Acute pulmonary edema and airway hemorrhage in a goat during sevoflurane anesthesia

C. Adami1, O. L. Levionnois1, C. Spadavecchia1

1Section of Anesthesiology and Pain Therapy, Department of Clinical Veterinary Science, University of Berne, Switzerland

Summary

A goat was scheduled for experimental surgery under general anesthesia. The first attempt of performing endotracheal intubation failed and provoked laryngeal spasm. After repeated successful intubation of inhalation anesthesia was delivered in high concentrations of sevoflurane. Suddenly hypertension and tachycardia were observed, followed by foamy airway secretion and then severe airway hemorrhage. The authors hypothesize that laryngeal spasm provoked respiratory distress and pulmonary edema. The delivered high concentrations of sevoflurane probably enhanced a hyperadrenergic response, predisposing to the development of airway hemorrhage.

Keywords: laryngospasm, airway hemorrhage, pulmonary edema, sevoflurane, inhalatory anesthesia.

Case History

A two year old, female, 55 kg Saanen goat was scheduled in an experimental setting for elective arthrotomy of the stifte joint. The experimental trial was performed with permission from the local committee for animal experimentation (Canton of Berne, Switzerland; permission number: 22/08). Clinical and hematological examination revealed no abnormal findings.

Results of anesthesia

A 14 G (80.0 mm) catheter was aseptically placed in the left jugular vein for intravenous (IV) drug administration and blood sampling. Following premedication with IV midazolam (0.3 mg/kg, Dormicum, Roche) and IV methadone (0.05 mg/kg, Methadon, Streuli), anesthe-
and an end-tidal gas analyser (Anandic Medical System, Datex-Ohmeda), and the auricular artery was cannulated for arterial blood pressure monitoring. Cardiovascular and respiratory variables were electronically recorded on a computer. Fluids (Ringer Lactate, Bischel) were delivered IV at 5 ml/kg/h. At the beginning of inhalational anesthesia, anesthetic depth was judged to be inadequate on the basis of the clinical evaluation (marked palpebral reflex and chewing were observed), therefore fresh gas flow was kept high (6L/minute) with a vaporizer setting at 5%. Hypertension (systolic arterial pressure (SAP) = 160 mmHg) and tachycardia (Heart rate (HR) = 130 bpm) developed rapidly within the first minutes of inhalation anesthesia.

A few minutes later 15 ml of frothy, foamy fluid was noted in the tracheal tube; this first secretion was attributed to pulmonary edema. Additional reddish, foamy fluid continued draining from the trachea, followed by a larger amount of pure blood. The bleeding started suddenly and was attributed to airway vasculature rupture. The goat’s head was lowered and the pharynx examined to check the position of the tube, which was judged to be correct. Sevoflurane delivery was immediately discontinued and the goat was briefly disconnected from the breathing system to allow suction of tracheal blood and fluids. Pure oxygen was then administered through the breathing system, to reduce bleeding. Atropine (Atropinum S.P.A.), 2 mg/kg IV, was administered IV to reduce fluid secretion. Vitamin K (Konakion, Roche), 5 mg/kg IV, and furosemide (Dimazon, Veterinaria AG), 2 mg/kg IV, were also given. Systolic arterial pressure progressively decreased to 110 mmHg. A light plane of anesthesia was maintained by administering IV propofol bolus, and controlled ventilation continued until no more fluids were drained out of the trachea. Then the animal was weaned from the ventilator and, as soon as it could maintain SpO2 above 97% breathing spontaneously, it was disconnected from the breathing system and allowed to breathe room air. The goat was then allowed to recover. SpO2 monitoring was continued for 10 minutes while breathing room air in order to ensure adequate hemoglobin saturation without oxygen supplementation. The total amount of fluids, including pure blood and foamy secretions, collected through active suction was 1200 ml. Although generalized weakness and mild hypothermia (T = 36 °C) were observed at awakening, the recovery phase was smooth and uneventful. Colloids were administered IV with a rate of 2 ml/kg/h during the following 2 hours until the goat was returned to the barn.

