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MALARIAL AFFECTIONS of the EYE

BY M. T. YARR, F.R.C.S.I.

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Moorfields; Member Ophthalmological Society, &c.*

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TEXT-BOOKS on diseases of the eye as a rule dismiss the subject of malarial eye-affections in a few words: in fact, I am only aware of one¹ in which anything like an adequate summary of the state of present knowledge of this important class of disease is given. When we consider the large amount of space often devoted in these works to diseases and congenital defects of excessive rarity—to the curiosities of ophthalmology, so to speak—the absence of detailed reference to malaria becomes astounding. On the other hand, a few able monographs on the subject are in existence, and a mass of evidence in the shape of articles on symptoms, notes of cases, &c., may be laboriously disinterred from the ophthalmological literature of the last thirty years.

Most medical men in practice in the tropics are familiar with a distinctively malarial class of eye-diseases, with characteristic symptoms, characteristic ophthalmoscopic signs, and above all, characteristic pathology; and few writers on tropical diseases omit more or less detailed allusions to the subject—indeed, so far back as 1833 we find a special “amaurotic” form of malarial fever described²—but only too often the value of their remarks is minimised by vagueness of description and unscientific phraseology, those convenient but antiquated terms, “amaurosis” and “amblyopia,” being employed to cover a multitude of sins of omission and inaccuracy. Some idea of the importance of these affections, as well as of their wide-spread prevalence, may be gathered when we find it stated by Poncet³—to whose able and laborious researches we owe most of

¹ “Traité Complet d’Ophtalmogie,” by L. de Weeker and G. Landolt (article by J. P. Nuel).

² Stosch, “Febris Intermittens larvata amaurotica,” Carper’s *Wochenschrift*, 1833, 11, No. 3.

³ *Ann. d’Oc.*, 1878, t. lxxviii., p. 201.

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our knowledge of the subject—that he found fundus lesions visible by the ophthalmoscope in no less than 10 per cent. of malarials examined by him in Algiers, and by the microscope found fundus lesions *the rule* in necropsies on cases of malarial cachexia and pernicious malaria. It is therefore evident that malaria—which, like syphilis, spares no organ—does not spare the eye. In the brief space at my disposal I can only endeavour to bring together, in the form of a concise summary, facts, hitherto to be found only in scattered form, supplemented by such observations as my personal experience in the tropics, at Netley, and at Moorfields enable me to make.

I purposely omit reference to malarial albuminuric retinitis and diseases of the anterior half of the eye, as not presenting, in my opinion, signs sufficiently distinctive to justify, in the present state of our knowledge, an attempt to group them under the head of affections characteristically malarial.

Malarial eye-lesions all originate in circulatory troubles and may be conveniently classified under the following heads:—

- (1) Neuritis.
- (2) Retinal Hæmorrhages.
- (3) Retino-Choroiditis.
- (4) Effusions into the Vitreous.

(1) *Malarial Neuritis.*

From one of my Hong Kong case-books I take the following brief notes of a typical case of malarial neuritis:—

Private A. F., admitted to hospital complaining of supra-orbital pains, dimness of vision, and photophobia.

During preceding twelve months had been in hospital seven times with malarial fever, no attack of exceptional severity; he is thin, anæmic, spleen slightly enlarged; temperature normal, urine normal. Vision $\frac{6}{60}$ each eye; fields normal; colour perception unimpaired; disc raised, reddish-grey, margins blurred; rest of visible fundus normal save for a slight haze. He was placed on light, nutritious diet—chicken, fish, beef-tea, and given quinine. A week later the visual acuity had improved to $\frac{6}{18}$, but reverted after three days to $\frac{6}{36}$, and varied between that and $\frac{6}{24}$ for some time; five weeks after admission it suddenly became $\frac{6}{12}$, at which it remained. The supra-orbital pain and photophobia ceased a fortnight after admission. After seven weeks in hospital he was sent to the sanatorium on the "Peak," and from thence invalided to England a month later with "malarial cachexia." Examined again before leaving, visual acuity $\frac{6}{12}$, fields normal, with the exception of a very slight contraction on the nasal side; colour perception normal; fundus normal, with the exception of a slight greyness on the temporal half of optic disc.

Signs and symptoms binocular throughout. No history of syphilis.

I have selected the above case as embodying most of the characteristics of the disease.

(1) It will be seen that the patient had suffered from repeated attacks of malarial fever. This is the case as a rule; it almost invariably occurs in those whose systems are broken down by many—it may be slight—attacks of fever. I have seen one case of neuritis commencing in the course of a pernicious attack, but such cases are not common.

(2) In the commencement, supra-orbital pain and photophobia are almost constantly present; night-blindness frequently.

