

Innovative Therapeutic Approach for Chronic and Acute Dermatitis: A Review of Recent Developments

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ABSTRACT

Purpose of Review: Contact with allergens or irritants can cause "contact dermatitis" (CD), a collection of skin conditions. Contact dermatitis, which is brought on by coming into contact with a chemical, results from them. Similar to other forms of allergy, the illness develops in two stages: sensitization in the first phase and a cutaneous inflammatory response in the second step. Symptoms of allergic contact dermatitis might include acute, subacute, or chronic dermatitis.

Recent Findings: The current study examines the pathophysiology and current management of allergic contact dermatitis and makes predictions about the potential for more effective treatment in the future. Managing now plays greater importance in preventing problems before they start, especially in high-risk work environments. This includes focusing on education and awareness, as well as continuing to avoid irritants, use protective barriers, and regularly apply moisturisers.

Summary: A detailed history, the detection of allergic contact dermatitis is made by a physical examination and patch testing. Once the allergen has been identified, avoidance is the cornerstone of treatment. Management of contact dermatitis must include both medical care and, where appropriate occupational adjustments in order to lessen exposure to the causative agents. The existence of this treatable medical problem should be known to physicians

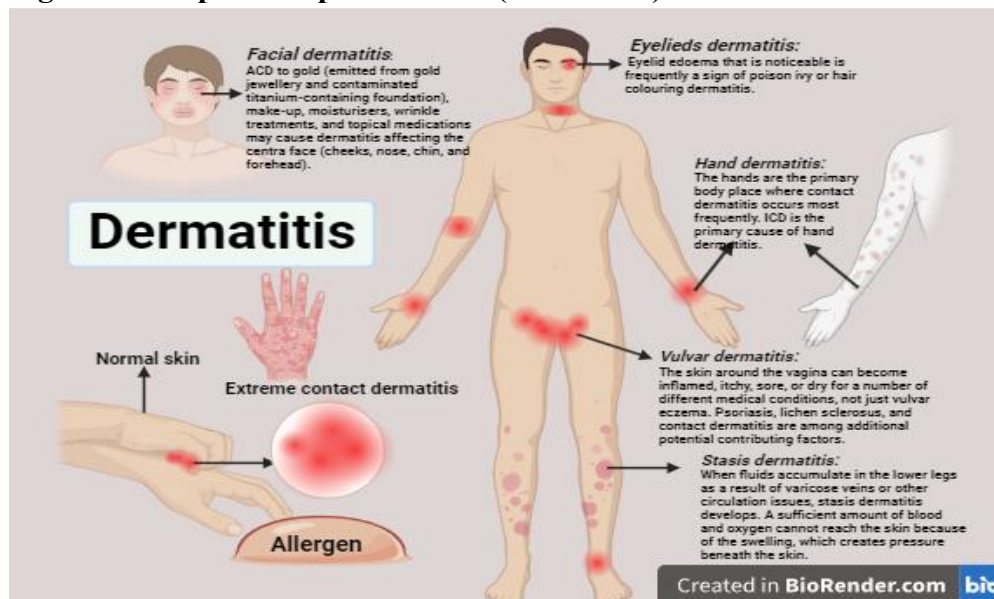
Keywords: Dermatitis due to contact, Atopic contact dermatitis, Irritant contact dermatitis, Allergens.

1. INTRODUCTION

Dermatitis is a multifaceted skin infection (Rios et al., 2005). A series of skin conditions known as contact dermatitis (CD) are brought on by allergens or other irritants that come into touch with the skin (Elmas et al., 2020a). A skin reaction called reactive eczema that flares up after coming into contact with a substance, usually a chemical but occasionally a biologic or physical agent (Al-Otaibi & Alqahtani, 2015). Allergic contact dermatitis (ACD) and irritant contact dermatitis (ICD) are viewed as the entity's two subgroups since they both exhibit the eczematous eruption that separates CD from other dermatological illnesses (Elmas et al., 2020a). ICD is an unspecific cutaneous disease response to direct tissue damage caused by one or more exposures to a noxious substance in contrast to ACD, which is an immunological response to antigens from external contacts and is believed to be a kind type-IV delayed hypersensitivity reaction (Elmas et al., 2020a). In comparison to allergic contact dermatitis (ACD), irritant contact dermatitis (ICD), which makes up 80% of all instances of contact dermatitis, is more common. ICD is an inflammatory response to an irritant that is non immunologically motivated. Clinically, these two types of dermatitis are often indistinguishable (Nassau & Fonacier, 2020). Chemical burns from hydrofluoric acid, hydrochloric acid, or alkali (such as solvents, water, soap, detergents, acid, alkali, etc) can cause ICD, this can develop either gradually over time from repeated contact with an irritating substance, or suddenly after just one exposure to the material (Al-Otaibi & Alqahtani, 2015). ACD is specifically classified as Type IV hypersensitivity, is a type of delayed hypersensitivity reaction. When someone with ACD is exposed to a specific allergen, their immune system reacts by causing an inflammatory response on their skin. Due to the fact that this reaction takes place after an extended period of time (usually 12-72 hours) following exposure with the allergen, it is known as "delayed" hypersensitivity. Exposure to compounds including chemicals, cosmetics, scents, and preservatives can result in allergic contact dermatitis (Nassau & Fonacier, 2020). CD can be acute, subacute, or chronic contact dermatitis are all possible, and each of these stages can advance to the next. The acute phase is marked by swelling, blistering, itching, and redness, as well as the development of crusts and scales. when the body is recuperating Fissures, hyperkeratosis, and dryness define the chronic phase. Atopic dermatitis and contact hypersensitivity are the two most prevalent types of dermatitis (Rios et al., 2005). Dermatitis and eczema are frequently used interchangeably to describe a erythema, vesiculation, and pruritus are the characteristics of a polymorphic noticeable flare-up of skin, especially in the early stages (Rios et al., 2005). Despite being pathogenetically unconnected and having a whole

