

Bedside ultrasound for early diagnosis and follow-up of postoperative negative pressure pulmonary oedema: case reports and literature review

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Dear Editor,

Negative pressure pulmonary oedema (NPPE) is considered an uncommon postoperative complication but a highly serious condition in patients undergoing surgery under general anaesthesia [1, 2]. Usually it occurs during the extubation or postoperative period. However, in general the outcomes are satisfactory with a total recovery within the first 24-48 hours, but there are more severe cases of NPPE that may be life-threatening [3]. In anaesthesiology and critical care, the main cause of NPPE is related directly to laryngospasms during the extubation after removal of the laryngeal mask [4]. Despite the fact that the fatality rate in those presenting this pathology is unknown, an early diagnosis and adequate follow-up delineate the patient prognosis.

In the era of ultrasound, patient safety has been the main reason to develop important advances in this area; in particular, the application of this tool to critically ill patients has become a fast guide instrument for multiple procedures in the least invasive way. In our medical centre it has been decided to implement the ultrasound as a technique for tracing NPPE. Herein we report two cases of NPPE in which ultrasound was used to diagnose this condition and assess the therapeutic response during the stay in the intensive care unit (ICU). Our aim is to educate on the use of this tool in the practice of anaesthesiologists and critical care physicians when it comes to diagnosis and follow-up of patients with NPPE.

CASE 1

In a 34-year-old male patient, without any important past medical history, who was scheduled for varicocelelectomy under general anaesthesia with laryngeal mask, anaesthesia was induced with remifentanyl and propofol, and maintenance with isoflurane and remifentanyl. Surgery lasted 45 minutes with no intraoperative anaesthetic or surgical complications. During the removal of the mask, the patient presented laryngospasm and oxygen desaturation of 61% that required management with positive pressure ventilation recovering an oxygen saturation of 98%. The patient was transferred to the URPA awake but could not sustain an oxygen saturation greater than 90% with a facial mask, so noninvasive mechanical ventilation was indicated. On arrival to the ICU an ultrasound scan was performed, showing B lines in all four quadrants (Figure 1) according to the BLUE protocol, and appropriate cardiovascular function with collapsibility of the inferior vena cava based on the FOCUS protocol, supporting the diagnosis of NPPE. The management was established with furosemide 20 mg with continuous positive airway pressure (CPAP). The initial chest X-ray showed (Figure 2) and blood gases noted: pH 7.34, pCO₂ 43 mm Hg (5.7 kPa), pO₂ 62.5 mm Hg (8.3 kPa), HCO₃⁻ 23.1 mmol L⁻¹, SpO₂ 92.2%, and PaO₂/FiO₂ 125. After 6 h the patient required two cycles of CPAP with alternating O₂ by nasal cannula, having

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FIGURE 1. Case 1. Lung ultrasound exploration showing multiple B lines

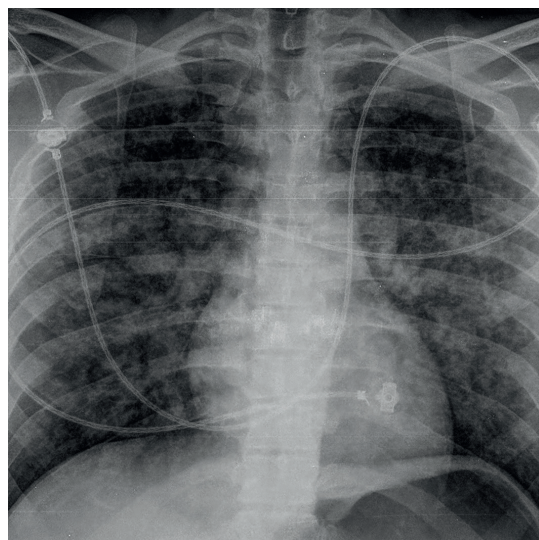


FIGURE 2. Case 1. Initial thoracic X-ray showing bilateral reticular lung infiltrates



FIGURE 3. Case 1. Control lung ultrasound without B lines after treatment

an adequate response. Follow-up of ultrasound scan at 24 h after the event showed no evidence of B lines (Figure 3), blood gases showed a $\text{PaO}_2/\text{FiO}_2$ increased to 260, and the patient was discharged.

CASE 2

A 77-year-old male patient was scheduled for an enterorrhaphy, without any important past medical history. Orotracheal intubation was performed for general anaesthesia, induction with propofol plus remifentanyl, relaxation with rocuronium, and maintenance with remifentanyl and sevoflurane. The surgery lasted 45 min, with no surgical or anaesthetic complications. Diclofenac and hydro-morphone were used for postoperative analgesia, and metoclopramide

and dexamethasone for nausea and vomiting prophylaxis. On arrival to the PACU the patient had SO_2 of 98% on nonbreathing mask at 4 L min^{-1} and Aldrete's score 8/10. After 5 min in the PACU, the patient became cyanotic and somnolent and saturation dropped to 60%. She was intubated, and vital signs reported a blood pressure of 210/119 mm Hg, heart frequency of 105 bpm, and SO_2 of 96%. Alveolar rales were auscultated in all four quadrants. A negative EKG and normal cardiac enzymes discarded an acute coronary event. Pulmonary oedema was suspected and immediately management with furosemide 20 mg i.v. bolus plus nitroglycerin, midazolam and fentanyl for sedation was started. After 15 min, nitroglycerin was suspended because of low

