QUININE AMBLYOPIA

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The following case history demonstrated several interesting points and for that reason is worth recording. The patient, Mrs. L., was a leading Wren aged 27 years, and was stationed at a Royal Naval Establishment on the east coast of Ceylon. She was married in June, 1944, and went up country for her honeymoon. Towards the end of this period, July 2, she developed general symptoms of malaise, frontal headache, pain behind the eyes and on moving the eyes, shivering at times and teeth chattering. On her return to Trincomalee on July 4 she was admitted to the sick quarters where her temperature was found to be 104°F. As well as general malaise she was complaining of headache, backache and nausea. She had left England in November, 1943, and had had no previous tropical diseases. I did not see her until July 24, 1944, when she was admitted to R.N. Auxiliary Hospital, Colombo, and came under my care. The following physical signs up to that date are taken from the notes made by Surg.-Commander H. L. Hoffman, R.N.V.R., Medical Officer in charge at Trincomalee.

General examination revealed no abdominal tenderness or enlargement of liver or spleen. On July 5 her urine contained bile pigments, but no albumen. On July 8 a blood film showed the presence of B.T. malaria parasites. The same day she started quinine and was given grs. x t.d.s. by mouth. On July 11 her spleen was palpable and she was also tender in the gall bladder area. Two days later on July 13 she complained of deafness and of seeing black spots in front of her eyes, which became bigger and paler turning greenish in colour. Her quinine therapy was stopped at once. She had had 120 grs. in all. She was put on tab-atebbrin until July 18th being given 12 grammes in all. On July 19 she was complaining of blurred vision, especially in the left eye and that her eyes were sore and hurt on movement. The next day her pupils were dilated and reacted sluggishly to light. On
July 21 her pupils were widely dilated. The left pupil did not react directly to light and the right pupil only reacted poorly. The left vision was no perception of light, and the right vision was counting fingers across the ward. The optic discs were swollen and there was a small flame shaped haemorrhage below the right disc. The external ocular movements were normal. There was no deafness now, blood pressure was 120/60, but she was complaining that her feet felt cold. On the following day the coldness had spread up to her knees. Her right vision was now only counting fingers at one metre. The retinal arteries were constricted and the veins engorged. There were some vague paraesthesiae in the legs and the following rather indefinite C.N.S. signs—blunting to pin prick in lower part of legs—poor sense of position in the right big toe—equivocal plantar responses and absent abdominal reflexes. She was put on tab. trinitrin grs. 1/100 six hourly; tab. digitalis leaf (grs. 1½) six hourly, given alternately at three hourly intervals.

On July 24 she was admitted to R.N. Auxiliary Hospital, Colombo, where she came under my care. Her pupils were three quarter dilated, circular, inactive to light, but reacted on convergence. There was no perception of light in either eye. The media were clear. The fundi looked pale and oedematous, veins engorged and arteries constricted. The edges of the discs were blurred, but there was no measurable swelling. The small haemorrhage just below the right disc already mentioned was still present.

The subsequent course of events was as follows:

July 27. Her discs appeared rather less swollen, but she still had no P.L. The trinitrin was stopped as it seemed to be upsetting her.

July 29. Examined by the Medical Specialist (Surg.-Lieut.-Comdr. J. H. Cobb, R.N.V.R.), who found she had severe circulatory disurbance of hands and feet which were pallid and cold. The peripheral nerves showed no defect. The abdominal reflexes were reduced but present, and plantars were flexor. He found no evidence of other neurological disease and thought acute disseminated sclerosis could be ruled out. Lumbar puncture and blood count were carried out and showed no abnormality apart from some anaemia.

July 30. She thought she could see slightly with her right eye; her pupils and fundi appeared unchanged.

August 1. Right pupil reacted briskly to light—vision hand movements in central field—field very constricted especially down and out. No P.L. in left eye and left pupil inactive directly to light.

August 2. Blood urea estimated 34 mgms. per 100 c.c.

August 3. Left pupil reacting to light directly, but not brisk. Vision hand movements in both eyes. Discs seemed less swollen and temporal edge of left disc could be made out.
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August 5. Discs definitely less swollen, temporal edges of both discs seen. Vision: right counting fingers at one metre, left counting fingers at one foot. Right pupil maintained its reaction to light well, but not the left. Discs clearer and arteries appeared less constricted. The retinal haemorrhage in the right eye had now absorbed. On confrontation, fields very constricted in lower temporal areas, practically to fixation in left eye.

August 13. Could now see to get about a bit, although vision seemed unchanged. Hands better colour and not cold to touch, but feet still cold to touch.

August 15. R.V. = 2/60. L.V. = Counting fingers at one foot.

August 17. R.V. = 3/60. L.V. = 1/60 pupil reactions seemed normal and fields seemed less contracted in lower temporal areas. Retinal reflex still very marked.


August 25. R.V. = 3/60. L.V. = 5/60. The right disc seemed definitely paler than the left.
October 3. Central vision unchanged. Peripheral fields charted on perimeter. Fields showed generalised contraction in both eyes, but more marked in left eye. Right field showed quadrant sector defect in lower temporal field. Left field showed marked loss of lower quadrants of temporal and nasal fields. The edges of the fields were "sieve like." This odd symmetrical field loss remained, and when her fields were last charted, November 28 (Fig.), she
still had this loss down and out but more marked in the left eye. A stereoscopic X-ray of skull showed no abnormality.

October 24. She developed dengue fever.

November 10. She had a recurrent malarial attack. B.T. shizonts were demonstrated in her blood and she was treated with tab. atebrin.

December 4. R.V. = 6/6 (less two). L.V. = 6/6 some letters. Binocular vision 6/6 full. Her discs were definitely pale but vessels seemed normal in calibre though the arteries might still be slightly constricted. Just prior to her return to England R.V. = 6/6 full, L.V. = 6/6 full and binocular vision 6/5 (less two).

Discussion

There are several very interesting points about this case. Most writers on quinine amblyopia have emphasised the suddenness of its onset. Often the first indication has been the patient’s complaint that he has suddenly become blind—perhaps immediately after waking from sleep, as in a case in the 1914-18 war described by H. E. Smith. By contrast, in the case now reported the onset was gradual, though the amblyopia was later complete. She first complained of visual symptoms of quinine poisoning on July 13. Quinine therapy was stopped at once but quinine amblyopia was not fully developed until July 21.

The present case also illustrates the variability of the toxic level for quinine. It did not seem to be a case of idiosyncrasy, so much as one in which the toxic level was unusually low. She had only grs. 120 in all. In the 1914-18 war the dosage of quinine was usually grs. 20 intramuscularly thrice daily, yet amongst the large number of patients treated for malaria in Macedonia the incidence of quinine amblyopia was comparatively rare. The practice in naval hospitals in which I served during this war was to give grs. 10 three times daily by mouth alternated with courses of atebrin.

The present concensus of opinion seems to be that quinine acts on the ganglion cells of the retina. But that does not seem to explain satisfactorily the course of events in this patient. She remained completely blind for over one week. Why do central ganglion cells recover and not the peripheral ones? Again she ended up with a lower bitemporal hemianopic defect. If drainage entered into the question one would expect an upper temporal lesion. This simulation of an optic chiasmal lesion has been noted before. Traquair in his book on perimetry mentioned that hemianopic defects may result, and includes some perimetric charts very similar to those in this case. But no explanation of this puzzling feature has yet been suggested.

REFERENCES

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doi: 10.1136/bjo.30.6.345

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