Case Reports/Case Series

Case report: Unilateral negative pressure pulmonary edema - a complication of endobronchial intubation

[Présentation de cas : l'œdème pulmonaire par pression négative unilatérale, une complication de l'intubation endobronchique]

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Purpose: We describe an unusual presentation of a case of fulminant unilateral pulmonary edema caused by unrecognized right endobronchial intubation that occurred during patient movement at the end of surgery. We review factors which may predispose to this complication.

Clinical features: During emergence from anesthesia at the conclusion of bariatric surgery, a 27-yr-old patient (163 kg, body mass index 61.5 $kg \cdot m^{-2}$) became hypoxemic despite vigorous spontaneous ventilatory efforts through a 7.0 mm endotracheal tube with 100% oxygen. Right mainstem endobronchial tube malposition was detected by auscultation. The tube was repositioned, followed by copious pink frothy pulmonary edema abruptly issuing from the tracheal tube. Chest radiography revealed dense left lung infiltrates, consistent with unilateral negative pressure pulmonary edema, caused by brief, but forceful, inspiratory efforts against an obstructed left bronchus. This condition resolved over the following 24 hr. The patient's trachea was then extubated, and the remainder of her recovery was unremarkable.

Conclusions: A high degree of airway anatomic variation, common tracheal tube insertion practices, unreliability of tube position detection methods, and the effects of patient positioning may all contribute to endotracheal tube malposition, including partial endobronchial intubation. Several modifications in airway management may help to prevent such complications of tracheal tube malposition.

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Objectif *: Nous décrivons la présentation inhabituelle d'un cas d'œdème pulmonaire unilatéral fulminant provoqué par une intubation endobronchique droite non détectée, survenue pendant le déplacement du patient à la fin de la procédure chirurgicale. Nous passons en revue les facteurs susceptibles de prédisposer à cette complication.*

Éléments cliniques *: Pendant l'émergence de l'anesthésie après une chirurgie de l'obésité, un patient âgé de 27 ans (163 kg, indice de masse corporelle 61,5 $kg \cdot m^{-2}$) est devenu hypoxémique malgré des efforts soutenus de ventilation spontanée via un tube endotrachéal de 7,0 mm avec de l'oxygène 100 %. Le positionnement inadéquat du tube dans la bronche souche droite a été détectée par auscultation. Le tube a été repositionné, mais suivi de l'irruption d'un œdème pulmonaire abondant spumeux et rose provenant de la sonde trachéale. La radiographie des poumons a révélé d'épais infiltrats dans le poumon gauche, compatibles avec un œdème pulmonaire par pression négative unilatérale et provoqué par des efforts inspiratoires brefs mais soutenus pour dégager une bronche gauche obstruée.*

Conclusion *: Une importante variation anatomique des voies respiratoires, les pratiques usuelles d'insertion de la sonde trachéale, le manque de fiabilité des méthodes de détection de la position du tube et les conséquences du positionnement du patient peuvent*

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tous jouer un rôle et provoquer un positionnement inadéquat du tube endotrachéal, y compris une intubation endobronchique partielle. Plusieurs modifications dans la prise en charge des voies aériennes pourrait nous aider à éviter de telles complications à la suite du mauvais positionnement du tube endotrachéal.

Although often benign, unintended endobronchial intubation may cause serious morbidity or an escalation of care.[1] Anatomic variation,[2–4] current unproven tracheal tube insertion practice recommendations,[5,6] patient positioning effects,[4,7] and unreliable tube position detection methods[1,7,8] likely account for the occurrence of tracheal tube malposition. We describe an unusual complication of intraoperative endobronchial intubation, i.e., unilateral left lung negative pressure pulmonary edema. The Vanderbilt University Institutional Ethics Board approved reporting of this case.

Clinical report

A 27-yr-old obese woman underwent open Roux-en-Y gastric bypass. Her additional medical problems included untreated obstructive sleep apnea, gastroesophageal reflux, recent upper respiratory illness (one month before surgery), depression, and latex allergy. Her preoperative medications included escitalopram and ranitidine. Her blood pressure was 136/70 mmHg, pulse 62 beats·min^{-1}, SpO_2 98%, height 1.63 m, weight 163 kg, and body mass index 61.5 kg·m^{-2}. Besides obesity, the patient's examination was unremarkable. Laboratory investigations, including hemoglobin (126 g·L^{-1}) and glucose (4.8 mmol·L^{-1}) and a chest radiograph, were unremarkable.

