Postobstructive pulmonary oedema

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A muscular 30-year-old man was admitted for an elective bilateral subcutaneous mastectomy. He had mild asthma relieved with salbutamol but was otherwise in good health, and smoked five cigarettes a day. He had undergone wisdom tooth extraction under general anaesthetic without complications.

He weighed 109.6 kg (body mass index 30.36 kg/m^2) and preoperative cardiovascular and respiratory examinations were normal. Peak expiratory flow rate (PEFR) was 620 litres/min, blood pressure (BP) 140/82 mmHg, arterial oxygen saturation (SpO_2) 98% on air and electrocardiogram (ECG) was normal. Blood tests showed a haemoglobin of 16.4g/dl, but all other parameters within normal ranges. Preoperatively he was given subcutaneous low molecular weight heparin for thromboprophylaxis.

Anaesthesia was induced with fentanyl 100 µg and propofol 200 mg, and a laryngeal mask airway (LMA) inserted. Intraoperatively, 1 litre of Hartmann's solution was administered with ondansetron 4 mg and morphine 8 mg. Anaesthesia was maintained with isoflurane and nitrous oxide in oxygen, and the patient was allowed to breathe spontaneously throughout. The patient's observations remained unremarkable.

On waking, the patient experienced laryngospasm, relieved by positive pressure ventilation. There was no suspicion of aspiration. After normal observations in recovery he returned to the ward.

That evening the patient developed a productive cough and shortness of breath on light exercise. No further medication had been given. He denied chest or wound pain, wheeze, palpitations or syncope. He was alert and cooperative with a Glasgow Coma Scale of 15. Chest wall movement was symmetric. His respiratory rate was 16 breaths per minute at rest.

There was no evidence of upper airway obstruction, and jugular venous pressure and trachea appeared normal. Sputum was pink and blood stained. Percussion of both lung fields was normal. On auscultation, his heart sounds were normal with no parasternal heave. Air entry was heard bilaterally, with widespread scattered crackles throughout both lung fields. There was no evidence of erythema, tenderness or swelling in the lower limbs.

He was clammy with temperature 37˚C, pulse 102/min, BP 130/72 mmHg, SpO_2 88.8% on air, blood glucose 7.5 mmol/litre and PEFR 480 litre/min. An ECG showed sinus tachycardia with normal axis and morphology. Venous blood tests were unchanged, but his arterial blood gas sample on room air revealed type 1 respiratory failure.

Chest X-ray showed symmetrical perihilar shadowing with a predominant air space component and relative sparing of the subpleural regions, in keeping with pulmonary oedema. The cardiothoracic ratio and vascular pedicle size were within normal limits, making cardiogenic pulmonary oedema unlikely. Treatment with 35% oxygen via a fixed performance mask was begun with good effect. Symptoms resolved spontaneously over the next 48 hours, and postobstructive pulmonary oedema (POPE) was diagnosed.

DISCUSSION

POPE occurs when a large negative intrapleural pressure is generated by inspiration against an obstructed upper airway. A wide range of obstructive causes have been reported, but most cases have been attributed to laryngospasm following the removal of a LMA or endotracheal tube post-general anaesthetic.

The incidence of POPE following general anaesthetic is 0.1% (McConkey, 2000). POPE usually develops within 24 hours of the precipitating event and resolves within 2–3 days. Most cases resolve spontaneously but fatalities have been reported. Early administration of oxygen and positive airway pressure can quickly resolve POPE in most cases.

The pathogenesis of POPE has been widely debated, but three mechanisms appear to be crucial:

1. The mechanical stress placed upon the lungs by an upper airway obstruction, up to −70 cmH_20 in young adults (Lang et al, 1990)
2. Negative intrapleural pressure has a direct effect upon the heart
3. The hypoxia caused by an obstructed airway prompts hypoxic pulmonary vasoconstriction.

CONCLUSIONS

POPE is an important cause of morbidity, prolonged hospital admission and mortality in young and otherwise healthy patients following general anaesthetic. When recognized, conservative treatment can be quickly implemented and aggressive diagnostic and therapeutic interventions can be avoided. HM

Lang SA, Duncan PG, Shephard DAE, Hung CH (1990) Pulmonary oedema associated with airway obstruction. *Can J Anaesth* **37**(2): 210–18

McConkey PB (2000) Post obstructive pulmonary oedema- a case series and review. *Anaes Int Care* **28:** 72–6

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