Abbreviations: ACD (allergic contact dermatitis), ICD (irritant contact dermatitis)

Figure 1: Graphical representation (Dermatitis)



different nature, both illnesses display common physical traits, particularly vesicle production, which lead to a breach in the vesicle development, the skin loses its ability to protect itself, as its natural barrier becomes damaged or weakened (Rios et al., 2005). The goal in treatment of ACD and ICD is to avoid coming into contact with the causative agent, which may be linked to exposure to allergens or irritants in the workplace or other settings. If the cause of the dermatitis is not found or eliminated, it has a tendency to become chronic and adversely affect the standard of life of the patients (Nassau & Fonacier, 2020). According to the most recent comprehensive investigation of the connection between AD and contact sensitization, patch testing in AD patients should only be investigated when ACD is suspected because the frequency of contact allergies among AD patients is comparable to that of the general population (Tramontana et al., 2023)..-

Symptoms

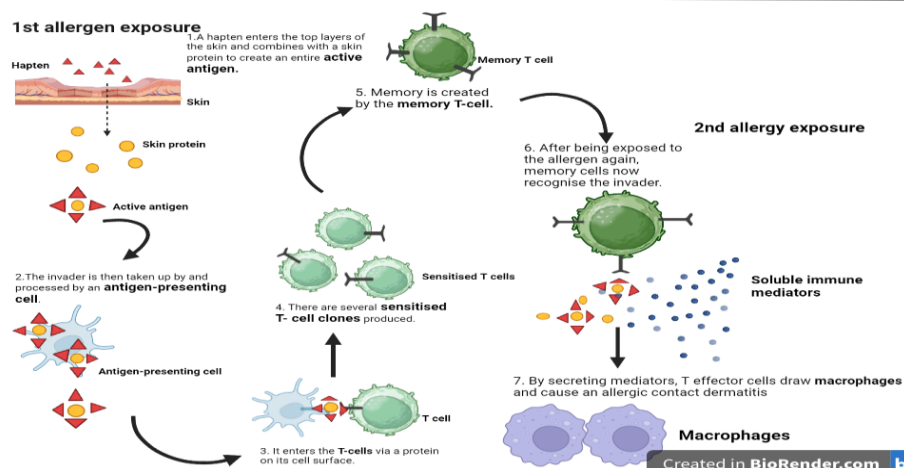
Signs of CD among a skin rash is that, it is:

- ☐ darker than your natural skin tone or from red to purple
- ☐ raised, swollen, or hive-like in the skin around it
- ☐ bumpy with a few tiny blisters or pimples
- ☐ oozing pus or liquid
- ☐ scorching or stinging in nature and painful
- ☐ scaling or flaky
- ☐ Itchy

2. MECHANISM

The mechanism which is involved in contact dermatitis when exposure is in two phases. A complete active antigen is produced when a hapten interacts with a skin protein in the epidermis. An antigen-presenting cell then ingests and processes the intruder. Through a protein on its cell surface, it enters the T cells. Many sensitised T-cell clones have been created. The memory T-cell generates memory. Memory cells now recognise the invader after being re-exposed to the allergen. T effector cells attract macrophages and produce an allergic contact dermatitis by secreting mediators. It is clearly represented in a given below diagrammatic representation.

Figure 2 (It has been explained briefly about the exposure of allergens which is one of the main factor in causing dermatitis)



3. CLINICAL FEATURES

A variety of skin symptoms can help identify the clinical signs of allergic contact dermatitis (ACD). The offending substance's chemical composition, the type and mode of exposure and the clinical and skeletal components of the area of skin damaged can all effect differences (Lachapelle, 2014a)(Elmas et al., 2020b) [6,7]. Erythema, edema, and vesiculation (and periodically bullae, depending on how severe the allergic reaction is) can be seen during the acute phase. A vibrant crimson or pinkish-red in colour with unclear boundaries are characteristics of erythema (Tramontana et al., 2023). Itching is the main symptom and may begin as early as 24 hours after coming into contact with an allergen. Although ICD is more often characterised by scorching and pain. They might also show up in uncommon ACD situations. Eczema is the most common way allergic contact dermatitis (ACD) shows up on the skin and develops 5-7 days after initial encounter with the causative allergen, while a cutaneous reaction only manifests after the second exposure within 24-48 hours(Tramontana et al., 2023)(Lachapelle, 2014a) [5,6]. Due to the significant extent of laxness of the skin's surface and its layers at these location, swelling is typically more serious when it affects the genitalia, face, and eyelids. Vesicles, which often appear a few hours after development of erythema and edema, have a tendency to rupture fast because of where they are located in the epidermis. This leads to numerous confluent erosions and, in more severe cases, abundant leakage(Lachapelle, 2014a) (Elmas et al., 2020b)[6,7].

