Letter to the Editor

KETAMINE FOR STATUS ASTHMATICUS WITH RESPIRATORY FAILURE

To the Editor:

In regards to Shlamovitz and Hawthorne’s case report on the use of “dissociative dose” ketamine in status asthmaticus with acute respiratory failure, I am hesitant to try the authors’ prescription before exhausting conventional and well-proven treatment modalities (1). In addition, the use of ketamine in anesthetic doses in patients in extremis is not without its pitfalls.

In asthmatics with respiratory failure, it is crucial for physicians, nurses, and respiratory therapists to understand that there are two, and only two, physiologic urgencies once the usual inhaled bronchodilators fail: first, the respiratory rate must be reduced, and second, the chest must be deflated. Reducing the respiratory rate increases the time available for exhalation, the latter being “mother’s milk” to asthmatics. The best way to do that is with a bolus of fentanyl—it is potent, has a rapid onset and short duration of action, and it works. Concomitantly, high enough levels of continuous positive airway pressure (CPAP) should be applied to stent open the airways and allow the hyperinflated chest to deflate, thereby restoring the mechanical advantage of the expiratory respiratory muscles. CPAP is the specific antidote for all kinds of physiologic airway obstruction, especially obstructive lung disease.

Although ketamine may have bronchodilatory properties that may or may not have some salutary role in the rescue of such acute asthmatic patients, decades of unequivocal clinical success with opiates and CPAP put ketamine a long way down the line in the armamentarium available to the emergency physician to treat asthmatics with respiratory failure. I do not understand the authors’ failure to utilize those methods, especially given the notation about their patient’s high respiratory rate and hyperinflated chest. Opiates and CPAP should have been employed even before epinephrine was given. As an unrelated and separate matter, equally puzzling was the decision to stick the patient for a blood gas, of all things. Blood gases have no decision-making role in the management of asthmatics with preintubation acute respiratory failure—defined clinically by failure of work of breathing (ie, fatigue). I do not know any simpler measure to clinically assess than that. For sure, blood gases have no role in the decision to intubate an asthmatic. The only possible role blood gases might have in asthmatic patients is in deciding whether or not a patient should be admitted to the floor or the intensive care unit. But even then, a much better and easier index to use for that purpose is simply the room air SpO₂ (peripheral oxygen saturation).

Finally, it appears that it is the authors’ purpose to suggest, specifically, an anesthetic dose of ketamine as rescue therapy. Their dissociative dose, 0.75 mg/kg (and I am assuming that is ideal body weight, not actual body weight), is most definitely an anesthetic dose in a patient with acute respiratory failure. I am a bit alarmed by that idea because as both a critical care physician and an anesthesiologist, the last thing I would do to a patient who is hypoxemic with the kind of respiratory failure described is administer an anesthetic dose of any agent (unless it was immediately before tracheal intubation). That is most certainly a paradigm that would be highly unusual in the entire realm of clinical medicine, and is not something to be undertaken lightly. And even though ketamine is known to maintain spontaneous ventilation and hemodynamics, the effect of an anesthetic dose on a patient in extremis is entirely unpredictable (speaking from large experience). On the other hand, if the authors administered ketamine as a last ditch effort to avoid intubation while they stood over her with laryngoscope in hand, that, in fact, might be an excellent idea (the ketamine serving as the induction dose for intubation if it failed to work to salvage the patient).

Although the patient described in the case report had an excellent outcome, ketamine has failed to consistently produce such results, as the authors’ own literature search revealed. Furthermore, it cannot even be concluded that bronchodilatation was the reason for its success in this case. Other conceivable mechanisms of actions include a relaxation of chest musculature or slowing of the respiratory rate, either one secondary to ketamine’s sedative/anesthetic effect.
The bottom line is that the efficacy and side effect profile of ketamine does not compare with the predictable efficacy and safety of opiates and CPAP. As the authors conclude, ketamine should be reserved for patients who fail standard therapies. Its use in anesthetic doses might have some role as rescue therapy in acute asthma with respiratory failure, but should be undertaken only with immediate readiness to address both respiratory and circulatory collapse.

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