

## Brief communication

# Hooch blindness: a community study report on a few indoor patients of toxic optic neuropathy following consumption of adulterated alcohol in West Bengal

Samanta SK<sup>1</sup>, Fariduddin K<sup>2</sup>, Mahapatra N<sup>3</sup>, Bhunia J<sup>4</sup>, Mondal P<sup>5</sup>

<sup>1</sup> Associate Professor (Ophthalmology), <sup>2-5</sup>Senior Resident, Ophthalmology, <sup>3</sup>Associate Professor (Pediatric Medicine), Calcutta National Medical College and Hospital, 32, Gora Chand Road, Kolkata 700014, West Bengal, India, <sup>4</sup>Senior Ophthalmologist, Central Hospital, S E Railways, Garden Reach, Kolkata, West Bengal, India

### Abstract

**Introduction:** Hooch blindness following consumption of adulterated alcohol has been known for centuries. **Objective:** To study cases of mass alcohol intoxication followed by Hooch blindness in eastern India. **Materials and methods:** Ten patients of toxic amblyopia collected from a community mass intoxication following consumption of adulterated alcohol were studied. The parameters studied were the findings of ocular examination, applanation tonometry, automated perimetry and visual-evoked potential (VEP). As a part of the special investigations fundus photography and OCT were done. Routine blood examination, fasting lipid profile, postprandial blood sugar estimation were done. All patients were treated with injections of methyl prednisolone and Hydroxy cobalamine, antioxidants and local neuro-protective agents. **Results:** Along with diminished vision (from NPL to 3/60), marked pallor of the disc without any other retinal change were noted. The amplitude on VEP was significantly reduced. However, visual improvement (up to 6/18) in 7 patients was observed within 6 weeks of treatment. **Conclusion:** Hooch blindness in India can be prevented by creating awareness among the target population and reducing the cost of country liquor.

**Key-words:** hooch blindness, toxic optic neuropathy, adulterated alcohol

### Introduction

Intoxication along with some side effects following alcohol consumption is known for centuries. But blindness following intake of adulterated alcohol did not get recorded till 1932.

To quote from the words of Mathewson & Alexander in 1932, "FORTY years ago blindness from methyl alcohol was practically- unknown. From that time onward it has become more and more frequent, so that in 1920 Edward Jackson made the statement that methyl alcohol caused as much blindness as all other toxic causes put together". Common agents used for adulteration are methyl alcohol, sugar cane juice, old battery water, heroine, henna, common grass etc. Death following consumption of adulterated alcohol has been reported from time to time from different parts of

Received on: 05.04.2011

Accepted on:09.08.2011

**Address for correspondence:** Dr Swapan Kumar Samanta, MBBS, MS, CCEH (ICEH, London), Associate Professor & Visiting Eye Surgeon, Department of Ophthalmology, Calcutta National Medical College, Kolkata 700014, West Bengal  
Postal address: 267 Abasbari, TAMLUK, Dist. Purva Medinipore, West Bengal  
PIN: 721636  
Tel: 03228266101, 09434023759  
E-mail: swapansamanta53@gmail.com

India (Krishnamurthy, 1968). But mass blindness following alcohol intoxication is rare (Bade et al, 1931). There had been an occurrence of mass poisoning in five villages under Tamluk Police Station, West Bengal, India (about 130 Km south west of Kolkata) from 3<sup>rd</sup> May to 6<sup>th</sup> May 2009 following consumption of alcohol adulterated with unknown poisonous material giving rise to a death toll of around 50, where the total estimated affected population was 600. They all had consumed the said beverage produced from the same source.

### Materials and methods

All the affected people were admitted to a local hospital (District Hospital) and the following treatment was given: IV infusion of 7.5 % sodium bicarbonate, a loading dose of 180 meq, maintenance dose of 180 meq/hr and 2 % absolute alcohol in dextrose (rate 9gm / hr.) with a loading dose 0.6 gm/ kg body weight and the maintenance dose of 66-154 mg/kg/hr. About 198 seriously ill individuals (all were male!) were from a low economic group of the age group of 14 to 80 years. The mean age was 50 (SD= 12) years with the history of consumption of said beverage in varied amount from 50 cc to 800 cc with an average of 300 cc. All of them attended the special Health Check Up Camp along with eye check up facilities organized for this purpose on 13<sup>th</sup> May 2009 at the community hall of the locality (Kanktia, Tamluk, West Bengal).

