

CHAPTER I

INTRODUCTION

Definition of Eczema

Eczema is a distinctive pattern of inflammatory response of skin induced by a wide range of external and internal factors acting singly or in combination (Rook and Wilkinson; 1979).

The term eczema has been derived from an old Greek word "ekzein". The word ekzein in Greek means to flow out or boil out (Domonkos; 1982) and (Calnan; 1977).

All eczema is dermatitis but not all dermatitis are eczema (Rook and Wilkinson; 1979 and Moschella and Hurly; 1985).

Eczema account for a very large proportion of all skin diseases.

The morphological changes of eczema are clear-cut and not easily confused with other gross changes of skin. The signs of eczema in the order of their evolution are :

- * Erythema and swelling of the skin, oozing and/or vesiculation.

- * Crusting and scaling, lichenification or thickening and evidence of repeated excoriation.
- * Hyperpigmentation and/or hypopigmentation, i.e., the effects of inflammation on the melanocytes.
- * The first three changes are those of acute eczema.
- * The latter two are seen if the process persists for several weeks or longer (Moschella and Hurly; 1985).
- * All stages may be seen on various aspects of the skin at one time as each lesion may progress independent of others.
- * However a progressive form of eczema may evolve into diffuse redness scaling, and swelling.

Classification of Eczema of the Hands

Although there are many types of eczema which occur in the body and can be classified into atopic dermatitis, contact dermatitis, seborrheic dermatitis, microbial eczema, stress eczema, autosensitization dermatitis and stasis dermatitis, (Domonkos; 1982).

Yet the classification of hand eczema is more or less a difficult task not only because more than one type may occur on the hand but also because no much is known

about the true aetiology (whether it is a purely dermatitis or eczematous changes due to other causes (Commander et al.; 1945)).

Different authors however attempted classification of hand eczema as Gay et al., 1945, who classified eczema of the hand into infectious eczematoid dermatitis, contact dermatitis, dermatophytid, bacterid, atopic dermatitis, pompholyx and rarely dermatomycosis and dietary insufficiency, and idiopathic eczema.

From the etiological point of view Rook and Wilkinson, 1979, divided eczema on the hand into two major groups namely :

The Exogenous Group

Where the cause of the eczema was outside the body. This group includes: Irritant dermatitis, allergic contact dermatitis and infective dermatitis.

While the second group was known as :

Endogenous Group

Here the cause of the eczema was believed to be from within the body. This group included :

- * Atopic dermatitis.
- * Nummular eczema, and
- * Pompholyx.

The same authors also classified the eczemas of the hand in accordance with the types of eczemas seen predominantly in certain age groups.

Classification of hand eczema according to age.

Eczema of the Hands before Puberty

- * During the first two years of life :
 - Manifestations of atopic diathesis, but this is rare.
- * A later age group :
 - Localised eczema of the thumbs, in the thumb suckers.
- * Between 3 - 5 years of age :
 - Atopic dermatitis patterns affecting the flexor surface of the wrists, fingers and back of the

hands. Also aggravated by common irritants such as playing with earth and sand. Also some causes of allergic contact dermatitis have been recorded but are rare.

* Between 5 - 10 years

- Atopic dermatitis becomes more prominent and may be disassociated from the eczematous group.
- Pompholyx, also post traumatic eczema involving usually the tip of one finger.

Eczema of the Hands in Adolescents and Adults

The common eczema of the hands seen in adults and adolescents are: Contact dermatitis, Atopic dermatitis, Nummular (discoid) eczema, and Pompholyx.

Despite these classifications, the distributions and specific features of eczemas of the hands are not of any diagnostic value except in some cases of contact dermatitis.

Meanwhile, as many as one out of three cases of eczema of the hands cannot be put under any of known categories. Thus such eczema are labelled as unclassified eczema (Calnan et al.; 1970).

Rook and Wilkinson; 1979, classified eczema of the hand to :

Atopic dermatitis, pompholyx, Nummular (discoid eczema) wedding-ring dermatitis, finger-tip eczema, dyskeratotic eczema, patchy vesiculosquamous eczema, Hyperkeratotic eczema of palms and fingers, phlyctenular eczema of the thenar eminence and xerotic eczema.

