Management of Contact Dermatitis: A Literature Review

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Abstract

An inflammatory skin reaction known as contact dermatitis can be brought on by a variety of factors. Most frequently, substances from the outside of the body could be an irritant or allergen and induce an inflammatory response. Contact dermatitis is a common disease in the community, counting for 20% of the general population. Hence, the management of contact dermatitis is very important as many patients do not know that they had contact dermatitis and do not know how to treat them. The findings of this research are that contact dermatitis could be managed by applying the rule of 4R which stands for recognize, remove, reduce, and restore.

Keywords: ACD, ICD, Contact Dermatitis

1. Introduction

Contact dermatitis is a response of the skin in the form of inflammation which can be acute or chronic, due to exposure to external irritants on the skin. There are two types of contact dermatitis, namely irritant contact dermatitis and allergic contact dermatitis. Irritant contact dermatitis (ICD) is inflammation of the skin, as a result of a response to exposure to an irritant, physical, or biological contact with the skin, without being mediated by an immunological response. While allergic contact dermatitis (ACD) is dermatitis that occurs as a result of exposure to allergens outside the body, supported by type 4 hypersensitivity reactions[1].

2. Pathophysiology

2.1 ACD

Allergic contact dermatitis is a disease that is strongly influenced by the external environment. The most common causes of ACD are age, workplace, and gender. This gender affects because ACD occurs most often in young women. ACD can be divided into 2 major phases, namely the sensitization phase and the elicitation phase[2]. The sensitization phase is the first phase where skin contact with allergens occurs. Meanwhile, the elicitation phase is the continuation phase of the first phase where the patient will be exposed to allergens again and will cause clinical manifestations. Usually, this second phase can occur in 1 to 3 days.

Strong experimental contact sensitizers such as 2,4-dinitro-1-fluorobenzene (DNFB), dinitrochlorobenzene (DNCB), trinitrochlorobenzene (TNCB), and oxazolone, which have unique chemical and immunological features but are not found in our daily environment, opened the path for this research to a better understanding of the pathophysiology of ACD

2.1.1 Sensitization Phase

Most of the hapten is derived from the lipophilic residues, which successfully penetrate the corneal barrier and make bonds with the nucleophilic residues[3]. The collected hapten will trigger the innate immune system and stimulate T Cells in this sensitization phase. Langerhans cells and dermal dendritic cells then capture the resulting hapten protein complex and channel it to the lymph nodes. The next process is the distribution to the bloodstream and skin tissue. The components distributed into the bloodstream are hapten-specific T cells generatic effectors and memory T cells. IL-1β, IL-1α, IL-8, IL-18, IL-23, TNF-α, and granulocyte-macrophage colony-stimulating factor (GM-CSF) are secreted in the patient's skin during this sensitization period [4].

2.1.2 Elicitation Phase

The second phase will occur when the patient is in contact with the same hapten that was exposed in the first phase. Hapten that enters this second phase will meet the effector and lymphocyte memory T. Unlike the first phase, Langerhans cells do not have as much role as in the first phase. In this elicitation phase, most of the T cells involved are non-specific t cells. The low threshold of the patient triggers a large number of heptanized cells. The entry of hapten will trigger Langerhans cells and keratinocytes to release chemokines and pro-inflammatory cytokines such as IL-18. TNF-a, and IL1B. After all of this, the patient will experience an eczematous reaction to the skin[4].

2.2 ICD

Physical and chemical substances can cause damage to epidermal cells and will make epidermal lipids disappear from the epidermal layer[5]. After that, there are trans epidermal water loss (TEWL) and increased permeability of the skin. This can be interpreted as the onset of irritant contact dermatitis and will result in the stimulation of lymphocytes and induce activation of T cells due to the presence of chemokines, cytokines, and adhesion molecules. Several studies have been carried out and from that can be concluded that acetone can extract lipids from the stratum corneum and lauryl sulphate (SLS), cobetaine and sodium dodecan sulphonate (SDS) can damage involucrin, keratin, profilaggrin, and some protein structures that are regulated for saturation of the stratum corneum, and disorganization of lipid bilayers[6].

In ICD, keratinocyte activation will occur after penetration of the irritant into the epidermal skin barrier. The activation of these keratinocytes causes a cytokine cascade. Proinflammatory cytokines such as IL-6, IL-1 beta, and tumor necrosis factor (TNF) alpha will activate the innate immune system. IL-1 beta, IL-6, and TNF alpha will also activate T cells. Mononuclear and polymorphonuclear cells present at the site of injury trigger the opening of CCL20 and CXCL8. Active mediators such as CXCL8, CXCL1, and CCL2 will also be stimulated by fibroblasts. This event will result in the removal of Langerhans cells from the epidermis and an increase in molecular adhesion which will attract more immune cells to the skin area[6].

