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# Pulmonary atelectasis manifested after induction of anesthesia: a contribution of sinobronchial syndrome?

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**Abstract**
A 31-year-old man underwent general anesthesia for sinus surgery. Anesthesia was induced with midazolam and butorphanol, and an endotracheal tube was orally placed with a bronchoscope, due to difficulty with temporomandibular joint opening. Ventilation difficulty and increased peak inspiratory pressure were noticed shortly after tracheal intubation, and bronchoscopy was performed for diagnosis. The bronchi were filled with a clear mucous secretion. Removal of the secretion improved respiration and decreased the peak inspiratory pressure. A chest roentgenogram taken prior to extubation showed right upper lobe atelectasis. A diagnosis of sinobronchial syndrome was made postoperatively. The etiology of the acutely developed atelectasis was unclear. However, the latent syndrome may have induced excessive airway secretion with stimuli such as endotracheal intubation.

**Key words** Pulmonary atelectasis · Sinobronchial syndrome · Chronic sinusitis

## Introduction

Sinobronchial syndrome is a complication of prolonged chronic sinusitis. The syndrome is defined as a coexisting pathology of chronic sinusitis and a nonspecific chronic inflammatory state of the lower airway [1,2]. Sinus surgery is performed to treat chronic sinusitis, usually without adverse outcomes, although when patients with the syndrome undergo the surgery, general anesthesia can expose them to special risks of unexpected pulmonary complications. However, anesthetic managements for these patients have been rarely described.

The following report is a case of a patient with undiagnosed sinobronchial syndrome who underwent general anesthesia for maxillary sinus surgery and developed severe airway obstruction and massive pulmonary atelectasis shortly after tracheal intubation. Preoperatively, the patient was considered to be a healthy adult. After the incident, the patient's history and symptoms were reviewed, and he was diagnosed to have sinobronchial syndrome.

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## Case report

A 31-year-old man (178 cm, 84 kg) had been suffering from chronic sinusitis for 16 years and was scheduled for bilateral endoscopic sinus surgery. Although he had a history of temporomandibular arthrosis, several years previously, he complained of only slight temporomandibular joint pain and could open his mouth without difficulty. His preoperative laboratory data, chest roentgenogram, and electrocardiogram were normal. He did not have a history of asthma, but had a mild form of food allergy to crab and shrimp. During the preanesthetic visit, the patient coughed up phlegm several times. He mentioned that he coughed up sputum daily in a similar manner, and he ascribed the cough to his chronic postnasal drip. His cough and sputum were not considered to indicate serious anesthetic risks at that time. He smoked 20 cigarettes a day (smoking index, 200). Despite a preoperative instruction to cease smoking, he had smoked until the day of the operation.

Ranitidine 20 mg was given orally 3 h before the induction of anesthesia. Anticholinergics such as atropine were not administered preoperatively. The peripheral artery oxygen saturation ($SpO_2$) was 97% in room air and increased to 98% after 3-min breathing with 5 l·min$^{-1}$ oxygen via a face mask. Anesthesia was induced with butorphanol 1 mg and midazolam 5 mg. After confirming of manual ventilation via the mask, vecuronium 8 mg was administered. Despite the adequate muscle relaxation, the anesthesiologist could not

