Severe coughing and pneumoperitoneum

Coughing and straining have been suggested as contributing to the formation of subcutaneous emphysema, mediastinal emphysema, and pneumothorax in adults and children with and without underlying lung disease.¹⁻⁴ Severe and protracted coughing, however, has not previously been associated with pneumoperitoneum. I describe a patient with subcutaneous emphysema, mediastinal emphysema, and pneumoperitoneum in whom coughing may have had a causative role.

Case report

A 23-year-old man presented to casualty complaining of feeling wheezy after having inhaled copper soldering flux fumes. He was examined, reassured, and sent home. Twelve hours later he returned complaining of dyspnoea and a severe, dry, non-productive cough. Clinically the chest was overinflated with poor bilateral expansion and widespread wheezing. The abdomen was distended but no discrete masses or organs were palpable.

An intravenous injection of aminophylline was given followed by an aminophylline infusion, and amoxycillin was started. Chest radiography showed subcutaneous emphysema, mediastinal emphysema, and pneumoperitoneum (figure). The subcutaneous emphysema worsened until it eventually almost closed both eyes. Salbutamol 2 mg thrice daily by mouth was started. Over the next few days the coughing decreased, his chest improved, and the pneumoperitoneum resolved. The subcutaneous emphysema slowly disappeared and he was discharged free of symptoms one week after admission.

Chest radiograph showing subcutaneous emphysema, mediastinal emphysema, and pneumoperitoneum.

Comment

The extra-alveolar air in this case probably arose by a mechanism similar to that described by Macklin and Macklin,³ who showed that when gas under pressure was applied to the trachea of cats it ruptured the overdistended alveoli, forming pulmonary interstitial emphysema that then disseminated towards the mediastinum to form mediastinal emphysema. From there it disseminated into the fascial planes of the neck to form a subcutaneous emphysema, ruptured through the mediastinal pleura to cause a pneumothorax, and escaped retroperitoneally to form a pneumoperitoneum. The pressures needed to cause a pneumoperitoneum are higher than those usually needed to cause a pneumothorax, and a pneumoperitoneum has not been previously noted without a pneumothorax when the source of the air is from the lungs.⁴

The extra-alveolar air in this case was probably due to a combination of the three factors that lead to alveolar breakdown.⁴ Firstly, because this patient had an overinflated chest with wheezing there may have been overdistended alveoli distal to obstructed bronchioles. Formation of extra-alveolar air has been reported under these circumstances,

particularly in asthma.³ Secondly, underlying lung damage due to the inhaled fumes may have encouraged breakdown of the alveoli wall. When this happens pulmonary interstitial emphysema forms, and this can progress to mediastinal emphysema, subcutaneous emphysema, pneumothorax, and pneumoperitoneum.⁴ Thirdly, Macklin and Macklin reported that a sudden increase in intrathoracic pressure during a Valsalva manoeuvre, such as coughing, could also cause pulmonary interstitial emphysema, mediastinal emphysema, subcutaneous emphysema, and pneumothorax in otherwise normal lungs or facilitate the movement of extra-alveolar air in damaged lungs.^{2,3} Although pneumoperitoneum and coughing have not been associated previously, there have been cases of so-called idiopathic or spontaneous pneumoperitoneum that might have been caused or exacerbated by coughing or vomiting; this could perhaps be determined by reading the case histories carefully.⁴

This case shows several important principles. Firstly, because the free air in the abdominal cavity was associated with subcutaneous emphysema and mediastinal emphysema it was almost certainly derived from ruptured alveoli in the thorax and not from a perforated abdominal viscus.⁴ Abdominal air may itself cause respiratory embarrassment by splinting the diaphragm. Secondly, the pressure in the pleural space should be relieved if necessary with an intercostal drain; but the formation of extra-alveolar air, from whatever cause, may be decreased or prevented by reducing coughing and all forms of straining. Furthermore, understanding of the mechanism of formation of extra-alveolar air would allow doctors to consider more critically current suggested practices such as clamping the intercostal tube of a patient with a pneumothorax who is about to cough.⁵

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Retroperitoneal fibrosis associated with sotalol

Retroperitoneal fibrosis is fairly rare, and the aetiology usually remains unknown. Occasionally, however, an aetiological factor may be identified: methysergide taken for migraine reportedly induced retroperitoneal fibrosis in 1-3% of patients,¹ and analgesics, sedatives, and antihistamines have also been incriminated.² Retroperitoneal fibrosis has also been reported in patients who have taken beta-blockers, including atenolol,³ oxprenolol,⁴ and metoprolol.⁵ We describe a case of retroperitoneal fibrosis in a patient who had been taking sotalol for five years.

Case report

A 55-year-old children's nurse was referred to this hospital in December 1981 for fever and a raised erythrocyte sedimentation rate. She had been treated with sotalol 80 mg twice daily for five years for uncomplicated hypertension. At the time of diagnosis intravenous urography had shown nothing abnormal. She was also taking a derivative of vitamin A (retinoic acid) 25 mg twice daily, which she had begun six months before admission for pustulosis palmoplantaris. She had otherwise been in a good health until three weeks before admission, when she complained of lack of appetite, nausea, and mild fever.

