Occupational Health / Safety in the World

Acute Methemoglobinemia—A Common Occupational Hazard in an Industrial City in Western India

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Abstract: Acute Methemoglobinemia—A Common Occupational Hazard in an Industrial City in Western India: Aruna DEWAN, et al. Poison Information Centre, National Institute of Occupational Health (NIOH)—Cases of acute methemoglobinemia reported to the Poison Information Centre at NIOH over a period of six years from 1993 to 1999 were analyzed. Of 1067 acute poisonings, there were 30 cases of acute methemoglobinemia, the majority of which were due to occupational exposures. The causative agents were mostly aromatic nitro amino compounds used as intermediates in the manufacture of dyes. Diagnosis of acute methemoglobinemia was made on the basis of clinical presentation and occupational history. A particular occupation viz. washing chemical contaminated plastic bags was responsible for many cases of methemoglobinemia. This report highlights the risk of acute poisoning among workers in the small scale chemical industry and also among self employed workers handling toxic chemicals. (*J Occup Health 2001; 43: 168–171*)

Key words: Methemoglobinemia, Acute poisoning, Small scale industry, Dye industry, Aromatic nitro amino compounds, Self employed workers

Acute methemoglobinemia often presents as a medical emergency requiring immediate treatment¹⁾. Signs and symptoms of methemoglobinemia reflect the quantity of methemoglobin present in blood and are due to the decreased oxygen carrying capacity of blood. The symptoms include slate gray cyanosis, headache, lethargy, dizziness, fatigue, syncope, dyspnoea, CNS depression, seizures, arrhythmia and shock. Patients with underlying medical conditions such as COPD, anemia or coronary artery disease are more susceptible to lower concentrations of methemoglobin. When the level of methemoglobin exceeds 50%, tissue hypoxia leads to

metabolic acidosis worsening the clinical picture²⁾.

Acute methemoglobinemia can be hereditary but most often it is acquired through exposure to a variety of chemicals and drugs, among which nitrites and aniline derivatives have been reported to be the commonest agents²⁾. Most of the chemicals involved are used either as dye intermediates or reducing agents in industrial processes. Sodium nitrite is used as a bleaching agent in the industry and because of its use as a meat preservative and similarity in appearance to table salt, it is frequently the cause of methemoglobinemia associated with contamination of food³⁾. Nitrites are also present in medicinal agents such as amyl nitrite, nitroglycerine and spirits of nitrite. Aniline and its derivatives are found in many household products such as inks, shoe polishes, paints and varnishes and is also an important industrial solvent. Nitrobenzene is a detergent-intermediate and also used in polishes. Poisoning by pure aniline or nitrobenzene is rare except in the chemical industry⁴⁾. Dinitrobenzene is a dye-intermediate and camphor substitute in celluloid production. Most of the time, acquired methemoglobinemia is reported to be due to accidental^{5–12)}, or suicidal^{13, 14)} causes and occupational methemoglobinemia is reported infrequently^{15, 16)}.

Interestingly, occupational exposures were encountered as the main cause of acquired methemoglobinemia among cases referred to the NIOH Poison Information Centre. Most of the cases were either self-employed persons or workers in small scale industry where hygiene standards are rarely observed. Self-employed workers are not covered by any occupational health and safety laws.

Materials and Methods

A Poison Information Centre was established at the National Institute of Occupational Health in 1993 with the technical collaboration of the International Programme on Chemical Safety (IPCS). The centre provides expert advice to medical personnel who manage acute poisoning cases due to various agents such as pesticides, industrial and household chemicals, and drugs.

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Laboratory support is also provided in pesticide and metal poisoning cases and this helps to reduce morbidity and mortality from toxic exposures. The mode of imparting information and consultation is mostly by telephone. During the communication, all possible details of the patient and poisoning incident are recorded and cases are followed to determine the outcome. Recorded data is periodically analyzed to determine regional trends and to suggest preventive measures whenever necessary. This toxico-vigilance function of the centre is all the more important when poisonings are unintentional. In the present report, cases of acute methemoglobinemia have been analyzed from the data recorded over a period of 6 yr from April 1993 to March 1999 to determine the causative agents.

Results

Over a period of 6 yr, a total of 1067 cases of acute poisoning were reported to the NIOH Poison Centre. The commonest agents responsible for poisoning were pesticides (68.4%) and others were chemicals (10%), drugs (8.3%), mixed poisoning (5.1%), plants (1%) and in 7.2% of cases the causative agent could not be identified. In 85.1% of cases the reason for poisoning was intentional (suicidal). In the above series, 30 cases of acute methemoglobinemia were reported in 16 incidents of which five episodes involved more than one

person (Table 1). All except one were young healthy adults of the 18–30 yr age-group. Methemoglobinemia was due to occupational exposure in 25 (83.3%) cases, accidental in 4 (3.3%) cases and only one case was intentional (3.3%). In the accidental group, one case of accidental poisoning happened to a two-yr old male child who was mistakenly fed nitrobenzene stored in a bottle of vitamin tonic. In the other episode, three workers residing within factory premises developed methemoglobinemia when they inadvertently used sodium nitrite in place of common salt on their food. In most of the episodes, the agents responsible for methemoglobinemia could not be identified but they are likely to be aromatic nitro- amino- compounds or nitrites as indicated by the occupational history. A particular occupational exposure was encountered in five episodes where methemoglobinemia occurred among self-employed workers while washing large plastic bags discarded by some local dye enterprises. The last episode occurred among seven workers in an industry manufacturing m-phenylene diamine from dinitrobenzene. Toxic exposure occurred during the task of removing iron sludge contaminated with dinitrobenzene and metaphenylene diamine and other intermediates without using any protective devices. The victims developed signs and symptoms of methemoglobinemia a few hours after finishing work and

