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 Kushta. 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: Kushta, 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 Kushta. 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.
MATERIALS AND METHODS

Material is collected from Brihatrayi and Laghu trayi. 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.

Pemphigus vulgaris

Severe, potentially fatal disease with interepidermal 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.

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.

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. 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.

Cicatricial Pemphigoid

Cicatricial pemphigoid is a rare, chronic autoimmune blistering disorder which can produce scarring. It can
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. Several different target antigen BP180, BP230, Laminin 5, alpha 6 beta4 integrin, laminin 332 and type 7 collagen.

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

**Charmadal (Bullous Pemphigoid)**

सकारात्मक संयंत्र संस्थान विकारात चिकित्साली यथा वसन्तसहस्रमुद्गोऽ
तथा तत्तथामध्यात संस्थानसहस्रमुद्गोऽ
स्फोटस्मपर्यंतकण्टः कण्ठातोऽदधायति
रक्तं दत्तातर्मलांगी
स्फोटः - Subepidermal bullous in erythematous base
कण्ठः - Tense blisters on urticarial base

**Kachhu (Dermatitis Herpetiformis)**

स्फोटं - Subcorneal blisters within the epidermis
स्फोटं - Binding of autoantibodies to dsg-1, which is principally expressed in the granular layer of the epidermis (superficial layer).

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 diseases. 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)**

स्फोटः - Subcorneal blisters within the epidermis
स्फोटः - Binding of autoantibodies to dsg-1, which is principally expressed in the granular layer of the epidermis (superficial layer).

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
Anubhandha Doshas. Periodical advice of Panchakarma procedures indicates the extent of the Dosha involvement in the Kushtha Roga. Shodhana Karmas are indicated in Bahudoshavaavastha. Vamana Karma is indicated for Kapha Pradhana and Doshotklesa Kushtha [in Charaka Chikitsasthana]. For this purpose, Raktaamokshana 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 Sthanasamsraya and Vyaktaasthana 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, Charmadal and having similarity with Pemphigus group of disorder on the basis of clinical features. All Acharya’s explain the etiopathogenesis of Vishphot and Charmadal are Rakta Dushtikar. 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.

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