OPHTHALMIC MANIFESTATIONS OF TEMPORAL ARTERITIS*

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TEMPORAL arteritis is the name given to a local and systemic symptom complex that attacks patients between the ages of 55 and 85 years. It is a benign self-limiting disease, insidious in development, with a course lasting over several months. As the name implies, a very regular feature is an inflammation of the temporal arteries of the scalp, but the disease is not confined to these vessels and any of the cranial arteries may be involved. Many arteries elsewhere may also become inflamed, particularly the aorta and its main branches. The clinical description given by Hutchinson (1890) is similar to the acute form of the disease as it is encountered to-day; he related that he had been called to see a man, upwards of 80 years old, because he had red streaks on his head which were painful and prevented him from wearing his hat.

The red streaks proved on examination to be his temporal arteries which on both sides were found to be inflamed and swollen. The streaks extended from the temporal region almost to the middle of the scalp and several branches of each artery could be distinctly traced. The condition was nearly symmetrical. During the first week when he was under my observation, pulsation could be detected in the affected vessels but it finally ceased. The tenderness then subsided and the vessels were left impervious cords.

No further references to the disease appeared until Schmidt (1930) described a patient who developed a complete right-sided paracentral scotoma 8 months after his temporal arteries had been inflamed. This patient also had an aneurysm of the internal carotid artery. Horton, Magath, and Brown (1932) described two further cases of temporal arteritis, and many other papers have since been published. A general survey of the literature, together with a description of seven cases was made by Cooke, Cloake, Govan, and Colbeck (1946). They considered temporal arteritis to be a widespread arterial disease, not uncommon but rarely recognized, in which characteristic arterial and striking local signs occur, and in which inflammatory and degenerative changes in the walls of the affected arteries produce a characteristic histopathological picture. The most important clinical features are anorexia, loss of weight, joint and muscle pain, pyrexia, painful arterial thrombosis, and very severe headaches occurring in elderly patients. The pathological features are those of a subacute inflammatory process commencing in the adventitia. The vasa vasorum then become involved and

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many vessels are occluded, causing areas of necrosis to appear in the media and internal elasticia. The inflammation spreads longitudinally and the internal elastic lamina becomes destroyed, though during healing new reduplicated layers are formed. The media becomes chronically inflamed, being infiltrated with lymphocytes, plasma cells, and large mononuclears. Occasionally a giant-cell reaction may be excited in the media. The intima is much thickened, and, in the smaller vessels, thrombosis is a common sequel. Prognosis is relatively good as regards life, though, with the generalized nature of the disease process, it may be fatal in some cases. Cardell and Hanley (1951) have found that twelve of the 27 fatal cases on record have died from cerebral vascular lesions. In their patient, who died from a brain-stem thrombosis, necropsy revealed changes in the ophthalmic, coronary, and other arteries as well as the temporal.

Various ophthalmic complications can be expected to occur during the course of the illness in about half of the cases. Vision was affected in six of the seven cases described by Cooke and others (1946): three became quite blind, one had permanent loss of vision in one eye, three cases had diplopia, and one a transient ptosis. Ophthalmic complications described by other authors include retinal phlebitis (Horton and Magath, 1937), obstruction of the central retinal artery (Jennings, 1938; Robertson, 1947; Anderson and others, 1948), occlusion of the superior temporal artery (Johnson, Harley, and Horton, 1943), periarterial retinal exudate (Kilbourne and Wolff, 1946), blurring of the optic disks (Le Beau and others, 1948), oedema of the disks with haemorrhages and exudates (Shannon and Solomon, 1945), blurring of the disks with narrow arteries (Peltola, 1947), papilloedema (Dick and Freeman, 1940; Bessière and Julien, 1950), peripapillary atrophy (Bain, 1938), blindness followed 3 weeks later by occlusion of the central retinal artery in the right eye and 3 days later by partial changes in the left eye (Curits, 1946), and macular degeneration with optic atrophy (Kaye, 1949).

