‘Numismatist’s pneumonitis.’ A case of acute nitrogen dioxide poisoning

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Summary: A case of acute nitrogen dioxide toxicity is described, together with its management and a review of the possible complications.

Introduction

Acute nitrogen dioxide (NO_2) poisoning has been described in many occupational settings; in farmers, as silo-fillers disease (Lowry & Schuman, 1956); oxyacetylene welders, miners, chemical workers (Jones *et al.*, 1973), missile workers (Yockey *et al.*, 1980) and firemen (Tse & Bockman, 1970). We report what we believe to be the first case of acute NO_2 toxicity in a coin collector.

Case report

A 77 year old retired building foreman and ex-50/day cigarette smoker with a history of mild chronic obstructive airways disease was cleaning some pre-decimal pennies at home, using a mixture of 50% concentrated nitric acid and 50% water in a tray. He accidentally dropped all the coins into the mixture at the same time. A vigorous reaction ensued, and clouds of a brown-coloured gas were given off. He picked up the tray, carried it slowly to the bathroom, and poured the liquid contents away. He described an attack of coughing at the time, productive of a small amount of brown sputum. On retiring to bed he was unable to sleep, and 2 h later had rigors, nausea and dyspnoea on slight exertion.

Twelve hours after the incident he arrived in the casualty department, where he was found to be anxious, dyspnoeic at rest and mildly cyanosed. His pulse was 100 beats/min, regular, and his blood pressure was 140/80 mm Hg. He was afebrile, with a respiratory rate of 36/min and bilateral inspiratory crackles at both bases. Investigations showed a total white cell count of $21.3 \times 10^9/\text{l}$ with 86% neutrophils, and normal blood urea and electrolytes. Blood gases

showed a PO_2 of 6.3 kPa and a PCO_2 of 5.3 kPa. The chest X-ray (Figure 1) revealed gross pulmonary oedema, and the electrocardiograph showed a sinus tachycardia. The sputum showed pus cells but no pathogens, and lung function tests showed a pattern consistent with severe fixed airflow obstruction, with a low carbon monoxide transfer factor.

He was treated with intravenous frusemide, and oxygen. On transfer to the ward, oxygen was continued, first at 60%, and then at 28%. The following day he was started on co-trimoxazole as his sputum was purulent, and on the sixth day he was started on prednisolone, 60 mg/d which was tailed off over the following 8 weeks.

Over the next few days he made a good clinical recovery, accompanied by rapid clearing of the chest X-ray (Figure 2), with improvement of his blood gases and lung function tests (Table I). He made an uncomplicated recovery, and 16 months later has normal

Figure 1 Chest X-ray on day of admission showing severe pulmonary oedema.

Figure 2 Chest X-ray 6 d post-admission showing substantial clearing of oedema.

exercise tolerance. His lung function tests continue to show a mildly obstructive picture, presumably reflecting his previous chronic bronchitis.

Discussion

Pre-decimal pennies consist of 95.5–97% copper by weight, the remainder being tin and zinc. The copper reacts with 50% concentrated nitric acid to form copper nitrate and nitrogen dioxide:

Table I Shows progress of blood gas results and lung function tests for 40 d after the acute injury

	Days post-admission								
	0	1	3	5	9	19	26	33	40
PO ₂ (kPa)	6.3	6.4	7.3	9.3	9.8	10.1	10.4	9.6	10.9
PCO ₂ (kPa)	5.3	5.6	5.5	5.1	4.8	4.6	5.3	5.6	5.5
FEV ₁ (l)		0.7			1.6		1.8	1.9	1.7
VC (l)		1.3			2.9		3.4	3.1	3.2
FEV ₁ /VC (%)		57			55		53	61	53
CO transfer factor		10.9			24.9		26.4	25.8	25.3

FEV₁ = Forced expiratory volume in 1 s; VC = vital capacity; CO = carbon monoxide. Predicted values for a man of his height and weight are as follows: FEV₁, 2.4 litres; VC, 3.7 litres and CO transfer factor, 22.9.

The syndrome following acute high-dose NO₂ inhalation is uncommon. An initial bout of coughing is followed, 3–36 h later, by dyspnoea, cyanosis, tachycardia and severe pulmonary oedema, due to NO₂ dissolving in the small airways to form nitric and nitrous acids which cause a chemical pneumonitis (Prys-Roberts, 1967; McAdams, 1955). A polymorphonuclear leucocytosis is usually present (Jones *et al.*, 1973). If recovery from this phase occurs, the patient may present 4–6 weeks later with a cough, cyanosis and severe dyspnoea due to bronchiolitis obliterans, which may be fatal (Yockey *et al.*, 1980; Tse & Bockman, 1970; McAdams, 1955).

The pulmonary oedema phase is managed with diuretics and oxygen therapy, but in addition most authors advocate a broad-spectrum antibiotic to prevent secondary chest infection, and steroids should be given in an attempt to prevent bronchiolitis obliterans (Jones *et al.*, 1973; Yockey *et al.*, 1980; Tse & Bockman, 1970; Prys-Roberts, 1967).

We would like to point out that, firstly, few serious coin collectors would clean their coins, and, secondly, this case illustrates the fact that even the most sedentary activities are not without their potential medical hazards.

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