**Discussion**

The case present describes the occurrence of pulmonary edema and airway bleeding as anesthesia-related complications in a goat. Pulmonary edema is a recognized complication of acute upper airway obstruction, as laryngeal spasm, in humans (Lang et al., 1990), horses (Tute et al., 1996; Senior, 2005) and dogs (Kerr, 1989). Inspiratory efforts against a closed glottis, as observed in this goat during laryngeal spasm, can result in rapid decrease in intrathoracic pressure which, in turns, causes an increased transmural pressure gradient for all intrathoracic vascular structures (Tute et al., 1996; Senior, 2005). In addition, negative intrathoracic pressure that develops during laryngeal spasm can increase both intracardiac pressures and bronchial vascular resistance through an interplay of different mechanisms, for example hydrostatic forces and neurogenic changes (increased pulmonary resistance, pulmonary blood pooling, altered capillary integrity) (Lang et al., 1990). As a result, bronchial vasculature can be injured, causing hemorrhage in the tracheobronchial tree; disruption of the more distal alveolar capillaries might occur as well (Bhavani-Shankar et al., 1997).

In this goat tachycardia and hypertension were observed at the beginning of inhalatory anesthesia before pulmonary edema occurred. It is recognized that intubation itself, due to mechanical stimulation of the airways, can increase blood pressure and sympathetic efferent activity (Tanaka et al., 1996) and provoke bronchospasm, which could persist during general anesthesia and further enhance the adrenergic response, even though the ETT is gently placed (Hirshman and Bergman, 1990; Lang et al., 1990). In the case presented here, the first attempt of intubation failed and a relevant mechanical stimulation of the larynx occurred until the ETT was successfully positioned; consequently, intubation itself might have played a role in initiating tachycardia and hypertension.

The systemic adrenergic response observed in this goat might have been provoked or aggravated also by hypoxemia, which probably took place during laryngeal spasm. Hypoxic Pulmonary Vasconstriction (HPV) develops in response to alveolar hypoxia and probably contributed to further increase the hydrostatic pressure and the vascular resistance at the level of the pulmonary circulation. However, the role of alveolar hypoxia in aggravating pulmonary edema is controversial (Senior et al., 2005). Beside
local HPV, cerebral hypoxia, which might have occurred in this goat as a consequence of hypoxemia, can also lead to sympathetic nervous discharge, resulting in systemic massive adrenergic response called hyperadrenergic state (Lang et al., 1990). In addition, hemodynamic changes associated with marked hypoxemia include also redistribution of blood flow from the systemic circuit to the pulmonary vessels (Lang et al., 1990), which could have contributed to the development of pulmonary hypertension and edema in this goat. Predisposed by the ongoing modifications of the pulmonary vasculature as described above, another factor besides laryngeal spasm, hyperadrenergic state and hypoxemia might have, enhanced the pulmonary hypertension, aggravating edema formation and leading to airway vasculature disruption and hemorrhage. Several studies showed that rapid increases in inhalation anesthetics concentrations can induce tachycardia and hypertension, due to increased catecholamine plasma levels and «neurocirculatory excitation» (Yli-Hankala et al., 1993; Weiskopf et al., 2002). Because anesthetic level was judged to be too superficial in the first few minutes of inhalational anesthesia, high fresh gas flow rates and high sevoflurane concentrations were employed to deepen anesthesia as soon as possible. Thus, a high wash in occurred and could have played a role in determining or enhancing the tachycardia and hypertension observed at the beginning of inhalatory anesthesia. In conclusion, it is believed that the laryngeal spasm observed in this goat induced a marked decrease in intrathoracic pressure which caused hypoxia, catecholamine release and bronchoconstriction. These changes led to pulmonary edema. It is also hypothesized that the high sevoflurane wash in at the beginning of inhalatory anesthesia enhanced the hyperadrenergic state, contributing to airway vasculature injury and consequent hemorrhage.

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References


Corresponding author

Dr. Chiara Adami
Section of Anesthesiology
Department of Clinical Veterinary Science
Vetsuisse Faculty
University of Berne
Längasstrasse n. 124
CH-3012 Berne
Phone: + 41 (0)31 631 27 91
Fax: + 41 (0)31 631 26 20
E-Mail: chiara.adami@knp.unibe.ch

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