Bruce (1950) reviewed the literature of temporal arteritis and collected from it 84 cases: of these, 34 had complained of disturbance of vision during the course of the illness. He analysed the causes of these visual disturbances and divided them into the three groups described by Wagener (1946):

(i) occlusion of the retinal artery within the optic nerve (6 cases, 18 per cent.);
(ii) ischaemic retrobulbar neuritis due to involvement of the retinal artery before it enters the optic nerve (thirteen cases, 38 per cent.);
(iii) an indeterminate group of fifteen cases (44 per cent.) including unexplained loss of vision, retinal phlebitis, photophobia, and subretinal haemorrhage.

Bruce found that as a result of the disease, blindness had ensued in both eyes in thirteen cases (38 per cent.) and in one eye in nine cases (27 per cent.). Various degrees of impairment of vision followed in seven cases (20 per cent.). Reasonable recovery of vision occurred in only two cases after a period of amaurosis. He comments that more than half of the patients whose eyes are involved during temporal arteritis may be expected to lose the sight permanently in one or both eyes and that few can expect a return of normal function.
In many cases it is possible to influence the course of the illness, though the cure has yet to be discovered. Pain in the scalp arteries can certainly be relieved, whilst administration of anticoagulants may prevent the formation of intravascular thrombosis in the affected arteries during the acute stages.

The purpose of recording a further series of cases of the disease is to emphasize how frequently temporal arteritis affects vision; indeed, it may present with ocular symptoms, as in each of the fifteen cases described below. It is important to recognize the condition early, since it may be possible by emergency medical measures to preserve sight in the otherwise fulminant group, details of which are given.

**Case Reports**

**Case 1, male, aged 65.**—*Progressive visual loss with bilateral papilloedema. Consecutive optic atrophy.*

A gradual onset of blurring of vision in the left eye was first noted towards the end of April, 1950. On May 3, 1950, the patient had left-sided papilloedema with visual acuity of 6/60 in the left eye. The right disk was normal and the visual acuity 6/18. There were no other abnormal physical signs except that the temporal arteries were swollen and tortuous, and slightly tender on the left side. Three weeks later he complained that the vision in the right eye had gradually deteriorated over the previous few days; right-sided papilloedema had developed with a visual acuity of hand movements only. The temporal vessels had become very tender to palpation and he complained of pain in his left ear and in the gums of the upper jaw. Because of the story of progressive papilloedema he was admitted for full investigation on July 13, 1950.

Blood pressure 145/60. Visual acuity in the right eye 6/60, in the left eye hand movements only. Right disk swollen, left pale and showing early atrophy. Pupils irregular, left larger than right. Left pupil did not react to light. No other abnormal physical signs in the nervous system.

A segment of the left temporal artery removed for biopsy showed that the vessel wall had been infiltrated by inflammatory cells. The only other abnormality found by pathological investigation was an increase to 50 mg. per cent. in the protein content of the cerebrospinal fluid. Air encephalography and left internal carotid arteriogram normal.

By August 17, 1950, vision in the right eye had failed and the patient was quite blind. During the subsequent 6 months he suffered from constant pain in the scalp involving particularly the right temporal and left occipital arteries. He also complained of severe dyspepsia and malaise. The acute condition then subsided, and in March, 1952, he was slowly recovering his general health, but was frequently troubled at night by a severe stabbing pain in the right side of the neck. As it was possible to reproduce the pain by pressure on the right common carotid artery it is probable that this vessel was now involved. The left temporal artery showed some pulsation, probably collateral, but there was no pulsation on the right side.

Although he has well-marked optic atrophy, his visual acuity has improved slightly. He can now count fingers with the right eye but sees hand movements only with the left.

**Case 2, married female, aged 63.**—*Thrombosis of left inferior nasal artery.*

On June 8, 1950, this patient complained of a sudden onset of blurring of vision in the left eye. There was mild conjunctival injection on the left side. She had hypertensive changes in both retinae, the left disk was blurred, and the left inferior nasal artery thrombosed. Visual acuity in the right eye was 6/12, and in the left counting fingers. She had mild hypertensive heart disease with a blood pressure of 180/95. There was a previous history of an embolic cerebral catastrophe when she was aged 57 but the only residual sign was a slight left-sided facial weakness. In September, 1950, she began to
suffer from severe pains in the head, and when examined on October 5, 1950, the left occipital and temporal arteries were inflamed and tender. There was no pulsation in the left temporal artery, though the right temporal artery was pulsating well. For the next 6 months she complained of many vague symptoms, particularly depression, lassitude, and dyspepsia, but by June, 1951, she had regained good health. In March, 1952, visual acuity was 6/24 in the right eye and 6/60 in the left.

