Section: Miscellaneous

Case Report

ACUTE ASPIRATION PNEUMONITIS DURING GENERAL ANAESTHESIA IN AN OBESE PATIENT UNDERGOING HUMERAL FRACTURE FIXATION: A CASE REPORT

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ABSTRACT

Pulmonary aspiration during general anaesthesia is a serious complication capable of driving up postoperative morbidity and mortality. Anything that slows gastric emptying or blunts airway-protective reflexes — obesity, pregnancy, paralytic ileus, reduced consciousness, general anaesthesia itself, or head injury — raises a patient's risk. In the early stages, aspiration pneumonitis and aspiration pneumonia can look very similar, yet telling them apart matters, since each calls for a different treatment approach. We present the case of a morbidly obese man scheduled for surgical fixation of a fractured humerus under general anaesthesia who developed a cannot-ventilate emergency and went on to aspirate; he was subsequently managed successfully in the intensive care unit.

Keywords: Aspiration pneumonitis; pulmonary aspiration; morbid obesity; cannot ventilate.

INTRODUCTION

Pulmonary aspiration is the entry of liquid or solid material past the larynx into the lungs.[1]. What happens next depends on what was aspirated: chemical injury to lung tissue is called aspiration pneumonitis, while a secondary bacterial infection of the lung is called aspiration pneumonia. Several factors raise the risk of aspiration under anaesthesia — reduced consciousness, blunted airway-protective reflexes, and delayed gastric emptying, the last of which is common in pregnancy, obesity, diabetes mellitus, and inadequate preoperative fasting.

We describe a 25-year-old man with a BMI of 39.1 kg/m² who underwent general anaesthesia for fixation of a fractured humerus, developed a 'cannot ventilate, cannot intubate' emergency intraoperatively, and went on to show pulmonary complications around the time of extubation.

CASE REPORT

Written informed consent for anaesthesia and surgery was obtained. The patient was positioned on the operating table and standard monitors were attached. Pre-induction vitals were BP 134/93 mmHg, heart rate 96/min, and SpO2 98% on room air. An 18G IV cannula was secured and Ringer's lactate infusion started.

The patient was placed in the RAMP position with 25 degrees of head-up tilt and preoxygenated with 100% oxygen for 3 minutes. Anaesthesia was induced with IV fentanyl 150 mcg, propofol 180+40 mg, and succinylcholine 100 mg. Bag-and-mask ventilation required two operators.

Direct laryngoscopy showed a Cormack-Lehane grade 2a view, and the trachea was intubated with an 8.0 mm ID endotracheal tube. Auscultation revealed normal air entry on the right but reduced air entry with wheeze on the left. The tube was withdrawn by one centimeter marking, and the patient was given IV hydrocortisone 100 mg, IV dexamethasone 8 mg, IV propofol 40 mg, and six puffs of salbutamol via the tube. Repeat auscultation was clear with equal bilateral air entry. The patient was ventilated in volume-control mode (tidal volume 580 mL, respiratory rate 14–16/min, PEEP 6 cm H2O), with peak airway pressures of 22–24 cm H2O.

Anaesthesia was maintained with sevoflurane, nitrous oxide, and 40% oxygen, with vecuronium for muscle relaxation. The patient remained stable intraoperatively.

At the end of surgery, neuromuscular blockade was reversed with IV glycopyrrolate 600 mcg and neostigmine 3.5 mg. The patient opened his eyes spontaneously and breathed regularly, but limb muscle power remained inadequate; a further dose of glycopyrrolate 200 mcg and neostigmine 1 mg was given, after which the patient gradually began following verbal commands. After extubation, however, no respiratory movement was felt on the reservoir bag. An oropharyngeal airway was inserted and bag-mask ventilation optimized. Auscultation revealed bilateral wheeze. Multiple positive-pressure breaths were delivered along with IV hydrocortisone 100 mg and salbutamol, but saturation did not improve and SpO2 fell to 42%. The patient was reintubated under direct laryngoscopy over a bougie with a 6.5 mm ID tube (using propofol 70 mg and succinylcholine 75 mg iv for induction); severe airway oedema was noted, and bilateral coarse crepitations were heard throughout the chest. Ventilation with 100% FiO2 brought saturation back up to 96–97%.

The patient was transferred to the ICU for mechanical ventilation on pressure-control mode with PEEP 12 cm H2O; bilateral crepitations persisted throughout the chest. He was started on IV ceftriaxone-sulbactam 1.5 g twice daily, IV amikacin 500 mg twice daily, IV paracetamol 1 g three times daily/as needed, and oral calcium with vitamin D3, a multivitamin, vitamin C, and pantoprazole 40 mg once daily. The following morning, sedation was stopped; the patient regained consciousness, breathed regularly, and was extubated again.

After this second extubation the patient was conscious, oriented, and following commands, with BP 136/90 mmHg, pulse 118/min, and SpO2 97% on 5 L/min oxygen. Two hours later he developed respiratory distress and tachypnoea with falling SpO2 and required a third intubation, after which he was placed on volume-control ventilation (FiO2 1.0, PEEP 7, RR 14). A chest radiograph obtained on postoperative day 1 (Figure 1) prompted a revised treatment plan: IV piperacillin 4.5 g three times daily, IV metronidazole 100 mL three times daily, IV dexamethasone 8 mg three times daily, a single dose of IV magnesium 1 g over 20 minutes, and IV dalteparin 5000 IU once daily, with nebulisation continued four-hourly.

