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REVIEW ARTICLE **OPEN ACCESS**

A Review on Atopic Dermatitis

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ABSTRACT
Various studies since ages have been proving that "AD is a skin condition characterized by inflammation, which typically starts at a young age and affects about 20% of the population over their lifetime. The cause of atopic dermatitis is still unknown, but the recent finding of filaggrin mutations suggests that it may be possible to prevent the development of asthma in later childhood. It is important for every physician to have a good understanding of the fundamental aspects of treatment for atopic dermatitis, as it can be challenging to manage". Thus, we have reviewed and discussed AD in terms of cause, pathology, risk factor, D/D, treatment, management, prognosis and complication aspects.
Key words: *AD, cause, pathology, risk factor, D/D, treatment, management, prognosis, complication aspects.*

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INTRODUCTION

Studies have concluded that "atopic dermatitis (AD) is a common type of skin condition characterized by inflammation".[1] Furthermore, studies have concluded that it is a "chronic condition that can recur and primarily affects young children".[1] There is also evidence that "some people are genetically more likely to make immunoglobulin E (IgE) antibodies in response to small amounts of common environmental proteins, such as those found in pollen, house dust mites, and food allergies".[1] One more important point is that studies have concluded that A can be inherited across generations. Furthermore, studies have concluded that the "term "D" is derived from the Greek words "derma" and "itis," which respectively mean skin and inflammation".[1] While studies have also come to the conclusion that the term eczema typically refers to the "acute form of the condition, dermatitis and eczema are often used interchangeably".[1] Other than this, studies have also shown us that due to the fact that "allergic sensitization and increased immunoglobulin E (IgE) are only present in about half of the patients with the illness, the term "AD" cannot definitively describe the condition".[1] Studies have concluded that this "chronic disorder that is associated with pruritus often starts in childhood and presents with dry skin, eczematous lesions, and lichenification as symptoms. Studies have concluded that it is speculated that AD is linked to other IgE-associated illnesses such as allergic rhinitis, asthma, and food allergies. Studies have concluded that the morbidity associated with AD is significant, and it would seem that the prevalence of the disorder has been steadily increasing over the course of the last several decades".[2] Thus, we have reviewed & discussed about AD in this paper.

CAUSE

According to studies, there are a number of different factors, some of which are genetic and others of which are environmental, that may contribute to the development of atopic dermatitis. Studies have concluded that these factors have the potential to cause abnormalities in both the epidermis and the immune system. Various other studies have concluded that the atopic triad comprises allergic rhinoconjunctivitis, asthma, and atopic dermatitis, and atopic dermatitis is one of the diseases that make up this triad. In addition to this, studies have concluded that the onset of all three of these diseases may occur simultaneously or in a specific order, which is referred to as the "A march." Studies have also found that people with the atopic triad have problems with the barrier function of their skin, upper respiratory tract, and lower respiratory tract. This is what causes their symptoms. Furthermore, studies have concluded that this insufficiency is the root cause of the "A" triad. Studies have further concluded that there is a greater than fifty percent chance that an atopic parent will pass their condition on to their offspring. Studies have also concluded that it is possible that up to eighty percent of the offspring will be affected if both of the parents are afflicted with the condition. Several studies have shown that these

genetic disorders cause changes in the epidermis that stop the filaggrin protein from working properly. Studies have concluded that this protein is then broken down into a natural moisturizing factor. Furthermore, studies have concluded that this natural moisturizing factor is produced when filaggrin is broken down. Some studies have also found that people with atopic dermatitis may be more likely to get keratosis pilaris, ichthyosis vulgaris, and allergic rhinitis if they have filaggrin mutations. These mutations happen in up to 30% of cases. Depending on the severity of the condition, studies have concluded that food hypersensitivity may possibly cause atopic dermatitis or make the condition worse in ten percent to thirty percent of patients. Additionally, studies have concluded that eggs, milk, peanuts, soy, and wheat are responsible for ninety percent of the reactions or flare-ups that are caused by these foods.[3,4] According to recent studies, smoking and the start of Alzheimer's disease in adults may be linked.[2]

