

Defecation-related Asthma

Satoshi Ano, Yuko Morishima, Yukio Ishii, Mio Kawaguchi,
Yosuke Matsuno and Nobuyuki Hizawa

Abstract

We herein report the case of a 39-year-old man with recurrent asthma exacerbations preceded by abdominal cramps with the urge to defecate. The patient had a history of near-fatal asthma associated with these gastrointestinal symptoms starting five years before his admission. He stated that, even when his daily asthma symptoms were under control, he suffered from attacks, especially when he had a strong urge to defecate. Although the contribution of increased parasympathetic tone to the onset of bronchospasms was likely, anticholinergics were not effective. Instead, the patient's symptoms successfully improved following the prophylactic use of laxatives, which might therefore be an appropriate therapeutic option for this type of asthma.

Key words: asthma, bronchospasm, defecation, exacerbation, laxative

(Intern Med 52: 685-687, 2013)

(DOI: 10.2169/internalmedicine.52.8943)

Introduction

Asthma is a chronic inflammatory airway disorder that is usually associated with exacerbations, including episodes of rapidly progressive asthma symptoms and airflow limitation. Asthma exacerbations vary from mild to life-threatening and may worsen within a couple of days or sometimes even within a few minutes. We herein present a case of asthma with severe acute bronchospasms associated with defecation. The precise mechanisms underlying defecation-induced bronchospasms remain unclear. However, physicians should be aware of this certain type of asthma in which increased parasympathetic tone or increased levels of chemical mediators are responsible for triggering bronchospasms.

Case Report

A 39-year-old man was admitted to our hospital with a history of recurrent asthma exacerbations that were preceded by abdominal cramps with the urge to defecate. The patient had a history of asthma starting at 28 years of age and was usually treated with inhaled short-acting beta-agonists. When he was 34 years old, he once experienced a near-fatal asthma attack. He had had signs of the common cold with a slight wheeze for a few days before the episode; however he

had been able to continue to work as usual. On the day of the attack at 7:00 am, he was awakened by abdominal cramping and pain with the urge to defecate. He developed increasing shortness of breath within several minutes while he was in the bathroom and was brought by ambulance to the emergency department. Upon arrival to the emergency room (ER), the patient was unconscious and exhibited mandibular breathing shortly followed by cardiopulmonary arrest. Fortunately, he was successfully resuscitated and since then has been placed on long-term control medications by his general physician. Even with the medications, the patient's frequent exacerbations did not improve. Therefore, he consulted our hospital for further evaluation. He had undergone abdominal surgery for neonatal gastric rupture at birth and for inguinal hernia twice, once at birth and at 32 years of age.

On examination, the patient was found to be totally asymptomatic with normal vital parameters and an SpO₂ of 98% on room air. Lung auscultation revealed normal breath sounds with no crackles or wheezes. Further physical examinations showed no abnormalities. Electrocardiogram showed a sinus rhythm without any abnormalities, and the patient's respiratory function was within the normal limits. There was no evidence of either hyperinflation or lung infiltrates on chest X-rays. The total IgE level was markedly elevated to 1,968 IU/mL (normal range: 0-170 IU/mL), and specific IgE

