Postobstructive Pulmonary Edema that Developed Immediately after the Removal of an Endobronchial Foreign Body

Atsunori Masuda¹, Fumihiro Asano¹, Akifumi Tsuzuku¹, Takuya Sobajima¹, Anri Murakami¹, Yoshihiko Matsuno¹, Kazuhiro Hirata², Kunihiro Matsunami² and Atsushi Imamura²

Abstract

The patient was a 5-year-old boy who was transported to our hospital for a paroxysmal cough, disturbance of consciousness, tonic-clonic convulsions and labored breathing. The patient’s respiratory failure persisted after the convulsions remitted, and the presence of an endobronchial foreign body was suspected based on the findings of chest CT performed the following day. A peanut was subsequently removed from the right main bronchus using a bronchoscope with tracheal intubation and bag valve mask ventilation. Immediately after removal, the patient rapidly developed exacerbated hypoxemia, and a reduction in right lung lucency was noted on chest radiography. He was therefore diagnosed with type II postobstructive pulmonary edema, and his condition improved within a short period of time.

Key words: bronchoscopy, endobronchial foreign body, negative pressure pulmonary edema, postobstructive pulmonary edema

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Introduction

Postobstructive pulmonary edema (POPE), also referred to as negative pressure pulmonary edema, is primarily associated with upper airway obstruction, including types I and II (1-3). Type I develops immediately after upper airway obstruction and is considered to be mainly caused by a strong negative pressure generated in the thoracic cavity due to forced inspiration against the site of upper airway obstruction. The negative pressure increases the pulmonary vascular volume and pressure load on the pulmonary capillary vascular wall, thus leading to fluid leakage into the pulmonary interstitium and alveoli. In type II, chronic upper airway obstruction is present in the background, and an increase in the end-expiratory lung volume induces a mild auto positive end-expiratory pressure (PEEP). When the chronic obstruction rapidly resolves, the auto PEEP disappears, subsequently inducing a relative negative pressure in the thoracic cavity. If this process is severe, the tissue fluid leaks into the pulmonary interstitium and alveoli.

We encountered a patient in whom hypoxemia and lung lucency reduction on chest radiography occurred immediately after the removal of an endobronchial foreign body; however, his condition rapidly improved within a short period of time. Based on the clinical course, this case was considered to involve respiratory failure associated with type II POPE. We herein report this case because, to our knowledge, there have been no previous reports of type II POPE developing immediately after foreign body removal.

Case Report

The patient was a 5-year and 3-month-old boy with a past

¹Department of Pulmonary Medicine, Gifu Prefectural General Medical Center, Japan and ²Department of Pediatrics, Gifu Prefectural General Medical Center, Japan

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Correspondence to Dr. Fumihiro Asano, asano-fm@ceres.ocn.ne.jp
medical history of admission for pneumonia of the left lower lung field two months prior to the current event and an allergy to both eggs and cow milk. He also exhibited mental retardation, with a developmental quotient of 67 at 3 years of age. After displaying intermittent coughing for four days, he developed paroxysmal coughing while playing with sand at school, followed immediately by a disturbance of consciousness, tonic-clonic convulsions and labored breathing. He was therefore transported by ambulance and admitted to the Department of Pediatrics.

The patient’s status on admission was as follows: height, 100 cm; body weight, 15.4 kg; consciousness level, JCS III-100; body temperature, 37.5°C; blood pressure, 129/83 mmHg; heart rate, 120/min; and respiratory rate, 30/min with labored breathing. Oxygen was administered using a reservoir-equipped oxygen mask at 10 L/min, and the SpO2 was 100%.

Blood testing performed on arrival showed the following results: aspartate aminotransferase (AST), 74 IU/L; lactate dehydrogenase (LDH), 346 IU/L; creatine kinase (CK), 166 IU/L; blood glucose, 265 mg/dL; and white blood cell count, 30,200/μL; all of which were increased. In addition, a venous blood gas analysis (with the administration of oxygen at 10 L/min) demonstrated a pH of 6.68, PaCO2 of 106.5 Torr, PaO2 of 86.6 Torr, HCO3- of 12.3 mmol/L, BE of -24.2 mmol/L and AnGap of 26.3 mmol/L, indicating respiratory and metabolic acidosis with CO2 retention. However, no abnormalities that may have caused the convulsions were observed.

