Clinical Report

Negative pressure induced airway and pulmonary injury

K Bhavani-Shankar MD, Nada Saliba Hart MD, Phillip S. Mushlin MD, PHD

Purpose: To describe negative pressure injury occurring during the use of a laryngeal mask airway (LMA) in which airway bleeding rather than pulmonary oedema was the major complication.

Clinical Features: A patient presented to the day surgery unit for resection of a ganglion cyst on her right wrist. She underwent general anaesthesia using an LMA, and experienced severe laryngospasm and transient hypoxaemia (oxygen saturation to 66%) seven minutes after incision. This resolved within 90 sec of succinylcholine administration. Nonetheless, the LMA was removed, a tracheal tube was inserted atraumatically and positive pressure ventilation was maintained until the time of emergence, when fresh blood appeared in the tracheal tube. The blood ultimately became frothy, resembling pulmonary oedema fluid. Haemoptysis, continued postoperatively and led to the hospitalization of this ambulatory patient.

Conclusion: Rapid development of large subatmospheric pressures, as can occur during severe laryngospasm, may disrupt the tracheobronchial vasculature causing airway bleeding. This bleeding should be distinguished from negative pressure pulmonary oedema.

Objectif : Rapporter une lésion causée par la pression négative survenue pendant l'utilisation d'un masque laryngé (ML). La présence de sang au niveau des voies respiratoires provenait d'une hémorragie et non d'un oedème pulmonaire.

Éléments cliniques : Une patiente se présente à l'unité de chirurgie ambulatoire pour la résection d'un kyste au poignet droit. Pendant l'anesthésie générale au masque laryngé, il survient un laryngospasme grave avec hypoxie transitoire (saturation en oxygène à 66%) sept minutes après l'incision. La situation se rétablit à moins de 90 secondes après l'adminsitration de succinylcholine. On retire le ML et on introduit de façon atraumatique une canule endotrachéale. La ventilation mécanique est maintenue jusqu'au réveil alors que du sang frais apparaît dans la canule orotrachéale. Le sang devient plus tard spumeux comme dans le cas d'un oedème pulmonaire. C'est l'hémoptysie continue qui nécessite en postopératoire l'hospitalisation de cette patiente ambulante.

Conclusion : De grandes pressions sous-atmosphériques soudaines, comme au moment d'un laryngospasme important, peuvent traumatiser la trame vasculaire trachéobronchique et provoquer ainsi un saignement des voies aériennes. Il faut distinguer ce saignement de l'oedème pulmonaire par pression négative.

From the Department of Anaesthesia, Harvard Medical School, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, USA
Address correspondence to: Dr. K Bhavani Shankar.
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LARYNGOSPASM complicates the use of a laryngeal mask airway (LMA) in 1–3% of cases, most commonly during induction or emergence from anaesthesia.[1,2] This complication can be rapidly and effectively treated by applying positive airway pressure, increasing the depth of anaesthesia, or by administering succinylcholine. Despite such measures, abrupt airway obstruction and its relief is occasionally followed by the development of negative pressure pulmonary oedema (NPPE).[3–10] We report a case in which airway bleeding (presenting as postoperative haemoptysis) rather than pulmonary oedema was the major sequel of laryngospasm.

Case report

A 23 yr-old woman presented to day surgery for resection of a ganglion cyst on her right wrist. Her health was excellent, excepting mild asthma and cigarette smoking. She used a ventolin inhaler on rare occasions, but required no other medications and had no drug allergies.

Preferring general anaesthesia, the patient refused a regional technique. Prior to induction, 1 mg midazolam, 50 μg fentanyl, 10 mg metoclopramide and 0.2 mg glycopyrrolate *iv* were administered. Induction with 2.5 $mg \cdot kg^{-1}$ propofol proceeded smoothly. Following a brief period of ventilation by face mask (size 3), an LMA (#3) was easily and atraumatically placed. The cuff was inflated with 20 ml air (inflation leak pressure of 20 cm water) causing the shaft to move outward by 1 cm. The shaft was in a midline position. There were no abnormalities on auscultation and inspection of the chest; inspiratory inflation pressures during controlled ventilation were normal, as was the capnogram (ie, no prolongation of phase II or increase in phase III slope[11]).