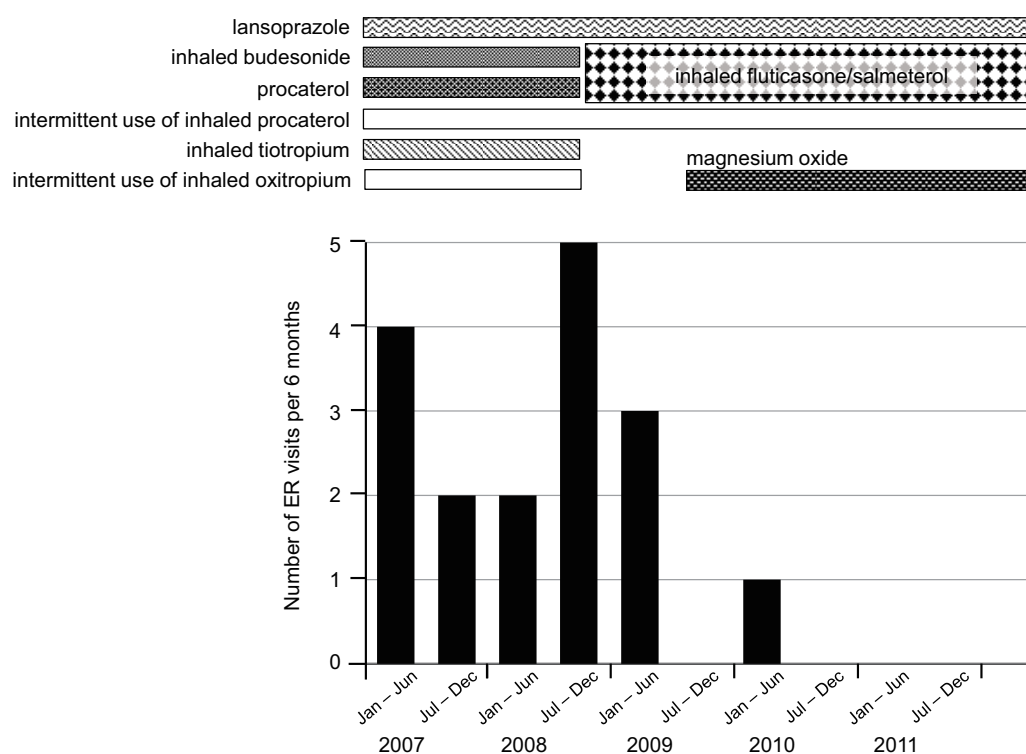


Figure 1. Frequency of emergency room (ER) visits. Long-term control medications, including inhaled budesonide, procaterol, lansoprazole and inhaled tiotropium with intermittent use of short-acting inhaled procaterol and inhaled oxitropium, did not reduce the number of ER visits. However, our patient visited the ER only once after he started using prophylactic laxatives in addition to the standard asthma treatment.

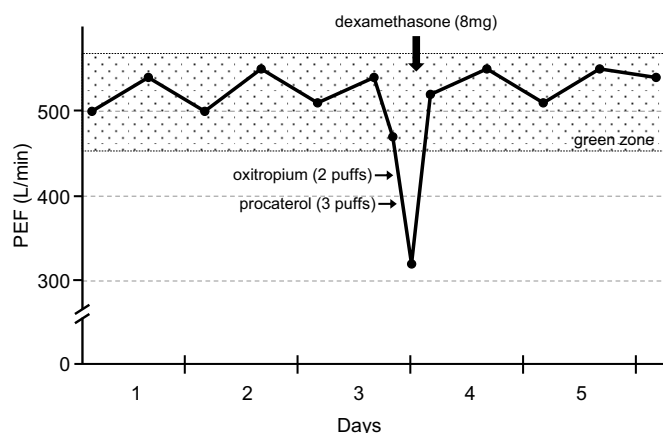


Figure 2. Recordings of the peak expiratory flow (PEF) chart showing a typical time course of asthma exacerbation in our patient. The PEF reading had been in the green zone with minimal variation. However, the PEF level suddenly decreased after the patient woke up with the urge to defecate. In spite of using a short-acting bronchodilator, within 30 minutes, the PEF level reached 57% of the value observed on the previous day.

tests were positive for various antigens, including house-dust, house-dust mites, *Aspergillus*, *Alternaria*, *Cladosporium*, *Penicillium*, *Candida*, cat dander, orchard grass, buffalo grass, cedar, Japanese cypress, milk and cheese. However, the patient appeared to have an allergic reaction to cedar pollen only.

First, he began to take long-term control medication for asthma, including inhaled budesonide, procaterol and lansoprazole. Since the contribution of increased parasympathetic tone to the onset of bronchospasms was considered the most likely possibility, anticholinergics (once daily inhaled tiotropium with intermittent inhaled oxitropium) were added. However, these medications did not reduce the frequency of acute exacerbations (Fig. 1). The patient continued to visit the ER with severe asthma exacerbations approximately every two months. He stated that, even when his daily symptoms were under control with long-term control medication, he suddenly suffered from asthma attacks with a runny nose, especially when he had constipation with the urge to defecate. A typical acute bronchospasm was recorded on the patient's peak expiratory flow (PEF) chart (Fig. 2). Although the PEF reading had been in the green zone with minimal variation, the PEF level rapidly decreased after the patient woke up with the urge to defecate. As soon as he noticed shortness of breath associated with abdominal cramps and pain, the patient took two puffs of oxitropium and subsequently three puffs of procaterol, neither of which were effective in relieving his symptoms. Within 30 minutes, the PEF level decreased to 57% of the value observed on the previous day, and he had to be rushed to the ER to receive intravenous corticosteroids. The onset of acute bronchospasms was not related to any particular time of the day or night, but rather was strongly related to the timing of the gastrointestinal symptoms.

The patient mentioned that the exacerbation episodes were often preceded by abdominal cramps and pain associated with constipation, not with diarrhea. Therefore, he was given laxatives to soften his stool and to make it easier to defecate. This simple intervention had an equivalent effect on controlling the defecation-induced bronchospasms. As a result, the patient visited the ER only once after he started using prophylactic laxatives in addition to standard asthma treatment (Fig. 1).

