OPTIC ATROPHY IN RHODESIA

AN ANALYSIS OF 50 CONSECUTIVE CASES

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In Southern Rhodesia we have encountered many cases of optic atrophy in Africans, and in a high proportion of them no clear cause could be found. As it is a serious disorder affecting young male adults predominantly, we decided to analyse a series of consecutive cases in which the aetiology was not immediately obvious.

Optic neuritis and atrophy has been known to exist for a number of years in Africa and has mostly been attributed to a deficiency of vitamin B. Wright (1928), in West Africa, referred to dimness of vision associated with pellagra, and Fitzgerald-Moore (1937), from West Africa, also gave a full description of this condition, which usually occurred after symptoms of glossitis, stomatitis, and pruritus of the external genitalia had set in. The vision suddenly became misty, and about two months after its onset ophthalmoscopic examination showed definite pallor of the disks, especially on the temporal side. In more advanced cases a complete optic atrophy developed, when the effect on the vision was usually profound. Fitzgerald-Moore found that in early cases of not more than six months' duration favourable results followed the administration of "marmite," but when the disease had lasted more than a year the response was much less.

In 1948 Dawson, Findlay, and Ward reported five cases of optic atrophy in native soldiers of the Gold Coast who lived on usual Army rations, and attributed it to a deficiency of the vitamin-B complex in the diet. Whereas most of the reports favour a nutritional basis, Boase (1952), who described optic atrophy as being common in the native of Uganda, thought that the cause was not a vitamin lack. Indeed, he failed to discover any obvious reason. Gelfand (1947) similarly referred to the many cases of optic atrophy encountered among the Africans in Southern Rhodesia, and regarded a nutritional disorder as an unlikely explanation for this.

Procedure

The cases with optic atrophy were taken consecutively from the native ophthalmic out-patient department at the Salisbury Hospital. They were predominantly male and were drawn from the industrial and farm-labouring class of the Salisbury area. Also included were labourers from all over British Central Africa and the neighbouring Portuguese East Africa. The diet of these people varies, but is based more or less on the following: 1-2 lb. (450-900 g.) of maize meal a day and 1-2 lb. (450-900 g.) of meat a week, with a variable amount of beans, ground nuts, and little or no fresh vegetables.

The types of optic atrophy were classified according to the ophthalmoscopic appearances based on the six descriptive categories of Duke-Elder (1940). (1) *Post-neuritic atrophy* following a papillitis and distinguished by the presence of post-inflammatory tissue on the disk. (2) *Simple or primary atrophy* in which the entire disk is greyish white and clear-cut, the remainder of the fundus being normal. (3) *Vascular atrophy*, distinguished by extreme attenuation of the vessels, as in occlusion of the central

retinal artery. (4) *Temporal atrophy*, following retrobulbar neuritis or compression, and distinguished by temporal pallor of the disk. (5) *Consecutive atrophy*, following choroido-retinal disease; this is typically seen in the last stages of pigmentary degeneration of the retina, and is characterized by a yellow waxy colour of the disk and extreme attenuation of the arteries. (6) *Glaucomatous atrophy*, distinguished by the presence of cupping of the disk. Groups 5 and 6 were excluded from the series, as the cause in these cases was not in doubt.

Method of Examination.—Each patient was first examined in the ophthalmic out-patient department, where his visual acuity, fields of vision, and pupillary reactions were recorded together with a description of the eye-grounds. If other ophthalmic disease was present this was also noted. The patient was then admitted to the medical ward, where a full examination was conducted. This included a history and physical examination, with special attention to the nervous and cardiovascular systems. Each case underwent lumbar puncture, the pressure was recorded, and the fluid was investigated for its protein, cell, and chloride content. Blood was taken for the Wassermann test, and a routine sugar and urea estimation was made on each blood sample. An x-ray examination of the skull was also carried out (antero-posterior and lateral views). The urine and stool specimens were examined chemically as well as microscopically. As expected in a study of this nature, allowances must be made for the difficulties ordinarily encountered in African practice as regards the accuracy of the clinical history.

Clinical Findings

Of the 50 cases in this series 32 were classified as post-neuritic atrophy, 12 were of simple or primary atrophy, and 6 had temporal and vascular atrophy. Two cases were followed to necropsy.

Case 3.—A waiter aged 48, from a Salisbury hotel, complained of a sudden loss of vision of the left eye some months before reporting to the clinic. He had a pale left disk with shallow cupping, extreme attenuation of the arteries, and an atrophic retina, the right fundus being normal. He died suddenly three months later. At necropsy a large haemorrhage into the left internal capsule was found. The cause of the atrophy was ascribed to endarteritis with occlusion of the left central retinal artery.

