TEMPORAL ARTERITIS

BY

JAMES W. BROWN AND FRANK HAMPSON

From the Grimsby General Hospital

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Temporal arteritis is a rare condition. The earliest reported cases are those of Horton, Magath, and Brown (1932) who described identical histological changes in the temporal arteries of two cases. Five years later these same authors described five additional cases. The first English papers are those of Curtis Bain (1938) and Jennings (1938). More recently the number of reported cases has been increased to some twenty by those of Bowers (1940); Sprague and MacKenzie (1940); Dick and Freeman (1940); Gilmour (1941); Hoyt, Perera, and Kauvar (1941); and Sproul (1942).

Little or nothing is known about the ætiology of temporal arteritis, and perusal of the reported cases substantiates the view that the disease is of a chronic infectious nature and is not related to either tuberculosis or syphilis. The condition occurs most often in the fifth or sixth decade and is commonest in women. Its principal symptom is headache, chiefly referred to the site of the affected vessels, and the pain is not only intractable to the usual remedies, but is also aggravated by mastication or by other movements of the jaws and face. The involved vessels feel thickened and nodular and gradually become devoid of pulsation. The disease lasts over a period of several months and most patients recover. Diagnosis rests upon a biopsy of the affected vessels, and sometimes the mere resection of a portion of the affected vessels seems to have relieved symptoms.

Horton, Magath, and Brown cultivated a strain of actinomyces from a portion of resected vessel, but did not believe that this was the cause of the disease. Sproul and Hawthorne (1937) and Gilmour (1941) showed that identical lesions may be present in the aorta and its branches, and the latter named the condition giant-cell chronic arteritis. This at least dispels the original and usual view that temporal arteritis is a localized and non-fatal malady, and indicates that temporal arteritis is but a local manifestation of a diffuse arterial disease. Among other sites where similar lesions have been described are the occipital, retinal, and radial arteries. It seems probable that the intractable headache of some cases may be due to involvement of the cerebral vessels. There does not so far appear to be any explanation for the predilection for the temporal arteries in this disease.

The histological picture of the published cases is remarkably uniform, and is that of an arteritis. The intima is greatly thickened and there may be thrombosis in the markedly reduced lumen of the vessel. The media is largely replaced by granulation tissue with fragmentation of the elastic lamina. Characteristically present in the media are giant cells with many nuclei.

REPORT OF A CASE

A. M., a labourer, aged 61, complained of rheumatic pains in the knees and shoulders for six months, and of severe headaches in the temporal and occipital regions for one month. His past history did not include any severe or important illness.

His present illness was ushered in gradually with pain in the knee and shoulder joints, and a general feeling of weakness, so that he found it difficult to get about. After a few months there was

spontaneous improvement in his joint symptoms, but he began to have very severe headaches involving the sides and back of his head. The headache was aggravated by movements of the jaw, by coughing, and by mastication. Coincidentally he noticed small tender nodules on his temples and over the occiput. The headache persisted with unabated severity and remained uninfluenced by any usual therapy. There was no disturbance of vision.

FIG. 1.—Low power view of part of a transverse section of the artery. (Stained hæmatoxylin and eosin; magnification $\times 250$.)

FIG. 2.—High power view of part of the same section showing a typical giant cell. (Stained hæmatoxylin and eosin; magnification $\times 550$.)

On examination he appeared to be rather thin and pale. Moderate arteriosclerosis was present. The heart was normal and the blood pressure 150/80. The lungs were emphysematous. The pupils reacted to light and to accommodation and the fundi were normal. The reflexes were all present and equal. There was some muscular wasting of the arms and legs. The joints were not swollen but coarse crepitus was present in knees and shoulders.

There was marked tenderness over the occipital and temporal areas of the scalp, particularly the latter. A number of small nodules were clearly visible and palpable over the temporal artery and similar nodules were palpable over the occiput. The temporal artery was thickened and pulsation was diminished. The nodules appeared to be in the walls of the vessel, and they were exquisitely painful on palpation; the skin overlying them was reddened. In the course of three weeks the pain subsided and the nodules disappeared. In the final stages pulsation in the affected vessels was barely apparent.

Laboratory Examinations. Blood sedimentation rate was 13 mm. in 1 hour (Westergren). Blood urea 60 mg. per 100 c.c. The red cell count was 3,670,000 per c.mm.; the hæmoglobin was 62 per cent (Haldane); the colour index 0.8, and the white cell count 7200 per c.mm. The Wassermann and Kahn reactions were negative. An electro cardiogram showed normal axis and no abnormality. An X-ray of the chest was also normal.

An initial diagnosis of periarteritis nodosa was made, but in the absence of any evidence of visceral involvement a biopsy of the temporal artery was performed, and a segment of the vessel removed. The course after operation was uneventful.

Pathological Report. The material submitted consisted of a small piece of an artery with grossly thickened wall, and a lumen that was practically occluded. The tissue was fixed in formalin, and transverse sections were prepared and stained by hæmatoxylin and eosin, Van Gieson's stain, and Verhoeff's elastic tissue stain.

Histologically, well-marked pathological changes were present, and these were found to conform very closely with the histological picture of temporal arteritis as described by Horton and Magath, and with the histological picture described in other arteries by Gilmour, and named by him giant-cell chronic arteritis.

The intima showed great hypertrophy, and the hypertrophied tissue was composed of proliferating muscle fibres supported in a mucoid ground substance. The cells in this tissue were arranged quite irregularly and the more usual arrangement into circular and longitudinal bundles was entirely absent. The elastic lamella was fragmented and in certain areas it could not be demonstrated. In a few areas in the intima giant cells could be seen, but they were very few as compared with those in the media, and much smaller in size.

The media showed chronic inflammatory changes with lymphocytic infiltration and giant-cell formation. These giant cells showed very large numbers of nuclei and appeared to be of foreign body type for the most part. In appropriately stained sections it could be seen that in some instances these giant cells were in intimate relationship to small fragments of elastic tissue.

The changes in the adventitia were relatively slight as compared with those in the other two coats, but signs of an inflammatory process were present here as well. There was some infiltration with lymphocytes and a few plasma cells could be seen, along with small numbers of epithelioid cells. The small branches of the artery outside the adventitia did not show any pathological changes. In both intima and media wide capillaries were present.

The picture is thus one of chronic granulomatous, giant-celled arteritis, affecting primarily the media of the artery, but showing changes in all three coats.

SUMMARY

A case has been described in which the clinical and pathological findings are those of an arteritis of the temporal arteries. Perusal of the reports of similar cases suggests that temporal arteritis is but a local manifestation of a general disease of the arterial tree.

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