PATHOLOGY

Studies have shown that, there are mainly 2 hypotheses which are as follows:-

1. **Immunological Hypothesis =** According to the "immunological imbalance theory, atopic dermatitis is caused by an imbalance of T cells, most specifically T helper cell types 1, 2, 17, and 22, as well as regulatory T cells". [5] Furthermore, studies have been proving that "the Th2 differentiation of naive CD4+ T cells is most prevalent after an allergic response, commonly known as atopic dermatitis, and particularly during an acute episode of eczema".[1] Thus , studies concluded that , this results in an increased production of interleukins, most notably IL-4, IL-5, and IL-13. This, in turn, generates an increased quantity of IgE, which prevents the differentiation of Th1 cells.[1]
2. **Skin Barrier Hypothesis =** According to the studies, a "more modern theory of skin barrier defects comes from the finding that people with changes in the filaggrin gene are more likely to develop atopic dermatitis" [6]. Furthermore, studies have shown that the "keratinocytes are kept together by structural proteins that are expressed by the filaggrin gene. This gene is located in the stratum corneum and stratum granulosum layers of the epidermis. Keratinocytes are found in the outermost layer of the epidermis. This contributes to the maintenance of the stratum corneum's hydrated condition as well as the structural integrity of the skin barrier. Less filaggrin is produced as a result of gene defects, which in turn cause transepidermal water loss and a failure of the skin barrier, both of which lead to the development of eczema".[7] There is evidence to suggest that an "impaired skin barrier, which results in dry skin, leads to increased penetration of allergens into the skin, which results in allergy sensitization, asthma, and hay fever". [7] Futhermore, studies have concluded that "dry skin is the result of an impaired skin barrier. Dry skin is the result of an impaired skin barrier".[1] Addition to this, studies revealed that " breakdown in the skin's natural barrier is the immediate result of dry skin. Emollients applied to the skin at a young age may be able to lower the risk of developing dry skin and active eczema, which may be regarded as a target for primary prevention of the development of eczema into allergic airway disease".[1]

RISK FACTOR

Studies have shown that "people who come from families that are affected by the condition have a significantly increased risk of having AD. For instance, the concordance rate of AD in monozygotic twins is around 75%, which indicates that the risk of the disease being passed on to the twin brother is 75% if the affected cotwin is present" .[8] In addition to this, there is a 75% concordance rate of AD in dizygotic twins. Dizygotic twins, on the other hand, face a risk that is only a third as high as the risk that is presented to monozygotic twins. As a result, this suggests that atopic genetic factors do play a role in the susceptibility to AD that is seen in people. While studies have found that monozygotic twins, who share all of their genes, do not always match, they do not always agree. This means that environmental and developmental factors must also be involved. This is because monozygotic twins share all of their genes with each other. This leads one to conclude that the two must have some kind of connection to one another. As a result, AD is a complex genetic disease that results from numerous interactions, not just between genes but also between genes and the environment. Studies have also shown that these interactions are what make AD such a difficult genetic condition to diagnose and treat. The disease may be caused by these interactions in a number of different ways. Additionally, studies also revealed that AD is a genetic condition that may be difficult to detect and find an appropriate treatment for because of these interactions between the genes.[1] Thus, according to studies 2 risk factors that needs to be understood and be careful was genetic and environmental criteria.[1]

GENETICS

Studies have shown that many genes have been associated with AD, particularly genes encoding epidermal structural proteins and genes encoding key elements of the immune system. A new and

interesting genetic finding is that Alzheimer's disease is strongly linked to changes in the filaggrin gene, which is found on chromosome-1 [6]. Studies have also concluded that the filaggrin gene is the strongest known genetic risk factor for AD. Studies have further concluded that around 10% of people from western populations carry mutations in the filaggrin gene, whereas around 50% of all patients with AD carry such mutations. On top of that, studies have shown that changes in the filaggrin gene make the filaggrin protein not work right, which hurts the skin barrier. Furthermore, studies have also concluded that the clinical expression of such impairments is dry skin with fissures and a higher risk of eczema. Studies have been showing that not all patients with atopic dermatitis have these mutations, and other genetic variants have also been identified [9]. Studies have also found that AD is caused by all of these genetic variants working together with risk factors from the environment and from early childhood. Studies have also found that AD is caused by a combination of several risk factors working together. These include genetic variants that might cause the condition and factors related to the environment and the development process.[1]

