damping-down method for the acute exudative lesions, expecting the surgeon to remove the cavity-bearing area at a later date.

Tracheo-bronchial disease is still a problem. Many patients in whom tuberculous disease of the walls of the bronchi can be visualized through the bronchoscope are not permanently cured by drugs. If such tracheo-bronchial disease is removable, we feel that localized segmental resection is eventually indicated.

We have termed these methods a "dynamic approach" to the problem of tuberculosis. With prompt diagnosis and adequate treatment more patients with pulmonary tuberculosis are now getting well than ever could have done so previously.

Summary

The trend of treatment of pulmonary tuberculosis in the United States is discussed on the basis of a questionnaire sent in 1953 to one hundred physicians in that country. Recommendations were sought as to the treatment of ten selected cases of the disease. The replies are analysed and compared with those received in answer to a similar inquiry in 1948 concerning the same ten cases.

The investigation has shown that since 1948 the use of bed rest plus drugs and without collapse therapy has enormously increased. Collapse therapy is, however, still favoured in cases of more advanced disease, but pneumoperitoneum seems to have largely replaced pneumothorax as the procedure of choice.

A CASE OF GIANT-CELL POLYPHLEBITIS

BY

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[WITH SPECIAL PLATE]

Since 1941 attempts have been made to bring under a single heading such apparently diverse clinical conditions as polyarteritis nodosa, lupus erythematosus, scleroderma, and dermatomyositis. Banks (1941) has suggested that there is a common denominator in these diseases. Klemperer, Pollack, and Baehr (1942) proposed grouping them under the term "collagen diseases," fibrinoid degeneration of collagen having been found to form the basis of all of them. Claims have also been advanced for including in the group such diseases as rheumatic fever, Buerger's disease, and even serum sickness and the calcinosis. In spite of attempts at generalization, however, the "collagen group" of diseases remains an assortment of heterogeneous clinical varieties, and the specific conditions which should be included or excluded continue to be a matter for speculation.

Polyarteritis nodosa is predominantly a vascular collagen disease. Recognized as early as 1866 (Kussmaul and Maier), it has received widespread recognition only in recent years. Though an appreciable number of cases have been reported in the world literature, so far only three cases occurring in India are on record, the first of which was reported by one of us (Chakravarti, 1951). It must be appreciated, however, that polyarteritis nodosa has hitherto been recognized as an entity only

on the basis of the clinical findings; that it has any relationship to infection, hypersensitivity, or allergy is far from being established, and experimental attempts to arrive at its aetiology have been without success. But, unless the aetiology of such diseases as this is established beyond doubt, cases of arteritis presenting aberrant manifestations differing from the recognized clinical picture will raise problems of identification and lead to claims for the discovery of new entities. Thus a variety of arteritis with a different age incidence, vascular distribution, and prognosis was described in 1932 by Horton, Magath, and Brown. They gave it the name of "temporal arteritis." Giant cells formed the distinctive feature in its pathology, and, though it was claimed that giant-cell arteritis and polyarteritis are identical, this view was not supported by Harrison (1948).

In the light of the above considerations we describe below a case in which the clinical picture resembled in some respects that of polyarteritis nodosa, yet differed from it in a number of essential features. Clinically it was considered to be an example of polyphlebitis rather than of polyarteritis, but histological examination of the vascular lesions revealed the presence of giant cells, so long considered almost characteristic of temporal arteritis. The case would therefore appear to be of a hitherto unrecognized type of arteritis.

Case Report

A married woman aged 28 years; mother of four children, the last of which was born six years ago. No history of miscarriage. Past history of colitis, lasting three months, seven years ago. Streaks of blood in sputum during a febrile bout lasting one week three years ago. Husband's two brothers died of pulmonary tuberculosis, six and three years ago respectively.

Present Illness

The patient had suffered from a low-grade, irregular fever and malaise for the past year. One year previously slightly painful, tender spots had appeared on the medial aspects of the thighs, followed by a similar spot on the anterior aspect of the right forearm. For six months there had been persistent and gradually progressive anorexia and asthenia. A constant dull pain in the left hypochondrium, with exacerbations when the patient lay on her left side and on walking, had been noticed for the past three weeks. Since the last attack of colitis seven years ago there had been frequent bouts of diarrhoea. There were no cardiac complaints, no subjective urinary symptoms or respiratory trouble, and no signs of mental disturbance.