Pre-medications included cefazolin 1 g *iv*, famotidine 20 mg *iv*, metoclopramide 10 mg *iv*, sodium citrate 30 mL *po*, and midazolam 2 mg *iv*. The patient was transported to the operating room and was positioned to optimize airway instrumentation. Oxygen 100% was administered by mask; fentanyl 250 µg and lidocaine 100 mg were administered intravenously, and cricoid pressure was applied. Next, rapid sequence induction of general anesthesia was performed with propofol 200 mg *iv* and succinylcholine 200 mg *iv*. A 7.0 Mallinckrodt® cuffed Intermediate Hi-Lo® (Mallinckrodt, Hazelwood, MO, USA) endotracheal tube (ETT) was easily inserted under direct visualization with a Macintosh 3 laryngoscope. At 21 cm, the ETT was taped at the lip, and breath sounds were equal bilaterally. Soon after, the SpO_2 decreased to 94% and auscultation revealed wheezing. Once albuterol (12 puffs) was administered via ETT, the wheezing resolved and SpO_2 increased to 98%. Vecuronium was administered, and pressure controlled ventilation was initiated with rate 12, inspiratory:expiratory time ratio 1:2, and pressure 30 cm H_2O producing expired tidal volumes of 700 mL. After the abdomen was opened and retractors inserted, 10 cm H_2O positive end expiratory pressure (PEEP) was added, and airway pressure was increased to 36 cm H_2O, with tidal volume = 600 mL. Anesthesia was maintained with desflurane 5.0–6.6% in 50% oxygen, additional fentanyl (250 µg), and vecuronium (5 mg). Oxygen saturation (98–100%) and expired tidal volume (600–615 mL) were stable throughout the three-hour uneventful surgery. At the end of surgery, three twitches were present by train-of-four monitoring at the ulnar nerve. Glycopyrrolate 1 mg *iv* and neostigmine 5 mg *iv* were administered, and the patient was breathing spontaneously with pressure support (20 cm H_2O), PEEP 10 cm H_2O, rate 23·min^{-1}, and tidal volume 400 mL. Fifteen minutes later, desflurane was discontinued, oxygen 100% administered, and spontaneous ventilation was supported using the same settings, with tidal volumes 650 mL. An oral gauze bite block was inserted to prevent biting of the ETT. Ten minutes later, the patient abruptly emerged with coughing and bucking and began reaching for the ETT. During emergence, oxygen saturation rapidly decreased to the mid-50% range. Endotracheal tube secretions were observed and were suctioned several times. Oxygen saturation slowly increased to the low 80's. Some wheezing was audible on auscultation, and albuterol was again administered. At that point, the patient was more awake and cooperative. Tidal volumes remained above 650 mL throughout emergence, and pressure support was reduced to 10 cm H_2O, with PEEP remaining at 10 cm H_2O. Repeat chest auscultation revealed diminished breath sounds on the left, and inspection of the ETT showed migration to 23 cm at the lip. The tube was withdrawn to 21 cm, immediately improving breath sounds on the left, but dramatically increasing ETT secretions, which then became frothy and light pink in color. Oxygen saturation increased to the low 90s, while F_iO_2 was still 1.0. The patient was transferred, with trachea intubated, to the postanesthesia care unit, and she was sedated with midazolam. Chest radiography showed diffuse left lung opacification, with the ETT tip sited 2 cm above the carina (Figure 1). The ETT was withdrawn a further 2 cm, and the patient remained on pressure support ventilation of 8 cm H_2O, PEEP 15 cm H_2O, 60% oxygen, with SpO_2 ranging 90–95%. Repeat chest radiography four hours after the event showed correct ETT position and

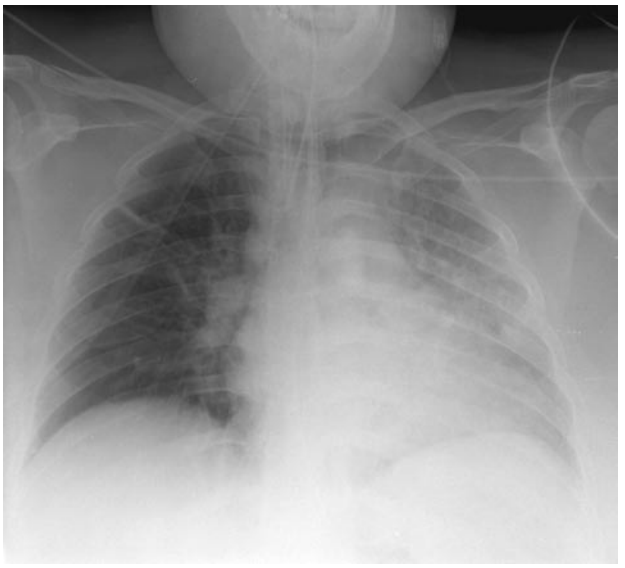

FIGURE 1 Immediate postoperative chest radiograph showing pulmonary edema restricted to the left lung.