4. CLINICAL TYPES

Acute ICD:

A severe irritant, such as concentrated acids, potent alkalis, and solvents like acrylonitrile, can cause acute ICD when the skin is subjected to it more than once(Patel & Nixon, 2022a) [8]. Erythema, oedema, and localised necrosis are some of the clinical symptoms that appear quickly after being exposed to the irritant (Slodownik et al., 2008) [9]. In extreme circumstances, such as while knelt in wet concrete, this may manifest as burns. The irritating reaction, which is frequently referred to as a "decrecendo phenomenon," swiftly reaches a climax and then starts to heal(Patel & Nixon, 2022a) [8]. There have been reports of the affected skin burning, stinging, and hurting. Among the various clinical symptoms of ICD are erythema, edema, bullae, and necrosis. While ACD may exhibit same clinical symptoms, it is distinguished by a "crescendo phenomena" in which symptoms deteriorate despite elimination of the offending allergen. In acute ICD, full recovery may take weeks despite a favourable prognosis (Patel & Nixon, 2022a) [8]. An illustration of this is getting a "cement burn" after being exposed to wet cement(Slodownik et al., 2008) [9].

Delayed Acute ICD:

ICDs is the most common variety is delayed acute contact dermatitis. Chemicals including calcipotriol, anthralin, benzalkonium chloride, and tretinoin may create a delayed inflammatory response that doesn't manifest for One-third of a day after the primary exposure (Patel & Nixon, 2022a) [8]. The prognosis is good, and the indications are comparable to those of acute ICD. The association between the exposure and the condition of dermatitis could be unnoticed by the practitioner and the patient (Slodownik et al., 2008) [9]. This could result in an ACD misdiagnosis. Clinical signs include skin that is sensitive to touch and wetness, which is comparable to acute ICD symptoms(Patel & Nixon, 2022a) [8].

Clinical Presentation

Depending on the skin area affected and the allergen or irritant that caused the condition, CD can exhibit clinically in a variety of ways [10]. The characteristics that assist in distinguishing between allergic and irritating contact dermatitis are listed in [Table 1]. Contact dermatitis usually appears as redness and scaling with clearly defined edges. While it can affect

any part of the body, it most often shows up on the hands, face, and neck. In some cases, frequent lip-licking can lead to irritant contact dermatitis on the lips, which may also cause thickened, cracked skin. In order to diagnose the patient and treat the dermatitis and avoid additional damage, it is critical to consider the patient's medical history [10].

TABLE 1: Irritant and allergic contact dermatitis can be distinguished by certain characteristics

FEATURE	Location	Symptoms	External appearance	Lesion boundaries
IRRITANT	Most often, the hands, Fingers	Burning, pain	Fissured and parched skin	Fewer definite borders
ALLERGIC	typically exposed skin, frequently the hands	predominant symptom is pruritus	Bullae and vesicles	Differentiated borders, lines, and angles

Information from reference [10]

