

SANCHIS
-OLMOS



Skeletal Tuberculosis

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SKELETAL TUBERCULOSIS

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BALTIMORE

THE WILLIAMS & WILKINS COMPANY

1948

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Made in the United States of America

COMPOSED AND PRINTED AT THE
WAVERLY PRESS, INC.

FOR

THE WILLIAMS & WILKINS COMPANY
BALTIMORE, MD., U. S. A.

TO
PROFESSOR FRANK R. OBER
AND
DR. JOHN G. KUHNS

*To all American Scientists, who have contributed
so widely to the progress of
Orthopedic Surgery*

FOREWORD

A book on bone tuberculosis appearing at this time may seem to some to be unnecessary, because bone tuberculosis in this country is diminishing so rapidly that the disease may become a rarity if not non-existent.

Herein lies the great danger that patients may suffer from tuberculous bone infections and go undiagnosed too long. It is, therefore, most important to keep the subject alive and up to date both from the clinical and scientific aspects since the problem of cure has not been solved ideally and the problem of diagnosis must be with us always.

This volume should be of use to all practitioners and students, since it covers so well the modern concepts of this subject. The author, who is a Spaniard, is to be congratulated on his production to which he has devoted a vast amount of work and time in this country, while studying the problem of bone tuberculosis and recording his ideas and conclusions.

FRANK R. OBER, M.D.

PREFACE

When I began my trip to the United States, I never suspected that I was going to write this book. In spite of the difficult work involved in carrying out the task, I gladly welcomed the kind suggestion of my colleagues in Boston that I do so. The work had two-fold purpose: on the one hand, it would permit me to set forth in the English language doctrinal concepts and scientific judgments not published heretofore in that language; on the other, it would give me the opportunity to review the subject with new clinical material, which would allow me to contrast the particular type of infection in the two continents while I revised my personal concepts.

Thanks to the aid of Professor Frank R. Ober, who held the Chair of Orthopaedic Surgery at Harvard University and who made available to me the abundant material of the New England Peabody Home for Crippled Children, I have been able to study the clinical aspects of tuberculosis of the skeleton with North American material. This fact is important since my statements, theories, and conclusions are based on cases whose clinical histories and subsequent developments can be examined at will by other colleagues. In order to facilitate discussion, I have given the case numbers as recorded in the files of the hospital, both in the illustrations and in the text, when it seemed of value to do so. Some pathologic aspects and the cause of deaths have been obtained from the autopsy records, furnished by Dr. Sidney Farber, Associate Professor of Pathology of Harvard University; this has made it possible for me to trace the development of the disease in its entirety.

The book has been written with the intention of emphasizing that which is new or little known and makes no pretence of being an exhaustive study of the subject. For this reason there are repetitions of ideas and concepts which I consider fundamental, while many technical details are only lightly treated.

The bibliography that I have used has been abundant. The greater part of it is listed at the end of the work. Almost all the books previously published on the same subject have been examined, and two of them were found especially useful: the book by Kremer and Wiese and the work of Gonzalez-Aguilar. I consider them the orthopedic specialists who have opened new roads, which I follow in part, attempting to lift the study of osteoarticular tuberculosis out of the rut in which it lay. My hope and ideal is that this study may be a step forward toward the common goal which permits us all to cure these patients with greater efficacy.

It was possible for me to complete the book during a few months' stay in Boston, since some aspects of the subject has already been discussed and

outlined in my monograph *Artritis tuberculosa de la cadera (Tuberculous Arthritis of the Hip)*.

The kindness of many good friends has helped my work to take on concrete form. Besides Professors Ober and Farber, already mentioned, I especially want to thank the staff of the New England Peabody Home, Drs. A. H. Brewster, Joseph S. Barr, E. F. Cave, P. W. Hugenberg, and others who aided my work and welcomed my ideas. Mrs. Smith and Miss Whealock, as well as their staff of nurses and secretaries, were extremely considerate while I was at the hospital.

Dr. William A. Rogers, editor of the *Journal of Bone and Joint Surgery* offered me a helping hand from the first day I arrived in Boston.

The Spanish Research Council, The Spanish Department of Cultural Relations, and the Minister of National Education of Spain have furnished me the means for prolonging my stay and bringing to completion the task I had begun.

I am indebted to Mr. Minos Generales of Boston University and to Mr. Tuckerman Day for examining the manuscript and giving suggestions for its improvement.

