

Bronchospasm After Intravenous Lidocaine

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IV lidocaine (1.5 mg/kg) administered to facilitate endotracheal intubation was associated with transient bronchospasm in a 17-month-old-female with mild intermittent asthma. Immediately after lidocaine administration, the patient developed diffuse bilateral expiratory wheezes and dramatic increases in peak inspiratory pressure. Over approximately 5 min the episode resolved and an uneventful anesthetic course followed. This is consistent with recent clinical studies suggesting that IV lidocaine may cause airway narrowing in asthmatics. Practitioners should be aware of this potential complication.

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Asthma is a major source of mortality and morbidity in both adult and pediatric populations,¹ and its incidence and severity continue to increase in the developed world.² Asthma presents anesthesiologists with a unique set of challenges, especially in those patients requiring endotracheal intubation, because stimulation of the airways can induce reflex bronchoconstriction.³ Fortunately, with proper management, most of these patients tolerate anesthesia well.⁴ To prevent reflex bronchoconstriction caused by endotracheal intubation, inhaled β_2 agonists, anticholinergics, aerosol and IV lidocaine are often administered as adjunct medications.⁵⁻⁸ IV lidocaine also significantly improves intubating conditions when used as part of an inhaled induction sequence in children that does not include neuromuscular blocking drugs.⁹ However, aerosolized lidocaine can increase airway resistance in asthmatics,⁵ and a recent study shows that IV lidocaine reduces airway diameter in adult asthmatics.⁶ Consistent with these latter observations, we report a case of bronchospasm associated with IV lidocaine in a child with asthma.

CASE DESCRIPTION

A 17-month-old, 9 kg female required anesthesia for upper endoscopy. She had no known drug allergies and no surgical history. Her medical history was significant for intermittent vomiting, seasonal allergies, and mild intermittent asthma. Her medications included nebulized albuterol and budesonide, used only during exacerbations. It had been several months since her last asthma exacerbation and use of asthma medications. On physical examination, her

lungs were clear to auscultation. Because of her history of asthma, she was treated prophylactically with 2.5 mg nebulized albuterol 10 min prior to anesthetic induction.

Oral midazolam (7 mg) was given 30 min before induction. An inhalation induction was performed using sevoflurane (up to 8% inhaled concentration) and 50% nitrous oxide. An oral airway was inserted and positive-pressure ventilation instituted by bag-mask ventilation with peak airway pressures of approximately 15 cm H₂O noted on the circle system pressure gauge. Approximately 10 min after induction began, IV access was obtained on a second attempt and 13 mg of lidocaine (approximately 1.5 mg/kg) was administered IV to facilitate endotracheal intubation. Immediately after lidocaine injection, the patient developed diffuse bilateral expiratory wheezes and a dramatic increase in peak inspiratory pressures to more than 40 cm H₂O with continued bag-mask ventilation by an experienced pediatric anesthesiologist (DOW). Nitrous oxide was discontinued and the patient was ventilated using 100% O₂. Oxygen saturation by pulse oximetry was maintained at 100% throughout, and both arterial blood pressure and heart rate were stable. No rashes or other signs of anaphylactic or anaphylactoid reactions were observed, nor was there evidence of regurgitation at this or subsequent times. Over approximately 5 min, the wheezing resolved without further interventions and peak airway pressures returned to approximately 15 cm H₂O. The trachea was intubated without the administration of further medications, with no wheezing noted after intubation. Anesthesia was maintained using sevoflurane and oxygen. The rest of the anesthetic course was uneventful, with no further evidence of bronchospasm. The upper endoscopy examination was normal, with no evidence of reflux or regurgitation. The trachea was extubated at the conclusion of the case when the child was responsive, without administration of further medications. No respiratory symptoms were noted postoperatively.