On chest radiography performed on admission, the left lung lucency was mildly enhanced (Fig. 1 left); however, no abnormality that may have caused the convulsions were observed on blood testing, head MRI or lumbar puncture. The patient displayed signs of choking when his posture was changed during the examination, and his respiratory condition became exacerbated, for which emergency tracheal intubation was performed. Treatment was thereafter conducted under respiratory management with synchronized intermittent mandatory ventilation (SIMV) + pressure support (PS) mode [peak inspiratory pressure (PIP): 15 cmH2O, PEEP: 5 cmH2O, pressure support: 8 cmH2O, frequency: 25, inspiratory time: 0.7 seconds, FiO2: 100→30%]. The presence of a foreign body in the airway was suspected, and the patient was referred to our department on the day after admission.

Holtzknecht signs were observed in the left lung on chest radiography performed during inhalation and exhalation at 11 AM on the day after admission, and the left main bronchus was interrupted on the coronal view of plain chest CT (Fig. 1 right), suggesting the presence of a foreign body in the airway. In addition, overinflation and a dilated hilum were noted in the right lung with a mild infiltrative shadow in the left upper lung field on chest radiography performed at 2 PM the same day (Fig. 2).

Bronchoscopy was subsequently performed using tracheal intubation and bag valve mask ventilation with the administration of oxygen at 10 L/min at 6 PM on the same day. The patient’s airway was observed using an ultrathin bronchoscope, and a pale yellow foreign body with a smooth surface was detected in the right main bronchus. Redness and swelling of the mucosa of the left main bronchus with partial granulation was also observed, suggesting that the foreign body had moved from the left main bronchus to the right main bronchus after admission. The foreign body was removed without being fragmented using basket forceps. Since the foreign body was large, it was removed with the tracheal tube, and ventilation was switched to bag valve mask ventilation. A new tube was then inserted after three minutes. The foreign body was identified to be a peanut.
radiography in association with a gradual improvement in rapid reduction in the right lung lucency was observed on tramuscularly, followed by bag valve mask ventilation, and a injected intravenously with 0.3 mg of adrenaline injected in-
ruled out, and an anaphylactic reaction was suspected. A to-
region were observed (Fig. 5).

bubbly, transparent, watery secretions originating from this mental bronchi in the right upper lobe were patent; however, bronchoscopy, no bronchospasms were noted and the seg-
moval, was noted in the right upper lung field (Fig. 4). On which had been absent immediately after foreign body re-
An infiltrative shadow accompanied by an air bronchogram, with a blood pressure of 129/89 mmHg and HR of 135/min. The SpO2 decreased from 98% to 80%, although his respira-
tion can also be excluded, as the patient did not receive an ex-
cessive transfusion. Plasma colloid osmotic pressure reduc-
tion can also be excluded because the patient’s level of nut-
rition obtained via normal oral ingestion was sufficient until the day before admission, no hypoproteinemia was observed on admission and he had no past medical history of liver disease. Furthermore, a diagnosis of neurogenic pulmonary edema can be excluded because no signs of head trauma, intracranial hemorrhage or mass formation were noted on head MRI performed on admission. We also investigated the possibility of an association with an allergy to peanut com-
ponents; however, this possibility was considered to be negative because the patient had no history of developing skin eruptions or respiratory symptoms after eating peanuts. In addition, no bronchospasms, wheezing suggestive of ana-
phylaxis or systemic symptoms, such as hypotension, were noted throughout the patient’s clinical course, and no in-
creases in the anti-peanut IgE antibody level were detected. Moreover, the differential diagnoses of pulmonary edema in-
duced by ARDS, aspiration pneumonia and/or chemical pneumonitis induced by rare conditions, such as dysfunction of the lymphatic system or altitude sickness, were excluded based on the patient’s clinical findings, particularly the prompt improvement in the infiltrative shadow. Therefore,

The patient was subsequently discharged 21 days after ad-
mission.