I shall never be able to express properly my gratitude to Dr. John G. Kuhns, Chief of the Orthopaedic Service of the Robert Brigham Hospital, for the enthusiasm and warmth he put into the translation, which he voluntarily accepted and which has taken precedent over his own work so many times.

My thanks are due also to the publishers, not only for their handsome presentation of the book, but for the interest which they have in Spanish and American scientific interchange. I hope that this book will be the first of a series to present to the English-speaking public the present state of Spanish medicine, which is just as universal as our religious, political, artistic, or cultural spirit.

It would be unfair to omit mentioning that the merit of having completed an undertaking which without the necessity of haste and in its proper perspective would have been a simple matter, but which was in my special case full of varied and unsuspected difficulties, is due to the persistent, unselfish, and cheerful collaboration unfailingly given by my wife.

SANCHIS-OLMOS

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PART I
GENERAL DISCUSSION

CHAPTER I

PATHOGENESIS

Tuberculosis of the bones and joints is conditioned in its origin, in its anatomic and clinical manifestations, in its evolution, and in its therapeutic indications by the variations in the biologic phenomena of tuberculous allergy. These show changes in the skeleton similar to those seen in the lungs; in fact, such changes appear in an extremely clear and definite form.

The appearance of an osseous or articular lesion is the result of a hematogenous dissemination from a primarily infected visceral focus. Thence arises a clear distinction between the two phenomena, primary infection and hematogenous dissemination, which, according to the theory of Ranke, represent the first and second period of infection. Theoretically, one could imagine that in a patient previously uninfected a wound penetrating to the skeleton could bring it in contact with Koch's bacillus. In this event there would be produced a bony or synovial primary complex—that is, myelitis or synovitis—which would give rise to a lymphangitis extending to the proximal lymph nodes, which would also become infected.

The concept of Ranke is disputed by many students of tuberculosis, especially by the school of Aschoff, which attempts to combine the two primary periods of Ranke in a single one of primary infection. As an osteologist, I accept Ranke's doctrine because the osteoarticular infection requires previous bacillary dispersion by way of the blood stream. The phenomenon of dissemination is one that can appear, according to the doctrine of Ranke, in the period of secondary allergy.

In the first period the organism is infected, reacting with the visceral focus, and in this phase of primary allergy the skeleton does not show the results of infection. For this to occur there must be a hematogenous bacillary dissemination, a completely distinct phenomenon which may either follow immediately on the first period or be delayed for several years. For the skeleton, then, I am able to accept the periods of Ranke. In the first period the skeleton remains immune, but is subject along with the rest of the organism to a possible infection coming from a hematogenous dissemination, occurring in the second period.

The concept of Ranke explains the phenomenon of Koch. If the skin of an uninfected animal is rubbed with an emulsion of the bacilli of tuberculosis, there originates a torpid ulcer with little tendency to heal, which does not lead to acute local or general reactions and which progresses accompanied by a caseous adenopathy of the neighboring lymphatic glands. If the experiment is carried out on an animal previously infected, the local lesion is of the acute type, healing with greater rapidity, and progresses

accompanied repeatedly by general manifestations and without glandular reactions.

Clinically one may observe the same phenomena as are seen experimentally. This is illustrated by the following cases:

A girl of 6 presented a productive-caseous osteoarthritic lesion of the left knee, which had been present for 2 years. The neighboring lymph nodes were enlarged. In order to study the pathologic aspects, I performed a biopsy in Scarpa's triangle, removing a caseous node the size of a hazel nut, previously shut off from all efferent and afferent lymphatic vessels. In picking it up with a forceps it was impossible to avoid crushing it slightly. The cutaneous operative wound healed normally by the 8th day. On the 10th postoperative day, the operative area became acutely inflamed and the temperature, which had been normal, rose to 102° F. The only treatment was the application of tincture of iodine. Ten days later the affected area had returned to normal and the patient was afebrile. During this time there was not the slightest clinical alteration in the affected knee or in any other part of the body. The crushing of the node involved in its removal provoked the same effect as the second phase of the experimental phenomenon of Koch.

The skeletal impregnation can occur during the phase of secondary allergy, with the characteristics of clinical sharpness typical of the period of hypersensitiveness. The bony focus may be the only manifestation of clinical tuberculosis. Similarly, the skeletal lesion may be the focus of infection of other organs or tissues, in some of which the infection may be fatal so long as the phase of general hyperergy persists. The following cases are illustrative.