5. EPIDEMIOLOGY

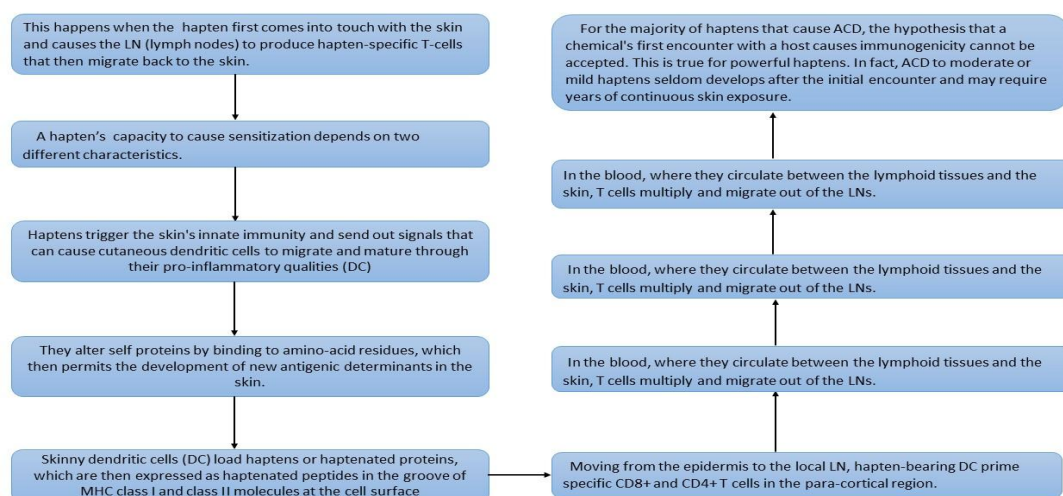
In addition to the misery of those who are afflicted, allergic and irritant contact dermatitis are widespread disorders that have significant well-being and socio-economic implications. More people experience irritant contact dermatitis than allergic instances, which make up about 80% of all cases (Dickel, 2023) [11]. There aren't many studies that have been carefully planned and carried out to establish the rate of dermatitis in the general population, however the point prevalence of dermatitis in the United Kingdom is thought to be around 20%, with atopic eczema being the majority. The most reliable research demonstrated a 2-percent prevalence of hand dermatitis in South Sweden and a lifetime risk of hand 20% of women are estimated to have eczema. More people experience irritating contact dermatitis than allergic dermatitis, and allergic dermatitis often has a worse prognosis unless the allergen is recognised and avoided (Bourke et al., 2009) [12]. Between 4 and 7 percent of all dermatology visits are thought to be for CD each year. Although it can happen to either gender and at any age, it is more common in women, particularly if they work in domestic duties (Tramontana et al., 2023) [5]. According to various published research, the prevalence of CD is uncertain with estimates ranging from 1.7 to 9.8 percent. According to recent literature, around 15 to 20.1 percent of patch-tested individuals in the regular individuals have contact allergies. The greatest risk factor for CD is occupation, and ICD, in particular, accounts for 90% of occupational skin illnesses in developed nations. Because of often contact with the most common allergens, it is believed that hairdressers, wet workers, food handlers, healthcare professionals, and construction and metal workers are more susceptible for development of contact allergies (Tramontana et al., 2023) [5]. The great majority of work related skin problems in the West are contact dermatitis, both allergic and irritating. Due to their frequent and intimate contact with common allergens, hairdressers, medical professionals, workers in the food industry and construction sector, and metal technicians experience a heightened risk of developing ACD. ACD can have a substantial detrimental effect on costs and productivity at work. Many employees with serious illnesses have to take extended leaves of absence, adapt how they do things at work, or even switch to a different profession depending on how bad their illness (Nassau & Fonacier, 2020) [4]. ACD seems to be more likely to affect women. Instead of being a function of sex, this variation is assumed to be the effect of exposure. For instance, women are thought to have greater incidence of nickel allergy as a result of wearing jewellery more frequently (Nassau & Fonacier, 2020) [4]. It has been shown that children are more likely to suffer from allergic contact dermatitis. Nickel, topical antibiotics, preservation compounds, perfumes, and rubber accelerators have all been recognised as major allergens. Patch testing should be done on children who have eczematous eruptions, especially those who have hand and eyelid eczema (Bourke et al., 2009) [12]. According to estimates, 4–5 percent of the citizens has a contact allergy to certain allergens, such as nickel, 7 and 1–3 percent of people have allergies to one or more cosmetic ingredient(s). Since almost all research used patch testing on selected groups rather than general populations, it is unknown how frequent allergy to other common allergens is in the general community (Bourke et al., 2009) [12]. The frequency of contact allergies among AD patients is comparable to that of the general population, according to a new systematic study of relationship between AD and contact sensitization, which suggests that skin sensitivity test in AD patients should only be explored when ACD is under consideration (Tramontana et al., 2023) [5].

6. PATHOPHYSIOLOGY

The main source of data regarding the pathophysiology of ACD (CHS) in animal models, where contact hypersensitivity is the term used to describe the epidermal irritation brought on by topical application of haptens to the skin. Thus, it is accepted that ACD and CS (CHS) are the same thing and that they describe T cell-induced skin inflammation due to hapten exposure [13]. It was once believed that the pathogenesis of ICD was a non-immunological reaction; nevertheless, it is now known that the immune system is crucial in the development of ICD. Alternatively, irritation results in epidermal cell damage,