Clinical Examination

On examination the patient was found to be sickly, of asthenic build, pale, and anaemic. In the left eye there was phlyctenular conjunctivitis. Temperature 99° F. (37.2° C.); pulse rate 128 and respirations 32 per minute. The fingers were not clubbed. There was no enlargement of cervical or axillary lymph nodes, but the inguinal nodes showed discrete enlargement; these were neither painful nor tender. The spleen was palpable three fingerbreadths below the costal margin; it was soft and acutely tender, but there was no evidence of perisplenitis. The liver was palpable two fingerbreadths below the costal arch; it was rather soft, but peritoneal or paravertebral tenderness was absent. No abnormality was detectable in the colonic or renal area or in the lungs. There was no obvious dilatation of the heart; a harsh systolic murmur was audible all over the praecordium, with maximum intensity in the left third space, in the parasternal line, conducted well out to the posterior axillary line, but no thrill or associated diastolic murmur; the pulmonary second sound was accentuated. Blood pressure 108/64 mm. Hg.

On the medial side of the right arm, just above the elbow-joint, a thick subcutaneous tender lump containing a non-pulsatile cord-like structure could be felt. Similar non-pulsatile cord-like structures containing tender nodules were present on the front of the right forearm and the medial aspects of the thighs; these were the painful, tender spots complained of by the patient. A nodule was also present in the course of the cephalic vein on the right arm. The superficial arteries felt normal and had pulsations in them, except the left radial and brachial arteries, which could not be felt. This was probably a congenital abnormality with adequate compensation by way of collateral channels, since the nutrition of the limb was well maintained. There was no subjective or objective evidence of peripheral vascular disturbance.

Laboratory Investigations

Blood: R.B.C., 3,750,000 per c.mm.; Hb, 60%; W.B.C., 11,000 per c.mm. (polymorphs 75%, lymphocytes 18%, monocytes 2%, eosinophils 5%); E.S.R., 144 mm. in 1 hour (Westergren); coagulation time, 2 minutes; bleeding time, 3½ minutes; blood culture (repeated after two weeks), no growth; blood urea, 24 mg. per 100 ml.; Wassermann reaction negative. Urine: albumin, trace; pus cells, few; red blood cells, occasional; culture, *Alcaligenes faecalis*; no acid-fast bacilli seen by concentration method (examined thrice at intervals of two weeks). Stool: few degenerated pus cells; occasional red blood cells; no protozoa; no acid-fast bacilli seen by concentration method (examined twice at intervals of two weeks). Sputum: no acid-fast bacilli demonstrated by concentration method (examined twice at intervals of two weeks). Peripheral blood tested for "L.E. cells" found to be negative. Tuberculin test just positive with 1 in 1,000 dilution.

Special Investigations

Skiagrams of chest (taken at intervals of two months) showed no evidence of any parenchymatous lung infiltration. No evidence of calcified lymph nodes could be seen in a flat radiograph of the abdomen. Radiological examination of bones and joints distal to the affected vessel in the forearm showed no detectable abnormality. Ophthalmoscopic appearances were normal. An electrocardiogram gave evidence of sinus tachycardia with a normal sinus rhythm and normal auriculo-ventricular conduction; there was no evidence of coronary disease or of ventricular enlargement.

Histological Examination

Subcutaneous Forearm Vein.—The vein was thickened, cord-like, and thrombosed. On section the outer coat showed haemorrhage and many new, thin-walled blood vessels throughout its thickness. In some areas there was fibrinoid degeneration, patchy in distribution. Infiltration by round cells, of which the principal cells were lymphocytes together with a few plasma cells and monocytes, was of almost uniform distribution. At the junction of the outer and middle coats there were multinucleate giant cells surrounding the whole circumference like a ring. The middle coat showed interruption by granulation tissue. The muscle fibres were separated from one another by vascular connective tissue and lymphocytes. Minute patchy areas of degeneration were also seen in the middle coat. The intima was thickened by cellular connective tissue, in which a few capillaries were also seen. The lumen was greatly narrowed, but no thrombus was present, and the appearance was suggestive of organization and recanalization of a previous thrombus. (See Special Plate.)

Cephalic Vein.—A small area of thickening was felt, but the vein appeared otherwise normal. On section the outer coat showed a few areas of focal collections of lymphocytes in which could be seen a few multinucleate giant cells; there were also small areas of haemorrhage and degeneration. In the middle coat the findings were the same as in the specimen from the forearm. The inner coat was more or less normal.

Inguinal Lymph Node.—There were patchy areas of fibrinoid degeneration, but no giant cells were seen. The follicular structure was unaltered and the capsule intact.

