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ACUTE METHYL ALCOHOL POISONING

(A review of an outbreak of 89 cases)

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INTRODUCTION

Early in August, 1967 more than 500 cases of acute methyl alcohol poisoning occurred in the City of Madras and the adjoining areas as a result of consumption of adulterated "French Polish". Though addicts have been consuming French Polish for their intoxicating effects due to its ethyl alcohol content no such incident happened before as it was being denatured with Pyridine and Coutchisine. Methyl alcohol, as such, is used only for fouling rectified spirit for industrial purposes in the preparation of paints, brake fluids etc. Methyl alcohol is never used in the preparation of French polish, but, had been done so inadvertently in this instance.

This review is confined to 89 cases, most of them acutely ill, who were admitted and treated in the Government Royapettah Hospital. It was stated that

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*****Asst. Professor of Medicine, Kilpauk Medical College and Asst. to Physician, Government Royapettah Hospital. Madras-14. a few died in their own houses, a few hours after consumption of this adulterated polish. But most of them exhibited symptoms only 12 hours later and were then hospitalised.

MATERIALS

Eighty-nine cases were admitted during the 8 days following the 7th August of which 77 were men and 12 women. Of these 32 cases were admitted in the first 9 hours i.e. the night of 7th and in the subsequent 24 hours 46 more cases were hospitalised and the rest came later. It was found that the maximum incidence occurred in the age group 20-40 years (63 cases). Majority of them were men (77) giving a ratio of male to female as 6:1. A large proportion of these cases were acutely ill and 32 of them died within a few hours after admission. The following vestigations were done wherever possible—CO₂ combining capacity, study of acuity and field of vision and fundus examination. Urine, blood and CSF were also analysed for methanol content in some cases. E.C.G. was taken in cases which showed arrhythmias. From the history and other evidences obtained it was found that the amount of French polish consumed varied from 2 to 6 ozs. They did not complain of vomiting soon after consumption but only 12-24 hours later.

Clinical Features

Majority of the patients complained of vomiting, abdominal pain and various visual disturbances at the time of admission and were found to be cold and clammy. Acutely ill cases developed hypotension, bradycardia, arrhythmias and became markedly acidotic. The terminal event was mostly respiratory arrest, the heart continuing to beat for a much longer time. In a few cases generalized fits preceded death.

Gastro intestinal symptoms

Vomiting (70) and abdominal pain (35) were the commonest symptoms and occurred 12 to 24 hours after consumption of the noxious agent. There was no diarrhoea in any of the cases. Acute abdominal muscle soreness with rigidity was seen in a few cases.

Neurological

Varying grades of neurological disturbances were noted—restlessness, delirium, drowsiness, coma and fits in a few cases. Non-specific signs like brisk or absent tendon jerks were present in 14 cases and 4 had terminal fits. Nuchal rigidity was present in 2 cases. Vocal cord paralysis was observed at the time of intubation. Ocular convergence was defective in two cases. Some of the cases exhibited extreme degree of opisthotonus and high arching of the back.

Visual disturbances

Majority of patients complained of varying degree of visual disturbances like blurred vision of a vague nature and a few described play of scintillating colours before their eyes (red and yellow). Photophobia was met with in 19 cases and was not of great severity. In 79 (89%) of the cases the pupils were

widely dilated and nonreacting. Fundus examination was done on 50 patients and showed hyperaemia of the disc (24), hallor (9), papilitis (3) and was normal in 14. Of the 24 cases with congested discs, dimness of vision was noted in 13, and the rest had normal vision. In the hyperaemic group the field of vision was normal in 10, mildly constricted in 10 and severely constricted in 2. (Fig. 1).

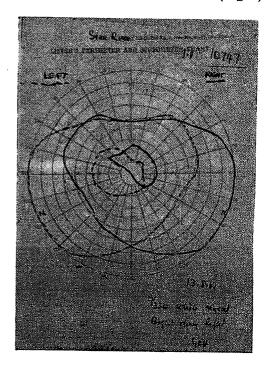


Fig. 1
Field chart showing typical tubular vision in a
case of methanol poisoning.

In 14 patients with normal fundi, 9 had dimness of vision and 5 had restricted field of vision. Appreciable impairment of the acuity of vision was thus noted in 54% with hyperaemic discs and in 36% of the cases with normal discs. Majority (66%) among the 9 cases who had pallor of the disc suffered from defective vision.

Cardiovascular

Varying degrees of fall in the blood pressure was recorded in 35 patients. Different types of arrhythmias were noted in 19 cases (complete heart block in 3, ectopic beats in 7, sinus bradycardia in 7 and diastolic gallop in 2).

Respiratory

In most of the cases (60%) the breathing was acidotic of which 43% succumbed. The clinical examination of the lungs was otherwise noncontributory.

Bio-Chemical Abnormalities

The CO₂ combining capacity of the blood was estimated in 35 patients. The mean value was 37.45 with a range of 15 to 50 vols. %. The lowest CO₂ combining power met with was 15 vol. % and this case recovered. Once the CO₂ combining power estimation was organised and the proper alkalinization given, the mortality came down steeply—i.e. only 4 out of the 35 such cases were lost. Blood urea and sugar were estimated in a number of cases but were found to be within normal limits.

Chemical analysis for methyl alcohol

Stomach wash analysis showed presence of methyl alcohol in 16. The mean value was 8.235 mg. w/v. Urine analysis was positive in 10, the mean value being 0.0351 % w/v. Blood was positive in 8 cases. The mean value was 6.411 % w/v. Methyl alcohol was not detected in the blood, urine and stomach wash at the end of 72 hours after admission. The analysis of the French polish seized by the police showed high percentage of methanol in all the specimens.

