STUDIES IN CONTACT DERMATITIS AND ITS MECHANISM

A Thesis

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By

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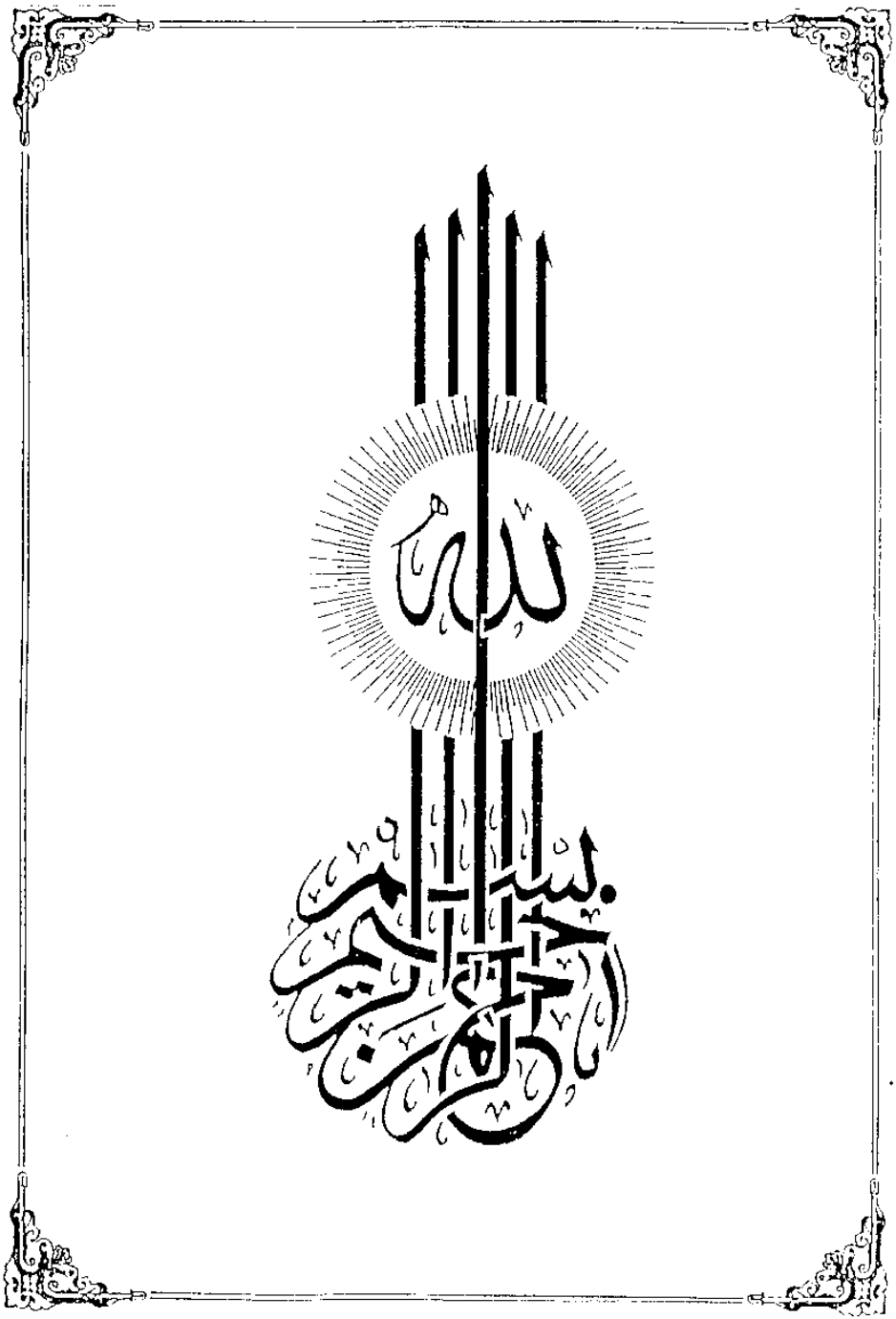

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Many workers had contributions to the history of contact dermatitis. The first hint on latent predisposition to contact dermatitis came from Devergie and Hardy in 1870 (Schwartz et al., 1957). The phenomenon of contact sensitization and its role in the pathogenesis of eczema was first recognized in 1895 by Jadasshon, who also devised the patch test as a diagnostic procedure (de Weck, 1971). The role of the regional lymph nodes in the establishment of contact sensitization was definitly established in 1956 by Frey and his collaborators (de Weck, 1971).

In spite of the ability of human skin to withstand the assaults of frequently hostile environment, chemicals can cause different skin diseases. The cutaneous reactions to these chemicals are almost as varied as the chemicals themselves. All are irritants to some degree, yet relatively few are known to be contact allergens. It is apparent to anyone studying the literature of contact dermatitis that there is a great confusion in the terminology and conventions employed **(Wilkinson et al., 1970).**

The aim of this study is to review the literature concerning the following points:-

- Definition and classification of contact dermatitis.
- Mechanism of contact dermatitis.
- Factors affecting contact dermatitis.
- Diagnosis.
- Patch test.
- Cement dermatitis as an example of contact dermatitis.
- Control of contact dermatitis.

Review of literature

REVIEW OF LITERATURE

CONTACT DERMATITIS

Definition:

Contact dermatitis is an acute or chronic dermatitis that results from direct skin contact with chemicals or other irritants (e.g. poison ivy) (Rees, 1979).