blood pressure. Chest X-ray (Figure 5) and cardiac enzymes were negative, gasometry revealed respiratory acidosis with a severe hypoxaemic $\text{PaO}_2/\text{FiO}_2$ 64 (pH 7.24, pCO_2 58.9 mm Hg [7.8 kPa], pO_2 64.2 mm Hg [8.5 kPa], HCO_3^- 25.7 mmol L^{-1} , BE -1.6 mmol L^{-1} , SpO_2 87.7%), and ultrasound scan according to protocol BLUE and FOCUS found pleural slip with B lines in all four quadrants (Figure 4), cardiac contractility qualitatively preserved, and adequate relationship between the right and left ventricle with normal collapsibility of the inferior vena cava. Lactate was 1.89 mmol L^{-1} . These findings allowed a diagnosis of non-cardiogenic pulmonary oedema in conjunction with NPPE. The patient was transferred to the ICU where the parameters of ventilatory sup-



FIGURE 4. Case 2. Lung ultrasonography showing multiple B lines

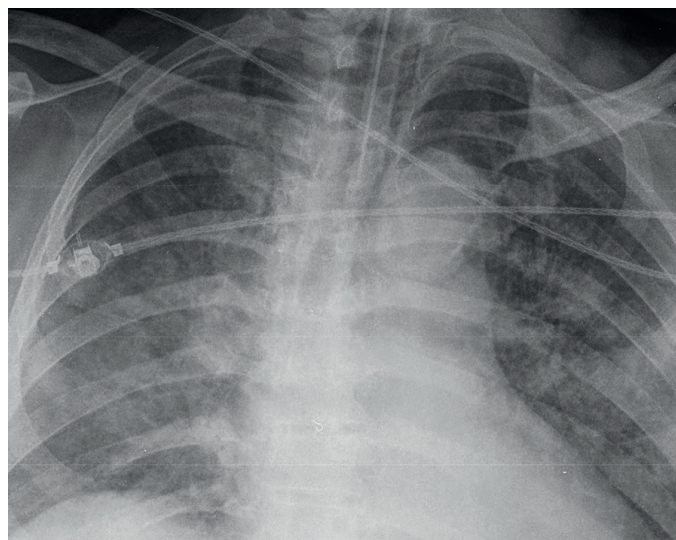


FIGURE 5. Case 2. Initial thoracic X-ray



FIGURE 6. Case 2. Control lung ultrasound after treatment

port were adjust, achieving a $\text{PaO}_2/\text{FiO}_2$ of 100 with SaO_2 96% 4 h later. Follow-up ultrasonography study was performed after 24 hours of the event finding no B lines (Figure 6) with laboratory studies showing normal arterial blood gases (pH 7.38, pCO_2 37.9 mm Hg [5.1 kPa], pO_2 82.8 mm Hg [11.0 kPa], HCO_3 22.3 mmol L^{-1} , BE -2.7 mmol L^{-1} , SpO_2 96.3%, $\text{PaO}_2/\text{FiO}_2$ 176). After 26 h of monitoring and regulating the respiratory parameters, control chest X-ray showed no pathological findings and a Pa/FiO_2 value of 217 was obtained. The patient was extubated with discharged 30 hours later with no immediate sequelae.

Negative pressure pulmonary oedema is considered a potentially life-threatening pathology. It is defined as a non-cardiogenic oedema characterised by a sudden increase or release of the inspiratory pressure posterior to an airway obstruction. NPPE type 1 is associated with a strong inspiration effort in the context of acute airway obstruction, while type 2 happens after the release of a chronic airway obstruction. Type 1 aetiology includes laryngospasm, epiglottitis, obstruction of the endotracheal tube, laryngeal tumour, and postoperative vocal cord paralysis. Conversely, type 2 is frequently caused by posterior adenoidectomy, tonsillectomy, and laryngeal mass resection [5]. The mechanism by which pulmonary oedema develops is the transmission of the intrathoracic negative pressure (up to -140 cc/ H_2O) to the intrapleural space, thus increasing venous return to the cardiac chambers, pulmonary venous pressure, as well as hydrostatic pressure and finally the formation of oedema [6, 7] – all in the presence of a hyperadrenergic state. According to severity, capillary injury can be reached with secondary

Actually, the incidence of NPPE varies from 0.1% to 11% [8], laryngospasm being responsible for 50% of cases, followed by obstructions of the upper airway by tumours, strange bodies, epiglottitis, and blockages

of the endotracheal tube. The laryngospasm is defined as the sustained closure of vocal cords with the loss of airway permeability; this protector reflex of the airway to prevents broncoaspiration usually presents in superficial anaesthetic planes. In the general population, the incidence of laryngospasm is approximately 1%, increasing to 10% in paediatrics with and hyperreactivity of the respiratory airways reaching up to 25% in children who have undergone tonsillectomy or adenoidectomy [9], of whom 4% to 11% can present NPPE [10]. The frequency of this complication is mainly found in young healthy adults ASA I and II, followed by children, and a smaller proportion in the elderly. It is known that NPPE is a potentially severe condition the early diagnosis and opportune treatment of which has to be established, and then ventilatory support with positive pressure should be started as soon as possible to prevent fatal outcomes. Nowadays, we know that 34% to 46% of the patients will need mechanical ventilation [6].