Case 696. This patient was a 5-year-old girl. In the fall of 1943 her mother noticed that the child's gait was stiff and awkward. She began to hyperextend the spine when walking or standing, and complained of pain in the back after sitting. A kyphos appeared suddenly about November 1. The child was sent home from school, and in January, 1944, she was admitted to the Children's Hospital, where a diagnosis of tuberculosis of the lumbar spine was made. She was sent to the Peabody Home on January 27. The tuberculin test gave a positive reaction. X-ray examination showed involvement of four lumbar vertebrae and a large paravertebral abscess. No lesion was seen in the lungs. There were calcified glands in the mesentery and the cervical region. The patient was treated in recumbency in plaster shells. Early in June, 1944, the vomiting began and there were daily elevations of temperature (100 to 103° F.). Malaise and indifference followed. The child developed tuberculous toxemia and died on June 30, with the picture of a tuberculous meningitis.

The autopsy showed a cured Ghon's lesion, pleural adhesions, and miliary tubercles in the lungs, liver, spleen, adrenal glands, meninges, and vertebral bodies. The bodies of the 7th and 8th dorsal and 2nd, 3rd, 4th, and 5th lumbar vertebrae were completely destroyed. The 12th dorsal and 1st lumbar vertebrae also presented lesions. There were three abscesses, one in the body of the left psoas muscle, another paravertebral, beginning at the level of the 10th dorsal vertebra and extending downward, and the third presacral, measuring 6 cm. in length and 5 cm. in width (Fig. 1).

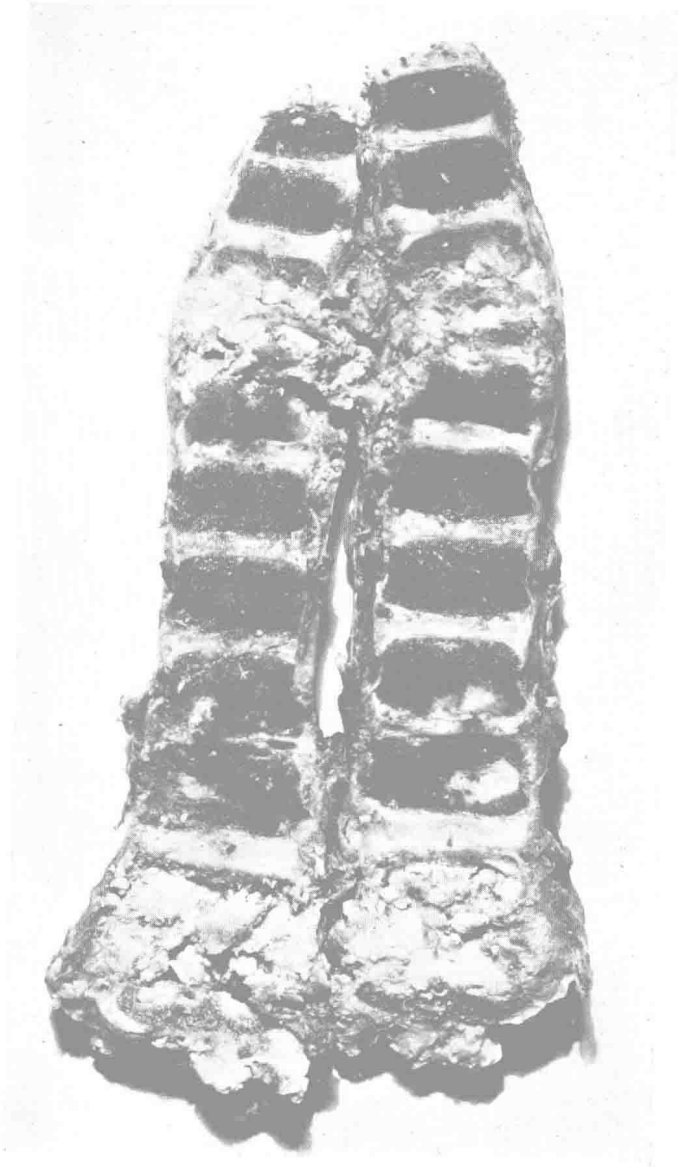


FIG. 1. Case 696 (see text)

Generally one finds lesions of the same pathogenic significance in the lungs (miliary lesions, infiltration, large hilar lymph nodes, and pleuritis), these lesions being the ones that usually provoke new outbreaks. Follow-