DISCUSSION

Bronchospasm is an infrequent⁴ but potentially serious perioperative complication. Several strategies have been used to prevent perioperative bronchospasm, including pretreatment with inhaled β_2 agonists or anticholinergics, avoidance of barbiturates for induction, and using an anesthetic technique which relies heavily on inhaled drugs and opioids.^{7,8,10} Considerable experimental evidence also supports the use

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of lidocaine. In dogs, IV lidocaine significantly attenuates methacholine and vagal nerve stimulation-induced bronchoconstriction.¹¹ The mechanism of benefit is likely multifactorial. Some studies find that lidocaine in high concentrations directly relaxes isolated airway smooth muscle *in vitro*, but these concentrations exceed those achieved with IV administration.^{12,13} Rather, lidocaine most likely attenuates neurally mediated reflexes that mediate bronchoconstriction.^{13–16}

In humans, IV lidocaine attenuates responses to inhalation challenges in awake subjects with airway hyperactivity.¹⁷ IV lidocaine also attenuates intubation-induced bronchoconstriction in some,¹⁴ but not all,⁷ studies of anesthetized humans. IV lidocaine has also been used as an adjunct to improve intubation conditions in the absence of neuromuscular blocking drugs. Warner et al. found that 1.5 mg/kg of IV lidocaine administered 90 s prior to laryngoscopy improved intubating conditions in children induced and anesthetized with halothane.⁹

Although lidocaine can attenuate reflex-induced bronchospasm, it may also cause airway smooth muscle contraction in the absence of reflex stimulation. In contrast to the above-mentioned studies demonstrating a direct relaxing effect of lidocaine on isolated airway smooth muscles, other studies find that it constricts isolated airways.^{18,19} In anesthetized dogs, lidocaine increases basal airway smooth muscle tone and enhances histamine and serotonin-induced bronchoconstriction.^{11,20} In humans, aerosolized lidocaine acutely increases airway resistance and decreases expiratory flow in awake patients with reactive airway disease.⁵ Recently, Chang et al. observed that, in awake asthmatics, IV lidocaine decreased airway diameter at total lung capacity (but not functional residual capacity) as measured by computed tomography scanning, resulting in a small but significant reduction in forced expiratory volume in 1 min.⁶ The mechanism responsible for this airway narrowing is not known. Chang et al. suggested that systemic lidocaine may act centrally, leading to reduced activity of the nonadrenergic, noncholinergic bronchodilatory system, the primary bronchodilatory mechanism of the human lung.^{15,16} They recommended auscultating the airways during IV lidocaine administration due to the potential to induce bronchospasm, but did not provide any clinical examples.⁶

We are not aware of prior reports of clinically apparent bronchospasm clearly associated with IV lidocaine during anesthesia. We infer a causative role of lidocaine based on biological plausibility suggested by the above reports, and the lack of other inciting causes. Lidocaine was administered approximately 10 min after start of induction, when a deep level of anesthesia had been established for several minutes (8% sevoflurane inspired with 50% N₂O) to the point of apnea requiring positive pressure ventilation. The event occurred immediately

after injection of lidocaine without other interventions, with oral airway in place and no evidence of upper airway obstruction. It is possible that the lidocaine formulation induced an anaphylactic or anaphylactoid reaction. However, no other signs of such a reaction, such as hypotension or rashes, were observed and the episode was brief and self-limited. The lidocaine formulation did not contain preservatives that could have caused a reaction. It is also possible that the bronchospasm occurred as a spontaneous exacerbation of the patient's underlying asthma. However, the self-limited nature of the symptoms, and the lack of further symptoms in the perioperative period (including the lack of a response to endotracheal intubation) argues against this possibility. We cannot exclude that unrecognized aspiration caused bronchospasm. However, there were no obvious signs of regurgitation on direct laryngoscopy and the endoscopic examination was normal. Also, we believe it is unlikely that aspiration sufficient to cause such significant bronchospasm would be self-limited, without any sequelae.

In conclusion, we present a report of intraoperative bronchospasm associated with the administration of IV lidocaine as an adjunct to facilitate endotracheal intubation. The mechanism is not known, but is consistent with prior clinical studies suggesting that IV lidocaine may cause airway narrowing in asthmatics. IV lidocaine is a useful adjunct to laryngoscopy in children undergoing inhaled induction, but practitioners should be aware that paradoxical bronchospasm is apparently possible with its administration in this setting.

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