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CASE REPORT

Nebulized Lidocaine as an Alternative Therapy for Reactive Airway Dysfunction Syndrome

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Abstract

Reactive airway dysfunction syndrome (RADS) is a variant of irritant-induced asthma that develops in subjects without prior bronchoobstructive disease, following high-level exposure to nonimmunogenic irritants. Recommended maintenance treatment for RADS is not different from asthma. But in some cases, severe symptoms may persist despite the bronchodilators and corticosteroids. We describe the first case of a patient with RADS, unresponsive to all medical agents, who was successfully treated with lidocaine.

KEYWORDS: Asthma, sodium hypochloride, lidocaine

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INTRODUCTION

Reactive airway dysfunction syndrome (RADS) or acute-onset irritant-induced asthma is a distinct subset of irritant-induced asthma that develops in subjects without prior pulmonary disease, within 24 hours of high-level exposure to nonimmunogenic irritants such as corrosive gas, vapor, aerosol or smoke [1]. The most frequently reported agents are chlorine, toluene di-isocyanate, oxides of nitrogen and sodium hypochlorite (bleach, 40%), which is also a common agent that is widely used for indoor cleaning in rural regions [2,3]. Non-specific bronchial hyperresponsiveness is characteristic of the disease. RADS usually persists for more than a few weeks after cessation of exposure and an accurate diagnosis depends on a compatible history in the absence of any other pulmonary disorder [4,5].

For patients with RADS who require long-term pharmacologic treatment for persistant symptoms that simulate asthma (cough, wheeze and dyspea), the stepwise method as described in the asthma guidelines is followed even though it has not been formally assessed in this setting [6]. Thus, treatment failure with this approach is commonly experienced [1].

CASE PRESENTATION

We report the case of a 51-year-old female with asthma-like symptoms persisting 1 year after she had an accidental exposure to sodium hypochlorite by inhalation. Immediately after that event, she developed shortness of breath and started to cough persistently. She was subsequently hospitalized because of severe respiratory distress. One week later, she was discharged from the hospital, but noted persistent shortness of breath, cough and increased airway excitability after exposure to nonspecific stimuli such as perfume, cold air and vapor. She was then treated with aerosol bronchodilators. However, her symptoms persisted and necessitated more than 10 emergency admissions per month after severe episodes of bronchospastic responses to many various environmental stimuli. During these visits, she was diagnosed with severe asthma, and she received intermittent injections of high dose corticosteroids. She was then referred to our emergency department and hospitalized. Despite intensive treatment for over three weeks, including nebulized beta-2agonists, anti-cholinergic drugs, nebulized magnesium sulfate, oral methylpredisolone, theophylline and a leukotriene receptor-antagonist, there was no obvious improvement in her severe cough, wheeze or bronchial hyperresponsiveness.

This patient was a lifetime non-smoker, had no preexisting respiratory complaint before the exposure to sodium hypochlorite and had no prior history of allergies to drugs, foods or aeroallergens. She also developed significant adverse effects as a consequence of systemic glucocorticoid administration. She became obese (body mass index, 33.3 kg/m^2) and showed characteristics of Cushing's syndrome. She was unable to speak in complete sentences because of coughing and wheezing. Her respiratory rate was 32-breaths/min. Chest auscultation revealed a diffuse wheeze. Other physical examination findings and blood test results were normal. Her peak expiratory flow value was 330 L/min (estimated value for her age and height was 403 L/min) and oxygen saturation was 98%. For lung function tests, she

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could not complete the required 6-second expiration due to her cough reflex; therefore the test was not acceptable. Chest X-ray film, computerized tomography scan results, skin prick tests for common inhalant allergens and larynx examination for vocal cord dysfunction were normal. A diagnosis of RADS was established based on the history and laboratory findings.

The worsening of the patient's clinical situation and secondary effects of steroid use necessitated steroid-sparing treatment. We observed her episodes of bronchial hypersensitiveness after nonspecific stimuli (such as deep breath, odors, cold and stress) began with an unpreventable severe cough reflex followed by a severe bronchoconstriction. We then attempted to suppress her cough reflex with the inhalation of a local anesthetic agent, lidocaine. Written informed consent was obtained from patient.

The patient's weight was 80 kg and a dose of 5 mg/kg nebulized lidocaine was administered. Four times daily, 5 mL of a 2% injectable lidocaine solution without preservatives was introduced 1 hour before meals to prevent possible aspiration resulting from oral and pharyngeal hypoesthesia. Her symptoms such as wheezing or cough dramatically improved following this treatment. After 2 weeks of treatment, the patient was able to reduce oral steroids and discontinue the drugs, and the effects secondary to exogenous hypercortisolism decreased. Subsequently, we reduced the dosage of lidocaine, and continued her treatment with a 2% lidocaine pump spray on demand. She was discharged from the hospital and after 2 years of follow-up, she is asymptomatic with no emergency room visits since she was discharged.

DISCUSSION

The approved treatment for patients with established RADS is not different from that of any other asthmatic; preventive measures for further accidental high-level irritant exposure are also suggested [5]. However, an asthma-like therapeutic approach to this syndrome may be inadequate in some persistent cases. In addition to relieving symptoms with bronchodilators, the treatment should be given to reduce the nonspecific bronchial responsiveness. Therefore, we suggest that a local anesthetic agent may suppress the neurogenic inflammation and reflex bronchial hyperactivity.

Lidocaine is a common local anesthetic that is frequently nebulized during bronchoscopy procedures. Nebulized lidocaine is an effective and safe therapy in subjects with refractory cough and mild-to-moderate asthma in adults and children [7,8]. Hunt and colleagues demonstrated in a randomized placebo-controlled study that nebulized lidocaine is a useful anti-inflammatory asthma treatment and it may even be an alternative to glucocorticoids. However, previous studies suggest that lidocaine produces an initial reflex-mediated bronchoconstriction in patients with asthma and hyperirritable airways. Thus, caution is needed especially for its first use [9]. We also recommended to our patient not to eat or drink for 1 hour after nebulization because of its anesthetic effect. Lidocaine toxicity occurs only when serum levels exceed 5 to 6 µg/mL, and it includes lightheadedness, tremors, hallucinations, muscle twitching, seizures, arrhythmias, paresthesias and respiratory arrest [7,10]. Patients with hepatic disease should be monitored closely because of decreased drug metabolism and elimination rates. A generally accepted safe range of nebulized lidocaine is between 100 and 200 mg per dose [10].

To our knowledge, the case described in this article is the first reported case of RADS where symptom relief is obtained using nebulized lidocaine. In conclusion, nebulized lidocaine was well tolerated in our patient and could be a useful alternative for patients with RADS. This observation merits further studies to confirm the benefits of lidocaine for this group of patients, to better define other possible adverse effects and to obtain a possible place in routine treatment of RADS.

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