3. Epidemiology

For the epidemiology of ACD, several studies have been carried out related to this ACD. ACD is a common disease as shown by several studies where the prevalence rate is 20% of the general population[7]. A 1998 to 2000 study conducted by the North American Contact Dermatitis Group using 5839 patient data concluded that 60% of patients had ACD out of the total dermatitis cases encountered in these patient data.

In a study conducted in the USA, Templet, Hall, and Belsito using a sample of 1034 patients and conducted for 8 years, it was concluded that 32% of cases had hand dermatitis, 54% had ACD, and 27% had ICD. In previous studies, it can also be concluded that there are some patients who experience hand dermatitis and also

face dermatitis. This could be caused by the contact between the hands and the patient's face which caused the patient's face to also experience dermatitis.

Meanwhile, it can be found that 80% of contact dermatitis cases are ICD [8]. The most common cause of ICD disease is occupational disease. In addition, the behavior of people who use body cleaning products or cosmetics can also be the cause of this ICD. However, this can be avoided by knowing what ingredients can trigger ICD, and not using products that can trigger ICD in patients [9].

Dr. Soetomo General Academic Hospital has also conducted contact dermatitis research. The study was carried out using a retrospective strategy based on secondary data that was already available. According to a study done at the Dr. Soetomo General Hospital in Surabaya between 2014 and 2017, the number of instances that occurred was fairly linear and did not exhibit a substantial increase from year to year. At the Outpatient Installation (IRJ), there were 228 instances of contact dermatitis in 2014, 344 cases in 2015, 252 cases in 2016, and 281 instances of contact dermatitis in 2017[10].

4. Etiology

Nearly any substance may cause aggravation dermatitis. In any case, the concentration and length of contact with the irritant decide the probability of ICD. Natural components, counting dry discuss and high temperature, can increase the aggravation impact of a contact operator. Solvents such as liquor and xylene, metalworking liquids, elastic gloves, sodium lauryl sulfate, hydrofluoric corrosive, alkalis, and plants are well-known causes of ICD [11]. Nonstop or tedious introduction to gentle irritants, that's, water and cleansing gels, is additionally a common offender for ICD and is known as aggregate ICD. Nickel, neomycin, cobalt, scent blend, thiomersal, and formaldehyde rank tall as the common allergens causing ACD[4].

The use of cosmetic ingredients is often one of the causes of contact dermatitis[12]. The cosmetics in question can be in the form of hair dyes, nail dyes, or ingredients that are used on the patient's skin. However, because the patient has used it for a long time and often, the patient is often not aware of it and even cosmetics rule out that is used as a trigger. In the use of cosmetics, not only the ingredients can cause contact dermatitis. The tool or applicator used can also be one of the causes of contact dermatitis in patients (Tan, Rasool and Johnston, 2021).

There is also some related factor that can cause ICD such as removed lipid surface and water-holding substances. The damaged cell membrane can also be one of the causes of ICD. It is caused by the increase of cytokine release due to the disruption of the skin barrier of the body. While the last thing that can cause ICD is a direct cytotoxic effect[13].

5. Therapy

There are “4 R” rules to manage dermatitis in patients. Those 4Rs are recognize, remove, reduce, and restore.

5.1 Recognize the causative agent

Before giving treatment to the patient, the doctor needs to identify what is the causative agent or irritant agent of the patient[14]. That is why patch testing and the patient’s substance contact history are important to diagnose the causative agent of the patient. Patch test has become a standard procedure in diagnosing the type of patients’ contact dermatitis as it is able to provoke the elicitation phase thus allowing us to determine the agents that cause the reaction[15]. Patch test can be conducted in adults and children which will be indicated when the patient has contact allergy in any site such as the extremities or the face. However, this test is contraindicated in patients with history of severe allergic reactions to suspected allergens, generalized active

dermatitis, extensive eczema, or dermatitis in the back region as the test will be carried out in the patient's back[16].

The most important role is to educate the patient and tell them not to get in contact with those substances that can trigger dermatitis[17]. It might be difficult to carry out everyday duties when ACD affects the palms (hand dermatitis), hence it is suggested to use suitable personal protective equipment and administer emollients. The prognosis for moderate contact dermatitis is greatly reliant on the capacity to avoid the irritating allergen. Even after changing occupations, severe contact dermatitis caused by employment might cause symptoms to continue for up to two years [1].

