GAS POISONING WITH FREON-12
(A Report of Three Cases)

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ABSTRACT

Three patients of accidental gas poisoning with Freon-12 are reported. Presenting features were transient loss of consciousness, bradycardia, hypotension and anaesthesia. All patients made a rapid and uneventful recovery without any residual effects.

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KEY WORDS :Gas poisoning; Freon-12

Introduction

Freons are a group of aliphatic halogenated hydrocarbon gases which exist in several forms (Table 1). They are colourless, odourless, and noninflammable gases which are in commercial use as referigerants, in fire extinguishers and as propellants in nebulizers, aerosol bombs, insecticides, deodorants etc [1,2]. Given their extensive industrial applications, there are however few reports in literature about their systemic or long term effects in humans. We report our experience of managing three cases of accidental exposure to Freon-12 gas.

TABLE 1

Structure of Freon Gases

Nomenclature	Chemical structure
Freon = 12	Dichlorodifluoromethane
Freon = 11	Trichloromonofluoromethane
Freon = 114	Dichlorotetrafluoroethane
Freon = 22	Monochlorodifluoromethane

CASE REPORTS

Three patients aged 32, 24 and 23 years respectively, accidentally inhaled Freon 12 gas which had leaked from an air conditioning system of a ship. The patients had been entrapped, in a small unventilated area for approximately 5-10 mins. They were admitted to our hospital half an hour later.

Transient loss of consciousness at onset lasted for 5 to 10 mins in two patients. On regaining consciousness, they complained of mild headache, confusion, inability to open mouth and complete loss of sensations, and had completely recovered within two hours. The third patient remained comatose for three hours (Glasgow Coma Scale score - 8), with normal sized, photoresponsive pupils and intact corneal reflex. He showed evidence of impending respiratory depression, and was given 60 mg of Doxapram, administered intravenously as a trial. This resulted in dramatic recovery, obviating the requirement of ventilatory assistance, which had been kept prepared.

Symptoms reported by the patients are shown in Table 2. There was no history of lacrimation, excessive salivation or irritation of eyes, nostrils, throat or skin.

TABLE 2

Symptomatology

Symptoms	Duration		
	First patient	Second patient	Third patient
Unconsciousness	10 m	15 m	3 h
Confusion	40 m	15 m	4 h
Headache	1 h	1 h	8 h
Lockjaw	6 h	8 h	24 h
Sensory loss	<1 h	<1 h	8 h

NOTE : m = minutes, h = hours

On clinical examination all patients were found to have bradycardia, hypotension (systolic pressure 84 mm to 96 mm Hg) and

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shallow breathing. Additional findings in the third patient were Cheyne-Stokes respiration, cyanosis, and cold, clammy extremities. No arrythmias were detected in any patient. Table 3 elucidates the signs seen in our patients.

TABLE 3

Clinical signs

Sign	First patient	Second patient	Third patient
Tremors	—	—	+
Trismus	+	+	+
Analgesia	+	+	+
Corneal reflex	+	+	+
Deep tendon jerks	Brisk	Brisk	Absent
Plantar response	Flexor	Flexor	Equivocal
Heart rate	62/min	58/min	55/min
Systolic blood pressure	96 mm Hg	92 mm Hg	84 mm Hg
Respiration	Shallow	Shallow	Cheyne-Stokes
Lungs	Clear	Clear	Clear

Laboratory investigations carried out included haematocrit, urine analysis, metabolic and biochemical parameters, these were all normal. Analysis of arterial blood gases was done on admission and thereafter six-hourly. The third patient had hypercapnia (pCO_2 = 66 mm Hg) and acidemia (pH 7.2). Serial ECGs were taken hourly for the first twelve hours and then every three hours, for the next twelve hours. Sinus bradycardia was present in the initial ECGs, the subsequent ECGs were normal. Chest X-ray done on day 1,2,3 and 7 were all normal.

Treatment instituted in all patients included close monitoring, frequent clinical assessment, continuous inhalation of 100% oxygen and intravenous hydrocortisone. Prophylactic broadspectrum antibiotics were given initially to combat possible chemical pneumonitis (and secondary bacterial infection). All patients recovered completely within 24 hours, but were observed for delayed effects as inpatients for a week. The second patient developed bilateral pleural rub on the third day, which disappeared over the next 36 hours. Follow up of the patients a fortnight later showed them to be symptom free with no residual signs.

Discussion

In the past, Freons were generally believed to be inert gases with low toxicity. Following a spate of sudden sniffing deaths in 1960, there was a renewed interest in the dangers of the sniffing fad (then prevalent amongst youngsters). This occurred due to inhalation of Freons from aerosol containers intended for frosting cocktail glasses. Over 170 cases of deaths due to Freons have so far been reported world wide [1,2].

Bass [3] reviewed the records of 110 sniffing deaths and inferred that sudden death was as a result of severe sudden cardiac arrhythmias. Subsequent experiments conducted on rodents suggested that the observed effects were possibly as a result of direct action on sino-atrial node and atrioventricular conduction. Taylor and Harris [4] demonstrated that Freons cause a reduction in sinoatrial rate, prolongation of P-R interval, fall in the T-wave amplitude and occasionally second or third degree heart blocks. They further concluded that sudden death in asthmatics was due to the cardio toxic effects of the propellant. Freons being heavier than air, lodge in the alveoli of asthmatics for a long time because of expiratory obstruction. None of our patients had bronchial asthma. Apart from slight bradycardia, no other abnormal electrocardiographic finding was evident in our patients. Sayers *et al* [5] showed that dogs and monkeys developed generalized tremors, salivation and lacrimation when exposed to concentrations above 20% volume in air. Tremors were only present in the third patient. Lester and Greenberg [6], demonstrated twitching and tremors at concentrations of 30-40%, and loss of reflexes at 50% in rats. At concentrations of 70-80%, corneal reflexes were abolished and the animals were in deep anaesthesia. Transient anaesthesia was a feature in all of our patients. The third patient also had absence of response to deep painful stimuli, with intact pupillary and corneal reflexes.

These findings are similar to those seen in the early planes of third stage of anaesthesia.

Although deaths due to Freon gas poisoning have been reported, we did not find any reference relating to documented histopathological changes in the tissues. Morita *et al* [7], have however reported the existence of fat droplets in hepatocytes of the mice exposed to Freon gas.

Very little is known of the immediate and long term toxic effects of Freons on humans. Our experience and observations could possibly be the first report of its kind in India, and in conjunction with other future reports should contribute to our knowledge of the toxic effects of these universally used halogenated hydrocarbons.

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