A small arteriole from the right forearm adjacent to the affected vein also showed no abnormality.

Discussion

In arriving at a diagnosis in this case there would, in the first place, seem to be little difficulty in excluding other known forms of acute phlebitis. Thrombophlebitis migrans, for example, is of short duration and is characterized by a polymorphonuclear leucocytosis. "Tropical primary phlebitis," which was described by Fisher, Fisher, and Lendrum in 1947, runs a self-limiting course, and recurrence is uncommon. Histologically the granulation tissue interrupting the vessel wall is composed of new capillaries, polymorphs, and polyblasts, some of the last-named containing phloxinophil inclusion bodies. Again, in thromboangiitis obliterans there may be an associated phlebitis, but the disease is afebrile and characterized by arterial obstruction in the limbs; moreover, giant cells are found only in the region of organizing blood-clot. Secondly, although the family history, together with the clinical course, might suggest chronic miliary tuberculosis, absence of positive evidence of tuberculosis and the fact that the lesions were limited to the systemic veins would rule out such a possibility.

It seems certain that the case belongs to the group of vascular collagen diseases. Of these, the conditions that seem worth considering in the differential diagnosis are: (1) lupus erythematosus, (2) giant-cell arteritis, and (3) polyarteritis nodosa. (1) In acute disseminated lupus erythematosus skin lesions may be absent, but, in addition to endocarditis, involvement of serous membranes and joints is an important feature. Moreover, this is rather a visceral involvement than a surface vascular manifestation. Clinical diagnosis is dependent on the demonstration of L.E. cells. (2) Giant-cell arteritis usually affects the aged, the lesions being chiefly confined to the scalp and cranial arteries; the veins are never affected. (3) Polyarteritis nodosa is essentially a condition of primary arteritis usually affecting males. Hypertension and kidney involvement form an essential part of the clinical picture, and leucocytosis is common. Histologically, giant cells are seldom encountered.

Our case, therefore, though most closely resembling polyarteritis nodosa in the widespread distribution of the vascular lesions, has definite points of difference. Thus there was no hypertension or renal failure even twelve months after the onset of the illness; leucocytosis was absent; lesions were limited to widely scattered veins; and, finally, giant cells were present. These would appear to be grounds for regarding the case as at least a variant form of the collagen group of diseases which has not hitherto been described.

The histological slides were sent to Professor J. H. Dible, of the Department of Pathology, Postgraduate Medical School of London, for his opinion, and he makes the following comments: "I have examined the tissues and have also discussed them with Dr. Harrison. He points out that giant-cell arteritis, so far as is known, never affects veins, and, moreover, it is essentially a disease of advanced years. I don't think either the history or the histology of the lesion fits in with syphilis. . . . I have never seen tuberculosis attack veins in this way. . . . The disease which seems most worth considering is polyarteritis nodosa. The limiting of lesions to the veins and the peculiar picture of giant cells are certainly unusual, but, then, so is the whole case. I don't think this disease, either clinically or histologically, is the same as those reported by Fisher, Fisher, and Lendrum (1947)."

Summary

A case is reported of a protracted febrile illness in a young married woman in which polyphlebitis, inguinal lymphadenopathy, disproportionate tachycardia, and a high erythrocyte sedimentation rate were notable

features. The condition in many respects resembled polyarteritis nodosa, but differed from it in that the vascular lesions were limited to the veins and in the presence of giant cells.

A full account of the clinical and laboratory findings is given and the differential diagnosis discussed.

The patient is alive and is being followed up.

We are most grateful to Professor Dible for his valuable opinion on the tissue slides. Our thanks are also due to Dr. C. V. Harrison, Reader in Morbid Anatomy, University of London, for his views on giant-cell arteritis, and to Drs. S. N. De and A. Mukherjee, of the Departments of Pathology at the Sir Nilratan Sarkar Medical College and School of Tropical Medicine, Calcutta, respectively, for their help and co-operation.

