

TEMPORAL ARTERITIS MASKED BY CONTRALATERAL PANSINUSITIS

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Temporal arteritis is a rare disease. It was first mentioned by Hutchinson (1891), but was first clinically described in 1932 by Hurton, Maggat and Brown. They believed it to be a localized inflammation of the temporal artery of one or both sides.

Kilbourne and Wolff (1946), who knew already about the clinical involvement of the cerebral and retinal vessels, beside the temporal arteries, suggested the name "cranial arteritis".

Gilmour (Crompton, 1959) relying upon histological findings introduced the name "giant cell arteritis".

Temporal arteritis is a general disease which involves not only the temporal arteries, but also the vessels of the retina, the ophthalmic artery, the branches of the aortic arch, the iliac trunk and branches, the renal, subclavian, mesenteric, coronary, pulmonary and others.

It is a panarteritis which involves all the 3 layers of the artery, beginning with the media and spreading to the intima and adventitia.

It is a disease of late middle age (beginning from 50-55 years) without difference of sex (Horton, Magath and Brown, 1934). The course of the disease appears usually in 4 stages:

The first stage is characterised by general weakness with elevated temperature, anorexia, pains in the arms and legs, muscles and joints, dizziness and nausea. Suddenly, within a few hours, strong headache appears. This severe headache is unilateral and only very rarely bilateral and is felt over the temples along the temporal artery. The scalp of the involved side is so tender that the patient cannot put his head on the pillow, or brush his hair and has difficulties in opening the mouth, as well as in mastication because of pains of the jaw. The temporal artery is thick, hard, curled and less or non-pulsatile. Palpation of the artery causes strong pains. After 4-6 weeks of headache, if the disease remains undiagnosed and untreated, the patient enters the 3rd stage — the stage of ocular complications, resulting from vascular damage of the retina and the optic nerve (Kilbourne and Wolff, 1946). The onset is usually abrupt and appears most frequently on awakening in the morning. The patient feels a shade over a part or over the whole eye. This shade becomes denser and within a few hours, sometimes minutes, he becomes partly or completely blind. Recovery may occur in a few hours or minutes, but in 25-30% the loss of vision remains permanent, due to occlusion of the central retinal artery with its branches, or the arteria ophthalmica or both. In the 4th stage of the disease the inflammation involves a large number of



Figure 1. The patient at admittance - The prominent and tortuous right temporal artery.

other vessels like the coronaries, the pulmonaries, the aortic arch, the iliac, renal, etc.

The prognosis of temporal arteritis is benign. After months of suffering with remission of syndromes, the patient recovers.

Etiology of temporal arteritis is unknown, but an infectious cause is suggested.

Laboratory findings show leucocytosis, normocytic anemia and a high sedimentation rate.

The histological picture is that of a collagenous disease with giant cells as the dominant characteristic (Poulley and Hughes, 1960).

Therapy - until the discovery of corticosteroids, local treatment was tried.

Novocaine infiltration around the inflamed vessels was given, excision of the temporal artery was performed (Ross, 1959). Since the use of steroids a potent, helpful remedy against temporal arteritis was put in our hands, and if used in time, ocular complications may be avoided (Turner and Van Horn, 1952).

Report of a case

A man, 53 years old, was admitted in June 1969 to the E.N.T. department of the Rothschild Hospital, Haifa, because of severe headache of almost two months duration. He was treated elsewhere for left pansinusitis, the antrum

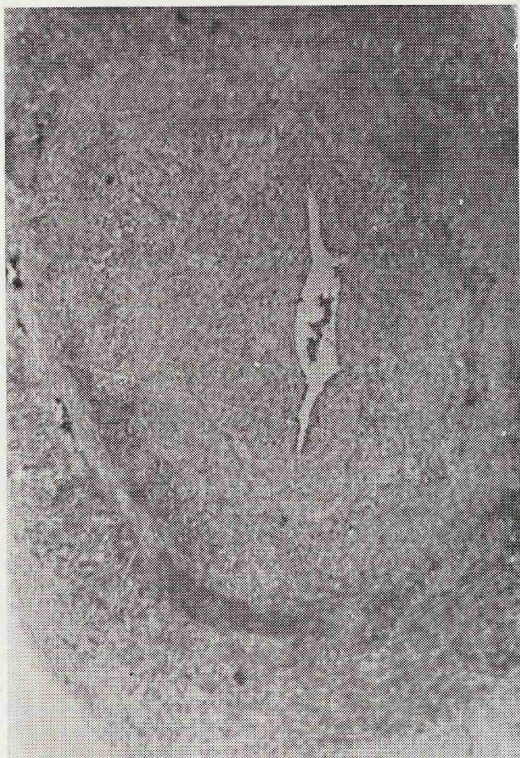


Figure 2. Section shows full thickness of the artery; inflammation in all the layers; marked thickening of the intima and area of fibrinoid necrosis in the media.

was washed out several times and he received antibiotics, but without effect. On admittance the patient looked very ill, weak and pale. He held his hand constantly over his brow and complained of severe headache. An X-ray of the sinuses, which the patient brought with him, revealed a homogenous clouded maxillary sinus on the left side, and a fluid level of the ipsilateral frontal sinus.

On examination, we could only confirm clinically and radiologically the diagnosis of sinusitis on the left side.

Laboratory findings showed: Hb - 9 gr%; leucocytes 14.200 and a sedimentation rate of 90/125 (Westergreen).

We began the treatment with washouts and high doses of antibiotics (penbritin and orbenin). After 5 days of this treatment, seeing no improvement and because of the headache which became more severe, we decided to perform a drainage of the frontal sinus.

The operation was performed under general anaesthesia, leading the incision in the midst of the superior palpebra. On opening the frontal sinus, pus and a swollen mucous membrane was found.

At the end of the operation a rubber drain was introduced through the nose and antibiotic therapy was continued. Two to three days after the operation we had the impression that the patient was improving, but this was most likely due to the heavy sedation he received after the operation. The patient began



Figure 3. Foreign body giant cell in the boundary between the media and the intima.

again to complain, but this time the pain concentrated only on the right frontal and temporal areas.

On closer examination our attention was drawn to the right temporal artery which was thick like a cord, curved, with nodules and almost pulseless. Every touch, even the slightest, of the artery caused the patient severe pain.

We suspected temporal arteritis. The patient was examined by the ophthalmological and neurological departments, but no other pathology was found. In order to confirm the diagnosis of temporal arteritis, an excision of about 7 cm of the artery was done. The histological examination revealed inflammatory changes in all the layers of the arterial wall, with foci of fibrinoid necrosis of the media. There was a marked thickening of the intima with narrowing of the arterial lumen. Several foreign body giant cells were seen in the partially destroyed internal elastic lamina. The inflammation tended also to the connective tissue around the artery. (Figures 2 and 3).

The diagnosis of temporal arteritis was made and treatment with corticosteroids (prednisone) was begun. After 3-4 days of this treatment, the patient felt better and the doses of prednisone were lowered. Three weeks later the patient left the hospital free of headaches. He was instructed to continue with small doses (10 mg daily) of prednisone to avoid a remission. Since then the patient was seen a few times and has no more complaints of headache.

SUMMARY

A case of temporal arteritis complicated by contralateral pansinusitis is presented. The effectiveness of corticosteroids as treatment of temporal arteritis is emphasized.

RÉSUMÉ

Un cas d'artérite temporale compliquée par une pansinusite controlatérale où l'efficacité des corticostéroïdes est soulignée.

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