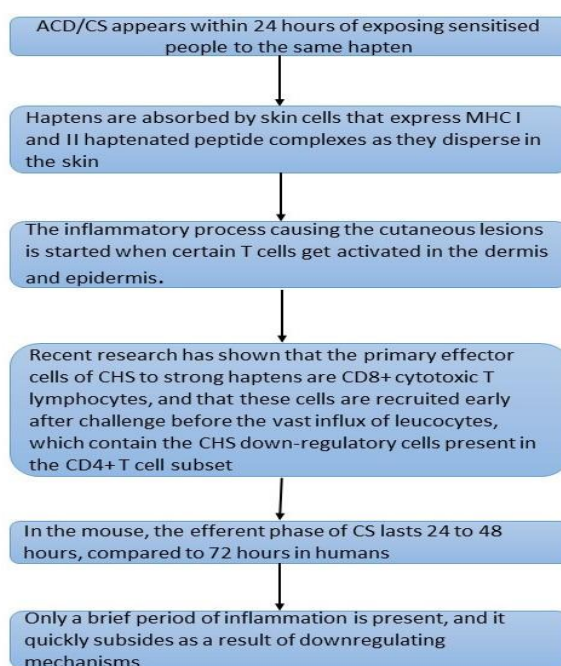
epidermal barrier disturbance or a combination of both. Damage to the epithelial barrier makes it possible for more irritations' permeability. "Signal" cells are keratinocytes. Transducers, which trigger cutaneous inflammation by transforming external impulses into secretion adhesion molecules, chemotactic factors, and cytokines (Patel & Nixon, 2022a) [8]. Skin-penetrating chemically reactive small molecules may elicit an immune reaction specific to the hapten and Integration of various immune cell types, including T cells, invariant NKT cells, NK cells, T regulatory cells, epidermal Langerhans cells and keratinocytes, in the process. In this process, Tr1 cells, characterized by their production of IL-10, and CD4+CD25+ T-regulatory lymphocytes are the primary T-regulatory cell types (Imbesi et al., 2011) [14]. Following uptake by antigen-presenting cells (APCs) like Langerhans cells and dermal dendritic cells, antigen-specific T cells (Th1, Th2, Th17, and T regulatory cells) become activated, undergo replication, and subsequently travel within the bloodstream before homing to nearby lymph nodes (Tramontana et al., 2023) [5]. Strong haptens, commonly referred to as "experimental haptens," have been studied since they are not present in the environment that humans normally live in. This has allowed researchers to learn more about the action through which a foreign matter can cause protective response. There are two main phases in the pathophysiology of CS (Nassau & Fonacier, 2020) [4].

Figure 3: Primary stage - Afferent stage (also known as Sensitization phase)



Information from reference [13]

Figure 4: Secondary stage – Efferent stage (also known as Elicitation phase)



Information from reference [13]

Causing factors

Allergens causing allergic contact dermatitis:

In the latest NACD series publication, two metals namely NiSO₄ (nickel sulphate-17.5%) and Co (cobalt-6.2%)—were associated with the most frequent positive allergic reactions; bacitracin (69.1%) and neomycin (7.0%) are two antibiotics; scent mix I (11.3%), mix II (5.3%), and myroxylon pereirae (7.0%) are three fragrances; Preservatives such as formaldehyde (7.3 percent), MCI/MI (13.4 percent), and MI (13.4 percent). Iodopropynyl butylcarbamate (3.9%), propylene (6.4%), and 1 percent (6.4%) and 2 percent (8.4%). Lanolin alcohol (4.1%), Carbamixture (4.6%) (TABLE 2)(Nassau & Fonacier, 2020) [4]. Two more subtypes of ACD are photoallergic CD and airborne CD. Several volatile allergens have the potential to produce airborne CD. In a clinical context, it manifests as traditional contact-induced eczema, which appears in exposed dermis region. The facial, neck, upper torso, hand, and arm regions, including areas like the eyelids (which may also be swollen), the area behind the ears, and beneath the jawline are the most frequently affected areas. Tearing, photophobia, and conjunctival erythema may accompany skin lesions. If solid particles are considered allergenic substances, eczema may develop on parts of the body that are covered by clothing that could get smeared over skin surface. Eczema frequently causes flexural folds. The majority of airborne CD cases are caused by plants, particularly those belonging to the Asteraceae family. Quinones (Rosewood), phenols (Anacardiaceae), and terpenes are additional frequent agents (Frullania, Pinus). Clinical symptoms typically disappear a few days after exposure to the allergen has been stopped (Tramontana et al., 2023) [5]. "In people who have already been sensitised, skin contact with a photo-antigen can cause photoallergic contact dermatitis (CD), a delayed-type cutaneous reaction of the Type IV hypersensitivity that is triggered by UV light. On the other hand, for airborne contact dermatitis, areas below the jawline and behind the ears are usually conserved. It impacts skin regions that are exposed to sunlight. Usually appearing as an exceedingly irritating eczematous dermatitis with the potential for expanding eruptions are skin lesions that usually appear within 24 hours of sun exposure. A diagnosis can be made with a photo patch test, which will be covered later. A potential consequence of photoallergic contact dermatitis is chronic actinic dermatitis (CAD), also known as actinic reticuloid (AR) which is characterised by abnormally continuous photosensitivity even after removal of the etiological culprit. Topical medications like perfumes, antimicrobials, and nonsteroidal anti-inflammatory medicines (NSAIDs) are most typical photo antigens (Tramontana et al., 2023)[5]. Finally, both ICD and ACD non-eczematous types of CD are characterised(Tramontana et al., 2023) [5].

