A CASE OF TUBERCULOUS RHEUMATISM
(GROCCO-PONCET TYPE)

BY

E. MONTUSCHI

The conception that rheumatoid (atrophic) arthritis and acute polyarthritis simulating rheumatic fever may in certain cases have a tuberculous aetiology with an allergic or bacillaemic pathogenesis, was born in Florence with the teachings of Pietro Grocco (1892), who held the chair of clinical medicine at the University.* Because of this, many papers on tuberculous rheumatism were published in Italy at the beginning of the century. A second crop of papers in the 1930's can be traced to the fact that Cesare Frugoni, a disciple of the Florentine school, became Professor of Medicine in Rome at that time. In 1932 Frugoni and Peserico, reviewing the subject of tuberculous rheumatism, proposed that the condition should be named "Grocco-Poncet" rheumatism. They expressed doubts of the claims of Reitter and Loewenstein (1933) that a tuberculous bacillaemia is commonly found in this condition, and also rejected the claims of the French authors that the condition itself is very common. The Roman school of medicine adheres to the view (Klinge, 1929) that rheumatism is a specific reaction of mesenchymal tissue to non-specific noxae, and believes that tuberculosis is one of the agents capable of producing the clinical and pathological changes characteristic of rheumatism. The observation of Bezançon and Weil (1930) and others of a clinical case of tuberculous rheumatism, in which an Aschoff nodule containing acid-fast bacilli was found at autopsy in one of the affected joints, appears to lend support to this view. No similar observation has since been reported, so far as I know. To A. Poncet (1903), however, and to the Medical School of Lyons, is generally given the credit for the introduction of tuberculous rheumatism in medicine. The French school certainly fought hard for its offspring and finished by attributing to it such far-reaching virtues as to induce much scepticism: "The first task when confronted by a case of rheumatism is to prove that it is not tuberculous" (Poncet and Leriche).

The "discovery" of a filterable phase of the tubercle bacillus (Fontes) lent impetus in Europe and in South America to the theory of a possible tuberculous aetiology of rheumatism. This discovery, however, remains unconfirmed (Topley, 1936). Then in the early 1930's came from Prof. Loewenstein's laboratory in Vienna a series of triumphant bulletins announcing the finding of Koch's bacillus in the blood stream in a variety of conditions, from disseminated sclerosis to rheumatism (Reitter and Loewenstein, 1933). Naturally a flood of papers followed in Central and Western Europe, and in 1934 Pic from Lyons, published an article under the title of: "Rheumatisme et tuberculose; Évolutions des conceptions médicales de Bouillaud à Poncet, de Poncet à Loewenstein." In England, Copeman and Clay (1935) published two cases of rheumatoid arthritis believed to be of tuberculous origin, and followed this with a series of twelve cases in which the most important evidence of tuberculosis was the report of positive blood cultures from samples sent by him to Prof. Loewenstein's laboratories in Vienna (Copeman, 1936). He quoted at the same time the 1933 memorandum of the Medical Research Council (Wilson, 1933) which entirely disproved Loewenstein's findings, and pointed out that the positive blood cultures were only "important evidence of tuberculosis" if the reader chose to believe in the reliability of Loewenstein's method, but that there was also clinical evidence of tuberculosis in support. Copeman's conclusion was that a tuberculous factor should be considered in all cases of atrophic arthritis in which the aetiology is obscure. His paper includes an extensive bibliography and is a useful guide to English readers on the subject. In the United States, Brav and Hench (1934), reviewing the subject in 1934, came to the conclusion that "there was as yet no adequate clinical method of identifying tuberculous rheumatism, no consistent roentgenographic evidence, no experimental nor laboratory evidence in its favour that is not highly controversial, and no consistent demonstration of its supposedly characteristic microscopic pathology." This is, to my mind, an unduly pessimistic conclusion, as individual cases have been published in which the clinical evidence in favour of a tuberculous aetiology appears overwhelming (vide Copeman's review).