Actually, the diagnosis is based on the medical record and context of the patient. It may also be supported by imaging studies such as chest X-ray and in some specific cases the CT scan of the chest, both of which have low specificity for NPPE [11]. Pulmonary ultrasonography has had an increasing impact on critical patients

and perioperative patients, due to its precision, early diagnosis, and its dynamic characteristics compared to conventional radiography [12]. Ultrasound also has the advantage of being possible at the bedside or in surgery rooms, applying validated protocols such as BLUE that pretends to find a suggestive profile of congestion with numerous bilateral B lines associated with pulmonary slip, which achieves a 97% sensitivity and 95% specificity in the diagnosis of acute pulmonary oedema [13]. For the above, ultrasound assessment could play a fundamental role in the early diagnosis and follow-up of these patients without transferring them to radiology service or exposure to constant radiation [13].

The general approach of postoperative respiratory insufficiency is oriented towards ruling out an acute coronary event, heart failure, or PE; in fact, the diagnosis of NPPE is made late. The two cases we have presented herein have different ages, in which the ultrasonography helps to achieve an early diagnosis based on the BLUE protocol, which determinates the findings of multiple B lines (B profile) in an asymmetric way, and in all four quadrants, which means the presence of alveolar-interstitial syndrome concentrating thus the possibility of different entities: hydrostatic pulmonary oedema by ventricular dysfunction or water overload, acute respiratory distress syndrome (ARDS), transfusion-related acute lung injury (TRALI), or NPPE.

The pulmonary ultrasound is a fundamental instrument in the diagnosis of interstitial syndrome, with a higher performance than chest X-ray.

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REFERENCES

1. Lichtenstein DA, Meziere GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. *Chest* 2008; 134: 117–125. doi: 10.1378/chest.07-2800.
2. Labovitz AJ, Noble VE, Bierig M, et al. Focused cardiac ultrasound in the emergent setting: a consensus statement of the American Society of Echocardiography and American College of Emergency Physicians. *J Am Soc Echocardiogr*

- 2010; 23: 1225–1230. doi: 10.1016/j.echo.2010.10.005.
3. Broccard AF, Liaudet L, Aubert JD, et al. Negative pressure post-tracheal extubation alveolar hemorrhage. *Anesth Analg* 2001; 92: 273–275. doi: 10.1097/0000539-200101000-00055.
4. Ghofailly LA, Simmons C, Chen L, Liu R. Negative pressure pulmonary edema after laryngospasm: a revisit with a case report. *J Anesth Clin Res* 2013; 3: 252. doi: 10.4172/2155-6148.1000252.
5. Udeshi A, Cantie SM, Pierre E. Postobstructive pulmonary edema. *J Crit Care* 2010; 25: 508.e1-5. doi: 10.1016/j.jcrrc.2009.12.014.
6. Bhaskar B, Fraser JF. Negative pressure pulmonary edema revisited: pathophysiology and review of management. *Saudi J Anaesth* 2011; 5: 308–313. doi: 10.4103/1658-354X.84108.
7. Fremont RD, Kallet RH, Matthay MA, Ware LB. Postobstructive pulmonary edema: a case for hydrostatic mechanisms. *Chest* 2007; 131: 1742–1746. doi: 10.1378/chest.06-2934.
8. Krodel DJ, Bittner EA, Abdalnour R, et al. Case scenario: acute postoperative negative pressure pulmonary edema. *Anesthesiology* 2010; 113: 200–207. doi: 10.1097/ALN.0b013e3181e32e68.
9. Gavel G, Walker RWM. Laryngospasm in anaesthesia. *Continuing Education in Anaesthesia Critical Care & Pain* 2014; 14: 47–51. doi: 10.1093/bjaceaccp/mkt031.
10. Visvanathan T, Kluger MT, Webb RK, Westhorpe RN. Crisis management during anaesthesia: laryngospasm. *Qual Saf Health Care* 2005; 14: e3. doi: 10.1136/qshc.2002.004275.
11. Silva PS, Monteiro Neto H, Andrade MM, Neves CV. Negative-pressure pulmonary edema: a rare complication of upper airway obstruction in children. *Pediatr Emerg Care* 2005; 21: 751–754. doi: 10.1097/01.pec.0000186430.92388.a6.
12. de la Quintana Gordon FB, Nacarino Alcorta B, Fajardo Perez M. Basic lung ultrasound. Part 2. Parenchymal diseases. *Rev Esp Anestesiol Reanim* 2015; 62: 337–349. doi: 10.1016/j.redar.2015.01.011.
13. Lichtenstein D. Lung ultrasound in the critically ill. *Curr Opin Crit Care* 2014; 20: 315–322. doi: 10.1097/MCC.0000000000000096.