Although both terms dermatitis and eczema are considered synonymous, dermatitis is usually used to denote an inflammatory condition of the skin caused by external agents. (Hjorth and Fregert, 1979).

Classification:

On aeticlogical grounds the following types of dermatitis are distinguished:

1- irritant dermatitis.

   (a) acute irritant dermatitis.

   (b) cumulative insult dermatitis.

2- allergic contact dermatitis.

3- phototoxic dermatitis.

4- photoallergic dermatitis.

(Seah and Wilknson, 1974; Hjorth and Fregert, 1979).

IRRITANT DERMATITIS

Synonyms:

- irritant eczema,
- non-allergic contact dermatitis,
- non-allergic eczema,
- orthoergic dermatitis, (**Meneghini, 1983**).
- toxic dermatitis (**Seah and Wilkinson, 1974**).

The international contact dermatitis research group is of the oponion that the term primary irritant dermatitis is redundant and that the word primary should be omitted (**Wilkinson et al., 1970**).

Definitions:

Irritant contact dermatitis describes the effect caused by a strong irritant on the skin, e.g. that caused by a strong alkali. The term "acute irritant dermatitis" may be used in contrast to "cumulative insult dermatitis", which is a dermatitis developing after repeated insults by weak primary irritants over a long period. It is to be preferred to "traumiterative dermatitis". "Wear and tear dermatitis" is an acceptable alternative. (**Wilkinson et al., 1970**).

The definition of an irritant must be rather broad: a skin irritant is any substance that, acting directly, damages the skin at the site of application for a sufficient time, in a sufficient concentration through non-immune mechanism. The term comprises so many different substances with a wide range of reactions. Actually any substance can be irritant under certain circumstances (Adams, 1983).

Irritant dermatitis represent 70-80 percent of cases of contact dermatitis (Epstein, 1983).

Mechanism of action of irritants:

Many chemicals penetrate the skin and some molecules may alter the skin cells. Dermatitis arises when these are not repaired. Many irritants induce damage through physico-chemical damage i.e. by gradually exhausting the horny layer, denaturing the keratin and altering the water-holding capacity. This increases the possibility of later biological damage to the living cells of the epidermis; cumulative insult dermatitis is often preceeded by a long period of chapping. Some irritants e.g. vesicant war gases, cause an immediate cell damage by their action on intra-cellular enzymes (Hjorth and Fregert, 1979; Adams,1983).

The mechanisms that evokes inflammation from irritants are varied. Direct cytotoxicity to epidermal and dermal cells may initiate inflammation. Alteration of normal cellular function or metabolism could also evoke inflammation, but mechanisms are not thoroughly understood. Undoubtedly a part of the mechanism involves chemical mediators, phagocytes and other components of the efferent limb of the immune response. Common mechanisms of inflammation from irritation and allergy could account for the similar clinical appearance of dermatitis resulting from low grade chronic irritation or allergy **(Dahl, 1981).**

Meneghini, (1983) classified the chemical irritants according to the aetiopathogenesis as:-

a- Oxidisers, such as hydrogen peroxide, permenganate, chromic acid and its salts etc...

b- Dehydrating agents, including strong acids, strong alkalies etc.....

c- Protein precipitants, as tannic acid, pycric acid, salts of heavy metals etc.....

d- Keratolytics, as salicylic acid, resorcinol etc....

e- Degreasing agents, alcohol, ether, chloroform, etc...

f- Various organic compounds.

Bland substances may become irritants when the skin is eczematous or has been subjected to maceration, friction, pressure, infection, excessive relative humidity and excessive perspiration (**Fisher, 1973**).

The major genetic condition leading to occupational irritant dermatitis is atopy (**Adams, 1983**).

Acute irritant dermatitis:

Acute irritant dermatitis is elecited by strong (absolute) irritants after a single application or a few brief applications. Depending on the substance applied and its ability to damage the skin not the affected individual (**Rostenberg, 1957; Hjorth and Fregert, 1979**).

Several different types of reactions may be seen. The lesions are red or brownish, sometimes oedematous and warm, sometimes with papules and vesicles or pustules, sometimes with necrosis and ulceration. (**Hjorth and Fregert, 1979; Adams, 1983**).

Any area of the skin will react. The initial reaction is strictly limited to the site of application. The concentration of the substance diffusing from its periphery almost immediatly falls below the threshold needed for provoking the reaction. This in contrast to the allergic

reaction. Within a few days, however, secondary lesions may appear without continued exposure. If the application is not repeated this form of primary irritant dermatitis clears up rapidly, unless there is necrosis **(Hjorth and Fregert, 1979; Adams, 1983).**

Examples of strong (absolute) irritants are: strong acids and alkalies, certain metallic substances and their salts, many organic compounds as DNCB.....etc.**(Adams,1983).** Other examples see appendix (1).

Cumulative insult dermatitis:

- Synonyms:- Traumiterative dermatitis,
  - Wear and tear dermatitis. **(Wilkinson et al.,1970).**
- It develops after repeated insults by weak irritants over a long period.

Housewives' dermatitis is caused by cumulative insults from cleaning, washing, cooking and bathing of children. The morphology of irritant dermatitis varies. The first stage represents a chemical damage leading to dryness and fissures, sometimes combined with enhanced percutaneous absorption. The second stage involves biological damage to the epidermis accompanied by dermal reaction.

The skin becomes red, swollen, warm and irritable. Sometimes, papules and vesicles with oozing and crusting follow. Scratch-marks and lichenification are common if