The Incidence of Eczema

The proportion of cases, for instance, of contact dermatitis varies with degree of industrialization of the community served and of the interest of the dermatologist and of the diagnostic facilities available. Another factor which facilitates the statistical calculation of incidence of eczema is the strict definition of certain types of eczema as atopic and the nummular in contrast to uncertainty in the nomenclature of other types as the seborrheic or pompholyx (Bowken; 1979).

The incidence of eczema in different countries is influenced by racial, climatic, economic and cultural factors, (Negreh and Rujan; 1976).

In a survey of cases referred to a Dermatology Department in the U.K., hand eczema, as defined accounted

for 21% of all cases of eczema. The figure can be expected to be higher in more heavily industrialized areas where the hands alone are frequently involved, (Bjornberg A.; 1968).

In the study of the incidence of hand eczema, it was found by Fregert and Bandmann, 1975, that the hands were involved in 64% of the men and 53% of women in European survey of 4000 cases of eczema.

Eczema was confined to the hands in 36% and 30% respectively. Women outnumbered men by 2 : 1 (Agroun; 1979).

Histopathology of Eczema

The histopathological features of eczema reflect a dynamic sequence of changes resulting from inflammation of the epidermis and the underlying dermal structures, these vary with the intensity and stage of eczematous process and are frequently modified by secondary events such as trauma and infection (Rook and Wilkinson, 1979).

Lever and Lever; 1983, stated that various types of dermatitis rarely present a histologic picture sufficiently diagnostic to allow their differentiation because similar

histological reactions occur in all forms of dermatitis. Spongiotic microvesicles or macrovesicles with oozing in acute dermatitis, acanthosis with parakeratosis in chronic dermatitis and a combination of these two reaction patterns in subacute dermatitis. The histologic picture described here as acute, subacute and chronic dermatitis, and the more or less distinctive features of various types of dermatitis will be listed in its section.

In Acute Dermatitis

The histologic picture is dominated by intraepidermally located vesicles or bullae, considerable intercellular oedema (spongiosis) and intracellular oedema may be present in the epidermis.

If the number of the vesicles is great and the intracellular aedema is pronounced, the histologic picture of reticular degeneration of the epidermis results which lead to bursting of the epidermal cells and formation of a multilocular bulla, the septa inside the bulla are formed by resisting cell walls, i.e., the bulla is multilocular. The spongiosis leads to stretching and eventual rupture of the intercellular attachments with the formation of primordial vesicles, these commonly occur in discrete foci in the mid-epidermal

region. On the palms and soles, the vesicles do not rupture easily and become large by coalescence (Civatte; 1957).

The vesicles and bullae as well as the oedematous portion of the epidermis may be permeated by an inflammatory infiltrate composed mainly of mononuclear cells formed largely of monocytes and histocytes or macrophages with a minority of lymphocytes. Lesions more than a few days old may also contain neutrophils particularly in the stratum corneum, the stratum corneum may be parakeratotic and contain aggregations of coagulated plasma, the substance of crusts.

The upper dermis shows vascular dilatation at the earliest stage and is marked in all stages, the papillary vessels are especially involved, also oedema and mononuclear cellular infiltrate mainly lymphocytes, although polymorphs and eosinophils may occur in the very acute eczema, (Kerl et al.; 1974). The infiltrate extends from the papillary blood vessels to the epidermis, (exocytosis).

In the Subacute Dermatitis

Spongiosis diminishes (inter cellular oedema in the rete malpigi) and is usually associated with acanthosis

(hyperplastic thickness of prickly cell layer) is associated with formation of parakeratotic horny layer, i.e. imperfect keratinization with retention of nuclei in the horny layer and associated with loss of granular layer. This often contains layers of dried-up serum and pyknotic nuclei of inflammatory cells. Later the rete ridges become elongated and broadened and hyperkeratosis replace parakeratosis, (Lever and Lever; 1983).

In Chronic Dermatitis

There is moderate to marked acanthosis with elongation of the rete ridges, there is also hyperkeratosis with area of parakeratosis, slight spongiosis may be present, but vesicles are absent. Intercellular vacuolization if present is a result of glycogen accumulation (Civatte, A.; 1957). The upper dermis shows inflammatory infiltrate in a perivascular arrangement. The capillaries in the upper dermis are increased in number and their walls are thickened and become tortuous with lichenification. There may be some increase in collagen manifesting itself as fibrosis in the upper dermis including the papillae, (Lever and Lever: 1983).