Discussion

Asthma exacerbations are episodes of acute worsening of asthma symptoms and airflow limitation that are sometimes rapid in onset and life-threatening. In the present case, the patient stated that the most important factor that precipitated his acute bronchospasms was the duration of abdominal cramps before defecation and that the longer a painful bowel movement lasted, the more severe the asthma exacerbation was likely to be. Acute bronchospasms are known to be provoked by various stimuli, including allergens, smoke, fumes, drugs, exercise, cold air and hyperventilation caused by strong emotional expressions (1). However, little is understood regarding the correlation between defecation and bronchospasms.

Gastrointestinal peristalsis and breathing are both well known to be controlled by the autonomic nervous system via the regulation of smooth muscle activity. Therefore, a dysfunction in the autonomic nervous system results in various disorders, such as constipation, diarrhea and asthma. There is only one case report written in English indicating that defecation is a trigger for acute bronchospasms (2). However, it is a reasonable possibility that a strong urge to defecate and bronchospasms share a common pathophysiological mechanism due to autonomic imbalance. In fact, previous studies have demonstrated increases in airway hyperresponsiveness and a high prevalence of asthma among patients with irritable bowel syndrome (3, 4). We assume that, in the present case, activation of the parasympathetic nerves during intestinal peristalsis associated with the Valsalva maneuver stimulated the autonomic nervous system to secrete acetylcholine, leading to bronchospasms as a response to the contraction of the bronchial smooth muscles. A history of past abdominal surgery might contribute to an increased reactivity of the visceral parasympathetic tone.

Since the Valsalva maneuver during defecation is considered to possibly enhance gastroesophageal reflux, which is known to cause asthma exacerbations, the patient was first treated with a proton pump inhibitor in addition to standard asthma treatment. These medications might have had a certain effect on stabilizing airway irritability; however, they resulted in clinically insignificant improvements. Considering the contribution of an enhanced parasympathetic tone to

the onset of acute bronchospasms, long-acting and short-acting anticholinergics were also added to the previous treatments. Inhaled anticholinergics were expected to be effective in inhibiting parasympathetic nerve impulses by blocking the binding of muscarinic receptors to acetylcholine. However, in contrast to that observed in a previous case report (2), the frequency of acute exacerbations did not decrease with anticholinergic therapy in this case. These treatments seemed to not be able to completely abolish the efferent parasympathetic nerve signals.

It is interesting to note that the patient's acute bronchospasms were evoked only by gastrointestinal symptoms and not by other parasympathetic stimuli such as pain, cold air, emotional stress, coughing, laughing, vomiting or urinating. Moreover, dexamethasone, not anticholinergics, was effective for relieving the patient's acute symptoms. A recent study showed that the mucosal release of serotonin and histamine significantly increases in correlation with mast cell infiltration in patients with irritable bowel syndrome (5). Since parasympathetic stimulation potentiates the release of serotonin and histamine and in turn these chemical mediators act as neurotransmitters as well as regulators of the inflammatory response, an enhanced parasympathetic tone together with increased levels of chemical mediators may have accelerated inducing bronchospasms and subsequent airway inflammation in this case.

In conclusion, we herein characterized the close relationship between asthma exacerbations and gastrointestinal symptoms such as abdominal cramps and pain with the urge to defecate. We found that defecation-related asthma tends to be severe and occurs suddenly. The use of prophylactic laxatives in addition to standard asthma treatment resulted in satisfactory improvements in this case; therefore, we speculate that this simple, noninvasive and inexpensive intervention may therefore be an appropriate therapeutic option for the treatment of this type of asthma.

The authors state that they have no Conflict of Interest (COI).

References

1. Global Initiative for Asthma (GINA). Global Strategy for Asthma Management and Prevention (updated 2011); URL: <http://www.ginasthma.org>
2. Rossman L. Defecation-induced bronchospasm. *J Emerg Med* **18**: 195-197, 2000.
3. White AM, Stevens WH, Upton AR, O'Byrne PM, Collins SM. Airway responsiveness to inhaled methacholine in patients with irritable bowel syndrome. *Gastroenterology* **100**: 68-74, 1991.
4. Yazar A, Atis S, Konca K, et al. Respiratory symptoms and pulmonary functional changes in patients with irritable bowel syndrome. *Am J Gastroenterol* **96**: 1511-1516, 2001.
5. Cremon C, Carini G, Wang B, et al. Intestinal serotonin release, sensory neuron activation, and abdominal pain in irritable bowel syndrome. *Am J Gastroenterol* **106**: 1290-1298, 2011.