ACNEFORM ERUPTION

Thesis

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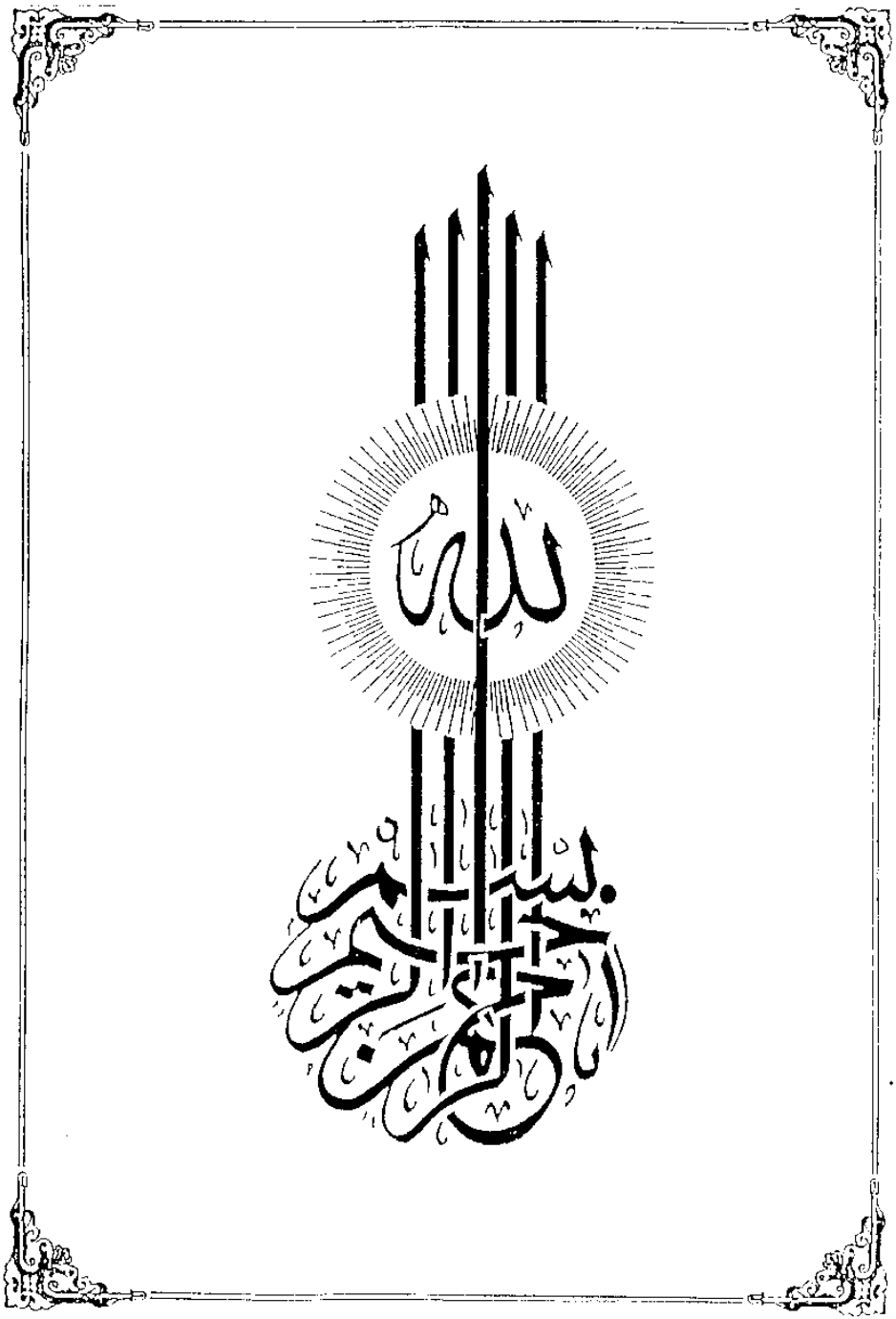

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INTRODUCTION

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The term acneform eruptions is applied to eruptions which resemble acne vulgaris. They are follicular reactions beginning with an inflammatory lesion, usually a papule or a pustule. Comedones are uncommon and they usually follow pustules (Plewig and Kligman 1975).

The eruptions are not necessarily confined to the usual sites of acne vulgaris and they are distinguished by their sudden onset, usually in a patient well past adolescence. (Domonkos, 1971).

Certain drugs and other chemicals are capable of aggravating already existing acne or inducing an acneform eruptions in predisposed subjects. (Fegeler, 1964).

The number of drugs that characteristically produced this type of lesion is rather small. The simple salts of iodine and bromine are by far the major compounds responsible for dermatitis medicamentosa of the follicular or acneform type. Generally these compounds are ingested as halogen containing cold remedies, expectorants, sedatives, analgesics and vitamins (Hitch, 1967).

Steroid acne has now become a common place in hospital practice owing to the extensive use of corticosteroids and ACTH to treat autoimmune diseases or to prevent homograft rejections. (Plewig and kligman, 1975), identical lesions can be produced by prolonged topical steroid therapy (Leyden et al., 1974; Baer et al., 1970; Plewig and Kligman, 1975).

Although the estrogen-progesterone oral contraceptives are being used in the treatment of acne vulgaris, some physicians believed that these preparations occasionally may produce an acneform eruption or, more likely, aggravate an existing acne vulgaris (Hitch, 1967).

Isoniazid induced acne form eruptions, especially in slow inactivators of the drug (Bereston, 1959 and Cohen et al., 1974).

The production of acneform lesions by several unrelated medications has been reported. Examples of such drugs are quinine, disulfiran, codliver oil, thiouracil, thiourea, trimethadione, and chloral (Hitch, 1967).