CHAPTER II

CONTACT DERMATITIS (C.D.) -----

Contact dermatitis is a dermatitis which results when the skin comes into contact with certain substances. It is divided into two group namely :

The primary irritant dermatitis, and
The allergic contact dermatitis,
(Domonkos; 1982 and Cronin; 1977).

Irritant dermatitis is known as primary irritant dermatitis. Primary irritant dermatitis is a reaction occurring on the skin, when exposed to certain substances which are irritant in nature. This type of dermatitis has no allergic factors involved, in it. It can affect any person's skin, if sufficient high concentrations are used (Domonkos; 1982), for a long period (Cronin; 1977 and Andrew's; 1982).

Allergic contact dermatitis is a skin lesion resulting from exposure of sensitized individuals, to contact allergens or sensitizers. It is a delayed hypersensitivity reaction in contrast to dermatoses that are of immediate urticarial variety, (Baer; 1973 and Stevenson; 1978).

Allergic contact dermatitis is due to allergic sensitization to various substances that produce inflammatory reactions in those, and only those, who have acquired hypersensitivity to allergen as a result of previous exposure to it, (Andrew's; 1982).

When those external agents are alive, we are dealing with infectious dermatosis such as impetigo, if they involve radiation we are dealing with the radio or photodermatitis, when inflammation is caused by external heat we are dealing with burns, (Lowney; 1975).

The term contact dermatitis includes those inflammations due to all remaining external agent, as chemical allergens, toxins and irritants and mechanical irritants (Lowney; 1975 and Lane et al.; 1975).

It is often very helpful to be able to determine from clinical features alone, whether an eruption is due to irritant or external causes. Lesions of contact dermatitis tend to begin in the area of contact, and the shape of the lesion will reflect the localization of exposure, (Fisher; 1973).

In contrast, a systemic eruption appears in the form of symmetrically located lesions, such lesions tend to be round and never linear as may be in lesions of contact dermatitis (Lowney; 1975).

Another characteristic of contact dermatitis is that the topographic features of the skin determine the location and intensity of the dermatitis. An ointment which causes a contact dermatitis, for instance is likely to accumulate in area of the skin, or under a ring producing an intense dermatitis there, while sparing exposed surfaces from which it is readily removed. A solid contactant however, will produce the opposite pattern, as the creases will be protected from contact, (Lane et al.; 1975). Regardless of exposure certain areas of the skin are more readily affected by contact irritants than others the preiorbital area and the dorsa of the hands are relatively sensitive while it is more difficult to induce a dermatitis on the scalp, palms and soles.

Another aspect of contact dermatitis is the nature of eruption itself. Most contact dermatitis are eczematous and superficial with epidermal involvement appearing relatively early.

Fine superficial erythematous papules and vesicles are often the first evidence of an acute contact dermatitis, (Moschella and Hurly; 1985). Most systemic eruption however, begin as deep erythema which produces scales only several days later. Even with the use of

these principles appearance alone can not always be relied upon to establish a diagnosis of contact dermatitis.

Dermatomycosis for instance, may mimic a generalized contact dermatitis by producing an eczematous dermatitis on exposed areas and on the periorbital skin, (Moschella and Hurly; 1985).

Another confusing feature of contact dermatitis is that an allergen may be absorbed from the site of contact and produce a generalized systemic eruption such as autosensitization which is a systemic eruption, superimposed on contact dermatitis, (Moschella and Hurly; 1985).

Classification of Contact Dermatitis

On aetiological ground

Rook and Wilkinson; 1979, classified the types of dermatitis into :

1. Irritant dermatitis.
 - a) Acute irritant dermatitis.
 - b) Cumulative insult dermatitis.
2. Allergic contact dermatitis.
3. Phototoxic dermatitis.
4. Photoallergic dermatitis.

Moschella; 1985, classified C.D. into two categories :

- Irritant contact dermatitis (which include all non immunologic mechanisms), and
- Allergic contact dermatitis (due to an acquired response).

These two types are also cross-categorized according to the kind of reaction produced.

Most allergic C. D. are of the classic eczematous type but urticarial and granulomatous reactions can occur. Irritant reaction may take these forms also but in addition may be manifested in many other ways (pustular reactions, follicular hyperplasia papules, blisters, dryness, redness and scaling).