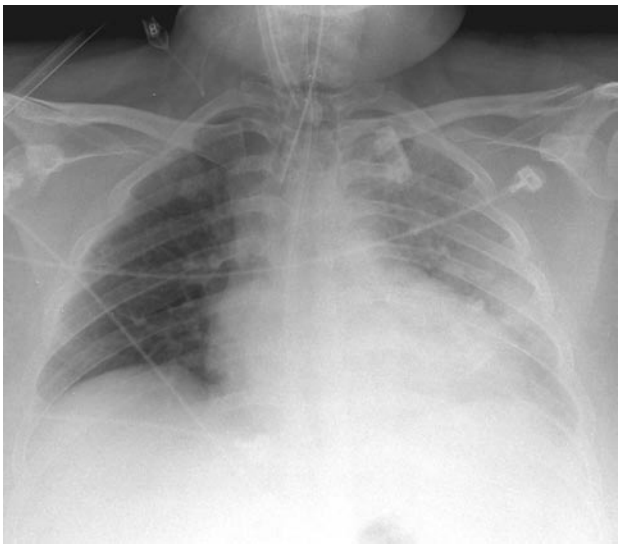

FIGURE 2 Repeat chest radiograph, four hours after the event, showing mild improvement in left lung edema.

improvement in left lung edema (Figure 2). Supplemental oxygen and ventilator support were reduced over the next sixteen hours; the patient's trachea was extubated, and the chest radiograph revealed substantial resolution of the edema (Figure 3). The remainder of the patient's course was uneventful, and she went home three days after surgery.

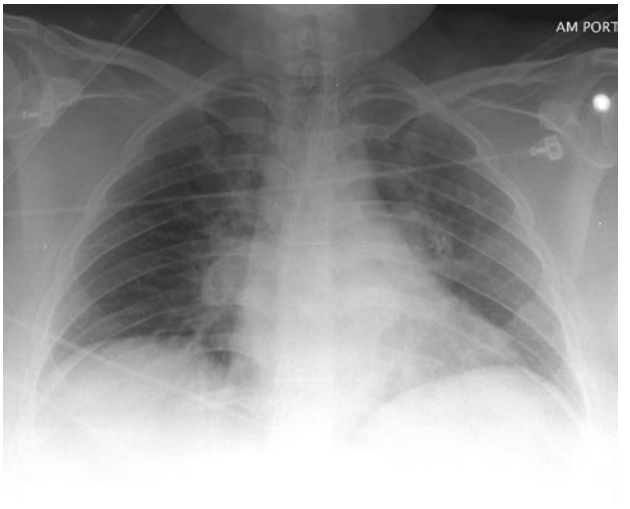

FIGURE 3 Repeat chest radiograph, 20 hr after the event and after discontinuation of mechanical ventilation, showing resolution of pulmonary edema.

Discussion

The patient we describe demonstrated typical features of negative pressure pulmonary edema (NPPE), but its appearance in only one lung distinguished it from the classic presentation. The likely mechanism involved migration of the ETT tip to a right endobronchial position (depth noted to be 23 cm). This would obstruct the left main bronchus during forceful inspiratory efforts as the patient emerged from anesthesia, thereby subjecting the left lung to significant negative pressure. In the presence of high F_IO_2, it is possible that absorption atelectasis, distal to the occluded bronchus, additionally contributed to left lung negative pressure. Endotracheal tube withdrawal to 21 cm immediately improved left lung ventilation, as evidenced by auscultation and SpO_2, and simultaneously permitted passage of copious pulmonary edema, which appeared abruptly.

In retrospect, there were clues that the ETT may have been positioned too distally upon initial placement, for instance, brief oxygen desaturation and wheezing occurred despite bilateral breath sounds. Additionally, observed peak airway pressures and expired tidal volumes suggested abnormally low compliance, although this may simply have reflected the patient's extreme obesity. Auscultation is not sensitive in detecting ETT tip placement in the right main bronchus. Sugiyama *et al.*[8] demonstrated normal breath sounds with endobronchial depths of 1.5–2.0 cm and

did not detect disappearance of breath sounds until a depth of 3.2 cm. Ezri *et al.*[7] corroborated these findings by showing that, in all of eight cases of endobronchial intubation, there were no signs of change in oxygen saturation, peak inspiratory pressure, or end-tidal CO_2 measurement. Extension of the ETT beyond the cuff (approximately 2.6 cm) and presence of the Murphy eye account for our and others' observations.[2]