Anaesthesia was maintained with isoflurane 0.6% in nitrous oxide 50%/oxygen 50%, using assisted ventilation for the initial 25 min. Thereafter, the patient was allowed to breathe spontaneously. An arm tourniquet was inflated to 250 mm Hg one minute before incision. Eight minutes later, the patient began to cough and exhibit evidence of increased sympathetic nervous system activity (heart rate increased from 82 to 126 bpm; blood pressure increased from 105/55 to 150/55 mm Hg). Our attempt to deepen anaesthesia with isoflurane was unsuccessful, as ventilation became progressively more difficult and ultimately impossible. The situation was not improved by administration of 150 mg thiopentone *iv*. Oxygen saturation progressively decreased from 100% to 66%. Since the LMA appeared to be positioned properly, we suspected that laryngospasm was the cause of the problem. Succinylcholine 80 mg *iv* was administered and, within minutes, oxygen saturation increased to 100%.

Nonetheless, because of the above mentioned problem, the LMA was removed and a #7.0 tracheal tube was inserted. Intubation was accomplished atraumatically while directly visualizing the vocal cords. At this time, we suspected that the problem had completely resolved. The anaesthetic continued uneventfully with isoflurane, 0.6%, in nitrous oxide/oxygen, using controlled ventilation for the remainder of the two hour operation.

During emergence from anaesthesia, however, bright red blood (approximately 15 ml) appeared in the tracheal tube. With time, the blood became frothy; initially it was red, then later pink. Auscultation revealed rhonchi (no wheezing or rales) which cleared after blood was suctioned from the tracheal tube. The patient was allowed to breathe spontaneously, had no respiratory distress, and was well oxygenated (SpO_2 = 100%; FIO_2 = 0.3). Her trachea was extubated and she received oxygen via face mask ($8\ l \cdot min^{-1}$).

In the Post Anaesthesia Care Unit (PACU), the patient continued to expectorate blood. She also complained of sharp bilateral chest pain in the superior mediastinal region of the anterior thorax. While breathing room air, her SpO_2 was 95% and the ECG was normal. Chest radiograph showed no abnormalities other than very mild diffuse airspace disease, which the radiologist interpreted as either mild pulmonary oedema or aspiration pneumonitis. A second radiograph, taken 4 ½ hr later, showed resolving airspace disease at the lung bases, which the radiologist felt was most consistent with aspiration pneumonitis stemming from the aspiration of blood. In spite of the rapid radiological improvement and the absence of hypoxaemia, the patient remained hospitalized for two days, owing to intermittent expectoration of blood (total volume approximately 100 ml). On follow-up at 18 mo, the patient was well, without recurrence of haemoptysis or chest pain.

Discussion

The present case involved intraoperative airway obstruction with a correctly placed LMA. The abrupt onset of obstruction was most likely caused by laryngospasm, precipitated by intense surgical stimulation during light anaesthesia. Consistent with this, succinylcholine improved ventilation and oxygen saturation within 90 sec: The capnogram and peak inflation pressures had returned to normal (10–14 cm water) and oxygen saturation was 100% within two minutes of succinylcholine administration. This rapid clinical improvement suggests that neither malpositioning of

the LMA nor bronchospasm precipitated the airway obstruction.

The appearance of frothy blood during emergence from anaesthesia initially suggested that negative pressure pulmonary oedema (NPPE) had occurred during the episode of laryngospasm, as inspiratory efforts against a closed glottis may generate substantial subatmospheric pressures within the thorax. Laryngospasm can trigger pulmonary oedema[4–10] but this has rarely been reported to complicate the use of an LMA.[3] In two cases, in which LMA insertion was difficult, chest retractions were readily apparent, hypoxaemia was severe, and chest radiographs displayed florid pulmonary oedema.[3] In our case, however, hypoxaemia was mild and transient, the radiological findings did not indicate severe pulmonary oedema, and the initial manifestation of pulmonary or airway injury was frank blood rather than frothy fluid in the tracheal tube. Furthermore, protracted expectoration of blood in the postoperative period unaccompanied by hypoxaemia in a patient breathing room air argues against severe NPPE as the predominant cause of the present complication.