Primary Irritant Dermatitis

There are many substances acting as primary irritants, that will produce a non-allergic inflammatory reaction of the skin. This type of dermatitis may be induced in any person if a sufficiently high concentration is used.

The only variation in the severity of the dermatitis from person to person, or from time to time in the same person, is the condition of the skin at the time of exposure to the primary irritant. The skin may be subject to maceration from excessive humidity, exposure to water, heat, cold pressure or friction. A dry skin is less likely to react to contactants. Repeated exposure to some of the more mild irritants may, produce a "hardening" effect. The process makes the skin more resistant to irritant effects of the given substance. Susceptibility to irritants varies in different persons mainly because of the differences of skin thickness of the exposed sites, (Domonkos; 1982).

There are many terms used when describing this type of dermatitis. They include: acute irritant dermatitis, cumulative insult dermatitis, traumatic dermatitis and wear and tear dermatitis (Fisher; 1973). Dermatitis from cumulative insults may develop after repeated insults by weak irritants over a long period. This type of dermatitis has also been named "wear and tear dermatitis", (Rook and Wilkinson; 1979).

Histopathology of Primary Irritant Dermatitis

When the skin comes into contact with harsh or strong chemical or irritants, it leads to necrosis. However, if these substances are of a milder nature, the following changes are noted :

- The damage occurs in the superficial living cells mostly, leaving the more deeper structures intact.
- There is exudation of fluids and polymorphonuclear leucocyte into the epidermis. These come from the blood capillaries, and lead to edema and cell degeneration of the epidermis.
- The horny and granular layers show minor or no changes. This is due to the fact that the cell bodies, and intercellular connections are rigid here.
- Intraepidermal edema is manifested due to spongiosis and inflammatory edema which separates the prickle cells, when acanthosis is seen, it indicates that mitotic proliferation has started. This process will replace the damaged layer.
- The papillae show some edema. Also some blood capillaries are seen to be dilated.

- A mild lymphocytic infiltrate is present in the papillary layer (Pinkus Mehregan; 1976).

Agent Implicated in Primary Irritant Dermatitis

Primary irritant dermatitis is caused by numerous agents, (Domonkos; 1982 , Hjorth and Feregert; 1972 , and Fisher; 1973), have grouped them as :

- A) Physical factors: Including heat, cold and ultra violet rays.
- B) Mechanical factors: Such as friction or mechanical pressure.
- c) Chemical agents: Which form the largest group.

This group is further divided into strong or absolute irritants, and relative or mild irritant.

The strong or absolute irritant, contain agents that are in high concentration, such as acids or alkalies.

The mild or relative irritants include a multitude of agents which need longer periods to produce the dermatitis. The dermatitis is most commonly seen in people who handle detergents, solvents, household cleansers, bleaches, amonia preparations, soaps, etc..

Both acids and alkalies are further subdivided into strong or corrosive, or weaker ones. Some of the famous acids known to produce primary irritant dermatitis are nitric acid, hydrochloric acid, sulfuric acid, oxalic acid, etc.. The alkalies commonly incriminated are, sodium hydroxide, calcium oxide, etc..

There are, however, numerous other agents which cannot be put under acid nor alkalies. These too, in turn may be corrosive or relative in their action on skin. Some of them are : Oil, cutting fluids, tars, salts of metals such as, copper, mercury, zinc, nickel, chromates, etc., (Fisher; 1973).

Also certain gases and dusts have been incriminated, i.e., teargas and dust from manufacture of explosives, (Madden; 1951 and Hitch; 1976).

This would be appropriate place to mention two eczematous lesion occurring on the hands. These are housewife's eczema and ring dermatitis. Both these skin lesions seem to have either an irritant or allergic or both factors involved in their etiology.

Housewife's eczema is an example of dermatitis due to chemical primary irritants, (Moschella and Hurly; 1985) .

Dermatitis from Cumulative Insults

Dermatitis may develop after repeated insult by weak irritants over a long period. This type of dermatitis has also been named "wear and tear dermatitis" (Hagerman G.; 1957).

The development of cumulative insult dermatitis is slow in contrast to irritant dermatitis. A characteristic of this type of dermatitis is the tendency to relapse on repeated brief exposures to mild primary irritants such as soap, detergents, and water (Plohm; 1978).