(3) Colour perception remains unimpaired, except in the rare cases ending in complete atrophy.

(4) The variations in the visual acuity in the course of the malady form the most characteristic symptom and distinguish malarial neuritis from all other forms. Sulzer, in a careful *résumé* of the subject,¹ lays stress on this point. A diminution of the visual acuity $\frac{1}{10}$ can rise in two or three weeks to $\frac{1}{2}$ or $\frac{2}{3}$, falling again perhaps in two or three days. Macnamara² described a case in the Calcutta General Hospital of a young girl of 13, admitted with a visual acuity = perception of light only—who was discharged five weeks later with normal vision.

(5) Fields intact, or only slightly contracted.

(6) The fundus changes visible with the ophthalmoscope include swelling of the papilla, which assumes a greyish-red colour, œdema of the circum-papillary retina with effacement of the papillary margins, and enlarged and tortuous veins. The peculiar colouration of the papilla—"teinte rouge-grisâtre"—due to parasites in its capillaries, is pathognomonic. In about a third of the cases tiny peripheral retinal hæmorrhages are also found.

(7) About 80 per cent. of cases terminate in a partial atrophy, indicated by varying diminution of visual acuity, irregular contraction of the field, and slight greyness of the disc; many end in apparently complete recovery, some rare cases go on to complete atrophy.

Pathology and Morbid Anatomy.—For our knowledge of this branch of the subject we are mainly, if not entirely, indebted to Poncet³ who systematically examined with the microscope the eyes in all cases of death from malaria at the military hospital of Philippeville (Algiers). His researches show conclusively that the changes in the disc and retina in this disease are due primarily to melanæmia with increased vascularisation, the subsequent atrophy or partial atrophy being explained by consecutive endoarteritis of the vessels. A perpendicular section through the retina shows the raised, swollen papilla ("tête de hanneton"), its little capillaries stuffed with leucocytes, each containing a central spot of black pigment—red corpuscles excessively rare; the same pigmented leucocytes when Poncet uses the word "leucocytes" he undoubtedly means (in most instances malarial parasites) fill the retinal vessels, and when hæmorrhages exist, they are found to be due to emboli of these leucocytes with consecutive extravasations.

The affection is always binocular, although it does not usually begin in both eyes at the same time.

Sulzer believes that a certain proportion of these cases have

¹ "Troubles de la Vision dans l'Impaludisme," *Arch. d'Ophthal.*, 1890.

² *Medical Times and Gazette*, May 2, 1868.

³ *Vide supra*. Also "Atlas des Maladies Profondes de l'Oeil," by Perrin and Poncet.

malaria as a predisposing cause only, the exciting cause being the indirect action of sunlight, and adduces certain cases seen by him in Borneo in support of his view, but his arguments are not convincing and the evidence seems inadequate.

(2) *Retinal Hæmorrhages.*

Two varieties of retinal apoplexy are found in association with malaria: (a) minute peripheral; (b) large peri-papillary and macular.

Minute hæmorrhages in the ciliary zone of the retina are frequent in acute attacks of fever: they are often so very minute and so far forward as to be easily overlooked. Poncet found them in all cases of death from malaria. They may accompany or follow neuritis,¹ but often form the only apparent lesion of the fundus. It seems probable that many of the transient disturbances of vision so commonly seen in malarial fevers are due to slight œdema of the retina, followed by these tiny hæmorrhages.

The large peri-papillary and macular hæmorrhages are much less frequent, and like the neuritis which they sometimes accompany, are usually seen only in malarial cachectics. These are of much graver import, always causing some impairment of vision and occasionally even absolute loss. I believe some of the cases of "sudden and persistent amaurosis" described by writers on malaria to be due to macular hæmorrhages. The only case of such sudden amaurosis I have seen was in Hong Kong, in a soldier suffering from advanced malarial cachexia who subsequently died; in the course of one night his vision in the right eye was reduced to perception of light, in the left to $\frac{6}{36}$; examination showed a large macular hæmorrhage in the right, with several small hæmorrhages between papilla and macula in the left; diffuse haziness of retina in both.

In a case of malarial cachexia invalidated from the Indian Frontier, which I recently saw at Netley by the courtesy of the Director-General, Army Medical Staff, there were several hæmorrhages arranged in a curiously symmetrical manner along the inferior temporal vessels in both eyes; V.A.: in R. $\frac{6}{12}$, in L. $\frac{6}{24}$; urine normal.

Microscopic examination shows these retinal apoplexies in malaria to be due to infarcts of parasites followed by extravasations (*vide supra*).

