EARLY LESIONS IN DISSEMINATED REACTIONAL CASES

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ABSTRACT*-Authors*** studied 50 patients with disseminated reactional lesions and classified both as reactional tuberculoids and reactional borderline (type I reaction of Jopling). After careful story taking and clinical and dermatological examination, it was clear that all of them had similar clinical story as regards the beginning of the disease, regardless of the immunological grade. The initial lesions (*mother lesions)* are hypo chromic macules or circumscribed areas of normal color skin with some sensory impairment, which should not be misinterpreted as those reported by Wade that circumscribe Immune areas". These lesions seems to precede a phase of bacillemia with destruction of bacilli, in the place they are, and the liberation of antigens with the consequent manifestation of delayed hypersensitivity, which is the reactional episode. Treatment would have some role to play only in those cases with low immunity and , thus, prone to new reactional episodes. Without treatment, these patients would continue to present new reactional episodes indefinitely.

Key-words: *Type I reaction - reactional tuberculoid - "mother lesion"*

1. INTRODUCTION

Today it does not cause much interest to discuss the clinical features of Hansen's Disease (HD) lesions. The understanding of their morphologic aspects and its significance is overcame by more recent issues such as immunology, bacteriology or the proposition of new therapeutic regimens. This is the reason why many of what is today considered as truth is based in short term clinical observation and end up originating misleading immunological correlations and inadequate interpretation of therapeutic results.

An example is the lack of consensus between reversal reaction and relapse, what has important practical implications. In this sense, if reversal reaction is considered a relapse, how could be explained the therapeutic results with MDT in PB patients who shows a reaction after release from treatment (RFT)?Anyway, in the clinical aspect of HD there are many manifestations which deserve, in our point of view, to be investigated and one of them is the mode of onset of disseminated reactional lesions in tuberculoid and borderline patients. The result of such investigation will be discussed in this paper.

2. METHOD

50 patients with disseminated reactional lesions and classified both as reactional tuberculoid and reactional borderline (type I reaction according to Jopling) were studied.

All patients presented similar clinical story as regards the beginning and evolution of the disease after diagnose and treatment. Because of this, we report here the cases of only two patients, one pertaining to that group showing Mitsuda test of 5 mm or higher and the other portraying those "reactional borderline" cases with Mitsuda test negative or lower than 5 mm.

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3. REPORT OF CASES

Case 1. I.B.L.F is a white, married, 56 years old, Brazilian woman. In august 1993 she told us that since 6 months ago she noted an anesthetic patch in the left elbow. Three months later a crop of elevated erythematous patches appeared in all her body. The initial anesthetic patch in the elbow become a plaque with elevated and erythematous borders surrounding a central area of normal looking skin. Skin smears were negative and Mitsuda Test equal 5 mm. Histopathological examination revealed a picture compatible with reactional tuberculoid Hansen's disease.

Case 2. F.L.S. is a white, married, 40 years old, Brazilian woman. She noted an anesthetic patch on the anterior aspect of her left thigh 5 years before her first consultation in our Institute. Six months ago many erythematous and varied size plaques appeared in all her body. Skin smears were positive and B.I was 0.6 without solid stained bacilli. The Mitsuda test was negative an the histopathology showed a reactional borderline picture. The remainders of clinical examination was inexpressive.

4. DISCUSSION

It became clear from our observations that the lesions which preceded the reactional outbreak, the "mother lesions", are hypochromic macules or circumscribed areas with sensory impairment.

The early reactional episodes are similar to those occurring in Pityriasis rosea of Gibed where an initial plaque termed herald patch precedes the other characteristic lesions of this dermatological condition.

WADE also mention the "mother lesion" in reactional cases. He consider these lesions as being lesions surrounding "immune areas", that is, a remission area of previous reactional outbreak (relapse lesions).

According to **Souza Lima** and **Souza Campos,** the reactional tuberculoid lesions arise in the five following circumstances a) in subjects apparently healthy; b) in subjects with atypical tuberculoid lesions that changes to reactional lesions; c) in subjects with indeterminate lesions which suddenly turn to be tuberculoid; d) in subjects with anesthetic patches on which reactional lesions are installed and e) in patients with chronic tuberculoid lesions that enter in reaction.

In the first case the initial lesion probably was not noted. It is also possible that cases with chronic tuberculoid HD turns to be a disseminated reactional case through a reversal reaction. However, this is not very frequent.

The most common occurrence is the initial lesion to be a flat, hypo pigmented or normal looking skin color lesion with sensory disturbance. It could also correspond to an indeterminated leprosy that, by an abrupt transformation, would change to a reactional tuberuloid leprosy. In the same way such transformation could occur in an atypical tuberculoid lesion, which is clinically undetermined but already showing in the histopathology a pre-tuberculoid infiltration.