The hands and forearms are frequently the initial sites of cumulative insult dermatitis. The hands are naturally exposed to a variety of repeated injuries, (GAUL, L.E.; 1958).

A dermatitis often start under the ring without any sensivity to metals. Ring dermatitis often spread to surrounding area is, for unknown reasons particularly persistent, (Rostenberg A.; 1957). Housewife's dermatitis and Ring dermatitis are two examples of dermatitis from cumulative insults.

1. House Wife's Eczema

Housewife's eczema is a common disease due to or at least initiated by excessive and prolonged exposure to soaps or detergent and water, (Domonkos; 1982).

Housewife's eczema also known as dishpan hands, or detergent hand is probably one of the commonest types of eczema of the hands, encountered, (Fisher; 1973).

Housewife's dermatitis is caused by cumulative insults from cleaning washing cooking and may be due to hormonal or other constitutional factors, (Laymon; 1970).

The eruption usually begins with dryness and redness of the finger. Dry scales with peeling are evident at the tips of the fingers "chapping" is seen on the backs of the hands and erythematous hardening of the palms with fissures develops. Frequently, dermatitis appears around and under rings, especially wedding bands, which are not removed when using soap and water.

The defatting action and maceration produced by prolonged immersion or frequent washing of the hands may be seen in many other handlers chiefs, physicians, and dentists all of them wash their hands frequently, (Andrw's; 1982).

Clinical Picture

The dermatitis occurs principally on the fingers, webs and dorsa of the hand of women who do house work and laundry.

This eczema may also affect other people especially those who are using excessive soaps and detergents such as; surgeon, medical personnel, soda clerks, bartenders, etc..

The condition starts usually with mild dryness, redness and scaling with continued exposure to soap and water, fissuring crusting and finally chronic eczema is produced, (Domonkos; 1982 and Fisher; 1973).

The condition is more commonly seen in the winter time.

In a study by Kalimo et al.; 1982, it was found that about 1% of all the hospital workers had had dermatitis cleaners, kitchen workers and nurses most

frequently. 54% of the patients who were clinically studied had suffered from periodic symptoms and 35% had current hand dermatitis. Those who had previous or present atopic dermatitis had most frequently developed dermatitis during the first year of their service. An atopic constitution seemed to predispose to the development of permanent or periodic hand dermatitis. The patients with sensitivity to nickel or fragrances had relapses in the majority of cases. During the study period, the incidence of new dermatitis cases was constant but the number of days sick leave showed a decreasing tendency.

Etiology

To determine the etiology of housewife's eczema, several authors have patch tested these patients and their numerous reports suggests that irritant and allergic factors play important role. Rothenborg and Skog E.; 1978, suggested that housewife's eczema is

caused by a multitude of factors. Among which soaps and detergents play an important role.

The soaps and detergents contain enzymes, chemicals and mild irritants, etc.. These agents irritate the skin and promote absorption of low grade allergens through cracks and fissures.

In 1970 Calnan et al., studied and patch tested 4000 patients with eczema*of the hands in five European centers. Of these, 1000 patients were engaged in domestic work only. 281 of them were women. The rest were men. From the women, 42% showed an allergic basis for the eczema they had. While in the rest, an irritant factor was implicated. This indicates that in housewife's eczema, both factors play an important role.

Before patch testing patients were clinically diagnosed as contact dermatitis, seborrhoeic, atopic, nummular, stasis or unclassified dermatitis. This was the primary diagnosis, (Calnan et al., 1970), of all the women doing domestic work only, half (51%) were diagnosed as contact dermatitis, a quarter (24%) were unclassified, and a quarter (25%) were seborrhoeic, atopic, nummular or stasis. However of the women with hand dermatitis doing domestic work only, 78% were diagnosed as contact der-

matitis before patch tests, 5% as atopic 1% nummular, 3% stasis and 13% as unclassified dermatitis. Definitive diagnosis is made after patch test.

Myriads of factors may contribute to housewife's eczema. These may be allergic sensitization to such food stuff's handled in the kitchen as garlic, onion, tomato, spinach, grape fruit, orange, fig., parsnip and cheese. There may be sensitization to rubber gloves plastic articles, house plants such as philadendron, to metals such as nickel and numerous other allergenic contactants. Candidiasis may develop especially in the interdigital weps, (Domonkos; 1982).