(3) *Retino-Choroiditis.*

In about 20 per cent. of acute intermittents, generally towards the end of the hot stage, patients complain of supra-orbital pains, tenderness on pressing the eyeballs, photopsies and photophobia. Examination then discloses a general hyperæmia of the fundus,

¹ Gueneau de Mussy, *Journal d'Ophthal.*, t. 1., p. 5, 1872.

mainly venous; red, slightly swollen papilla, surrounded by a grey veil; and general haziness of the retina which appears to have an undulating surface—"dunes" with intervening depressions.

This œdematous state of the ocular membranes—the first stage of malarial retino-choroiditis—generally subsides without leaving any appreciable trace. In a certain number of cases, however, more especially in those who have had repeated attacks of fever, and are falling into the condition of malarial cachexia, the symptoms persist, punctate peripheral hæmorrhages appear, and a chronic, slowly progressive retino-choroiditis is set up, ending in capillary atrophy of the choroid and much loss of vision. Out of thirty-eight cases of malarial cachexia which I examined at Netley, in April last, I found this condition of capillary atrophy of the choroid in three—nearly 8 per cent. I have been able to watch the progress of such a case in a man—a discharged soldier who had suffered much from malaria in Burmah—who has been attending Moorfields for the last twelve months; the fundus is now of an almost uniformly grey colour, as though powdered over with pepper, the disc is pale and the arteries reduced to fine threads; the distribution of the choroidal vessels is mapped out with extraordinary clearness, the vessels seem almost white with a central red streak; pigment-layer of retina and chorio-capillaries atrophied; vision $\frac{4}{60}$ in one eye, $\frac{1}{5}$ in the other; some myopic astigmatism, but the correcting glasses only improve to $\frac{6}{36}$ and $\frac{6}{18}$; fields irregularly contracted. When first seen he had only general haziness and loss of lustre of retina, with V. $\frac{6}{12}$ in each eye with correction.

The following description by Poncet¹ of the retina of an Algerian colonist who died from a pernicious attack of malarial fever shows clearly the nature of the circulatory changes in the acute stage of his affection. The patient was much emaciated, pale, and anæmic, and had had several attacks of fever.

"In all the capillaries such a quantity of pigmented elements is found that each vessel looks as though formed of a mosaic with little black points. Red corpuscles are rare. Each little black point is a pigment molecule in the protoplasm of a white corpuscle; very rarely the pigment is free in the capillary. Two points are very clearly demonstrated. First, the extraordinary number of leucocytes; second, the enormous quantity of pigment. It is easy to understand the obstacle to the circulation caused by this mass of leucocytes in the capillaries. This abundance of pigmented leucocytes (parasites?) is found in the entire retina, at the periphery as at the papilla, and explains the peri-papillary œdema and dirty grey aspect of the papilla in these cases of malarial cachexia."

The subsequent capillary atrophy of the choroid and partial atrophy of the optic nerve is due to chronic inflammation of the choroidal and retinal vessels, set up by the irritation of the plasmodia, ending in atrophic changes.

¹ Planchc, lix., fig. 1. Atlas de Perrin et Poncet.

(4) *Effusions into Vitreous.*

White Infiltration of Vitreous.—This rare and curious affection was first described by Seely,¹ and consists in an infiltration of the vitreous, forming in stages, causing almost complete loss of vision for a time, and giving a characteristic white reflex with reflected light. Seely attributes it to a serous infiltration, due to chronic paludism. In his two cases the progress of the disease was oscillating for several months; eventually the visual acuity became normal under prolonged quinine; in one case mobile opacities persisted.

Sulzer describes three cases of this disease. In the first two the ocular affection had existed some weeks before they came under his notice; in the first the eyes had been blinded during the night, with only a day's interval between each; in the second there was an interval of several weeks. The first was complicated by intense ciliary and supra-orbital neuralgia on both sides, with tenderness of the eyeballs and limited and painful motility. Both cases were malarial cachectics, with intense anæmia. Visual acuity was reduced to perception of light. The diffused vitreous infiltration, which at first gave a perfectly white reflex in all directions, was not completely absorbed in either case. During the eighteen months under observation this was replaced by moving flocculi arranged like a spider's web, with slight turbidity of the vitreous in the meshes, so that the fundus was only indistinctly visible, although the white atrophic colour of the papillæ could be made out. The right eye of the first case became blind, the left counted fingers at a short distance. The second counted fingers at 60 cm. when last seen. The third case was seen a few days after the onset of the disease, when only the right eye was attacked; it presented a diffuse and equal infiltration of the posterior segment of the vitreous with a faint peripheral red reflex on complete dilatation of the pupil; V.A. = fingers at 20 cm. Four weeks later the left eye was similarly attacked. In three months, under quinine, total reabsorption took place, leaving V.A. normal.