5.2 Remove the irritant/allergen

The most important thing in the healing process of therapy is the prevention of materials that can cause dermatitis. The next preventive way is to prevent complications from occurring. So, people affected by contact dermatitis should immediately visit a dermatologist to get the right treatment. While the last prevention is to improve the disease of patients who are already chronic and help people affected by this disease to be able to return to their activities in their environment again.

Actions that can be taken by patients are to look at the labels of the products they use. By looking at the label, sufferers can find out whether the ingredients in the product can irritate their skin or not. In addition, patients can use self-protection. The recommended protection can be in the form of barrier creams, emollients, gloves, etc. In everyday life, sufferers must do the right cleaning product for them. The right cleaning product is one that does not use fragrance and does not irritate the skin[17].

5.3 Reduce inflammation

The choice of this therapy should be based on the severity, acuteness, as well as morphology, and type of dermatitis. Basic therapy is needed as the main treatment in dealing with contact dermatitis. For acute dermatitis, it is recommended to use soaks with hand baths and wet dressings. Some solutions that can be used are potassium permanganate, aluminum acetate, saline, and synthetic tannins. This treatment is useful for removing dried crust and scales. In addition, the resulting cooling effect is also beneficial for the patient. However, it should be noted that the cold effect can cause hypothermia when applied to a large area. In some cases, there are indications of infection, and the doctor may prescribe antibiotics to fight the bacteria causing the infection. However, to avoid the occurrence of resistance, topical antibiotics are rarely used by doctors. As an alternative to a topical antibiotic, potassium permanganate is often used by doctors as a medication for dermatitis, cleaning wounds, and general disinfection. Its advantages include lower cost, good healing rate and reduced allergenic potential. However, it may leave stain to the skin and cause serious adverse effects such as chemical burns and systemic toxicity, hence the use of this substance must be used with cautions[18].

For the category of sub-acute dermatitis, the drug given must contain anti-inflammatory, moisturizer, and antipruritic. Topical treatments including urea and polidocanol are advised but polidocanol allergy has been recorded, particularly in older individuals with dermatitis of the lower legs. Additional anti-proliferative properties of ichthyol and tar formulations, on the other hand, include pyrenes that are known to cause cancer in animals. Higher systemic exposure also correlates with increased cutaneous effects. Anti-inflammatory drugs that are widely used to treat contact dermatitis is topical corticosteroids such as methylprednisolone or dexamethasone[19]. However, long-term use of topical corticosteroid is not recommended as it could damage the skin barrier[20].

Rich moisturizing ointments are recommended for persistent dermatitis. In situations of hyperkeratosis, such as tylotic hand eczema, keratolytic ointments are utilized. Tar preparations are also beneficial here because of

their antiproliferative properties. In persistent dermatitis, fissures are common. They are painful, take a long time to heal, and can become infected. The recovery process will be improved if the epidermis gets a softening treatment and sealing fissures treatment[17].

5.4 Restore skin barrier

Without steps to repair the skin barrier, contact dermatitis therapy is incomplete. Irritative contact dermatitis demands not only clinically normal skin but also functional normalcy, which includes a return to normal skin physiology and the absence of irritation. The maintenance of the skin barrier is very important as it is related to the effectiveness of the treatment and affects its prognosis[20].

After an incident of dermatitis, it takes weeks to months for the skin barrier function to restore. Chronic contact dermatitis takes longer to heal than acute contact dermatitis. Emollients have a crucial role in the treatment of contact dermatitis and the restoration of the skin's barrier function. Contact dermatitis therapy options have an impact on barrier function repair. While topical and systemic corticosteroids and retinoids have side effects, topical calcineurin inhibitors allow for normal recovery and UV phototherapy improves the skin's barrier function[17].

Restoration of skin barrier function can be achieved by the use of moisturizers and emollients. Moisturizers binds the water molecules to hydrate the stratum corneum as it contains humectants. While emollients prevent evaporation of water from the skin surface by forming a semi-occlusive layer on the surface of stratum corneum. Some emollients impact the skin barrier physiologically hence it could restore the barrier function by relieving itch and inflammation in ICD and ACD[19].

6. Conclusions

It can be concluded from the literature that reducing inflammation and restoring the skin barrier with drug prescription is needed as therapy for contact dermatitis. However, recognizing the causative agent and removing it from the body also play a big role in the therapy hoping that they will not cause another contact dermatitis in the future.

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