Allergic contact dermatitis caused by plants:

While ACD caused by plants is not common, it has been demonstrated that it can occur. In some instances, the reaction can be severe. It is interesting to note that some medicinal plants are used to treat allergies while others are used to treat inflammation, and in some cases they even exhibit anti-allergic properties. Normal contact with an allergic substance or plant can result in contact dermatitis, but sensitivity happens only after recurring exposure to a particular medicinal herb, food, or spice extract. Irritating contact dermatitis is one of the side effects of plants on the skin, which can be brought on by mechanical or chemical irritants present in plant sap, particularly from families like Ranunculaceae, Euphorbiaceae, and Asteraceae (Compositae) and Phytophotodermatitis, which can be brought on by plants containing furocoumarins, particularly those from the Umbelliferae, Rutaceae, and Moraceae plant families (Rios et al., 2005) [1]. Sesquiterpene lactones(Tanaka et al., 2001) [18] and furanocoumarins are two of the most well-known allergens present in plants, as already established. Other than sesquiterpene lactones, natural compounds like flavonoids (Havsteen, 2002) [15], alkaloids (Carboni et al., 1997)(Waclawski & Aldrige, 1995) [16,17], and terpenoids can also induce hypersensitivity reactions. It is crucial to note that Human blood has been found to have flavonol-specific antibodies, despite the fact that they are minimally antigenic and often does not result in immune reactions following consumption or for therapeutic administration (Havsteen, 2002) [1,15]. The most pertinent allergens derived from medicinal plants are included in Table 2. All of the plants on the list are utilised in food, medicine, or cosmetics. Each compound's most typical sources are listed in the Table 2 [1]

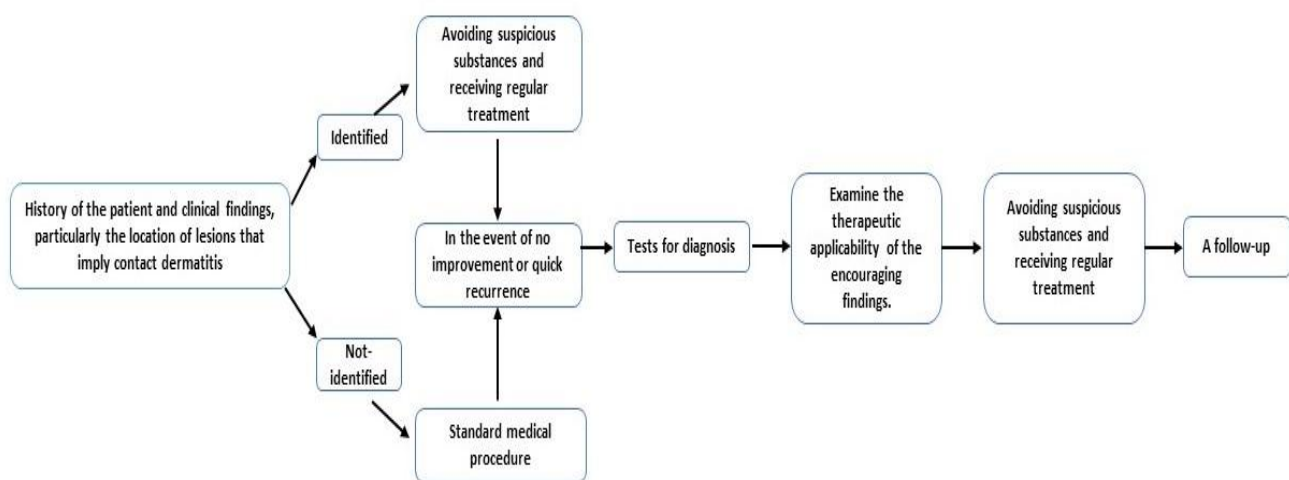
Allergen	Typical exposure source	Medicinal plant	Plant source
Fragrances • Balsam of Peru	• Cosmetics, fragrances, dental hygiene products	Alkaloids Atropine Quinine	<i>Atropa belladonna</i> <i>Cinchona sp.</i>
Formaldehyde-releasing preservatives • Diazolidinyl urea • Imidazolidinyl urea	• Personal care, hair care, and cosmetics products • Products for cosmetics, liquid soaps, moisturizers	Terpenoids Citral Sesquiterpenes	<i>Citrus sp.</i> <i>Parthenium hysterophorus</i>
Non-formaldehyde-releasing preservatives • Parabens • Iodopropynyl butylcarbamate	• Preservative in topical formulations, cosmetics • Preservative in cosmetics and topical formulations	Phenolics Cinnamon(oil) Curcumin	<i>Cinnamomum verum</i> Turmeric

Information from reference [4,13,16,17]

Management

The first step in treating those suffering from contact dermatitis is to locate and stay away from the responsible agent [19]. The incidence of contact dermatitis acts as a notice that workplace safety precautions probably need to be addressed (Al-Otaibi & Alqahtani, 2015) [3]. To alleviate the symptoms of dry skin and preserve healthy skin, moisturisers are frequently utilised (Patel & Nixon, 2022a) [8]. Drying and soothing acute, oozing sores may be assisted by calamine lotion and colloidal oatmeal baths, and the signs of acute contact dermatitis can be reduced by cool compresses [10]. Preservatives found in the steroid cream base may cause adverse reactions in some patients. It is advised to use steroid ointment because there is less chance of an allergic reaction and the medication can maintain skin contact for longer allergic response (steroid-specific allergies are rare). Moreover, wet the impacted regions first prior to rubbing the steroid is believed to assist in enhancing penetration and boost the efficiency of it [10]. There are, however, circumstances in which pharmacotherapy must be used because an allergy may not be detected or avoidance is not practicable (Bourke et al., 2009) [12]. In-depth patient education, health-psychological intervention, and expert employment consultants are also included in management (Patel & Nixon, 2022a) [8].