open the patient's jaw. Thus, an endotracheal tube was placed with the aid of a bronchoscope, 15 min after the induction of anesthesia. During the bronchoscopic procedure, the larynx became filled with copious clear mucus, which made the procedure difficult. Immediately after the tracheal intubation, the anesthesiologist noticed that the reservoir bag was unusually resistant to squeezing. While the lungs were manually ventilated with increased peak airway pressure above 35 mmHg, respiratory sounds were audible only in the bilateral lower lungs, but were absent in the middle and upper lungs. Wheezing was not heard, and the expiratory curve on the capnogram was not prolonged. End-tidal (Et) $CO_2$ was elevated to 55 mmHg. The $Sp_{O_2}$ rapidly dropped to 93% while the patient was ventilated with 6 l·min$^{-1}$ oxygen with 2% sevoflurane. As ventilation remained difficult, bronchoscopy was performed for diagnosis. Correct endotracheal tube positioning was confirmed. No gastric contents or foreign bodies were observed in the bronchi. Mechanical occlusion of the airway was thus ruled out, although the main bronchus discharged a large amount of clear serous secretion. Several segmental bronchi were completely blocked by the secretion and the bronchial epithelium was diffusely reddened. After 30 min of repeated suctioning with the bronchoscope, weak respiratory sounds became audible in the middle to upper lung areas. Wheezing was not auscultated. $Sp_{O_2}$ was 95%, with Et $CO_2$ 50 mmHg with 6 l·min$^{-1}$ oxygen. Arterial blood gas analysis demonstrated $P_{O_2}$, 190.8 mmHg; $P_{CO_2}$, 57.3 mmHg; and pH, 7.318. The otolaryngologists decided to cancel the operation. As sevoflurane was weaned from 2% to 1%, the patient began to move and coughed up a massive amount of clear sputum through the tracheal tube. Simultaneously, the peak inspiratory pressure decreased to 25 mmHg, and respiratory sounds became audible bilaterally, except from the right apical lung area. Et $CO_2$ decreased to 42 mmHg, and $Sp_{O_2}$ increased to 99% with 6 l·min$^{-1}$ oxygen. A chest roentgenogram was taken, and it showed right upper lobe atelectasis (Fig. 1). After the muscle relaxation was reversed and the sevoflurane administration was discontinued, the patient's trachea was extubated. Arterial blood gas analysis after the extubation showed $P_{O_2}$, 105.2 mmHg; $P_{CO_2}$, 45.3 mmHg; and pH, 7.36, while the patient was given 8 l·min$^{-1}$ $O_2$ via a face mask. The patient was transferred to his ward. A chest roentgenogram taken the next day was normal and the right upper lobe atelectasis was not present. The patient was discharged after his respiratory sounds and arterial blood gas analysis had returned to normal.

At the postanesthetic visit, the patient's family told us that he had a continual chronic cough with copious sputum. This information, along with the patient's prolonged history of sinusitis and the intraoperative bronchoscopic findings of the inflammatory lower airway with hypersecretion, led us to confirm the diagnosis of sinobronchial syndrome.

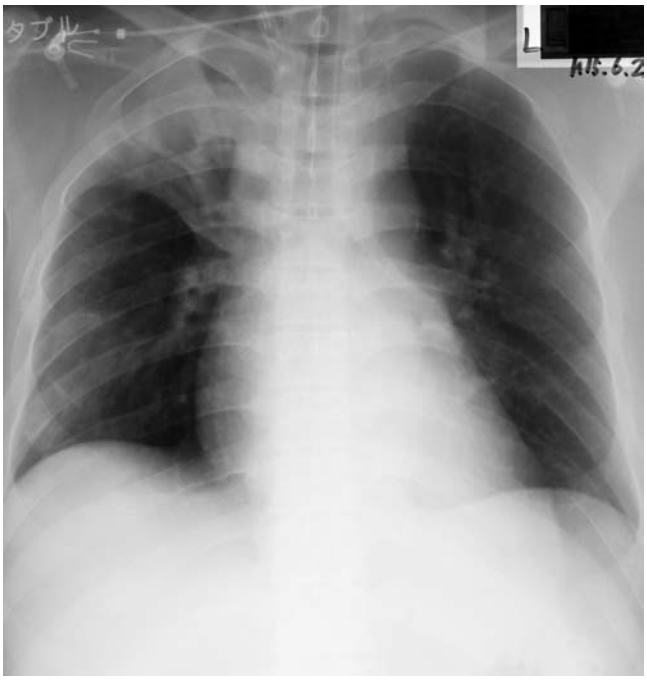

**Fig. 1.** Chest roentgenogram taken in the operation room prior to extubation. Atelectasis in the right upper lobe remained, despite previous efforts to aspirate the airway secretion

## Discussion

The most important finding in the present case was the unexpected airway hypersecretion, which led to the intraoperative ventilatory difficulty in a relatively healthy patient undergoing general anesthesia for routine surgery. The redundant secretion widely occluded the bronchi to the middle and upper lung segments and severely obstructed ventilation. As a result, acute lobar pulmonary atelectases developed.