Case 3, male, aged 65.—Papilloedema and thrombosis of right superior temporal artery.

On October 18, 1950, this patient had right-sided papilloedema with thrombosis of the right superior temporal artery. Visual acuity was 6/36 in both eyes.

By November 8 the central retinal artery had become completely thrombosed and the disk was swollen. Visual acuity in the right eye had deteriorated to counting fingers. There was also evidence of generalized vascular disease, and gross peripheral arteriosclerosis with a blood pressure of 210/120. There was some visible inflammation of the right temporal artery which was very tender when palpated. On January 11, 1951, he complained of feeling very unwell, had a severe headache, and could not bear the slightest pressure on the right temporal or left occipital arteries. Routine pathological investigations were all normal. A month later, when the generalized symptoms of malaise, dyspepsia, and insomnia were worse, a biopsy of the right temporal artery showed several arteriosclerotic lesions. The lumen was much reduced in calibre. Sections stained for elastic tissue showed that the internal elastic lamina had undergone degenerative changes. The membrane was fragmented, the remainder being thickened and hyaline. By March, 1951, his health had started to improve, he was eating and sleeping well, and there was only slight tenderness on the right temporal artery. The right disk had become pale and atrophic and the arteries were threadlike. Visual acuity in the right eye less than 6/60. There were signs that the cerebral arteries had become involved; he found it difficult to concentrate and became "very melancholy". The slight pain in the right temporal artery persisted until June, when he "turned the corner" and his melancholy became "a thing of the past". The right occipital artery then became very tender, particularly when he laid his head on the pillow and this persisted for 2 months, but by September, 1951, all the active arteries had ceased and he was in good health. The vision in the right eye, however, has not improved.

Case 4, male, aged 84.—Bilateral papilloedema. Progress of arteritis influenced by anticoagulants.

Progressive headache of 10 days' duration and bilateral papilloedema led to a suspicion of cerebral tumour. On December 6, 1950, the vision had started to deteriorate in the left eye and on December 15 the vision in the right eye had become affected for the first time. On December 16 visual acuity was 6/60 in the right eye and the left eye was blind. Both temporal arteries were tortuous, swollen, barely pulsating, and exquisitely tender; the skin lying over the arteries was reddened. There were no abnormal physical signs in the nervous system. The patient was a gross arteriopath but his blood pressure was 150/80. As the second eye was becoming involved, he was treated with heparin 10,000 units intravenously every 8 hours, together with nicotinic acid, 50 mg. three times daily for 7 days. Some vision in the right eye was preserved, the visual field on the right showing an irregular nasal hemianopia, and the visual acuity being 6/60. The left eye was blind. A left temporal artery biopsy showed typical changes of giant-cell arteritis with intravascular thrombosis. The arteritis persisted for 6 weeks and then gradually subsided. Unfortunately this patient was not examined again ophthalmoscopically as he fractured the neck of the femur on March 3, 1951, and died in another hospital a month later. It was reported from that hospital, however, that the vision was "apparently normal in the right eye, although he was blind in the left". Though the visual acuity was not recorded and there must be some doubt about the apparent recovery, some sight must have been preserved.
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Case 5, unmarried female, aged 77.—Macular haemorrhage.

On January 8, 1951, this patient had hypertensive changes in her retinae with a large haemorrhage below the left macula. There was a diffuse grey elevated area surrounding the macula. Visual acuity on the right side was 6/6, whilst on the left there was perception of light. Her left temporal artery was exquisitely tender on palpation but the overlying skin was not reddened. She gave a history of recent loss of appetite and pain in the head, this particularly affecting the left temporal and occipital areas of the scalp. The blood pressure was 210/130. A biopsy of the left temporal artery showed thickening of the intima and the internal elastic lamina had undergone considerable degenerative changes. The membrane was fragmented and the surviving pieces were thickened and hyaline. No active cellular area was seen. In April, 1951 both her vision and general condition had improved. The haemorrhage was absorbing, and the macula was pigmented. The left-sided arteritis had subsided but the right temporal artery was still slightly tender to pressure. By October the vision had cleared and except for some indigestion she was "marvellously better and a new woman". The left disk was pale, there was pigmentation of the macula and the haemorrhage had been absorbed. On April 3, 1952, the visual acuity on the right side was 6/12 and with the left eye she could count fingers easily. She was feeling very well and was free from headache. She occasionally complained of pain in the left side of her neck which suggested involvement of her left carotid artery.