ENVIRONMENT

Researchers have only discovered a "handful of environmental risk factors that have been found to be consistently connected with the condition, despite the fact that AD has been linked to a large range of putative environmental risk factors. For instance, there is persuasive evidence to suggest that our western way of life is somewhat to blame for some of the observed increase in the prevalence of eczema over the course of the previous few years. Despite this, neither specific environmental risk factors nor immediately applicable prevention interventions have been identified as a direct result".[10] When it comes to explaining the rapid increase in the prevalence of eczema, a large proportion of people believe that the hygiene hypothesis is the most plausible explanation .[11] According to this hypothesis, studies revealed that , an increase in susceptibility to AD disorders has occurred as a result of a decrease in early childhood exposure to classic illnesses, such as hepatitis A and tuberculosis.[12] The primary emphasis that contemporary medicine places on halting the propagation of infectious illnesses is directly responsible for the occurrence of this phenomenon. This trend has been linked to an increase in the prevalence of AD disorders.[12]

In addition, different studies have found that the youngest child has the lowest chance of getting atopic dermatitis. They have also found that kids who grow up on a traditional farm and are exposed to different microflora, like unpasteurized cow's milk, livestock, and livestock quarters, are somewhat less likely to get the disease and allergic diseases in general. These findings support the hypothesis.[13] On the other hand, studies have also concluded that the duration of time that a mother breastfeeds her child is likely to have a positive correlation with the development of the disease.[14] Additionally, a number of studies have shown that elevated social status in the parents is associated with an increased risk of AD in the offspring.[15] In spite of the fact that such observations are hard to interpret, they may also lend credence to the hygiene hypothesis or, at the very least, to the widely accepted view that eczema develops in genetically predisposed people who are exposed to a certain environment that is harmful to their health. This is despite the fact that such observations are difficult to interpret.[1]

DIFFERENTIAL DIAGNOSIS [1]

1. "Allergic Contact Dermatitis
2. Lichen Simplex
3. Lichen Planus
4. Psoriasis
5. Scabies
6. Tinea
7. Seborrheic"

TREATMENT [2]

1. "Topical Treatment
    a) Corticosteroids
    b) Calcineurin Inhibitor
2. Phototherapy
    a) UV-A
    b) UV-B
    c) UV-A +Psoralene (PUVA)
3. Systemic Treatment
    a) Oral Corticosteroid

b) Azathioprine
c) Cyclosporine A
d) Methotrexate"

MANAGEMENT

According to studies, the "four main components of treatment for eczema include avoiding triggers, daily skin care therapy, anti-inflammatory therapy, and various complementary therapies. Studies have also proved that the daily skin care routines for the arms should include the application of emollients twice per day, with the arm application taking place within three minutes after departing from a hot shower or bath in order to avoid arm dryness. Researchers also concluded that ointments are the form of topical treatment that has the potential to be the greasest, but they are also the kind of treatment that is the most occlusive. Other studies have concluded that emollients should be used after topical steroids have been applied in order to "lock in" the impact of the steroids. Studies have also concluded that topical steroids are the first-line therapies for acute flares and should be used before emollients. So, studies have found that people should pick a strength that is strong enough to stop an outbreak right away. Studies also concluded that they should also make sure to taper every other day and do maintenance therapy twice a week (for example, on the weekends) in the areas where the condition is most common. In addition, choose a potency that is powerful enough to immediately manage an epidemic. The condition will therefore be maintained under control as a result of this action. In addition to this, studies also revealed that the use of steroids may lead to a number of unwanted side effects, two of which are reversible i.e. skin atrophy & telangiectasia".[2]