Negative pressure pulmonary edema, first described in three patients (airway tumour, strangulation, and interrupted hanging) in 1977,[9] is now a well known pathologic phenomenon.[10] Negative pressure pulmonary edema typically results during forceful inspiration after acute upper airway obstruction in young healthy patients. A retrospective review of 31,826 anesthetic cases revealed an incidence of approximately one episode of NPPE per 1,000 patients.[10] Most often, they occurred in the post-extubation period following head, neck, and upper digestive surgeries, with laryngospasm being the most common cause of obstruction. Numerous other causes of airway obstruction leading to NPPE have been described, including tracheal aspiration of food,[11] tracheal compression by neck and mediastinal tumours,[12] and Ludwig's Angina.[10]

The mechanisms underlying NPPE have been reviewed in detail.[9,10,13] Normally, fluid balance within the pulmonary vascular bed and interstitial pulmonary space is maintained by Starling forces (intra-/extracapillary hydrostatic and oncotic pressures), capillary permeability, and lymphatic drainage.[9,10,13] Vigorous inspiratory efforts in the presence of large airway obstruction produce very large negative pressures in the interstitial space.[10,13] The corresponding increase in intra- / extravascular hydrostatic pressure gradient encourages an increased rate of transudation from pulmonary capillaries into the interstitial space. If the rate of interstitial fluid accumulation exceeds lymphatic drainage, then alveolar flooding (pulmonary edema) occurs. Negative intrathoracic pressure also contributes to development of NPPE by augmenting venous blood flow into the thorax while simultaneously increasing left ventricular afterload, thereby expanding the blood volume in the pulmonary vascular bed.[9,13] This further increases the pulmonary capillary to interstitial space pressure gradient, encouraging transudation. Another complicating factor is the development of hypoxemia during the onset of NPPE, triggering hypoxic pulmonary vasoconstriction, raising pulmonary vascular resistance, and increasing transmural hydrostatic pressures.[9,13] Hypoxemia and acidemia may also cause myocardial depression, further contributing to pulmonary congestion.[9,10,13] Localized hypoxemia, along with perturbations of Starling forces, may also adversely affect capillary integrity, further promoting fluid leakage into the interstitial space.[9,10] However, a recent study, reporting analysis of fluid produced by NPPE, strongly supports hydrostatic mechanisms as the primary underlying pathophysiologic explanation.[13]

Unilateral left lung pulmonary edema associated with right endobronchial ETT malposition has been described, once previously, in three patients undergoing emergency airway management during cardiopulmonary arrest, a scenario that is commonly associated with ETT malposition.[14] The authors postulated several mechanisms to explain lung re-expansion pulmonary edema and discounted the likelihood of NPPE, given that none of the patients was breathing spontaneously. In addition to the unusual appearance of unilateral NPPE, deep placement of an ETT (tip at carina or in main bronchus) is known to cause oxygen desaturation or bronchospasm (both observed in this case), as well as other consequences, including pneumothorax, resorption atelectasis, light anesthesia, and coughing. These can precipitate escalations of medical therapy and increased morbidity and/or mortality. Several variables determine ETT depth relative to the carina and glottis. Chong *et al.*[2] have recently demonstrated that there is great anatomical variation in the length of the human trachea. Vocal cord to carina distance measured in 130 adults averaged 13.4 cm in men and 12.0 cm in women, but ranged from 9.5 to 16 cm.[2] Similar extremes in this anatomic distance have been described by others.[3,4] Furthermore, external body measurements appear to be poor predictors of appropriate ETT placement depth.[3] Separately, the distance between ETT tip and distal cuff border varies depending on ETT size, style, and manufacturer.[2] Despite this, practice standards for ETT placement fail to account for these variables and include using preset depth at the teeth (which specific teeth are not described) or "lip line", advancing a specific distance beyond the vocal cords, and/or advancing the ETT to a specific distance above the carina.[5,6] These varied insertion guidelines, combined with insensitive methods of tube placement verification (auscultation, SpO_2),[7,8] are unlikely to reliably prevent endobronchial ETT malposition, as in this case. Flexible fibrescope examination is reliable,[7] but typically used only when malposition is suspected, which it was not in this case. External cuff ballotment was never performed in this patient; however, it has been reported to be a convenient, quick, and reliable predictor of ETT cuff position.[15,16]

Finally, patient position changes significantly affect ETT depth. Both head down positioning and abdomi-

nal insufflation independently cause downward ETT shift relative to trachea.[7] Head movement also shifts the secured ETT within the trachea, for instance, neck flexion produces downward movement and neck extension produces upward movement.[4] The patient movement that occurred during emergence could well have caused shift of an already marginally acceptable ETT position.

In summary, ETT misplacement, in this case, resulted in unilateral NPPE, leading to a substantive escalation of care. Unrecognized airway anatomic variation, current ETT placement practices, patient positioning changes, and insensitive methods of detecting endobronchial positioning likely contributed to this complication. Better understanding of these factors, along with modification in airway management practice and a heightened level of vigilance, may prevent this result and other less critical consequences of endobronchial tube malposition.

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