Case 6, married female, aged 80.—Thrombosis of central retinal vein.

On May 3, 1951, this patient reported that she had suddenly lost the vision of her right eye a week previously as the result of a thrombosis of the right central retinal vein. She could count fingers with the right eye, and the corrected visual acuity in the left eye was 6/18. She had arteriosclerosis, hypertensive heart disease, and auricular fibrillation. The blood pressure was approximately 200/110. There were prominent pulsating arteries in both temporal fossae which, on the right side particularly, were very tender to pressure. She had previously complained of a great deal of pain in the left temporal artery and related that she had had pain in the back of the head, 2 or 3 weeks before. She pointed to the course of both occipital arteries as the site of this pain. On May 31, 1951, there was still tenderness over both temporal arteries, but the pain was not so severe and the patient's sleep was less disturbed. By June she was quite well and there was no tenderness in the arteries, but the visual acuity in the right eye had not improved.

Case 7, unmarried female, aged 77.—Bilateral cataract. Transient worsening of vision due to development of cranial arteritis.

On June 4, 1951, this patient complained of a sudden worsening in her vision, which had been bad for years, together with giddiness and malaise. Her corrected visual acuity in the right eye was counting fingers, and in the left eye 6/36. There were bilateral cataracts and the fundi could not be seen. She had severe hypertensive heart disease with signs of failure, and generalized arteriosclerosis. Her blood pressure was 220/100. The right occipital artery was very tender on palpation, and this severe local pain persisted for 4 weeks. On September 10, 1951, the right temporal artery was palpable but not pulsating, whilst the left showed sluggish pulsation and was very thickened and tortuous. Two months later the arteritis had completely subsided.

In March, 1952, her right cataract was extracted, and 6 weeks later examination of the fundus showed that the retinal vessels were quite normal. There was some fine pigmentation at the macula with a few bright areas of exudate. In spite of this, with correction, she could see 6/12 and read J 4. Some pain in the scalp recurred at this time being particularly severe in the region of the right occipital artery. The area was injected with 2 per cent. procaine with immediate local relief.
Case 8, male, aged 69.—Paresis of left inferior rectus muscle.

On June 13, 1951, this patient gave a 4-week history of diplopia which was found to be due to weakness of the left inferior rectus muscle. Visual acuity in the right eye was 6/9 and in the left eye 6/18. Physical examination showed generalized arteriosclerosis with a blood pressure of 180/100. The patient had had some left-sided headache during the previous 2 to 3 months but it had not been severe enough for him to seek medical attention. The diplopia recovered within 4 weeks but at about that time the pain in the head became much more severe, and both temporal arteries had become prominent but not yet tender. In August, 1951, however, both occipital arteries became inflamed and he could not bear the slightest pressure on the left temporal artery. A month later his general condition had deteriorated. He was suffering from malaise, depression, anorexia, and severe pain in the head. The tenderness was especially marked in the left occipital artery, and the left temporal artery was thrombosed. Because of the severity of the pain in the region of the left occipital artery, he was admitted to hospital on November 2 for section of this vessel with the idea of alleviating the pain in his head. This operation was successful, and the biopsy showed a non-specific inflammatory change in the arterial wall. By January, 1952, his general condition had improved but he still had some pain in the left temporal area. There was no recurrence of the ocular symptoms.

Case 9, married female, aged 73.—Ocular pain.

This patient gave a long history of visual disorder. She had had corneal ulcers as a child, an iridectomy in 1931, and glaucoma in 1934. In August, 1951, she complained of severe pain in both eyes. Except for scarring, both eyes were quiet. She was a high myope. Visual acuity in the right eye was 6/24, but the left showed no perception of light. On August 20, 1951, she had severe hypertensive heart disease and arteriosclerosis, with a blood pressure of 255/130. On September 3, 1951, she had developed severe headache and examination showed tenderness localized to both occipital and temporal arteries. Her appetite was very poor and she felt ill. The condition of cranial arteritis persisted for 6 weeks but on October 22 the headache and pain in the eyes had completely subsided. No abnormal physical signs developed in the eyes.