Case 25.—This patient complained of sudden loss of vision and severe headaches six weeks before she reported to the clinic. The right fundus showed a white disk with the lamina cribosa obscured, the vessels, macula, and peripheral retina being normal. In the left fundus a white disk with clear-cut margin was found, the lamina cribosa being clearly seen. Radiographs showed enlargement of the pituitary fossa. No visual fields could be determined, as the blindness was absolute. She died one week after admission, the necropsy revealing a glioma of the left frontal lobe.

In two more cases the aetiology of the atrophy seemed to be reasonably definite.

Case 9.—A man aged 25 had very gross cupping of both disks and associated atrophy of the left optic nervehead. Full ophthalmic examination revealed no evidence of glaucoma and the medical examination was negative: his case was classified as primary cavernous optic atrophy (Schnabel, 1892).

Case 11.—A fat young man of 20 had complained of poor vision for some months. Examination revealed a bitemporal hemianopia with macular sparing and temporal pallor of the right disk. There was marked enlargement of the pituitary fossa. Unfortunately, on hearing that the treatment advised was surgical he asked to be discharged, and the exact nature of the lesion was not established. The provisional diagnosis was Fröhlich's syndrome.

Post-neuritic Atrophy.—Of the 32 patients exhibiting post-neuritic atrophy, 20 maintained that the onset of blindness was gradual and 12 said it was sudden (within two weeks being taken as the limit of sudden onset). The ages of the patients varied between 13 and 65, the average being 40. Most of these patients were males, only seven being females. The blood Wassermann reaction was positive in 13 cases

(40%) (Table I), but the reaction of the cerebrospinal fluid was negative in all. Eleven (34%) had changes in the cerebrospinal fluid (7 had an increase of protein, 7 a pleo-

TABLE I.—*Post-neuritic Atrophy Cases. Showing the Number with Blood Wassermann and Cerebrospinal-fluid Changes*

No. of cases	32 (100%)
No. of cases with C.S.F. changes (protein or cell increase)	11 (34%)
No. of cases with +W.R. in blood	13 (40%)
No. of cases with +W.R. in C.S.F.	—

cytosis, the main cell being the lymphocyte or monocyte, and in 4 the pressure was raised). Obvious signs of vitamin-B deficiency were present in only three of the patients, but as they had been blind for at least a year these changes could have occurred because of the difficulty of ensuring proper feeding.

TABLE II.—*Primary Optic Atrophy Cases. Showing the Number With Changes in the Blood Wassermann and Cerebrospinal Fluid*

No. of cases	11 (100%)
No. of cases with C.S.F. changes	2 (18%)
No. of cases with +W.R. in blood	4 (36%)
No. of cases with +W.R. in C.S.F.	0

Primary Optic Atrophy.—Of the 11 cases of primary optic atrophy—excluding Case 25, with the left frontal lobe tumour referred to above—seven claimed the loss of vision to have appeared suddenly and four gradually. Four of the seven with sudden onset gave a history of a severe blow to the eye, after which the vision was completely lost, but radiographs of the skull showed no sign of bony injury in the orbital region, and ophthalmoscopically there was no evidence of damage to the eye apart from the primary optic atrophy. The other eye was normal in all four cases. The ages of the patients varied from 15 to 55 years, the average being 29. The cases were predominantly male, there being only one female case. The blood Wassermann reaction was positive in four (36%), but in the cerebrospinal fluid the reaction was negative in all cases. In two there were changes in the fluid (both with a pleocytosis). Three cases showed skin changes of pellagra, but again the changes could have followed the blindness.

Discussion

Several features stand out from the above facts. Post-neuritic atrophy was by far the most common type of atrophy, the majority of the remainder being simple or primary atrophy. The most striking feature, however, was the high rate of positive blood Wassermann reactions coupled with the corresponding negative findings in the cerebrospinal fluid. The causes of the optic atrophy are considered below under four main headings.

1. General Inflammatory Conditions

Syphilis.—In a population with a high incidence of syphilis, this must be taken very seriously into consideration as an aetiological factor, and the suggestion is that a large number of cases of optic atrophy in Africa to-day are syphilitic in origin, though they show few or no signs of involvement of the nervous system. The incidence of syphilis as determined by the Wassermann reaction in a series of 1,500 consecutive admissions in the same hospital was 19% (Gelfand, 1947). Willcox (1949) estimated the incidence in the general African population in Southern Rhodesia to be about 15%. The percentage of positive blood Wassermann reactions in our post-neuritic atrophy cases was 40%, and in those with primary optic atrophy 36%. These figures seem significant when compared with the general incidence of syphilis. The Wassermann reaction of the cerebrospinal fluid was negative in every case.