Treatment

General measures to resuscitate the patient were carried out in all cases—

such as I.V. administration of fluids, steroids to combat shock, mephentermine and noradrenaline to combat the hypotension, and oxygen. In those who had respiratory arrest endotracheal intubation with oxygen under positive pressure from a Boyle's apparatus was given. A number of hand operated Pulmoflators were also put into service. Out of a total of 29 cases who had respiratory arrest only one could be revived and out of 16 who had cardiac arrest also none survived. Bandaging of the eye was resorted to in cases who complained of photobhobia.

Correction of acidosis

As acidosis was mainly responsible for the clinical ill effects of methanol poisoning 1/6 molar lactate was given intravenously to all patients immediately on admission.

Sodium bicarbonate was given orally in a dosage of 1 g. every 4 hours and after the estimation of the CO₂ combining power was available sodium bicarbonate was also administered intravenously 5—7.5% solution as a slow I.V. drip for those who were in severe acidosis. (CO₂ combining capacity less than 40 vol. %.). Repeated estimation of the CO₂ combining capacity of blood guided the dosage of I.V. sodium bicarbonate administration. This effectively reduced the morbidity and mortality. Moreover the visual disturbances subjective and objective also cleared rapidly.

Ethyl alcohol in small doses was given orally and in a few cases parenterally. Other supportive measures like I.V. glucose was given. Cases which showed retinal oedema with visual disturbances benefited with Cortisone, ACTH, B_1 and B_{12} injection, and the ocular disturbances improved considerably leaving no sequelae.

Mortality

In all 32 patients died in this series (36% of the total admissions). 29 out of 77 males (38%) and 3 out of 12 females (25%) expired. Mortality occurred only in the cases who had consumed over 4 ozs.

Autopsies:

Autopsies were performed in 9 out of 32 fatal cases. There was a general appearance of congestion and petechial haemorrhages in all the organs. The viscera and blood from these cases were sent for analysis for their methyl alcohol content. They are as follows:

shorter the latent period the more severe the toxicity.

Mode of action

The onset of symptoms following a latent period has given the suggestion that effects are due to break down products of methanol in the body—formic acid and presumably formaldehyde. Bennett, et al^1 mention the possibility of methanol specifically depressing the endogenous production of ${\rm CO}_2$ through interference with oxidative enzyme systems particularly those controlling aerobic glycolysis. Thus the peculiar cessation of respiration which characterised

TABLE I

Case No.	Period of survival after admission	Methanol content			
		Stomach	Brain	Liver and kidney	Blood W/V
1.	1 hour	200 mg.	120 mg.	% mg.	0.192%
3.	5 hours	20 "	45 ,,	35 "	0.008%
7.	45 minutes	112 ,,	192 "	120 ,,	0.115%
10.	3 hours	56 ,,	160 "	192 "	0.017%
11.	1 hour	156 ,,	80 ,	trace	0.12 %
15 .	4½ hours	10 ,,	20 ,,	20 mg.	0.02 %
34.	$1\frac{1}{2}$ hours	108 "	140 ,	112 "	
42.	5 hours	30 ,,	50 ,,	15 "	
73.	1½ hours	125 ,,	96 ,,	trace	0.076%

DISCUSSION

The lethal dose of methanol is extremely variable. Bennett, et al^1 observed that as little as 15 ml. of 40% methanol was fatal while as much as 500 ml. had not been so. Generally the latent period after consumption to the onset of symptoms is 24-48 hours though this too is variable as seen in our series. Symptoms were usually ushered by visual disturbances and most of the cases were severely acidotic at the time of hospitalization. In our series unlike that of Bennett, et al^1

death in acutely poisoned cases might well be the result of simple CO₂ deficiency and consequent lack of stimulation of the respiratory centre. Therefore the use of sodium bicarbonate may well represent a specific treatment. In our experience also it has been life saving. Roe² reports that in proportion to its content of iron, retina has greater O₂ consumption than any other normal tissue. Interference by formic acid and the iron containing enzyme may thus reveal the preponderance of ocular defects. When

methyl alcohol is completely metabolized the toxic products are found in the tissues. One of them, formic acid is corrosive in its action and adds to the existing acidosis. Perhaps the possible necrosis of pancreatic tissue may explain the signs of shock, abdominal pain and rigidity of the abdominal wall.

Mode of death

As recognised by previous authors all the patients died of respiratory failure while severely acidotic. Bennett et al.¹ reported that no patient who developed bradycardia survived. In our series out of 10 patients who had this sign 3 survived. We also noticed apparently good risk cases suddenly turned bad and died.

Alkalinization is the main treatment in these cases and even patients with very low CO_2 combining power as 15 or 16 vols. % have turned the corner by I.V. sodium bicarbonate therapy. In none of our cases alkalotic disturbances were noticed.

Ethyl alcohol has been given to inhibit the metabolism of methanol to toxic products. Tracheostomy was not considered necessary. Even though pulmoflators have been used the efficacy of the same could not be assessed. Vitamin B₁ was found to be helpful in some cases probably by aiding the Kreb's cycle. Fortunately in none of our cases there has been ocular or neurological sequelae.

SUMMARY

89 cases were admitted in Government Royapettah Hospital and 32 died in this episode of French polish drinking in which methanol was proved to be the toxic agent. The important features are, gastrointestinal upsets, visual defects, neurological and cardiovascular complications—all symptoms following a lucid period of 12-24 hours after consumption. While no claim for completeness of study is made, symptoms, bio-chemical changes, chemical analysis of gastric, blood and urine specimens, post-mortem findings and treatment with special reference to alkalinization are discussed.

ACKNOWLEDGMENT

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