Case 10, unmarried female, aged 78.—Diplopia.

On November 15, 1951, this patient complained of headache and of diplopia due to paresis of the left sixth nerve. She had had a right cataract extracted in 1947 and a left cataract the following year. Her corrected vision was 6/18 in the right, and 6/60 in the left. She had mild hypertensive heart disease with generalized arteriosclerosis and a blood pressure of 190/110. The pain in the head was localized to both occipital and temporal arteries. Recently she had lost weight and had had no appetite. She had not been able to sleep in bed as she found it impossible to rest her head on the pillow because of the pain in her scalp. For the same reason her hair could not be combed. The acute pain persisted for a further 4 weeks, but 2 months later the diplopia had improved, and the arteritis in the scalp had subsided. In March, 1952, an acute attack of pain in the right side of the neck developed and persisted for a week. This pain, which almost certainly originated in the right carotid artery, extended from behind the clavicle to the angle of the jaw. There were no further complications, and her corrected visual acuity improved to 6/12 in the right eye, and 6/9 in the left.

Case 11, married female, aged 59.—Unilateral papilloedema.

On November 6, 1951, this patient complained of blurred vision in the right eye. The right pupil was moderately dilated and reacted sluggishly to light and accommodation. The right optic disk was swollen but no other abnormality was seen. The visual acuity was 6/24 in the right eye and 6/6 in the left. The left disk was normal. Physical examination showed a condition of severe hypertensive heart disease with arteriosclerosis, and a blood pressure of 210/120. The temporal and occipital arteries on the right side
were very tender to palpation. On December 10, 1951, the vision had improved slightly but the patient still complained of severe headache; she admitted to having felt off-colour for several weeks, and to being tired and having no appetite. She had found it extremely difficult to lay her head on the pillow because of the pain, particularly on the right side. On February 7, 1952, the right occipital artery was still painful, but elsewhere the arteritis had subsided. The visual acuity had improved to 6/18 in the right eye and 6/5 in the left, but the right disk showed a severe consecutive optic atrophy.

Case 12, male, aged 64.—Alexia.

On November 22, 1951, this patient complained of difficulty in reading. There were hypertensive changes in both retinas but no other ophthalmic abnormality was discovered. The visual acuity was 6/6 in both eyes. The skin over the left temporal artery was exquisitely tender and reddened. The patient had hypertensive heart disease with auricular fibrillation. Routine pathological investigations showed no abnormality. A week later the condition had improved and he was able to read. This alexia was certainly the result of a vascular insult in the left parietal area, due either to an embolism from the heart or to a small thrombotic lesion in association with his cranial arteritis. In view of the active inflammation of the left temporal artery, the latter explanation is considered the more likely.

Case 13, married female, aged 75.—Bilateral papilloedema and consecutive optic atrophy.

On November 12, 1951, this patient gave a history of having become blind in both eyes a week previously, following a 17-day illness. She had awakened on the first day of the illness with misty vision in both eyes, had stayed in bed for 2 days when the condition improved slightly. Between the 3rd and 6th day she had been able to be up and about in her home but the vision had gradually deteriorated again in both eyes. On the 7th day she awakened to find herself unable to see and the vision has not improved since then. The patient had 1.5 dioptres of papilloedema on the right, 2 dioptres on the left and haemorrhages at both disk margins. There was advanced arteriosclerosis and a blood pressure of 190/100. It had been noted by her daughter during the onset of her visual symptoms, that when she had combed her mother’s hair there was extreme tenderness over the occipital areas on both sides. The daughter also reported that 6 days previously she had noted the right temporal artery to be red and very tender. The two occipital arteries were tender and not pulsating, whilst palpation of the right temporal artery caused great pain. The headaches persisted for a fortnight and then subsided, though the tenderness over the right temporal artery was still present a month later. It seems probable that had the underlying condition been recognized in time, the progressive course of the illness might have been favourably affected by treatment with anticoagulants.

Case 14, married female, aged 62.—Ocular pain, ptosis, and ophthalmoplegia.

