु सस्फोटं सरुग्दलति चापि यत् ।

तच्चर्मदलमाख्यातं संस्पर्शासहमुच्यते[13]

सस्फोटमस्पर्शसहं कण्डूषातोददाहवत् ।

रक्तं दलच्चर्मदलम्[14]

स्फोटं - Subepidermal bullous in erythematous base

कण्डू - Tense blisters on urticarial base

Kachhu (Dermatitis Herpetiformis)

सास्रावकण्डूपरिदाहकाभिः पामाऽणुकाभिः पिडकाभिरूह्या

स्फोटैः सदाहैरति सैव कच्छूः स्फिक्पाणिपादप्रभवैर्निरूप्या ।[15]

स्फोटैः------स्फिक्पाणिपाद - Tiny blisters and vesicles present over extensor surface such as knee, elbow and sacral region.

सदाहैरति, कण्डू - Intensely pruritic or burning tiny vesicles, which are usually scratched.

Treatment

The skin diseases are long time consuming, easily not curable and require patience to take medication for longer duration. *Kushta Roga* cannot occur without the vitiation of *Tridoshas*. Since the disease manifestation starts from the *Nidana*, first line of treatment should be *Nidana Parivarjana*. It stops in the further progression of the diseases by restricting the vitiation of *Doshas*. The therapy which aims at radical removal of causative morbid factors is called as *Samshodhana*. According to *Acharya Sharangadhara, Kushta Roga occurs* due to *Dosha Bahulyata*. These *Doshas* are *Tiryagami* and very difficult to treat by *Shamana Aushadhi. Acharya Vagbhata* says that, *Snehapanam* is given to the *Kushta Rogi* in the *Purvarupa Avastha*. *Acharya Charaka* states that, in *Vata Dosha Pradhana Kushta*, one should first administer *Virechana* and then give *Niruha Basti* with *Madhuphaladi Sidha Taila.*

Kushta is *Tridoshajanya Vyadhi*, therefore first predominant *Doshas* should be treated and then

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Anubhandha Doshas. Periodical advice of *Panchakarma* procedures indicates the extent of the *Dosha* involvement in the *Kushta Roga. Shodhana Karmas* are indicated in *Bahudoshaavastha*. *Vamana Karma is* indicated for *Kapha Pradhana* and *Doshotklesa Kushta* [in *Charaka Chikitsasthana*]. For this purpose*, Raktamokshana* is done at every six months, *Virechana* is to be done at every one month and *Vamana* is to be given every 15 days. *Shamana* therapy is very useful in treatment of *Kushtha*. After completing the *Shodhana Karma*, *Shamana Chikitsa* is indicated to pacify the remaining *Doshas*. In present life style when people do not have enough time from their busy schedule for *Shodhana* therapy in such cases *Shamana* therapy is to be advised. *Charaka* has described *Shamana* therapy with *Tikta* and *Kashaya Dravyas*. *Shamana Aushadhi* is more effective, when it is administered after *Samshodhana*. The use of external therapy is also important in *Kushtaroga* since the *Sthanasamasraya* and *Vyaktasthana* is *Twacha*. The importance of external therapy can be understood by the references of much different *Lepa* yoga in the classic.[16]

Discussion

Dermatological disorders described in modern medicine many be compared to *Kushtha Roga*. *Kushtha* is '*Kulaj Vyadhi'*. In today's era Dietetic (like a *Virudha Ahara* and *Mithya Ahara),* behavioral (like *Divaswapana, Vyavaya*, expose to cold and hot), environmental, genetic, and immunologic factors appear to play an important role in the pathogenesis of *Kushtha Roga* including psoriasis. 'Stress' is the main factor for manifestation of *Kushtha*. All the three *Dosha* plays major role in etiopathogenesis of *Kushtha,* but predominance of any one leads to classification of *Kushtha* in to *Maha* and *Kshudra*. *Vishphot* having predominance of *Vata Kaphaj* and *Charmadal, Pitta Kapha Doshaj*. Stress is the common factor for the manifestation of *Kushtha* a in this context Charak says skin has an internal relationship with *Mana*, hence stress gives negative impact directly or in directly on *Mana*.

