SYPHILITIC MACULAR ATROPHY
(Case report)

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Summary

A case of Syphilitic Macular atrophy in a 38 years old male is presented with a brief review of literature. Pathogenesis of the condition is discussed.

Introduction

Anetoderma or macular atrophy, is a localized defect in the elastic tissue of the dermis, also referred to as elastolysis. This defect produces a clinical picture consisting of a circumscribed area of soft, thin and wrinkled skin, which can be pushed easily into the subcutaneous tissue by the examining finger. Often the lesion protrudes slightly as a small outpouching.

Knowledge of macular atrophy of the skin may be said to begin with the year 1891 when Jadassohn grouped the macular atrophies on a clinical and pathological basis and introduced the term Anetoderma (loose or flaccid skin). Almost at the same time there appeared a report of a case by Schweninger and Buzzi under the title "Multiple benign tumors of the skin"[1]. Since then few isolated case reports of the condition have appeared in the literature[2-8].

Clinical features of the various reported case of macular atrophy are strikingly similar to the classic description by Schweninger and Buzzi[1]. Chargin and Silver[5] classified all recorded types of macular atrophy of the skin as follows :-

(a) Primary (not preceded by known dermatosis)
  (1) Anetoderma Jadassohn
  (2) Anetoderma Schweninger and Buzzi

(b) Secondary (preceded by known dermatosis)
  (1) Syphilis (Atrophia Mucosa Syphilitica)
  (2) Tuberculosis and Lupus Erythematodes
  (3) Leprosy (Dermatitis Atrophicans Leprosa - Oppenheim)
  (4) Secondary to other dermatosis

(c) Mixed type of atrophy (Macular atrophy and Acrodermatitis chronica atrophicans.)

Secondary macular atrophies have been shown to be preceded by definite known dermatosis. But in the end both primary and secondary macular atrophies clinically resemble each other, a common characteristic in all being the phenomenon of herniation. Combes[4] and later Chargin and Silver[5] suggested that a toxic substance, whatever its nature, may be assumed to play a role

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Received for Publication on 21—12—1976

in the production of the degenerative process. Scull and Nomland[7] believed that secondary macular atrophy irrespective of the causative disease, occurs subsequent to a subclinical destruction of elastic tissue in the upper dermis by an inflammatory infiltrate. They also showed, that both primary and secondary macular atrophy have similar histologic picture, the essential and most pronounced change being fragmentation, contraction and finally disappearance of elastic tissue. Ferrara[8] has reported a case of macular atrophy with apparently valid association with severe dental sepsis.

Butterworth[6] reported a case of primary macular atrophy in which he suggested the possibility that a low grade infection may be related in some way to macular atrophy.

Syphilis may be associated with macular atrophy and certain authors have spoken of syphilitic macular atrophy [2,3].

A case is reported of a syphilitic patient suffering from macular atrophy. The purpose of presenting this paper is to emphasize the difficulty in distinguishing between primary and secondary macular atrophy both by clinical as well as histological features.

Case Report

38 year old male patient was admitted to Base Hospital, Lucknow on 22-1-75 with complaints of multiple hypopigmented atrophic lesions over the trunk and extremities of 5 years duration. His trouble began in February, 1970 when he first noticed a few lesions over the trunk. New lesions gradually developed. Older lesions became flat after several weeks and eventually became scar like. The condition was asymptomatic. History revealed that patient had sexual exposure to an amateur in June, 1966 which was followed by a sore penis 2½ months later. He took five injections of penicillin from a private practitioner (quantity not known) with subsequent healing of sore. After this he was asymptomatic and in good genereral health. He denied any history of antecedent skin eruption stating definitely that the site of lesion was never red or pink but always the colour of the skin. He is married with one child who is alive and healthy. Family history was noncontributory and his parents were living and well.