Clinical examination showed nothing abnormal, and blood pressure was well controlled at 124/84 mm Hg. Haemoglobin concentration was 10.6 g/dl, erythrocyte sedimentation rate 77 mm in first hour, and serum creatinine concentration 71 µmol/l (0.8 mg/100 ml). There was no abnormality on

urine microscopy. Four months before admission her haemoglobin concentration had been 13.5 g/dl, erythrocyte sedimentation rate 28 mm in first hour, and serum creatinine concentration 84 $\mu\text{mol/l}$ (0.9 mg/100 ml) and she had been normotensive. Ultrasonography carried out because of abdominal pain showed bilateral hydronephrosis. Intravenous urography showed obstruction on both sides at the same level (L4). Retroperitoneal fibrosis was diagnosed and confirmed at operation. The fibrous tissue zone was 3-4 cm long on both sides. The obstructed ureters were liberated from the fibrous retroperitoneal tissue and transplanted to an intraperitoneal position. Biopsy specimens taken during the operation showed dense collagenous fibrotic tissue typical of retroperitoneal fibrosis. Postoperative recovery was uneventful. Sotalol was stopped and she was discharged well. Five months later she had no symptoms, was normotensive, and had a haemoglobin concentration of 13.7 g/dl and an erythrocyte sedimentation rate of 33 mm in first hour.

Comment

Retroperitoneal fibrosis has been described in association with beta-blockers including atenolol, oxprenolol, and metoprolol. So far as we know, our patient is the first report of an association with sotalol. Our patient had also been taking a derivative of vitamin A, but no cases of retroperitoneal fibrosis have been reported in association with this drug. No other cause for the retroperitoneal fibrosis was found in our patient. As Thompson and Julian⁵ reminded when reporting the association between beta-blockers and retroperitoneal fibrosis, however, we cannot exclude the possibility of an idiopathic case, since beta-blockers are so widely used.

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Chloramine-induced pneumonitis from mixing household cleaning agents

Housewives often mix domestic cleaning agents without being aware of the dangers. For example, mixtures may liberate substantial amounts of irritant gases. We describe a case of severe pneumonitis caused by chloramine released from a mixture of ammonia and household bleach containing sodium hypochlorite.

Case report

A 27-year-old woman mixed about 500 ml of 4.5% household ammonia with the same amount of 5% sodium hypochlorite bleach in a small, poorly ventilated bathroom. Harsh fumes evolved immediately, causing burning in her eyes and throat. Within a few seconds she started choking and coughing violently and later felt nauseated and vomited. Twelve hours after exposure, while still dyspnoeic and coughing, she was admitted to hospital.

Results of pulmonary function tests after inhalation of chloramine fumes

Time after exposure	Pulmonary function values (as % of predicted normal ¹)								Oxygen pressure/carbon dioxide pressure (kPa)		
	Vital capacity	Residual volume	Functional residual capacity	Total lung capacity	Residual volume/total lung capacity	Forced expiratory volume in 1 s	Rate ₅₀	Rate ₂₅	Fractional inspired oxygen: $\pm 50\%$	100%	21%
48 hours	46	137	85	66	46	42	36	29	6.8/3.6	19.6/4.1	13.0/4.9
9 days	93	122	106	99	27	105	100	100			

Conversion: SI to traditional units: 1 kPa $\approx$ 7.5 mm Hg.

On admission the patient was dyspnoeic and tachypnoeic (32 respirations/min). Pulse rate was 130/min and blood pressure was normal. Rales and wheezing were heard throughout both lungs. Chest radiography was unhelpful. Blood gas analysis showed hypoxaemia with oxygen pressure 6.7 kPa (50 mm Hg). Humidified 70% oxygen, terbutaline sulphate inhalations, and intravenous methylprednisolone 1 g thrice daily were administered. Twenty-four hours after exposure she was still tachypnoeic, with no improvement in blood gas values. She was therefore transferred to the intensive care unit and continuous positive airway pressure ventilation was instituted. Pulmonary function values 48 hours after exposure showed a severe restrictive and obstructive pattern with a distinct right-to-left shunt (table). These findings were consistent with chemical pneumonitis. A definite infiltrate in the right lower lobe was first seen only on the third day after exposure.

The patient recovered uneventfully after treatment with parenteral penicillin and was discharged nine days after admission. Pulmonary function returned to normal.¹

Comment

Mixing solutions of ammonia and sodium hypochlorite produces acrid monochloramine (NH_2Cl) and dichloramine (NHCl_2) fumes.² On contact with mucosa chloramines decompose to ammonia and hypochlorous acid. The latter combines with moisture, forming hydrochloric acid and liberating nascent oxygen, which is a potent oxidising agent causing most of the cellular injury. Corrosive effects of ammonia and hydrochloric acid also contribute to chloramine-induced respiratory tract damage.

There are no reported cases of serious injury from inhaling chloramine fumes, transient cough, dyspnoea, nausea, and vomiting being described.^{3,4} Hence inhaling chloramine fumes is considered less hazardous than exposure to other lung irritants, as expressly stated in recent reference texts.^{2,5}

The extent of injury from noxious gases depends on the concentration of inhaled gas as well as the duration of exposure. Chloramine concentrations attained by mixing small or moderate amounts of cleaning agents are probably too low to cause severe injury. This might explain the absence of serious sequelae to chloramine inhalation in other reports. Our case shows that when copious amounts of ammonia and hypochlorite are combined the extent of the bronchoalveolar injury may be considerable. In this respect chloramine is probably no less dangerous than chlorine.

An important observation in our patient was the delayed radiographic appearance of pneumonitis. Pulmonary function tests were more helpful in diagnosing this complication, being commensurate with the clinical findings early in the course. Chloramine exposure from mixing household ammonia with cleaning agents containing hypochlorite is a fairly common domestic accident. The hazards of this poisoning should not be underestimated.

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