The Journal of Laryngology and Otology
July 1996, Vol. **110**, pp. 663–664

Clinical Records

A complication of indoor pistol shooting

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Abstract
The aetiology of chronic rhinitis and nasal obstruction is often elusive and obscure. Many different causes have been documented including allergens, cigarette smoke, cold dry air, viruses, sulphur dioxide and industrial pollutants. This is the first case we have been able to identify of rhinitis associated with exposure to high levels of lead through indoor pistol shooting.

Key words: Rhinitis; Lead, toxicity.

Case report

A previously fit 37-year-old car mechanic presented to the Accident and Emergency department with a two-month history of bilateral rhinorrhoea, headache and facial pain which had become intolerable over the preceding three hours after a session of pistol shooting. A provisional diagnosis of rhinitis with sinusitis was made and he was immediately referred for an ENT opinion.

On further questioning he complained of bilateral hip pains, abdominal cramps and poor concentration at work occurring over the previous four months. For six years he had been participating in indoor pistol shooting in an unventilated range. Over the last four months he had increased his shooting from 150 to 600 rounds of cast ammunition three times a week. He also mentioned that a friend who also participated in indoor shooting had complained of a similar illness which was thought to be due to excessive lead exposure.

On nasal examination there was a mild degree of mucosal inflammation typical of rhinitis. A computed tomography (CT) scan was arranged along with a full blood count, liver function tests, serum lead and zinc protophorphyrin levels. The patient was told that his symptoms might be attributable to his shooting and should improve with the nasal decongestants he was prescribed. An eight week follow-up appointment was made and he was advised to stop shooting in the meantime.

At follow-up the patient said that he had been unable to stop shooting but he had used a facial respirator to protect his nasal and oral airway from inhalation of particulates. He had noticed a remarkable improvement in his symptoms. Blood results confirmed excessive lead exposure (serum lead 2.1 micromols/l, normal <1.5 micromols/l, zinc protoporphyrin (ZPP) 418 nanomols/l, normal <500 nanomols/l). Repeated serum lead levels at follow-up were also suggestive of chronic exposure to lead (Serum lead 1.5 micromols/l, ZPP 1084 nanomols/l). Liver function tests and a full blood count were normal, with no evidence of basophilic stippling. His CT scan showed osteomeatal complex disease and thickened ethmoidal cells to account for his nasal symptoms.

Discussion

Exposure to lead in indoor firing ranges occurs secondary to inhalation of suspended lead particulates. The primary source of lead is produced when it is sheared off as the bullet passes through the weapon and as it hits the target. It also originates from the ignition of primer material containing lead azide or styphnate within the percussion cap (Valway *et al.*, 1989).

Lead toxicity may present with many systemic symptoms making diagnosis difficult. Symptoms such as irritability, nausea, abdominal and joint pains, lethargy and muscle weakness are well recognized. Rhinitis has never been previously reported. Analysis of blood lead is the measurement of choice for assessment of lead toxicity. Assays of ZPP in blood correlate well with blood lead levels (Grandjean and Lintrup, 1978). Lead toxicity can cause anaemia, reticulocytosis and basophilic stippling. However, basophilic stippling may not be present in peripheral blood but up to 50 times more lead may be bound to erythrocytes in the bone marrow (Albahary, 1979).

Lead toxicity in indoor ranges has been recognized as a hazard since 1979 (Fischbein *et al.*, 1979; Ozonoff, 1994), suggesting that preventative ventilation of indoor ranges may still be inadequate. In our case a causal relationship between lead toxicity and rhinitis cannot be confirmed, due to other nasal mucosal irritants that may have arisen from the gun smoke and at work. Other compounds generated from gun powder include nitrogen, hydrogen, carbon dioxide and complex organic compounds of nitrogen (Fischbein *et al.*, 1979) but their role in the aetiology of rhinitis is not well documented (Ballenger, 1991). Other possible sources of high exposure to lead in this particular case could have been from tetraethyl lead from car exhaust fumes or the lead particles suspended from sanding or

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Accepted for publication: 7 March 1996.

grinding car bodywork.

It is interesting that no preventative ventilation measures nor monitoring of excessive lead within the air had been undertaken at this particular indoor shooting arena. Therefore, in arenas considered at risk there should be a strict government legislation on ventilation systems. There should also be regular surveillance of atmospheric lead levels within arenas. If ventilation is proven to be inadequate with correspondingly high atmospheric lead levels then periodic serum lead and ZPP levels should be undertaken along with improved ventilation. In addition, airway protection should be mandatory in poorly ventilated arenas. In the United Kingdom, the Health and Safety Executive places a duty on employers which extends to the protection of persons at the place concerned and to those who are not employees. Nevertheless, there appears to be no strict surveillance of recreational activities that put people or employees (e.g. the police) at risk of lead toxicity even though there should be a good standard of ventilation in indoor ranges (H.M.S.O., 1985).

References

Albahary, C. (1979) Lead and hemopoiesis. *American Journal of Medicine* **52:** 367–378.

Ballenger, J. J. (1991) Chronic rhinitis and nasal obstruction. In *Diseases of Nose, Throat, Ear, Head and Neck* (Ballenger, J. J., ed.), Lea and Febiger, Philadelphia, USA, pp 140–142.

Fischbein, A., Rice, C., Sarkozi, L., Kon, S. H., Petrocci, M., Selikoff, I. J. (1979) Exposure to lead in firing ranges. *Journal of the American Medical Association* **241:** 1141–1144.

Grandjean, P., Lintrup, J. (1978) Erythrocyte Zn-protoporphyrin as an indicator of lead exposure. *Scandinavian Journal of Laboratory Investigation* **38:** 669–675.

H.M.S.O. (1985) *Approved Code of Practice Control of Lead at Work.* 0-11-883780X piii.

Ozonoff, D. (1994) Lead on the range. *Lancet* **343:** 6–7.

Valway, S. E., Martyny, J. W., Miller, J. R., Cook, M., Mangione, E. (1989) Lead absorption in indoor firing range users. *American Journal of Public Health* **79:** 1029–1032.

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