It is clear, therefore, that the condition is uncommon and that damage to the good reputation of tuberculous rheumatism has been done by the exaggerated claims which continental authors have made on its behalf, and by their attempts to bolster up their claims in more recent times with bacteriological findings which have been disputed and remain as yet unconfirmed (Wilson and Topley). The case I am presenting furnishes, I think, sufficient evidence for a diagnosis, on clinical grounds, of acute rheumatism (simulating Bouillaud's disease) of tuberculous origin. To the English-speaking readers it has an added interest in that the reports of W. S. C. Copeman and of Brav and Hench limit themselves to the consideration of a possible tuberculous aetiology in certain cases of rheumatoid (atrophic) arthritis, but do not describe, nor consider, cases of acute rheumatism of tuberculous origin.

* Grocco's original paper "Osservazioni su due forme cliniche dell'infezione tubercolosa," *Lo Sperimentale*, 1892, is not easily available in London. An important quotation from Grocco's paper, translated into French, can be found in Frugoni, C., and Peserico, E., "Rheumatisme et Tuberculose," *Acta rheumatol.*, 1932, **4**, 9–14.

CASE HISTORY

A male aged 19 years, was admitted to the pleural effusion unit on March 3, 1943, from another hospital. Sudden onset of illness on Dec. 25, 1942, with pain in right hemithorax, malaise, and unproductive cough. Mid-January, 1943, developed pain in left hemithorax also with malaise, sweating, and a little fever. No joint pains. No sore throat. Treated at home by panel doctor up to Feb. 11, 1943, when he was admitted to hospital. There, diagnostic aspiration from left pleural cavity showed straw-coloured clear fluid with high protein content, and a predominantly lymphocytic cell count; the fluid was sterile (Loewenstein-Jensen culture also negative).

Past history was uneventful: chicken-pox, measles, and whooping-cough in infancy; not subject to recurrent colds or sore throats. He is an only son; parents are alive and well. No history of contact with known case of pulmonary tuberculosis. Had been a clerk up to March, 1942, when he became a sheet-metal worker. Hours of work from 8 a.m. to 6 p.m., with frequent overtime. This increase in his working hours often left him very tired.

On admission the patient was a well-built youth with an obviously toxic facies; moist, white, "transparent" hands; and recent loss of weight (on Feb. 3, 1943, weight 145 lb.; highest known weight 164 lb.; weight on Nov. 1, 1943, 170 lb.). There was a malar flush, slight cyanosis of finger-nails, but no clubbing of fingers. Occasional unproductive cough and some pain in the right hemithorax, of pleuritic type. T. 98·8° F.; P. 80–95, regular; B.P. $\frac{110}{80}$. The trachea was pushed to the right and there was diminished excursion of the left hemithorax. The apex beat was visible and palpable inside the nipple line. The heart sounds were pure, but the second pulmonary sound was loud and reduplicated. There were signs of a left pleural effusion of moderate size, and a loud, pleural-friction rub was heard at the right base. Apices clear. Abdomen well retractable; liver not palpable. No signs of free fluid in the peritoneum. Urine clear; no albumin or sugar. A skiagram of the chest showed normal heart shadow, small left pleural effusion, peaking of the right dome of diaphragm; no parenchymatous lesion observed. Tuberculin patch-test (Evans and Lescher) clearly positive after 48 hours. Enthrocyte sedimentation rate: 25 mm. first hour (Westergreu).

Although there was no fever the disease was obviously still active nine weeks after its onset, and the patient was kept on absolute rest. On Feb. 6, 1943, his condition appeared to deteriorate; the temperature rose to 100° F. and the patient was listless and refused food. The physical signs in the chest were unchanged. On Feb. 9 pain and swelling appeared in the first interphalangeal joints of the first and second fingers of the left hand. The swellings were tender, not red, and there was some limitation of movement. Within the following twelve days these joints became involved in succession: right wrist and right hand, left ankle, left wrist, and right ankle; vague pains in elbows and knees but no visible swellings nor limitation of movement. As the other joints became involved, the first appeared to heal. During this time there was moderate fever (99°/100° F.); the pulse was regular at between 90–95 per minute, and the patient was clearly very ill. He started to improve by Feb. 22 when his temperature became normal and no swelling or tenderness could be found in the joints. A week later the improvement was marked. The patient has since made a slow but steady recovery. He is now up and about half day and is allowed to perform some light engineering work in the ward. The left pleural effusion is clearing up, there remaining only some pleural thickening at the left base, while an adhesion holding up the middle part of the diaphragm is still seen in the skiagram of the chest. The heart is now central, of normal size and contour. No bruits have been heard at any time. Electrocardiogram is normal.