Examination of the skin revealed numerous lesions on the trunk, particularly over the abdomen, back and also on the proximal part of both upper and lower extremities (Fig. page No. 290). The lesions were isolated or in groups. Each group consisted of small and large lesions; four to ten in number. Lesions varied in size from bean to coin size. No erythema or telangiactasia could be detected in any of the lesions. The palpating end of the finger was able to press the balloon like lesions into and below the level of the surrounding skin and gave the feeling of forcing a soft tumor through a sharply marginated ring as in hernia. On withdrawal of the finger the lesions assumed their original shape. This phenomenon of herniation was a characteristic and constant clinical feature. The surface appeared wrinkled when looked obliquely. The colour of the lesion was that of normal skin or hypopigmented.

Laboratory Studies

Routine blood examination for Hb, TLC, DLC and ESR were within normal limits. Urinalysis was normal. Blood urea and fasting blood sugar were within normal limits, and so was serum calcium. Liver function tests revealed normal results. Blood serology results are shown below:—

Date	WR	Kahn	VDRL
27—1—75	Reactor	Reactor (32 Ku)	Reactor (16 Dil)
3—2—75	Reactor	Reactor (32 Ku)	Reactor (8 Dil)
10—2—75	Reactor	Reactor (64 Ku)	Reactor (16 Dil)
3—3—75	Reactor	Reactor (16 Ku)	Reactor (8 Dil)
17—3—75	Reactor	Reactor (32 Ku)	Reactor (32 Dil)

Mantoux test was negative. X-ray chest gave no evidence of tuberculosis or any other disease. ECG readings were normal.

Skin biopsy showed epidermis which was slightly thinner over the lesion and some flatening of the rete pegs. Collagen was swollen and homogenized. There was moderate cellular infiltration limited to the upper cutis. There was minimal round cell infiltration around the blood vessels and glands of the skin. Weigert's elastic tissue stain revealed fragmentation and reduction of elastic tissue throughout the dermis. Clumping of the broken fibers was seen in the deeper part of the dermis. Elastic tissue fibres in the deeper vessels were present.

Clinical Course:—Patient was given a course of antisyphilitic treatment with Benzathine Penicillin 2.4 mu weekly for 4 weeks. Total number of lesions, over 150, did not increase since treatment was given and over a followup period of about two years.

Discussion

Macular atrophy in syphilitic patients has been observed in two forms. In some the atrophy is merely associated with syphilis whereas in others it replaces a prior syphilitic lesion. Transformation from a syphilitic papule to atrophy occurs through various stages which usually requires a period of less than two months.

The papule first becomes flats and is gradually transformed into a livid red lesion with peripheral pigmentation. The skin becomes thin and weak. It soon becomes concave ; the surrounding pigmentation becoming less marked as the atrophy becomes more pronounced. Thus three stages can be differentiated:—

(i) Infiltrated papule
(ii) Change of colour
(iii) Atrophy

While macular atrophy usually follows papular lesions, it has also been observed replacing macular lesions. The localization necessarily varies according to the location of the syphilitic eruption which it replaces. It is usually seen on the trunk, extremities and abdomen. However, lesions have been observed on the head, thighs, breasts and face. Size of lesions vary from that of a pea to a 5 cent piece.

Atrophy usually appears during the second or third year of syphilitic infection. Many exceptions to this have been reported. In some instances the atrophy has occurred 10-29 years after the infection.

That syphilis is regarded as an important etiological factor in macular atrophy goes without saying. There are numerous reports of cases in which macular atrophy appeared on pre-existing syphilitic eruption[2,3]. On the other hand there are cases wherein only a positive Wassermann, or syphilitic history was present with a well pronounced macular atrophy. In such cases macular atrophy did not seem to have replaced any active lesion. Syphilis can hardly be regarded as a common cause of macular atrophy since macular atrophy is rare but syphilis common. If syphilis had a direct effect upon elastic tissue, macular atrophy would be much

more common. It is possible that patients with syphilitic macular atrophy, have a special disposition of the skin to such a change; the syphilitic infection acting then as a precipitating factor.

The essential change in all types of macular atrophy is seen in the elastic tissue which shows fragmentation, contraction and finally disappearance. While the inflammatory reaction most often precedes the destruction of elastic tissue, complete loss of tissue may occur without any evidence of inflammatory reaction. On the basis of this observation, it has been suggested that in addition to inflammation some toxic substance, may also play a role in the production of the degenerative process[5].

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