Bronchial secretion is a frequent cause of atelectasis. Segmental or lobar collapse often results from bronchial obstruction by secretions. Preoperative pulmonary infection, smoking, and immobility are the risk factors for perioperative pulmonary atelectasis. Abdominal surgery, high concentrations of oxygen, and reduced functional residual capacity with mechanical ventilation increase the risk [3,4]. Our patient was lacking these conditions, yet the airway was severely obstructed by secretions after the induction of anesthesia. Initially, possible causes of the sudden airway obstruction were sought, such as malfunction of the anesthesia breathing system, bronchial intubation, pulmonary aspiration,

asthma and pneumothorax. These were ruled out, and the bronchoscopy performed for diagnosis revealed that segmental bronchi were widely occluded by airway secretion. Because removal of the secretion improved the ventilation, we suspected that bronchial occlusion with mucus was the cause of the airway obstruction.

The etiology of the acutely induced airway secretion was unclear. As was diagnosed later, the patient had been suffering from sinobronchial syndrome. This manifested itself in a hyperresponsive airway coupled with chronic inflammation. Stimuli such as the fiberoptic bronchoscopy and the tracheal intubation could have triggerred excessive mucus production. Furthermore, anticholinergics were not administered preoperatively. Although the routine use of anticholinergics as premedication is considered unnecessary, in this case, the patient would have benefited from the antisialagogue effect of atropine. In additon, the patient's habitual smoking could also have contributed to the increased airway secretion.

Patients with sinobronchial syndrome are, characteristically, adults with a prolonged history of sinusitis who manifest bronchial symptoms of chronic lower airway inflammation, such as cough, expectoration and shortness of breath on exertion [5]. With chronic sinusitis, the numbers of mucosal glands in the maxillary sinus are reported to be increased [6], as are the numbers of goblet cells in the airway epithelium, due to coexisting chronic bronchitis. Mucus production is highly and acutely aggravated by stimuli such as irritation of the surface epithelium [7]. The syndrome is reportedly related to HLA antigen Bw54, a gene carried only by East Asians, which partly explains why the syndrome is exclusively reported in Asia, and why the disease rates in Western Europe and the United States are low [5]. Although diseases previously reported in relation to sinobronchial syndrome, such as primary ciliary dyskinesia (Kartagener's syndrome) and Young's syndrome are rare disorders, chronic sinusitis is common and, reportedly, in 10.4% of Japanese patients with chronic sinusitis, there are complicating chronic lower airway symptoms [5]. Thus, a large portion of the population could be latently at risk for sinobronchial syndrome.

The most important element of the anesthetic management of a patient with sinobronchial syndrome is the preliminary diagnosis. Patients with chronic sinusitis are at risk for the syndrome. Clinical signs suggesting airway inflammation, such as chronic cough and airway secretion, can be reliable clues for the diagnosis. Conservative therapy, such as low-dose administration of erythromycin, is reported to reduce the symptoms of chronic airway inflammation [8]. Thus, preoperative consultation with otolaryngologists regarding the treatment is mandatory. Regional anesthesia may be preferable in intractable cases, as previously reported in the anesthetic management of patients with Kartagener's syndrome [9].

In summary, we have reported a case of severe airway obstruction and intraoperative pulmonary atelectasis in a patient with sinobronchical syndrome. To avoid serious pulmonary complications in such patients, we emphasize the importance of preoperative diagnosis. Clinical signs such as cough and sputum can be reliable clues for the early diagnosis of this syndrome.

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