During the episode of acute polyarthritis treatment was confined to an occasional dose of nepenthe to relieve pain, and to light splinting and warm wool around the affected joints. A throat swab gave less than 5 per cent. haemolytic streptococci. Antistreptolysin titre of serum 30 units. Blood count showed moderate secondary anaemia (Hb. 80 per cent.). White cell count was 7,600 per cmm. Differential white cell count was: polymorphs, 68 per cent.; basophils, nil; eosinophils, 1 per cent.; lymphocytes, 31 per cent.

DISCUSSION

The diagnosis of this case, in the absence of any bacteriological proof of tuberculosis, rests on clinical grounds. The differential diagnosis is evidently between tuberculosis and rheumatic fever. The main point against a rheumatic aetiology is that it is difficult to imagine a bilateral rheumatic pleurisy persisting throughout many months, complicated by an episode of acute polyarthritis, not affecting the heart. Then, the onset of the illness was abrupt, with pleural pain not preceded by a sore throat or even vague joint pains. The episode of acute polyarthritis was short, especially as no salicylates were given. Since the end of February there has been no recurrence of general pain or swelling. Pain also appeared to be less severe in the affected joints for the degree of swelling than would have been expected in a comparable case of true rheumatic fever. A curious feature was that, while the proximal joints (shoulders and hips) were apparently not affected at all, and the elbows and knees were only subjectively involved, the wrists, hands, ankles, and feet, more distally placed, showed swelling and tenderness with limitation of movement. The low haemolytic streptococci in the throat, the white cell count, and the low antistreptolysin titre of the serum, all bear witness against a rheumatic aetiology. The diagnosis of tuberculous pleural effusion rests on the acute onset, on the chronicity of the condition, on the fact that both pleurae were involved, on the severity of the illness with a relatively low degree of fever, on the character of the pleural fluid, on the white cell count, and upon a positive skin reaction to tuberculin. It is, of course, the whole picture and not any single feature which is diagnostic. Is there any reason, apart from obstinate prejudice, for postulating a different aetiology for the polyarthritis? I can find no such reason.

While the diagnosis of acute tuberculous rheumatism appears to be fairly certain, I feel that it would be idle, in the absence of bacteriological and pathological proof, to make any pronouncement on the pathogenesis of this case, whether it be a tuberculous bacillaemia or an allergic reaction to tuberculous toxin. Clinical proof of bacillaemia would

have been the development of true tuberculous arthritis in one of the affected joints, but this did not happen.

The experience at Sidcup of over 350 consecutive cases of tuberculous pleural effusion (Heaf and Ellingworth, 1944) shows that transitory tuberculous bacillaemia is not uncommon in the course of this disease. Apart from the more obvious cases of discrete or frank miliary dissemination, distant bone and joint lesions and tuberculous bacilluria, the incidence of lung foci of presumed haematogenous origin is relatively common. I have also twice observed transitory monarticular swellings—one of a knee, the other of an ankle—with complete resolution within three weeks, and I confess to a great reluctance in ascribing these swellings to allergy. A sudden allergic flare-up due to exogenous re-infection—if such an event ever really occurs—can be excluded with some degree of confidence, as it is a strictly observed policy at Sidcup not to admit or retain open cases of pulmonary tuberculosis. Finally, the low eosinophil count is not in favour of an allergic reaction. On the other hand, we all know that allergic phenomena do occur during the course of tuberculous disease. While confessing to a personal bias towards the bacteriaemic pathogenesis, the experimental aspects of rheumatism, allergic phenomena and their related problems are so complicated and full of pitfalls that I feel that further discussion of these points would be unfruitful. It is therefore best to limit this discussion to the observed clinical facts, and the facts, I think, lead to a diagnosis of " tuberculous rheumatism " in this case.

SUMMARY

A brief historical outline of the vicissitudes of tuberculous rheumatism is given, with the reasons for naming this condition Grocco-Poncet rheumatism. It is recalled that a tuberculous aetiology has been described not only in cases of rheumatoid (atrophic) arthritis but also in cases of acute polyarthritis simulating rheumatic fever. The conclusion is reached that tuberculous rheumatism is a true clinical condition, but that the claims made on its behalf by the Lyons' Medical School and by other continental authors are grossly exaggerated.