Kligman and Mills (1972) suggested that cosmetics may cause a slow-grade persistent acneform eruption, consisting of small, closed comedones with occasional papulopustules.

Acneform eruptions may be induced by exposure of the skin to the fumes generated in the manufacture of chlorine and its products (Domonkos, 1971).

Many oils and tars produce acneform eruptions at the site of contact with the skin, and the individual lesion may be identical to that seen in acne vulgaris (Hitch, 1967).

Acneform eruptions were also noted in patients who received cobalt irradiation (Trunnell et al., 1972).

AIM OF THE WORK

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This work has been performed to review the pathogenesis, the clinical picture, the pathology, the prevention and the treatment of some of the most important causes of acneform eruptions.

REVIEW OF LITERATURE

ACNEFORM ERUPTION ASSOCIATED WITH ISONIAZID THERAPY.

Bereston (1959) was the first to report on isoniazid-induced acneform eruptions in 16% of 2,600 patients receiving a combination of isoniazid and paraminosalicylic acid. A large but unspecified number of these patients were between 40 and 70 years of age, well beyond the age when acne vulgaris usually occurs, but only 11% of Bereston's acne patients were slow inactivators of the drug. (i.e = abscence of the inactivating enzyme acetyl transferase).

In 1954 when Hughes et al., showed that pyridoxine-responsive neuropathy occured predominantly in slow inactivators, it became generally accepted that the slow inactivators of the drug were the ones who predisposed to toxic reactions (Kalow, 1962).

In 1963, in a general review of reactions to isoniazid, Honeycutt and Huldin did not report the acneform eruptions, although pellagra, morbilliform maculopapular and purpuric eruptions as well as exfoliative dermatitis were mentioned, in addition there was frequent accompanying of toxic or alleric involvement of other organ systems.

The toxic reaction to isoniazid in the usual therapeutic doses (5 mg/kg) were reported to be relatively rare compared to other antituberculous agents.

The german literature reported the existence of acneform eruptions due to isoniazid (Riebel, 1963; Hesse, 1966).

In 1974 Cohen et al., studied seven patients with acneform eruptions associated with isoniazid therapy, one of whom had pellagra. Five patients, from 32 to 48 years of age, who had extensive eruptions were slow inactivators, as determined by the serum level of isoniazid six hours after administration and by serum half-life determination. Two patients, who were under 30 years of age and who had pre-existing acne vulgaris and mild flares were found to be slow inactivators by serum half-life determination.

The authors mentioned that, the following factors should be considered in the diagnosis of isoniazid-induced acne:

(1) The occurence in older persons.

(2) Abscence of recent or remote history of acne vulgairs.

(3) Sudden, extensive, or exanthematic efflorescence of lesions.

The presence of all of these factors supports a diagnosis of drug-induced acne. However, in the case of younger patients who have acne vulgaris at the onset of therapy and who sustain mild flares while taking the drug, there is more difficulty in deciding that the drug caused the eruption. Although Bereston (1959), in describing isoniazid-induced acne, did not give the age incidence of his patient, he did indicate that only 9% of patients had a history of acne vulgaris in the remote past and that 1.5% had active acne immediately prior to therapy.

Cohen et al. (1974) mentioned that perhaps higher blood level of free isoniazid in slow inactivators facilitated the development of the eruption.

Other drugs that are structurally related to isoniazid such as hydralazine, monamine oxidase inhibitor, phenelzine sulfate, also provoke side effects exclusively in slow inactivators (Price Evans, 1965).

Levantine (1972) stated that paraaminosalicylic acid, streptomycin and isoniazid may produce acneform eruption.

Cohen et al. (1974) described the eruption as monomorphic spiny keratotic plugs admixed with closed comedones and papules, no nodules or cysts appeared and healing is without scarring.

The eruption is of abrupt onset and appear on the forehead, cheeks, chin and nose and may spread to the whole trunk including the buttocks (Ebling and Rook, 1979).

A biopsy specimen of the persistent lesions showed open and closed comedones and absence of inflammatory infiltrate, which is characteristic of the early phase of isoniazid-induced acne. The open comedones showed spiny follicular plugs consisting of demodex folliculorum organisms, colonies of corynebacterium acnes, no inflammatory infiltrate.

In a later phase, the eruption showed prominent inflammatory infiltrate, no comedones, and hyperplastic follicular epithelium. These features are identical with those of acne vulgaris in the

healing phase. Compulsory discontinuation of isoniazid was not necessary. Some early lesions cleared in response to conventional therapy for acne, while others persisted and required surgical extraction. (Cohen et al., 1974).

ACNEFORM ERUPTION RESULTING FROM TETRACYCLINES ADMINISTRATION

The report of Weary et al. (1969) was the first to implicate broad spectrum antibiotics as being capable of producing an acneform eruption, in addition the authors offered a possible explanation for the occurrence of this unusual idiosyncrasy, in which they stated Since these drugs are Widely used to treat acne it is conceivable that the same reaction may occur in occasional individuals and be masked because of a basic similarity to the initial acne eruption. It is therefore important for the physicians who use broad spectrum antibiotics for therapy of acne to be aware that an occasional case may respond in such a paradoxical fashion to the medication.

A report was made for a 36-years old white female with an acneform eruption which has resulted on five occasions from ingestion of several types of broad spectrum antibiotics, one of the five episodes was induced by giving the patient one of the suspected antibiotics (Tetracycline). On each occasion discontinuation of the antibiotic resulted in gradual and complete resolution of the eruption over a period of several weeks. The patient was free in the interim period. Clinically, the lesions were symmetrically distributed in the upper and mid back chest, upper abdomen, neck and face. Several hundred lesions were present. The lesions were asymptomatic except for slight pruritus during the time of evolution and resolution.