We suggest that negative pressure injury (NPI) rather than NPPE was the main problem in the present case. We suspect that this phenomenon may occur more commonly than has been recognized previously. In our case, airway pressure changes may have injured tracheobronchial vessels to a greater extent than pulmonary vessels, predisposing to airway bleeding when the patient coughed during emergence from anaesthesia. The blood ultimately appeared frothy as it mixed with pulmonary gases during positive pressure ventilation. Initially, this was interpreted as florid pulmonary oedema, a diagnosis that in retrospect seems unlikely, as haemoptysis continued postoperatively without hypoxaemia. Aspiration of blood was felt by the radiologist to be the most likely explanation for the mild transient abnormalities noted on the chest radiographs.

The circulation of the lungs and airways includes a low pressure (pulmonary) and a high pressure system (bronchial vessels, systemic arterial pressures). The bronchial circulation supplies tracheal, bronchial and bronchiolar walls, and forms a vascular network in the tracheobronchial mucosa as far as the terminal bronchioles. During laryngospasm, subatmospheric intrathoracic pressures that develop can increase both intracardiac pressures and bronchial vascular resistance through an interplay of hydrostatic (pulmonary), neurogenic (hyperadrenergic), and cardiac mechanisms.[4,12] As a result, bronchial vasculature may be injured, causing focal haemorrhages in the tracheobronchial tree, as described using endoscopy.[12] Hence, haemoptysis could be the primary presenting feature of tracheobronchial injury, whereas disruption of more distal alveolar-capillary systems would present as pulmonary oedema. It would appear that, in our patient, the pulmonary component (NPPE) was minimal and the effects of tracheobronchial vascular disruption predominated. Conceivably, cigarette smoking and bronchitis, which may increase the friability of vascular mucosa,[13] could have increased susceptibility to negative pressure injury. The duration of haemoptysis may have reflected the time required for the vascular injury to heal.

Other possible causes of the haemoptysis include aspiration pneumonitis, airway trauma, tuberculosis, polyp, arterio-venous malformation and tumour. Aspiration of gastric contents is possible, as the LMA does not form an occlusive seal over the laryngeal inlet. However, this seems unlikely as the hypoxaemia resolved rapidly. Militating against trauma-induced bleeding were an absence of blood in or around the larynx during laryngoscopy and a lack of trauma on intubation.

It could be argued that, in cases such as ours, bronchoscopy is indicated to exclude pulmonary or airway pathology. On the other hand, general guidelines provided by Haponik et al suggest deferring bronchoscopy when haemoptysis occurs in patients < 40 yr, if no lesions are present on chest radiograph and episodes of bleeding are brief (< 1 week) or sporadic.[14] Since our patient arrived in PACU with a presumed diagnosis of NPPE, we felt that bronchoscopy was unnecessary. The persistence of haemoptysis prompted us to reconsider bronchoscopy, to characterize the extent and nature of any airway injury and to rule out co-existing pathology. However, this procedure was not performed as the bleeding decreased substantially in the early postoperative period, and the PACU attending staff felt that haemoptysis would be self-limited since it resulted from a transient negative pressure injury. Indeed, the absence of radiographic abnormalities or symptoms during an 18 mo follow-up interval indicates that our patient's haemoptysis was not caused by preexisting pulmonary or endobronchial lesions but rather by a negative pressure injury.

Conclusion

Airway bleeding in the present case most likely resulted from injury to tracheobronchial vessels as large subatmospheric airway pressures developed during laryngospasm. This negative pressure injury (NPI) was accompanied by transient hypoxaemia and frothy haemoptysis, which was initially misinterpreted as negative pressure pulmonary oedema.

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