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SPONTANEOUS MYXOEDEMA DEVELOPING AFTER THYROTOXICOSIS

BY

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Primary adult myxoedema most commonly occurs from idiopathic primary atrophy, but may also follow thyroidectomy, excessive radio-iodine dosage, or destructive thyroid disease such as lymphadenoid goitre (Hashimoto's thyroiditis). It is also frequently stated in textbooks that adult myxoedema may arise in patients who have previously had frank thyrotoxicosis ("burned out toxic goitre"), but it is exceedingly hard to discover records of such cases. Crotti (1938) describes one, but gives the impression that he has seen many more: he intimates that the progression from hyperthyroidism to hypothyroidism takes many years. McGavack (1951) describes the case of a patient who received only iodine treatment for his thyrotoxicosis but developed myxoedema some four years later. In view of the paucity of cases recorded, we are reporting one which was first treated for thyrotoxicosis with antithyroid drugs for 21 months; an active thyroid gland was still present at the end of this time. Twelve months later the patient was definitely myxoedematous.

Case Report

A married man aged 40 first presented in November, 1950, with an 18-months history of increasing fatigue, intolerance of hot weather, and 3 st. (19 kg.) loss of weight. He also complained of some dyspnoea on exertion and of some prominence of the eyes in the past two months. His appetite was very good. On examination at that time he was a thin, flushed, and anxious man (Fig. 1 A) with a fine tremor of the fingers, an increased peripheral circulation, and a slightly enlarged soft thyroid gland with a bruit. There was no true exophthalmos, lid retraction, or lid lag, but a definite "stare." There were no other physical signs. Investigation confirmed the diagnosis of hyperthyroidism (Fig. 2). The basal metabolic rate ranged from 140 to

160% of normal according to the Boothby standards (52 to 60 cal./square m./hr.). A radio-iodine test yielded only 10.7% excretion in 48 hours ("T" index of thyroid uptake = 105 compared with the normal of 3-13) (see Fraser *et al.*, 1953).

After a short course of an anti-thyroxine drug (butyl-4-hydroxy-3:5-diiodobenzoate) (Fraser and MacLagan, 1953), and after an episode of pyrexia which was found to be due to otitis media, the patient was discharged on methyl-thiouracil, 600 mg., and thyroid, 1 gr. (65 mg.), daily. He responded rapidly to treatment, gained weight, and remained well controlled on diminishing doses of thiouracil from January, 1951, to October, 1952. Antithyroid drug treatment was then stopped, and a radio-iodine test done a few days later showed that the gland was still active in that the 48-hour excretion was 16.7% (T=62). He was then given a short course of potassium iodide and re-tested in February, 1953, when the 48-hour radio-iodine excretion had risen to 77% (T=2.6). This was attributed to excessive ingestion of iodides as the patient appeared

Fig. 1.—Photographs taken in November, 1950 (A), and in January, 1954 (B).

	1950	1951	1952	1953	1954			
	JAN.	DEC.	JAN.	SEP.	OCT.	FEB.	MAY.	JAN.
B.M.R. %	+43 +45 +52							-27 -31 -33
Blood Cholesterol.	177mg.%							391mg.%
48hr. ¹³¹ I Excretion.	10.6 %				16.7%	77%	79%	83 %
"t"	105				61.7	2.6	1.4	2.0
E.C.G.								
Weight in stone.	9.9	10, 12	12, 3	12, 9		12, 11		13, 11
Anti-Thyroid Drug.								

	1950	1951	1952	1953	1954			
	JAN.	DEC.	JAN.	SEP.	OCT.	FEB.	MAY.	JAN.

Fig. 2.—Results of investigation.

euthyroid, but when the test was repeated three months later it yielded a similar result, and when he was seen again in October, 1953, he had all the features of myxoedema—full puffy facies, husky voice, and dry skin—and his weight had risen to 13 st. (82.5 kg.) (Fig. 1 B). On admission for reinvestigation it was found that his blood cholesterol had risen to 391 mg. per 100 ml., his basal metabolic rate had fallen to -28 to -33% (25-28 cal./square m./hr.), his protein-bound iodine was 2.8 µg. per 100 ml., and he had

H. G. TRIMBLE: CURRENT THERAPY IN TUBERCULOSIS IN THE U.S.A.

FIG. 1.—Case III: Minimal bilateral tuberculosis with right pleural effusion.

FIG. 2.—Case VIII: Moderately advanced tuberculosis with solitary 3-cm. cavity at right apex.

FIG. 3.—Case IX: Moderately advanced acute bilateral tuberculosis, more extensive on left.

FIG. 4.—Case II: Far advanced tuberculous pneumonia.

FIG. 5.—Case X: Far advanced bilateral fibrocaceous lesions with cavity on left.

A. CHAKRAVARTI and G. CHAKRAVARTI:
GIANT-CELL POLYPHLEBITIS

Section of vein showing thickened and infiltrated wall with collections of giant cells (arrowed). $\times 65$.