From all that we have been discussing about these anesthetic, flat, hypo pigmented or normal looking skin color lesions, it seems that there really are those who correspond to indeterminate HD and other that, despite of continuing with the morphology of indeterminate HD, they already presents an histopathological picture of pre-lepromatous, pre-borderline or pre-tuberculoid types, what make them lesions of these clinical forms.

Among these, are the lesions previously termed "maculo-anestetic"which were subject of so much discussion with authors from India and that, finally, were included among the tuberculoid macular type in the Madrid Classification.

Cochrane already admitted the existence of cases not inderterminate which presented large numbers of hypopigmented and hypo esthetic macules. He thought that these cases took part of a group that he termed dimorphous-macular (pre-dimorphous) from which would arise the reactional tuberculoid cases that he used to term as low resistant tuberculoid. Chochrane thought that those cases would begin as borderline and then, by a hypersensitivity reaction, they would make a shunt through the the borderline zone of the spectrum and would transform themselves in tuberculoids. Ridley is in agreement to this opin-

ion. He admits the existence of TT cases with delayed hypersensitivity which certainly correspond to the reactional tuberculoids and called them secondary TT being TT in the sense that they have evolved as a result of reactional processes which often start in the borderline part of the spectrum.

The characterization of these early lesions helps to clinically interpret the reactional picture presented by the patient. Thus, among the multiple lesions that a reactional patient presents, there are those predominant, which are erythematous papules and plaques and other single or in a low number of lesions which are larger in size and that occasionally present a different morphology. These later are also plaques with irregular shapes which have a hypo pigmented or a normal skin color center and sensory impairment. These lesions should not be mistaken with those which circumscribe "immune areas" as reported by WADE and that correspond to the lesions of a reactional episode already regressed which are "respected"by lesions of a new one. Such lesions, termed "holey"and "swiss cheese"lesions by several authors today, may have the same connotation given by Wade but some of them are indeed the flat initial lesions which are participating of the reactional episode.

As regards to relapsed tuberculoid lesions, hitherto nobody has considered the possibility of such lesions being actually relapsing from the edge of apparently regressed lesions, instead of being "respecting immune areas". If this is true, they have the same explanation of that initial lesions. The relapsed tuberculoid lesion would be, indeed, the first lesion of a new outbreak.

These facts should be admitted since all the reactional cases exhibit this kind of lesion, regardless of their skin smears result, histopathology picture or immune level assessed by the Mitsuda test. Therefore, this means that all cases begin in the same manner. Thus, at the beginning, the reactional cases presents one or more circumscribed, hypo pigmented or normal skin color, flat areas with sensory impairment. Suddenly, the edge of these lesions become erythematous and raised and their central area may or not take part of the reactional process. Later, new erythematous papules and plaques occur throughout the skin. Some of these lesions may become ring shaped and this occur when the regression starts by their center which tends to become more flat and light. New outbreaks increase the number of lesions. The more recent outbreaks lesions are smaller in size that the ones of the previous reactions.

It is also possible that lesions of new reactional episodes will progressively lose some of their characteristic. Their limits may fade out and become irregular. If we consider that the relapsed lesions actually correspond to reactivation from edge of old and already regressed reactional lesions, as the number of episodes increase, more bizarre will be the dermatological picture presented by the patienty.

Changes in the morphology of lesions was shown by WADE when he described, for the first time, his "borderline"cases and the relapse lesions. It is interesting to remember that he wrote that he has never seen the so called "lepromotous transformation" in such cases. In the other hand, RYRIE in Malaysia admitted that his ulcerative reactioal cases changed to lepromatous pole due to a "tissue mania". Wade, however, examining the patients of Ryrie could not observe such transformation in any of his cases. Although many authors believe in this `transformation", it could hard be possible that it indeed occurs. Lepromatous cases have chronic lesions since the very begging of the disease and there are no reports of purely reactional cases that suddenly stop to present new outbreaks and begin to show lepromata due to a supposed deterioration of their immunity.

It seems that the initial lesions in the reactional cases forerun a bacillary phase with the destruction of Bacilli in the places where they have settled in the skin with consequent antigen release which will elicit a delayed hypersensitivity reaction. If there will be new outbreaks, they will be due to new bacillemia. In these cases the bacilli are those that were not completely destroyed just before the installation of a previous outbreak. Treatment may play some role in cases prone to suffer new outbreaks due to their lower immunity. Without treatment these cases will continue to present new outbreaks indefinitely.

NOTICE: Legend in English can be found in the original article in Portuguese (pág. 37).

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