Ten patients out of 16 grossly visually challenged were admitted to Calcutta National Medical College and Hospital, Kolkata, West Bengal. All the admitted patients were examined carefully and thoroughly by a slit-lamp. Visual acuity (uncorrected, corrected and a pinhole vision) was measured and the fundus was examined by direct and indirect ophthalmoscope and with +90 D lens. Intra-ocular pressure was checked with the applanation tonometer. Visual field was assessed with an automated perimeter. The fundus photograph was taken. Visual evoked potential test was done in all cases to assess the functional condition of the optic nerve.

Systemic evaluation included central and peripheral nervous system was examined. Treatment in every patient was the same which was as: Injection Hydroxycobalamine (1000 mg) x IM stat and after 1 week. Anti-oxidant tablet once daily with local neuro-protective agents were give for 3 weeks. The best corrected visual acuity was tested in all cases during discharge of the patients. All the patients were followed up after six weeks.

### Results

Total 10 patients were specially investigated. All the victims of adulterated alcohol consumption were male (100 %). Median age was 55 years, the youngest one being of 40 years and the oldest of 70 years. By occupation all were daily laborers with average monthly income between of IRs 600 (approximately equivalent to US \$ 11.0). All were chronic alcohol users for more than 10 years. Average daily alcohol consumption was about 300 ml (country liquor). Alcohol was taken with food by 6 patients and without food by 2. The amount of alcohol that was taken on the day of event was at least 300 cc. Fundus examination revealed temporal pallor in 4 (40 %) cases, moderate pallor in 2 (20 %) and marked pallor in 4 (40 %). Applanation tonometry showed a normal intra-ocular pressure (IOP) in all cases. Automated perimetry was normal in 5 (50 %) cases and showed altitudinal visual field defect in 2 (20 %) and could not be done in 3 cases due to poor vision. The VEP was normal in 2 (20 %) cases, showed a prolonged latency period in 4 (40 %) and no wave formation in another 4 (40 %) cases. The OCT study was normal in 4 (40 %) cases, showed thinning of neuro-retinal rim in 4 (40 %) and a slight foveal flattening in 2 (20 %) cases. Triglyceride, Cholesterol, post-prandial blood sugar level, urea and creatinine were within normal limits in all cases.

Visual improvement after 6 weeks was noted as perception of light to counting fingers close to face in 2 patients (20 %), 1 line improvement in 3 (33 %), 2 line improvement in 2 (20 %) and > 2 line improvement in 2 patients (20 %). There was no visual improvement in one patient.

## Discussion

Toxic optic neuropathy is not an irreversible phenomenon. Methanol by itself has low toxicity; however, its metabolites cause most of the toxic effect. Accumulation of formic acid in tissues causes systemic metabolic acidosis and ocular toxicity in patients of methanol poisoning. Visual loss is dependent on the initial dose of methanol ingested and on the interval between ingestion and start of therapy, but the latter is more critical for the outcome. Fundus findings in cases of acute methyl alcohol poisoning vary from peri-papillary edema, hyperemia of optic disc and venous engorgement, to pallor of optic disc, attenuation and sheathing of vessels depending upon the time of presentation after alcohol consumption (Barceloux et al, 2002).

Early investigators had tried spinal fluid exchange therapy, sweat baths, thyroid extracts, adreno-corticotrophin hormone, gastric lavage and alkali treatment without any significant clinical response (Pappas, 1982). Ethanol and fomepizole have been used as specific antidotes to methanol in cases of toxicity (Stelmach et al, 1992). Both the agents block the conversion of methanol to formic acid, which is an active toxic metabolite. Bicarbonate administration and hemodialysis were also used to correct the systemic acidosis caused by accumulation of formic acid (Roe, 1950). Folinic acid is administered to enhance the metabolism of formic acid already produced in the body. These forms of therapy prevent the formation of toxic metabolites and its subsequent catastrophic complications but do not have any significant value in treating established methanol toxicity (Sivilotti et al, 2008). The treatment modalities already mentioned are beneficial in cases of acute toxicity, as the interval between ingestion and start of therapy is more critical to the outcome than the total dose of methanol ingested (Krishnamurthy et al; 1968; Stelmach et al, 1992). In the present study we used inj. hydroxy methyl cobalamine, neuroprotective agents and antioxidants for the treatment of these patients so from the present study it is seen that hydroxy cobalamine and antioxidant has got significant role in the treat-

ment of toxic neuropathy due to methyl alcohol poisoning. Along with the subjective visual improvement, the optic disc also showed decrease in disc edema and clearing of disc margin.

## Conclusion

It may be logical to believe that cases presenting early would develop less manifest primary optic atrophy following treatment. We, therefore, recommend all patients to be treated by this regimen irrespective of the amount of visual debility and the time of presentation following methyl alcohol poisoning.

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Source of support: nil. Conflict of interest: none