Short Communication

Interventional Cardiology

Effects of postoperative nebulized furosemide and budesonide on the postoperative sore throat in patients undergoing maxillofacial surgery: A randomized controlled trial

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Description

As an intravenous traditional diuretic, a common loop diuretic, furosemide has been used for inhalation therapy in respiratory medicine in recent years. According to relevant studies, furosemide aerosol inhalation may be useful in relieving dyspnea in patients with Chronic Obstructive Pulmonary Disease (COPD) and acts with a bronchodilator effect on the airway epithelium [1], nevertheless, all published studies have shown great variability in response. Vahedi, et al. [2] concluded that inhaled furosemide provides relief for induced dyspnea in people with COPD and it was noted that there was a significant improvement in mean FEV1 and FVC. Likewise, Jensen, et al. [3] reported statistically significant alleviation of exercise-induced dyspnea in people with COPD following inhaled furosemide therapy. However, the study has come to the opposite conclusion by Ghaysouri, et al. [4], which may be related to the different studies using different indicators of COPD improvement (Including the use of the patient's subjective perception through the visual analog scale and the numerical rating scale, and the objective analysis of lung function values to measure dyspnea) and the different clinical courses of the participants at the time (clinically stable or who are going through an acute exacerbation). It should be noted that the correlation between pulmonary function values and symptoms is not strong. Thus, assessment of the patient's subjective dyspnea score is essential for assessing the patient's stage of COPD properly. The difficulty with quantifying the sensation of dyspnea is that it is experienced differently among patients and may be caused by a multitude of factors. Apart from that, the explanation for this wide variability in treatment effects is unclear, but several other mechanisms can be postulated to explain the lack of treatment effects in some individuals. Inconsistency in aerosol administration is one of the most obvious possible causes. Control of inspiratory flow or inspiratory volume during aerosol administration is difficult to standardize. Inspiratory flow and tidal volume are key factors in the efficiency and location of particle deposition; if the flow is high, particles are deposited in the oropharynx and upper airways rather than deep in the lungs. In addition, when administered through an inhalation nebulizer, exhalation causes aerosol loss into the room, resulting in the actual inhaled dose varying depending on the proportional difference between inhalation and exhalation times. Finally, the variability in response to nebulized furosemide may also be due to differences in the sensitivity of pulmonary stretch receptors to the relief of dyspnoea in different individuals. For these reasons, Panzini, et al. [5] used a laboratory model of dyspnoea in which graded hypercapnia was delivered to healthy subjects during restrictive ventilation, while controlling inspiratory flow and tidal volume during aerosol administration. It

was concluded that inhaled furosemide alleviated the challenge of respiratory distress associated with experimental dyspnoea, but that treatment effects were equally variable between subjects. Almost all subjects experienced some relief of dyspnoea following furosemide inhalation and none reported worsening of respiratory distress. Currently, inhaled furosemide is not a standard therapy for dyspnea relief and improvement of pulmonary function values in people with COPD. Additionally, all studies assessed the temporary nature of inhaled furosemide and the short-term impact on dyspnea and pulmonary function values, as opposed to any long-term effects [6]. To date, while the therapy of inhaled furosemide is promising for dyspnea relief and improvement of pulmonary function values in people with COPD, there is still insufficient data to draw definitive conclusions regarding the effectiveness of inhaled furosemide in this case.

In addition, Murad, et al. found furosemide to be useful for bronchial asthma [7]. The results of the study showed that inhaled furosemide significantly reduced the increased levels of inflammatory cells in Airway Hyperreactivity (AHR), IL-6, TNF-α and Bronchoalveolar Lavage Fluid (BALF) as well as inflammatory cell infiltration in the lungs of ovalbuminous asthmatic mice and it enhances the anti-asthmatic effect of albuterol. However, the underlying mechanism is not known.

Furthermore, nebulized inhalation of furosemide dilutes sputum to facilitate expectoration, while suppressing inflammatory reactivity and playing an antiviral effect [8]. The coronavirus infection has evolved into a global crisis. Following initial viral infection, the host's inflammatory response often leads to an overproduction of inflammatory cytokines (e.g. IL-6 and TNFα) that develop into a 'cytokine storm', causing severe lung tissue damage. Wang Z, et al. found that nebulized inhalation treatment with furosemide reduced the production of pro-inflammatory cytokines (including IL-6 and TNF-α) and promoted the production of anti-inflammatory cytokine products as Interleukin-1 Receptor Antagonist (IL-1RA, arginase) [8]. Thus, furosemide may be an agent capable of treating the cytokine storm of coronavirus disease, that is safe, easy to synthesize, handle and store, and available in reasonable quantities globally. Therefore, furosemide may be a candidate for inhalation therapy against COVID-19.

Our study also confirmed that nebulized inhalation of furosemide reduced the degree of sore throat and significantly reduced postoperative hoarseness in patients after maxillofacial surgery, as well as influencing foreign body sensation in the pharynx.

Conclusion

Due to the above results, it needs to be further explored whether nebulized inhaled furosemide can be used in the field of cardiac interventions for the treatment of airway hyperreactivity due to contrast drug allergy, or airway edema due to cardiogenic pulmonary edema. Nevertheless, even though several studies are currently examining the potential benefits of inhaled furosemide, there is still a lack of sufficient clinical evidence to support its use as one of these therapies, and the mechanisms of action of these potential therapies are not yet fully understood. Therefore, further research is essential. In our opinion, this article provides clinicians with new ideas for clinical treatment and prompts them to focus on the possibility of using old drugs in new areas.

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