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Chlorine-induced anosmia. A case presentation

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Abstract
Chlorine gas is known to be a potent irritant to the eyes, nose, throat and lungs with severe exposure resulting in pulmonary oedema and even death. There have been no case reports, however, of chlorine inducing anosmia. We present such a case, along with a review of the literature on the toxicology of chlorine and its effect on the nose.

Key words: Chlorine; Anosmia; Smell

Introduction

Gas! Gas! Quick boys! – An ecstasy of fumbling
Fitting the clumsy helmets just in time,
But someone still was yelling out and stumbling
And flound'ring like a man in fire or lime –
Dim through the misty panes and thick green light,
As under a green sea, I saw him drowning.
'*Dulce et Decorum est.*' (Wilfred Owen 1921)

Chlorine gas was first used in chemical warfare in Ypres on 17 April 1915 due to its properties as a known respiratory irritant causing coughing, choking, hypoxia and pulmonary oedema. It is reported as being a potent irritant to the eyes, skin and mucous membranes including the nose (National Poisons Information Service). However, we have found no cases of permanent anosmia due to chlorine exposure in the literature. We present a case of chlorine-induced anosmia from an industrial accident.

Case study

A 62-year-old, previously healthy, engineer accidentally poured a drum of sodium hypochlorite into a tank of hydrochloric acid. The chemical reaction which ensued released copious amounts of chlorine gas i.e.:

$$NaOCl\,(aq) + 2HCl\,(aq) \rightarrow Cl_2\,(g) + H_2O\,(1) + NaCl\,(aq).$$

The patient inhaled three to four breaths over twenty seconds, then felt his nasal passages become blocked. He then found it difficult to breathe through his mouth and developed acute dyspnoea and pains in his chest.

He was admitted via Accident and Emergency where a diagnosis of pulmonary oedema secondary to chlorine gas inhalation was made. He was treated with oral prednisolone (60 mg decreased over three weeks) and was discharged from hospital four days later.

After discharge he became aware of his complete lack of smell and reduced sensation of taste. At three months following the incident he was seen in the ENT clinic, when it was noted that he had complete anosmia on testing. His nasal airways were clear, the nasal mucosa looked healthy and there was no evidence of nasal polyps.

At a two year follow-up, he still complained of difficulty breathing on exertion, and still had complete lack of smell. On formal testing he was unable to smell or identify any of the following odours (afferent nerve stimulated):

Methanol	(olfactory and trigeminal),	Coffee	(olfactory),
Acetic acid	(olfactory and trigeminal),	Onion	(olfactory),
Peppermint	(olfactory and trigeminal),	Clove	(olfactory),
Acetone	(olfactory and trigeminal),	Vanilla	(olfactory),
Cinnamon	(olfactory),	Eucalyptus	(olfactory).

Discussion

Anosmia leads to a variety of problems ranging from an inability to smell dangerous fumes such as domestic gas leaks to the more trivial inability to judge the amount of perfume to be worn.

Henkin (1982) showed that 66 per cent of Americans had a period of reduced olfactory acuity at some time in their lives, which translates to a figure of about 16 million people. Four per cent of these cases were due to chemical exposure. Various chemicals have been reported in the past as being responsible for causing anosmia. These include, cadmium (Rose *et al.*, 1992), chromium (Watanabe and Fukuchi, 1981), sulphur dioxide and ammonia (Harada *et al.*, 1983), petroleum fumes (Latkowski *et al.*, 1981) and carbon monoxide (Hansen, 1970).

Chlorine is listed in various health and safety documents as causing a burning sensation in the nose (Trevethick, 1980), irritation of the mucous membranes of the respiratory tract and eyes (Occupational Exposure Limits, 1993) and cough, retrosternal discomfort, hoarseness, dyspnoea and stridor (National Poisons Information Service), but we have not found anosmia reported.

As previously quoted, chlorine was one of the first gases used in chemical warfare in the First World War. As well as the known respiratory symptoms, it was also seen to cause furring of the tongue, headaches, 'nervous insomnia' and morning vomiting (Sandall, 1922). There have been several large industrial chlorine accidents reported in the literature (Ramachandran *et al.*, 1990; Schwartz *et al.*, 1990). Patients complained of unproductive cough, oropharyngeal pruritis, burning of the eyes, nose and throat and there were several cases of pulmonary oedema, but there were no reports of anosmia.

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A connection between anosmia and chlorine has been made in animal toxicology studies. Wolf *et al.* (1995) showed that chlorine exposure-dependent lesions in rats and mice were confined to the nose. They observed vestibulitis and epithelial metaplasia of the squamous, transitional and respiratory epithelia of the nose and specific effects on the olfactory epithelium. Here they found an accumulation of eosinophilic proteinaceous deposits with variable loss of the sensory cells. This was most prevalent in the anterior extension of the olfactory epithelium extending into the dorsal meatus, and it was thought this corresponded to a concentration gradient of chlorine in the nose. Similar work by Jiang *et al.* (1983) illustrated, by light and scanning electron microscopy, a partial to complete degeneration of olfactory cells with evidence of exfoliation, erosion and squamous metaplasia. A possible mechanism for this cellular injury caused by chlorine was suggested by McNulty *et al.* (1983). They suggested that as the chlorine dissolves in the cellular water (of the nasal epithelium), the two stage reaction (in fact an equilibrium of the two) occurs producing hydrochloric acid and hypochlorous acid which then breaks down to release nascent oxygen or possibly oxygen free radicals. It is the oxygen that is believed to have the cytopathological effect.

Shroff *et al.* (1988) quote the same mechanism in the causation of acute inflammation of the conjunctivae, nose, pharynx, larynx, trachea and bronchi in humans following chlorine exposure. Surprisingly, Small and Murray (1987) showed that swimming in chlorinated water has no effect on nasal function nor does it cause nasal irritation where you would expect the above reaction to occur.

Our case demonstrates that anosmia can occur from chlorine exposure. Olfactory mucosa of experimental animals has been shown to be severely affected by chlorine and a possible mechanism has been suggested.

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