In October, 1951, this patient developed a severe stabbing pain in her left eye. The pain spread from the eye to the face and a tooth was extracted from her upper jaw on the left side, as it was thought to be the cause of her pain. She had mild diabetes mellitus but otherwise her previous history was negative. At the beginning of November, 1951, she experienced considerable pain in the left side of the neck and this spread to the back of the head. The tenderness became very severe, so that she found it difficult to lay her head on her pillow. A week later she developed diplopia, with left-sided ptosis and complete paralysis of the left 3rd nerve. The visual acuity was 6/6 in the right eye, and 6/9 in the left. The blood Wassermann reaction and Kahn test were negative. On December 13, 1951, the left temporal and occipital arteries were acutely tender but the patient felt that her condition was improving rapidly. Except for absent ankle jerks there were no abnormal physical signs in her nervous system. The condition of diabetes was well-controlled, and the blood pressure was 130/88. There was no evidence of
generalized arteriopathy. By January 10, 1952, the arteritis had subsided and the visual symptoms had cleared up.

Case 15, male, aged 64.—Thrombosis of left superior temporal artery.

On January 16, 1952, this patient gave a 24-hr history of blurring of vision in the left eye after stooping. On examination no abnormality was seen in his fundi. The corrected visual acuity was 6/5 in the right eye and 6/9 in the left. While the fields were being recorded the patient frequently complained of blurring in the lower temporal field but there was no definite contraction to be charted. He was thought to be suffering from transient narrowing of the left superior temporal artery of the retina. On February 7, 1952, the visual acuity in the left eye had improved to 6/6. During the previous few days he had developed a pain in the scalp, due to active arteritis of the left temporal artery, which was distended and pulsating, and also of the right occipital artery in which the pulsation could not be felt. The blood pressure was 130/70 and there were no abnormal physical signs elsewhere. The patient felt well and none of the usual accompanying symptoms of cranial arteritis had yet occurred. On March 3, 1952, the visual condition had not deteriorated but the headache persisted, the patient did not feel well and was suffering from dyspepsia.

Discussion

The aetiology of temporal arteritis is as yet not known, though it is generally considered that the disease is infective, the nature of the inflammation, the general systemic disturbance, and the self-limiting course of the illness all being in favour of an acute or sub-acute infective process. As the condition does not affect patients under 55 years of age, the blood vessels of the older age groups would appear to be more susceptible to the infection than those of younger subjects.

Pathologists are undecided whether to classify temporal arteritis as a separate disease, or to group it with polyarteritis nodosa, eosinophilic arteritis, thrombo-angiitis obliterans, and the collagen diseases. Since the cause of these various diseases is still not understood it does not seem possible to clarify the relationship between them, although there is some overlap in the histopathological appearances. It certainly appears, on clinical grounds, that temporal arteritis is a disease sui generis, and that Cooke and others (1946), Crosby and Wadsworth (1948), Harrison (1948), Cardell and Hanley (1951), and Cloake (1951) are correct in differentiating it from other diseases of the blood vessels. From the clinical viewpoint, particularly as it affects the ophthalmologist, the differential pathological diagnosis is not, at present, of great importance, since no specific treatment exists for any one of these various illnesses.

The various eye symptoms and signs in temporal arteritis are brought about by the disease process affecting the arterial supply of the optic and oculomotor nerves, and the retina, and sometimes causing complete thrombosis within the affected vessel. In the less severe cases the blood supply may be cut down by temporary swelling of the vessel wall and, as the inflammation subsides, function is restored provided that the degree of ischaemia has not been too great. This variation has been noted in the
present series of cases, the worst results occurring when the optic nerves or retina were involved. In nine of such cases, five did not improve at all, three showed some minimal improvement, and only one made a moderate recovery. All the three cases in which the oculomotor nerves were involved recovered within a period of 8 weeks. Attention is drawn to a catastrophic group in which both retinal arteries are affected and total blindness invariably ensues. Although visual symptoms may be the first warning that the patient has temporal arteritis, the course in this fulminant group is generally spread over several days, during which time anticoagulant treatment may influence the intravascular thrombosis so that some degree of sight may be preserved. Three fulminant cases in which the course of the visual loss was spread over several days were seen in the present series (Cases 1, 4, 13), and descriptions of similar cases appear in the literature, though in one case reported by Robertson (1947) vision in both eyes was lost within 2 hours. Cloake and others (1946) encountered one such case and were able to make a detailed study of the histopathology post mortem. The patient was a woman aged 73 who, in the 10th month of her illness, suddenly lost the sight of her right eye. Four days later the left eye was affected and within 24 hours she was totally blind. One month later the optic disks were pale and atrophic. She died 14 months after the beginning of the illness, and autopsy showed the widespread nature of the condition. The optic nerves showed marked softening; both retinal arteries were equally affected, the lumen in each being obliterated by cellular fibrous tissue obviously formed during the organization of a thrombus; the adventitia and media were infiltrated by lymphocytes and plasma cells, the internal elastic lamina was fragmented and calcified, and a few giant cells were engaged in phagocytosis of these calcified fragments. The changes could be traced forward into the branches of each artery. Similar pathological changes in a similar case are described by Cardell and Hanley (1951).