Studies have shown that "sensitive areas like the axilla and groin may need topical nonsteroidal agents, such as the calcineurin inhibitors tacrolimus and pimecrolimus. Besides this, research has also shown that crisaborole, a new non-steroidal drug, works as planned by stopping PDE-4 from doing its job. Furthermore, studies have concluded that this makes crisaborole one of the most recent non-steroidal agents. In addition to this, studies have concluded that if topical agents are unable to effectively treat atopic dermatitis, systemic agents may be required. Studies have also shown that these systemic agents include phototherapy with UVA, UVB, azathioprine, mycophenolate mofetil, and cyclosporine. Besides this, research has shown that the monoclonal antibody dupilumab can block the IL-4 receptor and, as a result, the effects of IL-4 and IL-13. Other complementary therapies include the use of bleach baths (0.5 cup bleach in a full 40-gallon tub) one to two times weekly to decrease S. aureus colonization, low-allergen maternal diets while breastfeeding, and the use of probiotics and prebiotics in pregnant mothers and at-risk infants, which has shown a 50% decrease in the frequency of atopic dermatitis at ages 1 to 4 years old compared to the use of a placebo".[16,17,18]

Studies have shown that "crisaborole topical ointment has only been given the green light for usage in the treatment of AD ranging from moderate to severe cases. This medication, which has been found to reduce skin symptoms, may work by inhibiting phosphodiesterase, which is an enzyme. Other than this, studies also concluded that it is conceivable that probiotics will be helpful to select patients. In addition to this, it is thought that the bacterial products may enhance the immune system and prevent the creation of an allergic IgE antibody response. In addition, it is also recommended from various studies that women take probiotics while they are pregnant as well as when they are nursing their children after the baby is delivered. In addition to this, studies also concluded that this is due to the fact that probiotics assist in the maintenance of healthy gut flora, which is critical for the development of a healthy newborn. Furthermore, studies have found that taking a bath with bleach may reduce the risk of bacterial superinfection. In turn, this may help to alleviate some of the symptoms that are associated with Alzheimer's disease (AD)".[2]

PROGNOSIS

Studies have shown that the majority of patients diagnosed with Alzheimer's disease (AD) really do see at least some degree of recovery during the course of their illness. However, at the same time, studies have also shown that patients who have AD may also have other diseases, such as asthma and allergic rhinitis, which may not get better even while they are getting treatment for AD. This is because studies have shown that AD is a systemic condition that affects the whole body. Other than this, studies have also shown that the great majority of people who get Alzheimer's disease (AD) as children continue to be sick throughout their lives. Studies have also shown that relapses and remissions are the defining characteristics of the condition. During relapses, it is often necessary to take medication in order to control the condition. People who are routinely exposed to allergens such as cigarette smoke, nicotine, pet dander, fumes, pollen, soap, detergent, and wool will continue to experience symptoms, and as a result, their quality of life as a whole will suffer. Other common allergens include dust mites, pollen, and

fumes. Not only is an itching that does not go away and that is persistent over a long period of time uncomfortable, but it is also difficult and expensive to treat. An eruption called Kaposi varicelliform, which is associated with a primary herpes infection, is one of the most well-known outcomes of AD. This rash is caused by the herpes simplex virus. The vesicular lesions first manifest themselves in the eczematous region, but they are able to rapidly spread to the healthy skin that is located in the areas that surround the eczema. With a course of treatment with acyclovir, the severity of the condition may be lessened. Patients who are afflicted with Alzheimer's disease are at an increased risk of suffering from skin infections that are brought on by bacteria such as staphylococcus and streptococcus.[2]

COMPLICATION [1]

1. "Kaposi Varicelliform Eruption
2. Bacterial Infection
3. Uriticaria"

CONCLUSION

We have come to the conclusion that the quality of life may be negatively impacted by AD, a persistent skin disorder. There are remedies that do not require the use of medication that may significantly improve a person's quality of life, and although the condition can be treated with medical treatment, there are other remedies that can be used. In order to educate the patient about non-medical remedies for treating atopic dermatitis, both the pharmacist and the nurse may play an important role. The patient has to be instructed to avoid wool and wear soft clothes instead, with cotton being the preferred option. Because heat may cause perspiration and make irritation worse, the temperature in the home should be maintained at a modest level. It is recommended that a humidifier be used in the home in order to prevent dryness. It is recommended that all clothing be washed with a gentle detergent that does not include any bleach or fabric softener. Before venturing outside, one should lather on copious amounts of sunscreen and moisturizer. The patient should maintain a diet diary and avoid eating foods that bring on the attacks. The patient should avoid doing things that cause them to sweat excessively. Last but not least, patients who have asthma should take their medicines as prescribed and avoid contact with allergens.

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