Feuerman; 1969, believes that potassium dichromate sensitivity plays a major role in housewife's eczema since he was able to demonstrate a positive patch test in 47 of 50 patients with typical eczema.

Soaps and detergents probably contribute more to the pathogenesis of hand dermatitis than any other factor. It is true that the soaps and detergents may act secondarily to some other primary irritant or real allergen in which case avoidance of soap and detergents

will not ameliorate the symptoms, Stoughton , 1965 and Domonkos; 1982, found no difference in the course of dermatitis whether or not soap was used.

Hand dermatitis frequently begins with the onset of cold weather when the absolute humidity is low. Chapping is commonly observed under such condition, (Domonkos; 1982).

2. Ring Dermatitis

Many people complain that they have a tendency to develop a rash under their rings. The initial rash is at the site of the ring finger. When the rash appears, many patients change the ring to the other hand. Hot weather with associated sweating seems to be an important factor (Gaul; 1958). Both irritant and allergic factors have been incriminated. Though gold and silver do not seem to cause allergic contact dermatitis (Fisher; 1973). But cheaper metals such as nickel and chromates present in ornaments may lead to the development of the dermatitis. Gaul, 1958 , believes that rings seem to irritate more commonly the skin surface than do other metallic contactants on the skin else where. He attributed this to the fact that the hands have numerous opportunities for exposure to salts,

such as eating salted foods like nuts, popcorn, etc.. The salt from these food stuffs may be deposited under the rings, leading to a reaction with the metal, which is then enhanced by moisture and sweat. In time, the dermatitis manifests itself.

If dermatitis from rings is not treated, it may spread to the hand and other fingers (Fisher; 1973).

This presentation of hand eczema is most common in young women, often occurring soon after marriage. Characteristically a patch of eczema appears under the wedding ring and spreads in a rather typical manner to involve the adjacent side of the third finger and the palmar surface adjoining the finger. It may remain there for some months if the ring is not removed. Sometimes discoid patches appear else where on the finger or hand.

Despite the sharp localization and demarcation of the initial eruption these patients can not be shown to be sensitive to gold or copper. Transference of the ring to the other hand is often followed by eczema at the new site and once affected, patients will over that wearing of the ring for only a few minutes, without washing, causes irritation. This kind of hand eczema

is commonly believed to be an irritant reaction to alkalics and detergents collecting beneath the ring.

A nervous habit is also an additional factor and may explain the onset of the condition in some patients at time of stress or anxiety, (Rook and Wilkinson; 1986).

Diagnosis

Diagnosis of hand eczema due to soaps or detergents is mainly by exclusion of the offending substances. The alkalis in the soap and cleansers are true sensitizers. Patch testing does not elicit a true allergic reaction to the alkali. However, it must be remembered that perfumes and antibacterial substances that are allergenic may be incorporated in the substances.

Photosensitivity to some of the antiseptic soaps may occur. Patch testing to soaps is performed with a 2% aqueous solutions placed on the skin for 48 hours. The reaction is not allergic but rather irritation. Patch tests to other substances should be performed simultaneously to determine possible multiple sensitization that may occur, (Domonkos; 1982).

Management

The results of the survey done by, (Agroup; 1979), seem to warrant the following advice to patients with housewife's eczema. Protect the hands against chemical substances by wearing synthetic rubber gloves over cotton gloves next to the skin. Rubber gloves should not be worn for more than a few minutes because of the macerating action of the accumulated perspiration in the gloves.

If the whole cotton gloves are worn most of the time, the patient is deterred from frequent wetting of the hands, (Domonkos; 1982). The various corticosteroids creams are helpful and should be applied lightly every two or three hours.

For the acutely inflamed swollen oozing and weeping dermatitis compresses of ice cold milk or Burrow's solution applied for "20" minutes every 4 hours will relieve the symptoms, after the use of the compresses, corticosteroid cream without neomycin, should be applied, (Domonkos; 1982).

Histopathology of C.D.

