

Methanol-Induced Blindness: A Case Report.

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SUMMARY

A case of irreversible blindness in a young adult following methanol ingestion is reported. Forty-eight hours after drinking an unspecified quantity of alcoholic beverage the 21 year old male student experienced sudden visual loss. When seen in our hospital 2 days later each eye of the patient had visual acuity if No Light Perception; dilated, fixed pupils; tortuous retinal veins, A-V nipping, retinal and optic disc oedema. A diagnosis of bilateral optic neuropathy secondary to drug toxicity was made. Treatment could not reverse the blindness. Blindness in this patient is avoidable. This report should alert both clinicians and the public about this unnecessary cause of ocular morbidity. *Niger Med J. Vol. 49, No. 1, Jan. – Mar., 2008: 20 – 21.*

Keywords: methanol, blindness.

INTRODUCTION

Blindness and low vision have many causes. Apart from age-related diseases such as cataract, nutritional deficiency and some drugs including chloroquine, tobacco and alcohol could cause blindness. Ayanru had documented that monotonous diet of cassava and its derivatives was a significant cause of defective vision in Mid-Western Nigeria¹. Recently from Ibadan, Ajayi and Bekibele reported on chloroquine-associated blindness². Although alcohol amblyopia is well known in medical literature³, blindness due to use of alcoholic beverages has not been frequently reported in Nigeria⁴. This article reports on a case of irreversible blindness due to methanol ingestion in a 21 year old male student.

CASE REPORT

A 21 year old male student presented on 16/3/2003 at the Guinness Eye Center Onitsha with a 4 day history of sudden bilateral visual loss; bilateral painful eyes and severe headache. His illness started 48 hours after he drank an unspecified quantity of what he thought to be alcoholic beverage ('hot drink') in a neighbour's house. His neighbour, who offered the drink and also partook of it, died suddenly 24 hours after.

Examination showed a healthy-looking young man with visual acuity of No Light Perception (NLP) in each eye; each pupil was dilated and fixed and the retina had tortuous veins, A-V nipping, peri-papillary retinal oedema and optic disc oedema.

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The haematological and blood chemistry results were normal. A diagnosis of bilateral optic neuropathy secondary to drug toxicity was made. The patient was admitted and treated with iv infusion 5% dextrose-saline solution – 1 liter per 12 hours for 72 hours; iv dexamethasone 8mg 6 hourly for 72 hours; subsequently oral prednisolone 20mg 6 hourly for 11 days. Vitamins C and B complex were also administered orally.

Further history obtained from the mother two weeks after presentation revealed that what the patient drank was 'spirit' – the local name for methanol (methyl alcohol). This time the visual acuity had improved to Count Fingers (CF) at 1 metre in each eye and the pupils were reactive. He was discharged on oral prednisolone 20mg twice daily and vitamins C and B complex. He maintained regular 2 weekly follow up.

However two months after presentation the visual acuity in each had dropped to light perception (LP) only. Each pupil was dilated and fixed; the optic discs were atrophic with the pallor of the left disc being clinically worse. The optic disc and retinal oedema had resolved. He was re-admitted and restarted on intravenous steroids in addition to intravenous vitamin B12 injections, oral vitamins B6, B-complex and C. He was also given oral ibuprofen for retro-ocular pains. At discharge 4 days later the visual acuity in each eye was Hand Motion (HM), the pupils reacted very sluggishly to light. At the last visit one year after the onset of the illness, the visual acuity in each eye was HM.

DISCUSSION

Methyl-alcohol (methanol) is a common industrial solvent that is very readily available in the open markets in Nigeria where it is known as 'spirit'. Being inflammable it is domestically used as priming fuel for cigarette lighters and some lamps known as 'gas lamps'. Locally brewed alcohol ('ogogoro') is also widely consumed in Nigeria. Although the chemical contents of the local brew are not fully known there are no reports of blindness resulting from its use.

Part of the ophthalmic features in our patient was bilateral optic neuropathy which presented as disc edema in the first week of the illness and progressed to marked optic atrophy 2 months after the onset of the illness. Bilateral optic atrophy has been reported as a prominent feature of tropical amblyopia in Nigerian^{1,5}. Tropical nutritional ataxic neuropathy due to chronic cyanide intoxication from prolonged ingestion of cassava products was also associated with optic atrophy⁶. In 1994 optic nerve disease suspected to be due to a combination of chronic consumption of cassava diet and locally brewed rum reached epidemic proportion in Cuba⁷. Although cassava remains a dietary staple and locally brewed alcohol is still consumed freely

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in Nigeria, it is unlikely that our patient's problem was due to a cumulative effect of chronic consumption of alcohol or cassava products.

In recent years adulteration of drugs and alcohol beverages have reached epidemic dimensions in Nigeria. It is however likely that our patient drank alcoholic beverage adulterated with methanol. Methanol is known to cause blindness and death depending on the quantity ingested⁸. It is conceivable that our patient's friend who died has taken a larger quantity of the drink.

A recent report from Germany described a young man who became blind from accidental ingestion of methanol⁸. The patient also developed bilateral optic atrophy. Methanol is destructive to neural tissue and it is a known cause of cerebral infarction as well as optic neuritis⁸. Our patient presented initially with features of optic nerve inflammation which on resolution became atrophic.

In spite of its lethal effect, end organ damage from methanol poisoning could be minimized if specific antidote is administered early. Intravenous ethyl alcohol has been reported to be useful in treatment of methanol intoxication. It acts by displacing methanol from receptor⁸. However the application of the antidote depends on obtaining accurate history of methanol use early and the availability of the drug. Intravenous ethyl alcohol is not available in our hospital and our patient presented late.

In conclusion blindness from the situation embodied in this report is avoidable. The essence of this report is to alert both the clinicians and the public about this unnecessary cause of ocular morbidity.

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