Fig. 3 Flowchart of probable contact dermatitis patient approach.



Information from reference [20]

Prevention

A comprehensive strategy that takes into account both exposure of causative agent and the exposed individual is necessary for the elimination of workplace contact dermatitis (Wigger-Alberti & Elsner, 1997) [21]. There is several ways to prevent contact dermatitis generally, especially in workplace environments, including heavily automating processes, removing the need for employees to prevent their skin sensitive to allergies or irritants, replacing harmful substances with less irritating and less allergenic ones, and using potent allergens in closed systems (Table 5) (Dickel, 2023) [11]. Understanding the three basic types of prevention is necessary. The goal of first-line defence is to delay the emergence of illness among healthy individuals. Secondary prevention is used who as the condition to stop the recurrence of contact dermatitis. As part of third-level prevention, an individual with a chronic condition is treated and put back into the workforce (rehabilitation) (Wigger-Alberti & Elsner, 1997) [21]. Eight fundamental components of preventative planning have been presented as part of a multidimensional approach: To prevent skin exposure, use engineering controls or chemical substitutions like personal protection such as wearing the right clothing or creams, personal hygiene and environmental hygiene, management of conceivable irritants to the skin and allergies in the work environment, and initiatives in education to encourage Pre-employment and regular health screenings, knowledge of allergies and irritants that could exist, approaches to motivation to support safe work conditions and behaviours (Wigger-Alberti & Elsner, 1997) [21].

TABLE 3: According to the principle of Dickel H has explained the preventative actions based on the "STOP" principle to lessen activities that damage the skin. (Amended in accordance with [11])

	MEASURES	EXAMPLES
"S"	Personal actions	Use of appropriate personal protective equipment (PPE; e.g., skin products, protective gloves), health education, and training in skin protection
"T"	organization-wide actions	Distributing skin-stressing activities among numerous people and regularly switching activities to cut down on exposure to irritants and moisture
"O"	Substitution/elimination	Skin-damaging contact chemicals can be replaced or banned by laws and regulations.
"P"	Technical actions	Automation or process encapsulation/shielding to prevent contact

7. DIAGNOSIS

Case study serves as the foundation for a comprehensive susceptible assessment. It encompasses details regarding a patient's occupation, pastimes, medication usage, topical skincare products or treatments, and clothing fabrics worn. Additionally, a physical examination is essential, which involves evaluating the appearance and location of skin or mucous membrane lesions. This initial procedure is crucial for formulating hypothesis or pinpointing potential sensitizing agents (Tramontana et al., 2023) [5]. Since there is no standard diagnostic procedure, ICD is a diagnosis of exclusion. The history, clinical examination, and exclusion of ACD with negative patch testing are used to make the diagnosis. a complete record of their professional and pertinent information is essential, as well as domestic exposures contains the exposure's frequency, intensity, and length.to the infected skin region as well as skin irritants (Patel & Nixon, 2022b) [23]. The diagnosis of ACD must be based on a comprehensive history. It is crucial to clarify the origin of the lesions, their course through time, and any potential causes. Due to the fact that an allergic reaction is not always quick, it may be challenging to identify suspicious chemicals. It may be challenging for the patient and healthcare professionals to identify exposures due to this delay in reactivity, which can last up to 72 hours (Tramontana et al., 2023)[5]. The lesions' distribution and placement can help with the diagnosis. In many cases, particularly when the dermatitis has been present for a while, it is challenging to pinpoint any suspicious substances at all (Nassau & Fonacier, 2020) [4]. For diagnosing ACD, a patch testing is considered ideal. This skin test uses a standardised approach to apply an allergen to the skin while keeping it covered, as a way to replicate how the body reacts during the trigger phase of type IV hypersensitivity (Tramontana et al., 2023) [5]. Acute, subacute, or chronic dermatitis may be the first symptom of ACD (Tramontana et al., 2023) [5]. Erythematous papules and vesicles are the main symptoms of acute ACD. Bullae may be seen in severe cases. The lesions that characterise chronic ACD are typically erythematous, itchy, and may show signs of longer-lasting inflammation, such as lichenification, scaling, and fissuring. Superinfection can happen when the epidermal barrier is damaged, as is the case with chronic ACD. It is more challenging to define subacute ACD because it might present with a variety of symptoms. Distribution is useful in making an ACD diagnosis. Certain distributions, including those on the hands, neck, lateral face, or eyelids, should prompt the consideration of ACD with regards to personal care and cosmetics. The top 10 principal sites of are listed in (Table 4[1,26]).