Penoff² also described diffuse opacities of the vitreous in malarials, but they were complicated by affections of the uveal tract.

I have never seen a case of white infiltration, and such cases of vitreous opacities in malarials as I have seen have been preceded or accompanied by uveal or other lesions only indirectly due to malaria.

It will, I hope, be clearly understood from the foregoing necessarily imperfect sketch, that the classification I have attempted to make is based only on the prominence of one set of signs or another in the majority of cases; for instance, a sharp line of demarca-

¹ W. W. Seely, *Transactions of American Oph. Soc.*, 18th Annual Meeting, 1882, p. 345.

² *Centralblatt für pract. Augen.*, 1879, p. 80.

on cannot always be drawn between neuritis, retinal hæmorrhages and retino-choroiditis; many cases occur in which all three affections are associated or form stages in one process.

It remains only to enumerate some of the rare or obscure affections mentioned by writers on malaria.

Sudden and persistent Amaurosis without visible Fundus change.—Well authenticated instances of this are on record: they can only be attributed to some obscure focal brain-lesion.

Periodic Amaurosis.—See remarks on œdema of retina and retinal hæmorrhages. Possibly also due to quinine.

Sudden Amaurosis ending in Atrophy.—Possibly due to hæmorrhage into the sheath of the optic nerve: frequently cases of quinine amaurosis.

*Persistent Central Scotoma.*¹—I have never seen a case that was not due to macular hæmorrhage.

Periodical Blue Vision.—Baas² describes a case of malarial fever in which this curious symptom was present. I am unable even to conjecture an explanation.

The treatment of malarial eye troubles is the treatment of malaria; most essential of all is the early removal of such cases to a non-malarious country. A prolonged course of iodide of potassium is generally of benefit in hastening the absorption of vitreous opacities.

Heurteloup's artificial leech to the temples, protection from light, blisters, are useful in relieving local symptoms.

Quinine Amaurosis.

Before concluding, it is necessary to say a few words on *quinine amaurosis*.

Slight degrees of this, caused by spasmodic contraction of the arteries, are amongst the familiar experiences of all practitioners in malarious countries. A certain amount of amblyopia is almost invariably present in cases of quinism, synchronous with the aural symptoms. In very susceptible individuals this may amount to absolute blindness, persisting for hours and even days. In severe cases varying amounts of concentric contraction of the field remain, central vision, colour, and light senses being as a rule unaffected. In slight degrees of quinine amaurosis the ophthalmoscopic appearances are normal; in severe cases, with persistently contracted fields, pallor of the disc and thready vessels are found.

I am indebted to the kindness of Mr. Treacher Collins for the details of the following remarkable case. The patient has been attending Moorfields at irregular intervals for the last four years. I first saw him three years ago.

W. J., aged 29, came to the out-patient department on June 8,

¹ Teillais (de Nantes), *Ann. d'Oct.* xcvi., p. 234.

² *Klinische Monatsblätter für Augenheilkunde*, 1885, p. 240.

1894. He stated that three months before he had taken six-penny-worth of quinine (120 grs.) for a headache; vomiting, lasting eighteen hours, followed, with acute head-pains but *no* deafness; his "sight seemed to go all at once," and he could not distinguish light from darkness with either eye; four days after sight began gradually to return, and in three weeks' time he could read; he returned to his employment as a carman and felt little trouble till a week ago, when sight began to fail again. On examination, V. $\frac{6}{9}$ each eye (telescopic); fields concentrically contracted to the size of threepenny pieces; discs a dead white; vessels threads, fundi anæmic, white lines along arteries. When next seen, on June 28, 1894, V. was reduced to hand movement. After this it began very slowly to improve under nitro-glycerine tabloids; slight momentary improvement could always be obtained by inhalations of nitrite of amyl. When last seen, eight months ago, V. $\frac{6}{18}$ and J.₂ in R.; $\frac{6}{9}$ and J.₁ in L. (telescopic); field and fundus unchanged.

Such cases are happily of extreme rarity, in this country, at all events. Mr. Swanzy, in the last edition of his work on "Diseases of the Eye," states that he has seen only one case of quinine amaurosis with permanent serious defect of vision.

De Schweinitz made some interesting experiments on dogs in order to determine the lesion in quinine blindness,¹ showing conclusively first, that the prolongation of quinine-amaurosis produces true atrophy; and second, that thrombosis of the central vessels may be expected in severe cases.

Treatment.—Stoppage of quinine; this, with tonics and nitro-glycerine, generally leads to ultimate cure. I need hardly add that in treating ocular affections in malarials, the possibility of the symptoms being produced by quinine should never be overlooked.

¹ *Transactions of the American Ophthalmological Society*, 1891.