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Optic atrophy during chlorpromazine therapy

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Chlorpromazine hydrochloride, a synthetic phenothiazine, has been widely employed in psychiatric illnesses, and an extensive literature exists on its side-effects; many reports describe ocular changes, including an oculo-dermal melanosis with deposition of pigment in the retina (Feldman and Frierson, 1964; Zelickson and Zeller, 1964; Cairns, Capoore, and Gregory, 1965; *Brit. med. J.*, 1967), depigmented spots in the fundus (Zelickson and Zeller, 1964), and corneal and scleral pigmentation and lenticular opacities (Greiner and Berry, 1964). Chlorpromazine has not so far been incriminated as a cause of optic atrophy, the only instance of optic atrophy during phenothiazine therapy having been reported in a mentally-retarded girl with epileptic tendencies who was treated with perphenazine and subsequently with large dosage of thioridazine (Bonaccorsi, 1967).

The development of optic atrophy during therapy with large doses of chlorpromazine deserves attention because of the serious and permanent nature of the injury.

Case report

An artist aged 27 was seen in the mental clinic in October, 1967; a diagnosis of catatonic schizophrenia was made, and he was given oral chlorpromazine 500 mg./day in four divided doses. Despite treatment he became withdrawn and intensely negativistic and refused food and drink, and was admitted on November 17, 1967, to the Dacca Medical College Hospital in a state of severe dehydration. Apart from clinical and biochemical evidence of dehydration (*e.g.* hypochloraemia and hyponatraemia and a prerenal azotaemia), no major physical abnormality could be detected. He was found to be negativistic and showed stereotypy and waxy flexibility. He did not respond to questions or commands.

Examination The patient was slightly anaemic. Pulse 120/min.; blood pressure 90/60 mm. Hg. Heart and lungs no abnormality. Liver and spleen not palpable. Skin normal with no changes due to malnutrition. CNS, including cranial nerves and fundi, no abnormality. CSF normal.

Hb 10·4 g./100 ml. Mean cell volume 87 μm^3, eosinophils 3 per cent., monocytes 2 per cent. Erythrocyte sedimentation rate 10 mm./1st hr. Wassermann reaction negative. Electrolytes showed serum sodium 122 mEq/litre, potassium 3·5 mEq/litre, chloride 92 mEq/litre, urea 97 mg./100 ml. Stools and urine no abnormality.

X ray of skull, including the pituitary fossa, reported as normal. *X* ray of chest no abnormality.

Treatment Biochemical correction was rapidly obtained with intravenous fluids and the blood urea came down to 22 mg./100 ml.

He was given nasal feeds and oral chlorpromazine 500 mg./day in divided doses, reduced after a month to 150 mg./day.

Within 5 weeks he was behaving and reacting normally and had developed a voracious appetite.

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Ocular damage He now had no complaints except of photophobia and blurring of vision. He was seen by an ophthalmologist on December 12, 1967, when his pupils were widely dilated and non-reactive. He had no perception of moving objects but some perception of light. Fundoscopy showed complete optic atrophy in both eyes and disseminated retinal pigmentation. There were no pigmentary changes in the media or the sclera. Chlorpromazine was stopped forthwith.

Result At this time the CNS was normal apart from the findings described above. There was no evidence of peripheral neuritis or malnutrition, and the anaemia had improved on iron therapy. There was no history of alcoholism or any family history of blindness.

Discussion

It has not been possible to assign the optic atrophy to any of the known causes including pituitary tumour. The fact that the patient was a recognized and reputed painter before his illness shows that he had had perfect vision. The authors are not aware of any neurological condition or any deficiency or avitaminosis which could involve the optic nerve alone without the other systemic features. Nor is it possible to incriminate a heredofamilial or organic mental illness. In view of his occupation, he was investigated for lead poisoning, but of this we could find no evidence. Nor was there any indication that he had taken any poisonous substances including methyl alcohol. Transient blurring of vision may occur because of the atropine-like action that most phenothiazines are supposed to have. Ocular changes consisting of the deposition of fine particles in the cornea and lens were reported by Mathalone (1967). These patients may also have a dermal melanosis which is seen on the exposed parts of the body and could be a photosensitivity reaction (Cameron, 1967).

Although the exact mechanism of ocular changes, including the optic nerve atrophy described above, cannot be satisfactorily explained, the associated melanosis suggests an enzymatic interference. Toxic amblyopia has also been reported with pheniprazine. The important thing is that in some cases these side-effects are reversible, the determining factor being the duration and amount of therapy. In our case a serious irreversible complication followed a dosage which was not too large by psychiatric standards.

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