Purely from an ophthalmic consideration there were several features which favoured a syphilitic basis. The fields showed concentric contraction rather than central scotomata. Occasional ocular nerve palsies were noticed, and, unlike the usual case of optic neuritis with a good prognosis for vision, secondary atrophy generally developed and bilateral blindness was a common sequel.

A large number of the patients gave a history of having had injections, though few could say whether these were

antisyphilitic or not. Some of them mentioned a previous history of venereal disease, though very few had stayed to complete the course of treatment, and, as approximately 36.5 kg. neoarsphenamine was used in one year (1952–3) in the treatment of African patients in Southern Rhodesia, it is reasonable to suggest tentatively that a number of these cases represent a neurorecidive reaction following inadequate treatment for their original venereal complaint.

The association of normal neurological findings with a clear cerebrospinal fluid in the great majority can only be taken to mean that these cases represent a very localized retrolubular or papillary neuritis in which the brain and meninges escape implication. This parasyphilitic manifestation with no other evidence of nervous involvement has been reported outside Africa (McIntosh *et al.*, 1913; Head and Fearnside, 1914).

Neurotropic Viruses.—The possibility of a neurotropic virus was borne in mind. Boase (1952), of Uganda, puts forward this as a suggestion only, but we thought it unlikely because of the absence of any sign of infection such as fever and because of the frequently normal cerebrospinal-fluid findings. Disseminated sclerosis is an extremely rare condition in Africa, and no case in this series was thought to be due to it. It is included here for convenience.

2. Endogenous Toxins

Following Acute Infective Diseases.—Malaria, pneumonia, dysentery, and typhoid are all common acute infections in African hospital practice. Optic atrophy, however, has not been reported as an important complication of these diseases. In our series only four cases gave a history of severe illness prior to the onset of the blindness. None of these were admitted to hospital in the acute phase, and no particular infection was traced on admission.

Septic Foci.—In only one patient was this considered to be a possible cause. She complained of a severe bout of toothache involving the first premolar tooth on the upper right side, which caused oedema of her face and right orbit. Only after she had noticed, one month later, the loss of vision of the right eye did she have the tooth removed; the blindness, however, remained.

Metabolic Changes.—Anaemia, diabetes, etc., did not seem to play any part in the aetiology in this series. The blood counts were on the whole within normal limits and no case had an abnormal blood sugar or a raised blood urea.

3. Nutritional Causes

It seems unlikely that poor nutrition has *per se* produced many of the cases of optic atrophy in this series. The majority showed no sign of malnutrition on examination.

The staple diet of the native population is maize meal—of which 1–2 lb. (450–900 g.) a day is eaten, and meat (almost always beef) 1–2 lb. (450–900 g.) a week. This is often supplemented by dried beans, 1 lb. (450 g.) a week, and salt 4 oz. (110 g.) a week. Ground-nuts, sweet potatoes, fresh vegetables, sugar, and bread occasionally are added. Taking 7 lb. (3.2 kg.) of maize meal, 2 lb. (900 g.) of meat, and 1 lb. (450 g.) of dried lima beans a week as an average, this would give an approximate daily intake of 74 g. of protein, 34 g. of fats, and 385 g. of carbohydrates; giving 1,994 calories.

The vitamin-B intake was estimated as adequate. Although not showing any particular deficiency that might be expected to result in an optic atrophy, this intake is probably too low to sustain manual labour.

Defective vision associated with symptoms of pellagra, glossitis, scaly itching of the scrotum, and mental and other nervous manifestations as described in Africans by Wright (1928) from Sierra Leone and from Nigeria by Fitzgerald-Moore (1937) were not seen. Nor did our series bear much relationship to a series of cases of optic atrophy published by Wilkinson and King (1944) from the Far East. In these series of cases the primary foodstuff was rice—in contradistinction to the basic maize meal of our cases.

As a contributory cause, however, it is considered that malnutrition may play a part. In Cases 29, 36, 42, and 48 unilateral optic atrophy followed a severe blow in the region of the eye concerned. In no instance was the blow sufficient to have left any radiological evidence of bony damage, nor was any ocular damage seen apart from the optic atrophy. It seems possible that where a severe concussion injury has caused damage to the optic nerve the nutritional level may be too low to repair the damage and atrophy may result.