Cardiology review on postoperative day 2 recommended 2D echocardiography, D-dimer, NT-proBNP, and troponin I testing; the patient was started on subcutaneous enoxaparin 0.8 mL twice daily with CVP-guided fluid therapy. The echocardiogram excluded moderate-to-severe pulmonary embolism, though a mild embolism could not be ruled out, and showed an LVEF of 40%. A subsequent CT pulmonary angiogram was negative for pulmonary embolism.

The patient was extubated for a third time after 48 hours, fully conscious and haemodynamically stable, maintaining SpO2 99% on a simple face mask at 5 L/min. That evening he was transitioned to high-flow oxygen therapy (30 L/min, FiO2 60%), maintaining saturation of 98%. FiO2 was subsequently weaned and he returned to a simple face mask. He remained in the ICU for six days before being discharged home.

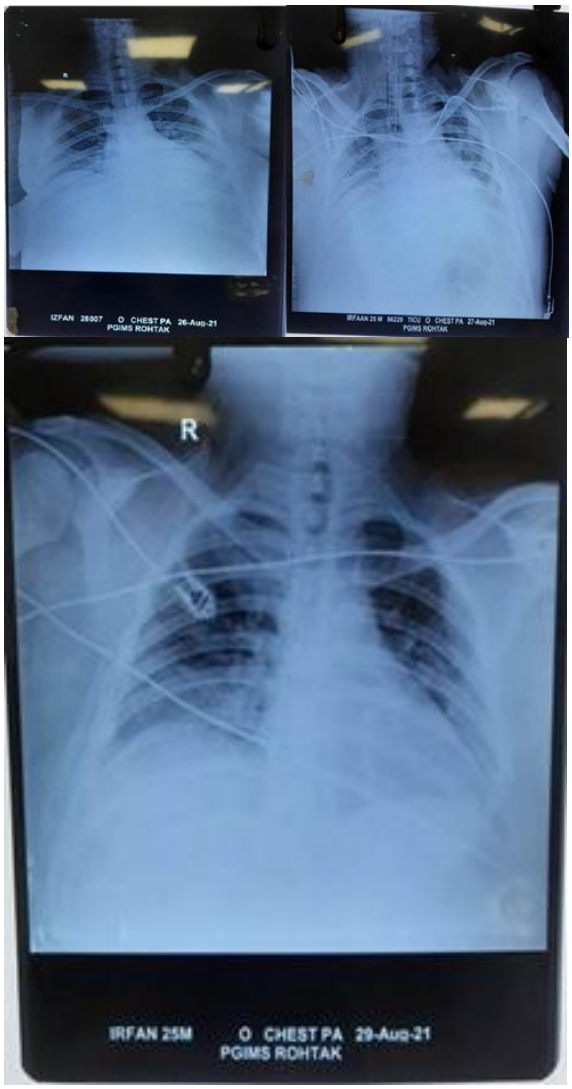

Figure 1: Chest radiographs of the patient before surgery and on postoperative days 1 and 3, showing findings consistent with aspiration pneumonitis.

DISCUSSION

General anaesthesia is generally safe, but aspiration remains one of its most feared complications, occurring in roughly 1 in 2000–3000 cases and more frequently during emergency surgery.[2] Mendelson's original 1946 report put aspiration-related mortality at around 3%, but subsequent studies have reported figures as high as 6.6%.[3-5] The clinical picture varies widely: some patients show no symptoms at all, others develop mild hypoxia, and still others progress to respiratory failure, ARDS, or sudden death.[1]

What determines severity is largely the nature and volume of the aspirate — a small volume of low-pH gastric content can cause more damage than a larger volume at a higher pH — and this spectrum ranges from aspiration pneumonitis and aspiration pneumonia to aspiration of particulate matter.[6]

In aspiration pneumonitis, acidic gastric contents chemically injure lung tissue directly; in aspiration pneumonia, the damage instead comes from a secondary bacterial infection.[7]

General anaesthesia itself lowers the threshold for aspiration by blunting laryngeal reflexes, and certain patient factors compound this risk further. Delayed gastric emptying makes obese patients particularly vulnerable. In this case, bronchospasm at the time of extubation led to a cannot-ventilate situation; aspiration most likely occurred as a result of ventilation at high pressures causing gastric insufflation.

These risks argue for careful preoperative screening, particularly before emergency surgery, and for airway management plans tailored to the patient's risk profile. Rapid sequence induction, cricoid pressure, and antacid prophylaxis are all recommended for high-risk patients undergoing general anaesthesia.[8,9]

When gastric contents are seen in the oropharynx or airway, prompt suctioning and head-down positioning are essential. If aspiration does occur, distinguishing pneumonitis from pneumonia is critical, since the two require different treatment and carry different implications for morbidity and mortality — even though this distinction can be difficult to make early in the disease course. A chest radiograph can offer useful clues to the underlying pathology.[6] Management is largely supportive, centred on maintaining adequate oxygenation through non-invasive or invasive ventilatory support as the clinical picture demands.

Because the acute injury is chemical rather than infectious, routine antibiotic use is not recommended in the early phase; empirical antibiotics, in fact, have been linked to a higher rate of later ventilator-associated pneumonia, so antimicrobial therapy should instead be targeted to a confirmed pathogen.[10] Corticosteroids are similarly best avoided in the acute phase. Pentoxifylline and thymoquinone have shown promise in animal studies, but human trials have yet to confirm their benefit.[11,12]

Bronchodilators and positive end-expiratory pressure remain the mainstay of treatment, with therapy individualized to the patient's clinical course.

CONCLUSION

Anaesthesiologists should stay alert to the factors that predispose patients to aspiration and be well prepared with strategies to optimize outcomes should aspiration occur.

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