Conclusion

In every *Samhita*, etiological factors explained are *Raktadushtikar.* Acharya Sushrut along with eating unhealthy food mentioned as a etiological factor in *Kushtha* The present review has mainly focused on different aspects of etiopathogenesis of *Kushtha Roga* as well as *Vishphot, Chrmadal* and having similarity with Pemphigus group of disorder on the basis of clinical features. All Acharya's explain the etiopathogenesis of *Vishphot* and *Charmada*l are *Rakta Dushtikarak*. By this study we can conclude that *Ayurveda* has effective treatment for psoriasis. The cost therapy is minimal. The side effects are minimal. Natural sources are easily available and easy to perform the medicine.

References

1. http://en.wikipedia.org/wiki/Obesity.in.Indiadated-27|03|2017.
2. http://W Sharma T, Halayudh Kosha Hindi Commentary, Varanasi, Choukhambha Surbharati; 2001. P87.
3. Kabir Sardana and Pallavi Ailawadi Textbook of dermatology and sexually transmitted diseases with HIV infection, CBS Publisher and Distribution Pvt. Ltd 2019 First edition.
4. ABC of Dermatology, Sixth Edition. Edited by Rachael Morris-Jones. © 2014 John Wiley & Sons, Ltd. Published 2014 by John Wiley & Sons, Ltd.
5. Kirk A. James, Department of Dermatology, University of North Carolina at Chapel Hill, Mailing Address: 5101 Thurston-Bowles Bldg, CB#7287, Chapel Hill, NC 27599-7287, Phone: N/A, Fax: (919) 843-5766, ude.cnu.dem@semaj_krik;
6. 5.Khandpur S, Verma P. Bullous pemphigoid. Indian J Dermatol Venereol Leprol 2011;77:450-455.
7. Tolaymat L, Hall MR. Cicatricial Pemphigoid. [Updated 2022 Sep 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan.
8. Beiu C, Mihai M, Popa L, et al. (December 15, 2019) Epidermolysis Bullosa Acquisita: A Case Report of a Rare Clinical Phenotype and a Review of Literature. Cureus 11(12): e6386. doi:10.7759/cureus.6386.

ISSN: 2456-3110 **REVIEW ARTICLE** May 2023

9. Kabir Sardana and Pallavi Ailawadi Textbook of dermatology and sexually transmitted diseases with hiv infection CBS publisher and distribution pvt. ltd 2019 First edition.
10. https://dermnetnz.org/topics/junctional-epidermolysis-bullosa.
11. Huang, S.; Hsu, S.; Motaparthi, K. Vesiculobullous Diseases. Medicina 2022, 58, 186.https://doi.org/10.3390/medicina58020186.
12. Tripathi B, Charak Samhita (5th ed.) Chikitsa Sthana 7/25.Delhi, Chaukhamba Sanskrit Pratishthan, 2014.
13. Srikanthmurty K.R Ashtang Hridaya of Vagbhata: edited with Sarvanga sundari (9th ed.) Nidan.14/27). Varanasi, choukhambha surbharati ;2008.
14. Tripathi B, Charak Samhita (5th ed.) Chikitsa Sthana 7/24.Delhi, Chaukhamba Sanskrit Pratishthan, 2014.
15. Srikanthmurty KR. Ashtang Hridaya of Vagbhata: edited with Sarvanga sundari (9th ed.) Nidan.14/28). Varanasi, Choukhambha Surbharati ;2008.
16. Dutt A, Sushrut Samhita of Sushrut Nidan Sthan 5/14. Varanasi, Chaukhamba Sanskrit Sansthan;2006.p.87
17. Rozy Sharma et al. Review on the disease Kustha and its management in Ayurvedic literature ISSN: 2456-3110 Review Article Mar-Apr 2021 Journal of Ayurveda and Integrated Medical Sciences, Mar - Apr 2021.
18. HO.int>nutrition>topicics>obesitydated-30|03|2017.

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