Table 1. Details of patients presenting with Methemoglobinemia

Episode No.	No. of persons	Sex	Reason	Agent	Occupation	Outcome
1	1	M	O	N.I.	Fertilizer factory	R
2	1	M	O	N.I.	Dye factory	R
3	1	M	O	Aniline	Dye factory	U
4	1	M	O	N.I.	Dye factory	R
5	1	M	O	N.I.	Dye factory	R
6	1	MC	A	Nitrobenzene	—	R
7	1	M	O	p-anisidine	Dye factory	U
8	3	M	A	Sodium nitrite taken in place of common salt in food	Chemical factory	R
9	1	M	S	Paint solvent	Painter	U
10	5	F	O	N.I.	Plastic bag cleaning	R
11	1	M	O	p-chloroaniline	Dye factory	R
12	2	M	O	N.I.	Plastic bag cleaning	R
13	2	M	O	N.I.	Plastic bag cleaning	R
14	1	M	O	N.I.	Plastic bag cleaning	R
15	1	M	O	N.I.	Plastic bag cleaning	R
16	7	M	O	Metaphenylene diamine and Dinitrobenzene	Dye factory	3 dead 4 R
Total	30	—	—	—	—	—

M=Male, F=Female, MC=Male Child, O=Occupational, A=Accidental, S=Suicidal, N.I.=Not Identified, R=Recovered, U=Unknown.

three of the workers died within three days of exposure. Methemoglobin estimations could not be carried out as this facility was not available.

Discussion

There are many reports from developed countries which indicate that the commonest causes of acquired methemoglobinemia are drugs¹⁷⁻¹⁹⁾ cosmetics^{10, 20)} or substances of abuse^{21, 22)}. There are very few reports of occupational methemoglobinemia from these countries^{15, 16)} which may be due to better regulation of manufacturing processes.

In India, the chemical industry is growing rapidly and is highly concentrated in the western part, mainly in the states of Gujarat and Maharashtra. Most of the enterprises are small in scale. Ahmedabad, the largest city in the state of Gujarat, is surrounded by a large number of industrial units manufacturing dyes and dye intermediates. Workers in these units are at high risk of developing acute methemoglobinemia due to multiple reasons viz. lack of awareness, non-availability of protective devices and poor work practices. In spite of this, there is paucity of any documented data on occupationally caused methemoglobinemia from India. One reason of for this could be a lack of reporting since the majority of industrial poisoning cases are treated by private practitioners and only severe cases seek admission to state run hospitals. Another reason could be the absence of Poison Information Centres where general physicians can seek help in the case of poisoning emergencies. Such centres are just being started in India and one such center has been established at the National Institute of Occupational Health in Ahmedabad.

The cases referred to in this paper presented at different hospitals in the city which in turn consulted the center for guidance. The signs and symptoms reported were: slate gray cyanosis, altered sensorium, dizziness and vomiting. Occupational history and staining of exposed parts and clothes with different coloured dyes were some of the important factors in establishing the diagnosis. Since diagnostic facilities for methemoglobin estimation were not available, the resident doctors were advised to carry out a simple bedside test involving the comparison of colours between patient's blood and the normal blood on a filter paper. The blood in methemoglobinemia cases appeared chocolate brown in colour compared to normal blood²³⁾. Since all cases had signs of hypoxia besides cyanosis, it is expected that their methemoglobin levels must have been above 30%. All cases were given methylene blue at a dose of 1–2 mg/kg (i.v.) which has been suggested as the drug of choice for treating severe methemoglobinemia^{24, 25)}. Sometimes methylene blue elicits late Heinz body anemia 8–10 h after the treatment even when methemoglobin is strongly reduced. In our series, we were not able to determine the occurrence of

these complications.

The present report points to the risk of acute poisoning due to unsafe handling and disposal of chemicals and also highlights the significance of dermal absorption in acute occupational exposures. Chronic exposures to methemoglobin forming agents are also likely as most of these workers go back to the same jobs. The long term haematological effects of such exposures need further investigation because methemoglobin inducers may cause denaturation of hemoglobin resulting in hemolytic anemia²⁶⁾ and anemic persons are more prone to the effects of methemoglobinemia. This is very relevant to third world countries where anemia is common due to malnutrition and parasitic infestations.

Our personal experience shows that there is gross under reporting of industrial poisonings due to compensation issues and legal procedures. Added to this, the poisoning cases reported to our center are purely voluntary. Therefore, the actual magnitude of the problem is likely to be many times higher than that reflected by the small number of cases reported in this paper. However, this paper is a definite pointer to poor work practices and the high risk of hazardous chemical exposures in unorganized and self-employed work sectors.

To prevent such toxic workplace exposures, it is imperative to create awareness among workers and employers as well as exercise strict regulatory controls. The use of protective devices and good hygiene practices and periodic monitoring of workers for sub-clinical exposure needs to be encouraged. Occupational health and safety needs of self-employed workers have to be attended to which may even need amendment of existing labour-laws in this country. There is also an urgent need for establishing a network of regional poison information centers.

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