TABLE 4: The primary cause of dermatitis is where it occurs on the body.	
Site for Dermatitis:	n(%)
Hand	1230 (22.0)
Scattered generalized	995 (17.8)
Face	946 (16.9)
Eyelids	535 (9.6)
Trunk	307 (5.5)
Lips	274 (4.9)
Arm	230 (4.1)
Scalp	225 (4.0)
Leg	207 (3.7)
Foot	120 (2.1)
Total n	5591

The spread and pattern of the dermatitis are crucial to the diagnosis. ICD never spreads and always begins where the irritant made contact with the skin (Al-Otaibi & Alqahtani, 2015) (3). The most precise method for diagnosing allergic contact dermatitis and replicating its underlying mechanism is through epicutaneous patch testing' (Nassau & Fonacier, 2020) (4). Today, a variety of patch test units, includes employing devices like 'Finn Chambers' and 'van der Bend square chambers' for the same purpose, are commercially available. The International Contact Dermatitis Research Group 28 advises applying the patch test system on top half of the back and removing it after 48 hours. Readings should be taken 20 minutes after removing the strips. Additionally, readings may need to be repeated after 72 or 96 hours. In some cases, a re-evaluation of certain test series after seven days might be necessary to ensure that delayed reactions are not overlooked(Patel & Nixon, 2022a) [8]. The International Contact Dermatitis Research Group has established standards for patch testing's gradings, procedures, and nomenclature (Bourke et al., 2009) [12]. Occasionally, extreme reactions can cause burning and itching, leading to the premature removal of the patch test(Patel & Nixon, 2022a) [8].

Treatment

The only effective remedy is to get rid of the offending allergy (s). Before a firm diagnosis can be made, symptomatic therapies, primarily topical corticosteroids, can assist to lessen the symptoms (Lachapelle, 2014b) [22]. The use of causal and symptomatic therapy (Paravina et al., 2019) [24]. Eliminating the contact allergen is the sole etiological therapy for ACD that is currently available. Patients should be informed about the name of the substance causing the issue and any potential possible sources of the allergen. List the substances that react with one another. Steroids applied topically are used during the early stage, and as the skin heals, ointments and cold creams progressively take their place. Lesions retreat. Systemic corticosteroids may be necessary for short period if ACD is widespread and severe [25]. Additionally, a brief mid- to high-dose topical application corticosteroids is the foundation of the treatment of ACD. There are several classes of the topical corticosteroid drugs which is used in formulation at different potency (Tramontana et al., 2023)[5,27].(Parikh et al., 2017).

Examples

CLOBETASOL PROPIONATE it comes under the class 1, which is used to formulate the formulation cream at the concentration of 0.05%. It contains a ultrahigh potency (Tramontana et al., 2023) (Parikh et al., 2017)[5,27].

FLUOCINONIDE it comes under the class 2, which is used to formulate the formulation cream, ointment or gel at concentration of 0.05% It contains a high potency (Tramontana et al., 2023) (Parikh et al., 2017) [5,27].

BETAMETHASONE VALERATE it comes under the class 3, which is used to formulate the formulation ointment at the concentration of 0.1%. It contains a high potency (Parikh et al., 2017; Tramontana et al., 2023) [5,27].

DESOXIMETASONE it comes under the class 4, which is used to formulate the formulation cream at the concentration of the 0.05%. It contains a moderate potency (Tramontana et al., 2023) (Parikh et al., 2017)[5,27].

HYDROCORTISONE VALERATE it comes under the class 5, which is used to formulate the formulation cream at the concentration of 0.2%. It contains a moderate potency(Tramontana et al., 2023) (Parikh et al., 2017)[5,27].

DESONIDE it comes under the class 6, which is used to formulate the formulation cream at the concentration of 0.05%. It contains a low potency (Tramontana et al., 2023) (Parikh et al., 2017) [5,27].

METHYLPREDNISOLONE ACETATE it comes under the class 7, which is used to formulate the formulation cream at the concentration of 0.25%. It contains a low potency (Tramontana et al., 2023) (Parikh et al., 2017) [5,27].

8. DISCUSSION

The current available research suggests that a variety of different cells in the body can play a role in the development of allergic contact dermatitis. For diagnosis, a complete history and physical examination are required. ACD, in particular, has the capacity to mimic a variety of dermatological conditions. It is common to misdiagnose the creature in this situation. A thorough medical record and physical examination are necessary for prognosis, in addition to a suspicious approach. Patch testing could be beneficial for identifying the causes, particularly in ACD; Numerous improvements have been made in our understanding of ICD. The significance of the TNF- α gene polymorphism is one of the exciting new fields of research, yet even though there is lack of standard examination and the ICD is frequently a exclusionary diagnostic and confocal microscopy usage. The cornerstone of treatment is avoiding the offending agent and adopting precautions.

9. CONCLUSION

ACD, in particular, endures the potential to imitate various dermatological conditions. It is common to misdiagnose the creature in this situation. For diagnosis, a complete history and physical examination are required, in addition to a suspicious approach. Patch testing could be beneficial for finding the responsible parties, particularly in ACD; nonetheless, the significance of the identified however, all potential drugs should be compared to the clinical history and results. Before making the definitive diagnosis of contact dermatitis, other illnesses need to be ruled out. The accuracy of the diagnosis may be improved by being aware of the disorders that CD imitates. The cornerstone of treatment is avoiding the culprit and adopting precautions...

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