**Treatment**

No specific treatment yet exists for temporal arteritis, but palliative measures modify many of the more severe symptoms. It is of the greatest importance that patients should be reassured. The agonizing and prolonged pain together with the general symptoms causes them, not unreasonably, to adopt a hopeless outlook, and they must be told that the illness is a self-limiting one and that when the inflammation has subsided they will regain good health. This reassurance, which should be reinforced with regular sedation, plays the most important role in management of the condition. The antibiotics will not modify the disease, though one case with a positive blood culture which responded to aureomycin was reported by Boquien and others (1951). The sulphonamides are contraindicated. Aspirin is the drug of choice for the general relief of the severe pain, and excision of about an inch of an acutely inflamed scalp artery will immediately relieve the local
pain, or the peri-arterial injection of 2 per cent. procaine (Roberts and Askey, 1948; Keen, 1950) will give temporary relief. The condition of the blood must be observed throughout the illness, because patients frequently develop a profound anaemia and on occasion even have to be transfused.

Patients with superadded ophthalmic symptoms may be treated on the same general lines but all concerned in the case and particularly the patient, who is always depressed and retarded, must be warned of the significance of the slightest worsening of vision. Should this occur, and should it be thought that fulminant eye changes are imminent, the patient must be admitted to hospital as an emergency case, and treatment with anticoagulants must be started immediately. The anticoagulants at present available are neither easy to control nor free from danger, but heparin appears to be the drug of choice in ophthalmic conditions because it acts rapidly and undesirable side-effects are less frequent. Duff, Falls, and Linman (1951), who analysed the results of dicoumarol and heparin therapy in the management of occlusive vascular disease of the retina, concluded that eyes with venous occlusion do slightly better than those not so treated. No significant advantage was obtained from prolonged therapy and they recommended intensive short-term treatment with heparin. No reports are yet available on the effects of the new anticoagulants “Paritol” and “Tromexan” in ophthalmic work, nor of the value of anticoagulants in temporal arteritis. Heparin proved efficacious in our single partially successful case, a dose of 10,000 units being given intravenously 8-hrly for a week. There must be careful supervision of the clotting times and prothrombin levels. Should any spontaneous bleeding occur, protamine is an antidote for heparin. Either nicotinic acid, 50–300 mg. thrice daily, or priscol, one or two tablets thrice daily, should be given for their vaso-dilatory effects. Massage of the globes together with inhalations of amyl nitrite relieved the prodromal visual symptoms on three occasions in the fulminant case of temporal arteritis reported by Cardell and Hanley (1951), and this type of local treatment should certainly be tried.

Preliminary unpublished reports claim that dramatic changes are produced by the use of cortisone in temporal arteritis, but further work must be carried out before it can be recommended for use when the ophthalmic vessels are affected. It may prove to be of great value as a short-term measure while the retinal vessels are involved in the fulminant group. If the patient can be got through the acute phase of visual involvement successfully, the vision in the fulminant cases is not likely to be affected at a later date even though the generalized arteritis may continue for many months.

Summary

(1) Temporal arteritis is not a rare disease.
(2) It is a self-limiting disease during which vision is frequently affected.
(3) It may present with ophthalmic symptoms. Reports are given of fifteen patients who sought medical aid on account of disturbance of vision.
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(4) The various ophthalmic manifestations of the conditions are described.

(5) A fulminant group of cases in which vision was rapidly lost is discussed, and methods of treating this group are suggested.

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REFERENCES