One of our cases (No. 46)—that of a soldier aged 37 who complained of the fairly sudden onset of blurring and dimness of vision soon after arrival in Egypt—showed points of similarity to a series of cases from the Middle East (Spillane and Scott, 1945). The majority of these cases retained good physical health, and the skin, heart, lungs, and gastro-intestinal tract were not affected. Within a year of this soldier's return to Rhodesia his vision was reduced to R. 6/36, L. 6/24. His disks showed evidence of post-neuritic atrophy, no central scotoma was demonstrated, his fields being apparently full, but the use of a Bjerrum screen at 2 metres was not made.

There was no significant increase in the number of cases admitted towards the end of the dry season and the beginning of the rains, when food supplies became exhausted. It is at this time of the year that we see, for example, many cases of pellagra, night blindness, and scurvy.

4. Exogenous Toxins

Methyl alcohol, arsenic, and quinine are probably the main toxic agents with which the indigenous population may come into contact. As has already been mentioned, arsenic may play an important part after inadequate treatment of syphilis. Quinine and methyl alcohol blindness were not encountered in this series.

It is possible that a native medicine—not divulged by the patient—may play a larger part than is suspected at the moment. We failed to discover any definite addicts such as the case of optic neuritis in a Qat addict in Somaliland described by Baird (1952).

Summary

Fifty consecutive cases of optic atrophy in Africans from British Central Africa have been analysed. Purely ophthalmic causes, such as chorio-retinal disease with consecutive optic atrophy, and glaucomatous optic atrophy, have been excluded from the series, as the cause in these cases was not in doubt.

Thirty-two of the series were found to be of the post-neuritic atrophy type and 12 were of the primary optic atrophy type. Vascular and temporal atrophy made up the balance of cases.

Various aetiological factors have been considered. In four patients—two of whom died—it was possible to ascribe a definite cause. Of the remainder, it was thought that syphilitic optic atrophy, but with no other neurological lesion, was a probable cause in the majority. A disorder of nutrition does not seem to be an important cause of optic atrophy in Central Africa.

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SHIGELLA FLEXNERI ENZOOTIC IN CAPTIVE RHESUS MONKEYS

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The occurrence of dysentery among the rhesus monkeys at the Clifton and West of England Zoological Society's gardens in Bristol proved to be associated with a type of *Shigella* organism which is rarely found in the human population in Great Britain. As the outbreak presents some other interesting features it is reported here.

Macaca mulatta, the rhesus monkey, is kept in the "monkey temple" at the Bristol Zoo. This is an erection in the centre of a concrete pit, well away from direct contact with the public, although the latter can drop food to the monkeys if they wish. The first colony to occupy this temple arrived in 1929. There were 80 monkeys, and many of them died from a disease then regarded as ulcerative colitis, which closely resembled the condition we are now describing as shigellosis. In 1932 the colony began to die from tuberculosis and all had to be destroyed. In the spring of 1933 a second colony arrived, this time without intestinal disease; but again they acquired tuberculosis, and the remaining monkeys had to be destroyed in 1935. In 1936 a third colony of 70 young ones came. They bred well, and showed no signs of intestinal disease or of tuberculosis. However, furious fighting broke out in 1950 and many died of wounds. They were mostly quite old by now, so they were destroyed, except for eight young ones which were kept, and the new colony about which this paper is written was put in with them. In 1949 one monkey of the third colony had died with diarrhoea, and *Sh. flexneri* had been isolated from its faeces, but was not typed. The eight remaining monkeys appeared fit, and it was hoped they did not harbour the organisms.

Cook (1953) reports on rectal swabs taken from 339 freshly imported monkeys received by the Ministry of Supply. From these *Shigella flexneri* type 103 (4a) was cultured once, type 103 Z (4b) 40 times, and type Z twice; *Shigella sonnei* 24 times; *Shigella schmitzi* 50 times, and various other types 6 times. Cook states that bacillary dysentery, pneumonia, and tuberculosis are the main scourges of captive monkeys in this country.

The Epizootic

The present (fourth) colony of 103 monkeys was imported by air from Calcutta on March 19, 1951. They were mostly under 2 years old. Eight days after arrival the new young monkeys began to die. The weather was very cold and snowy. Post-mortem examination was carried out in all cases (Table I). The first eight died of pneumonia; then diarrhoea was noticed, and in the three months up to the end of June a further 18 died. Faeces from 15 of these were examined *post mortem*, and *Sh. flexneri* was isolated from 8, and typed in 6 cases as *Sh. flexneri* 103 (4a). In December, 1951, three more monkeys died, the faeces of two of which were examined, and from these the same organism was isolated.

Early in 1952 sporadic deaths occurred, reaching a peak in April with four deaths, one producing *Salmonella typhi*.