A personally observed case of tuberculous pleural effusion is described, in the course of which an acute polyarthritis occurred, simulating rheumatic fever, with complete resolution. The diagnosis of tuberculous rheumatism in this case is discussed and upheld. No conclusions as to the pathogenesis of this case, whether through bacteriaemia or allergy, are arrived at, but the writer's personal bias towards a hypothesis of tuberculous bacteriaemia is confessed.

The L.C.C. is in no way responsible for the views expressed in this paper.

REFERENCES

Bezançon, F., Weil and Others (1930). *Bull. Acad. de Me'd., Paris*, **104**, 676.
Brav, E. A., and Hench, P. S. (1934). *J. Bone and Joint Surg.*, **16**, 839.
Copeman, W. S. C. and Clay, L. D. (1935). *Lancet*, **2**, 1460.
—— (1936). *Reports on Chronic Rheumatic Diseases*, No. 2, London, civ. 24.
Fontes, C.: quoted by Topley and Wilson. P. 291.
Frugoni, C., and Peserico, E. (1932). *Acta rheumatol.*, **4**, 9, 14.
Grocco, P. (1892). " *Osservazioni su due forme cliniche dell'infezione tubercolosa*," *Lo Sperimentale Florence*.
Heaf, F. R. G., and Ellingworth, C. (1944). *Brit. J. Tuberc.*, **38** No. 1.
Klinge, F. (1929). *Beitr. 2 Path. Anat. V.Z. Allg. Path.*, Nov. 19, civ. 205.
Pic, A., Delore, P., and Chapuy, A. (1934). *Journ. de Me'd de Lyon*, **15**, 283.
Poncet, A., and Maillaud, M. (1903). " *Rheumatisme et Tuberculeuse (Pseudorheumatisme d'origine bacillaire)*," 8°, Paris.
Reitter, C., and Loewenstein, E. (1933). *Beitr. 2 Klin. D. Tuberk.*, **83**, 225.
Topley, W. W. C., and Wilson, G. S. (1936). *The Principles of Bacteriology and Immunity*, London.
Wilson, G. S. (1933). " Tuberculous Bacillaemia," M.R.C. Rep. No. 182.

BOOK REVIEW

The Arthropathies; a Handbook of Roentgen Diagnosis. By Alfred A. de Lorimier, A.B., M.A., M.D. Colonel Medical Corps, American Army; Commandant, the Army School of Roentgenology, Memphis, Tenn.; Formerly Director, Department of Roentgenology, Army Medical School, Washington, D.C. $5.50. The Year Book Publishers, Inc., Chicago.

This work is one of a series of six covering the whole field of diagnosis by the use of x-rays written by acknowledged experts in each branch. It is dedicated to all doctors possessed of scientific enthusiasm having primary interest in the patient; secondary, but closely equivalent, interest in the underlying pathology; and least interest in their personal gain in handling the case.

The foreword stresses the importance of the radiographer having the assistance of the clinical and laboratory data in attempting to arrive at a diagnosis, a matter of great importance too often thought to be quite unnecessary. Abnormalities as well as diseases are dealt with, and illustrations are on a lavish scale, for the most part reproductions of x-ray photographs but also some useful anatomical diagrams. In the study of the film the appearance of the soft tissues is first described, followed by those of the bones and joints; the appearances are differentiated into those that are likely to be seen and those that may possibly occur, and the difference between the early and late stages are clearly described. This is followed by the incidence, history, physical and laboratory findings, and clinical course, thus supplying a clear picture of the morbid condition under study, concise but astonishingly complete. The development of bones and joints in the embryo is described and the bearing this may have in pathological changes in later life. Every known disease or disorder of joint structures seems to have been included in the author's survey, with a bibliography to each, so that fuller descriptions may be consulted where necessary.

Many radiographers will appreciate the general discussion of technique, the effects of difference in kilovoltage and other factors; and the directions for routine analysis of the appearances in the film are a valuable guide to the practitioner in arriving at a diagnosis. The reproductions of typical radiographs are on the whole good and clear in detail. The system of placing the letter identifying the particular feature in the picture after the description of that feature is contrary to the usual practice and apt to be confusing at first, but this is a minor detail.

A book of this kind is essential to any doctor interested in the diseases of joints, and this volume can be cordially recommended for its compactness, clearness of description, wide scope, and its reasonable price.

C. W. B.