Biopsies are of little help in C.D., most types of eczema show identical pathological changes, and allergic and primary irritant C.D. cannot be distinguished with certainty (Rook and Wilkinson; 1979). This difficulty arises from the fact that a biopsy is usually carried out days or weeks after appearance of dermatitis. By that time, regenerative processes complication have altered the original picture of the dermatitis, and the findings are those of a nonspecific subacute or chronic dermatitis. However, different histological responses can be obtained to patch testing with an allergen in a sensitized individual (Lever and Lever; 1983).

Thus in allergic C.D. the application of the patch test consists of vasodilation and extravasation of mononuclear cells from the papillary and subpapillary capillaries into the dermis.

The mononuclear cells migrate towards the epidermis and entering it, produce basal spongiosis about 8 hours after application of test. As they migrate upwards between the epidermal cells spongiosis develops also in upper epidermis after 12 to 24 hours, (Lever and Lever; 1983).

The areas of spongiosis soon develop into spongiotic vesicles. In contrast to patch tests with allergens those with primary irritants induce damage to the epidermal cells within a very few hours, although the speed with which the damage occurs and the degree of damage depends on the type and the concentration of the primary irritant, (Hall et al.; 1967).

With moderately strong primary irritant such as 10% DNCB, (Dinitrochloro benzene), both intracellular and intercellular oedema throughout the epidermis is evident after 3 to 6 hours mostly in the basal layer in contrast to that in allergic C.D. which is located in the middle portion of the malpighian layer. Within 24 hours, epidermal necrosis characterized by cellular vacuolization and nuclear pyknosis occur, (Epstein, 1958). This may result in subepidermal blister formation, (Kerl et al.; 1974). The upper dermis in the irritant C.D. is severely damaged and is chemotactic for polymorphonuclear leucocytes.

In contact allergic dermatitis, monocytes and macrophages are prominent constituents of delayed hypersensitivity reaction. There is possibility that interaction (Dumond; 1967), between lymphocytes and

monocytes macrophages exist and may well underlie the inflammatory reaction of delayed hypersensitivity.

The nature of this interaction is explained by the following hypothesis. First is that monocytes and macrophages are simply passive participant in immunological reactions initiated by lymphocytes, (Dumonde; 1967). Second, is that sensitized lymphocytes reacting with antigen generate non-antibody biologically active substances lymphokines which serve roles in the mechanism of cellular surveillance.

A third possibility is that monocytes and macrophages are involved in the sequestration and processing of antigen and pass the antigenic fragments to immunologically competent cells which are then stimulated to produce antibody, (Buraunet ; 1969).

In allergic C.D. the simultaneous appearance of lymphocytes and monocytes in the epidermis and dermis, the frequent membrane contact between these two cells, and the later appearance of macrophages rich in secondary lysosome lead to speculation about still another possibility namely, that antigen antibody complexes formed on the surfaces of antibody producing lymphocytes in delayed hypersensitivity

reactions are remained phagocytized and digested by the monocytes, (Buranet; 1969).

This process might serve by removing surface burden of antigen-antibody complexes, not only to stimulate the lymphocytes to produce more antibody and permit reaction with more antigen, but the monocytes and macrophages which by digesting antigen antibody complexes might release inflammation promoting substances which enhance the reaction. Basophilic leucocytes also appear to be involved in mechanism of C.D. because of their conspicuous accumulation in contrast to their scarcity in primary irritant C.D.. There are several reports on increased basophils in allergic C.D. and in delayed reactions following the intradermal injection of metal salts in sensitized persons (Apsegreen et al.; 1963).

Local basophilia in photo-allergic C.D. (Fregeret; 1966), has also been reported. Basophils may play a role in allergic C.D.. Through their histamine content. Histamine increases vascular permeability and promotes diapedesis of leucocytes.

Ultramicroscopic examination shows that irritant and allergic responses can be distinguished by the

behaviour of langerhans cells, which are activated by lymphocytes in the allergic response (Rook and Wilkinson; 1979).

In a histological study of lysosomes in C.D.. Weissmann in 1960 stated physical as well as chemical agents can labilize lysosomes with release of their enzymes, and that lysosomes are implicated in the pathophysiology of C.D.. As a primary irritant, this may result from physical and/or chemical alterations of the lysosomes.

In allergic C.D. the antigen-antibody reaction occurs on adjacent to the lysosomal membrane causing its disruption.

Wissman; 1960, has also localized antigenic macromolecules within subgroups of lysosomes and further suggested that the lysosomes may be the site of antigen degradation.