CASE NOTES

AMBLYOPIA DUE TO ETHYL ALCOHOL*

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Case Report

A dairy farmer, aged 42, was referred for admission to hospital by the consultant physician on June 19, 1957. He complained of deterioration of vision in the past 6 weeks and said that for the past 2 weeks he had been unable to read the paper. His visual acuity on admission was 4/60 in the right eye and 1/60 in the left.

He stated that he drank about 8 pints of beer a day, but his wife said that he usually had this amount before breakfast, and that his daily consumption would amount to 2 gallons of beer and cider and sundry spirits.

He stated himself that he smoked approximately 10 cigarettes a day. He said he was in good health. There was no family history of poor vision and he had not apparently been in contact with any poisons.

Examination.—He was a large gaunt man whose clothes hung rather loosely on him. His speech was slow but probably normal. His memory was quite good, although obviously better for distant than for recent events.

Apart from the pupillary reflexes his eyes were considered normal. The optic discs were both of good colour and the maculae within normal limits.

Central Nervous System.—The knee and ankle jerks were absent and there was loss of vibration sense in the lower extremities. There was a slight general weakness of all muscles which were flaccid and poor in tone. Coordination was slow and a coarse tremor of the extended fingers was present. He had marked hyperkeratosis of the hands.

Visual Acuity (21.6.57).—1.5/60 in the right eye and 3/60 in the left with correction.

Visual Fields.—The peripheral fields were full. The central fields are shown in Fig. 1 (opposite).

Laboratory Investigations (19.6.57):

Blood.—Erythrocyte sedimentation rate 36 mm/1 hr; white blood count 7,300/cmm.; total polymorphs 4,700/cmm.; haemoglobin 14.1 g/100 ml. (95 per cent.); packed cell volume 44 per cent. red blood cells; M.C.H.C. 32 per cent.; platelets present; film normal.

Wassermann reaction negative; Kahn test negative.

Cerebrospinal Fluid.—Clear and colourless; pressure 100 mm.; Queckenstedt test normal; white cells nil; red cells nil; protein 135 mg./100 ml.; globulin increased.

Wassermann reaction negative.

Lange count 1100000000.

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FIG. 1.—Central visual fields in June, 1957.

Fractional Test Meal (including histamine test meal).—Within normal limits.

Lumbar Puncture (10.7.57).—Cerebrospinal fluid clear and colourless; pressure 170 mm.; white cells nil; red cells nil; protein 127 mg./100 ml.; globulin increased.

Liver Function Tests (4.7.57).—Within normal limits.

Treatment.—The patient was allowed two small bottles of beer a day. On 27.6.57 he was started on treatment with vitamin B group tablets forte, two three times daily, after meals, intramuscular Cytamen 50 μ g. daily, and vitamin C tablets 200 mg., three times daily, after meals.

On 1.7.57 the patient reported an improvement in visual acuity, and on 3.7.57 the visual acuity was 6/18 with +0.50 D sph. in the right eye, and 6/18 with +0.25 D sph. in the left.

On 3.7.57 he was started on 6 ml. Parenterovite intramuscularly daily which was continued until his discharge from hospital on 19.7.57. The visual acuity continued to improve and on discharge it was 6/12 in the right eye and 6/9 in the left uncorrected.

He was discharged on Multivite tablets, two three times daily after meals. On 24.7.57 the visual fields showed a remarkable improvement (Fig. 2), and the visual acuity was 6/9 in each eye uncorrected.

FIG. 2.—Central visual fields in July, 1957.

Discussion

This case presents several unusual features. It is rare to find a "toxic amblyopia" due to ethyl alcohol in this country. It is usually stated to be associated with another agent, tobacco, in causing a field defect.

Traquair (1949) wrote as follows:

"Neither ethyl alcohol nor methyl alcohol can be credited with special toxic effects upon the visual nerve path. . . . amblyopia traceable to alcohol, apart from tobacco, is almost unknown in British Hospital Clinics and the experience of British ophthalmologists does not support the view that alcohol is a potent factor."

Duke-Elder (1945) stated:

"The condition is always associated with a long history of alcoholism and frequently with peripheral neuritis. A recent deterioration of the physical and mental health enters largely into the aetiology, a matter in which malnutrition, owing to long-standing alcoholic gastro-enteritis usually plays a prominent part".

This is supported by the response to treatment in this case, which, apart from almost complete abstinence from ethyl alcohol, consisted of vitamins and the usual hospital diet.

Bell, Davidson, and Scarborough (1956) state:

"Diets which produce vitamin deficiency disorders lack not only a number of vitamins but may be deficient also in proteins, essential amino acids, and minerals, and it must not be assumed that deficiency disorders such as beri beri and pellagra are due to the lack of a single vitamin. Also there may be variations in the individual response to deficiency, since a diet lacking in vitamins of the B complex may produce beri beri in one and pellagra in another".

The picture in this case, though indirectly due to ethyl alcoholism, appears to be directly due to avitaminosis, and resembles the retrobulbar neuritis seen in prisoners of war in Japanese camps.

The question remains whether ethyl alcohol has any direct toxic effect on the optic nerve; it is quite probable that it has in such extreme cases as this.

The second unusual feature is the rapid response to vitamin therapy. In tobacco amblyopia, return to normal vision frequently takes 6 weeks of abstinence with vitamin therapy. In the present case, after 3 weeks' treatment, the patient's vision was almost within normal range, but although the visual acuity returned to almost normal, he still showed evidence of peripheral neuritis.

The third feature is the raised cerebrospinal fluid protein. This would not appear to be a commonly reported phenomenon in this condition, as I have not been able to find any reference to it. However, Dr. A. M. G. Campbell (personal communication) has seen a high cerebrospinal fluid protein in association with alcoholic consumption, and this may be a common feature of alcoholism. It would appear to be similar to the peripheral oedema found in cases of beri beri, the protein diffusing through the blood-brain barrier of the choroid.

Finally, although the patient was an habitual heavy drinker, the liver function tests were normal. These findings are in accord with the present-day teaching of the effects of alcohol on liver function. The reserve capacity of the liver is such that many tests do not yield abnormal results until the liver tissue is extensively and perhaps irreparably damaged.

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