Discussion

The characteristic finding of this case was the rapid oc-
currence of hypoxemia immediately after the removal of the endobronchial foreign body under positive pressure ventilation, in association with an infiltrative shadow representing pulmonary edema appeared in the right upper lobe then disappeared on chest radiography.

Pulmonary edema reflects a status of morbidly increased tissue fluid and is generally caused by the following four events (3): 1) increased hydrostatic pressure in the pulmonary capillary bed (or conversely, decreased pressure in the interstitium), such as that observed in patients with heart failure, 2) decreased osmotic pressure in the plasma due to the loss of plasma proteins as a result of undernutrition or terminal hepatic cirrhosis, 3) increased capillary vascular wall permeability due to a reduction in the colloid osmotic pressure difference caused by inflammation and 4) a decreased return of fluid to the circulation via the lymphatic system.

Bhaskar et al. (3) reported a diagnostic flow chart for as-
seSSing postoperative respiratory distress based on the classi-
fication of the causes described above. Applying this chart to the present case, a diagnosis of cardiogenic pulmonary edema can be ruled out because the patient had no past medical history of heart disease, no changes were noted on ECG, no cardiomegaly was detected and the infiltrative shadow was localized on chest radiography. In addition, the possibility that the pulmonary edema was caused by over-
flow can be excluded, as the patient did not receive an ex-
cessive transfusion. Plasma colloid osmotic pressure reduc-
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duced by ARDS, aspiration pneumonia and/or chemical pneumonitis induced by rare conditions, such as dysfunction of the lymphatic system or altitude sickness, were excluded based on the patient’s clinical findings, particularly the prompt improvement in the infiltrative shadow. Therefore,
the patient was ultimately diagnosed with type II POPE based on the evidence of acute airway obstruction caused by the peanut, the fact that the abnormal region on noted on chest radiography was consistent with the location of the foreign body and the exclusion of the above differential diagnoses.

POPE develops primarily in association with upper airway obstruction, including types I and II. In both types, the pressure in the capillary blood vessels rises, thereby disrupting the balance of Starling’s law, which determines the direction of water movement through the capillary vascular wall, thus pushing out tissue fluid from the pulmonary capillary blood vessels to the tissue, thus resulting in pulmonary edema (3). Type I develops immediately after the onset of upper airway obstruction, with the main cause thought to be a strong negative pressure load on the thoracic cavity due to forced inspiration against the site of upper airway obstruction. The negative pressure subsequently increases the pulmonary vascular volume, thus increasing the pressure load on the pulmonary capillary vascular wall, which causes fluid leakage into the pulmonary interstitium and alveoli. Type II is caused by chronic upper airway obstruction in which an increased end-expiratory lung volume leads to the development of a mild auto PEEP. When the source of chronic obstruction is removed, the auto PEEP resolves, the pulmonary volume and pressure normalize and the intrathoracic pressure becomes negative. Reportedly, fluid leakage into the pulmonary interstitium and pulmonary alveoli subsequently occurs, with negative pressure pulmonary edema potentially developing when the process is severe (4). In the present case, overinflation of the right lung was observed on radiography prior to bronchoscopy and then disappeared after foreign body removal, suggesting that the foreign body in the right main bronchus acted as a check valve generating an auto PEEP in the right lung, which was resolved by removing the foreign body. Consequently, artificial respiratory management with PEEP was applied before the procedure, and the foreign body was removed under positive pressure respiratory management employing a back valve mask, which may have led to the development of type II POPE.

The grade of POPE shadows on chest radiography varies, with such shadows normally developing in the bilateral lungs, although unilateral lung development has been reported (5). It has also been reported that hypoxia and abnor-
malities on radiography may develop rapidly (within one hour in many cases) and subsequently improve promptly (within 24 hours in most cases) in patients with POPE (6), consistent with the clinical course of our patient. In addition, no atelectasis was noted in the right upper lobe on bronchoscopy in this case, although bubbly secretions originating from this region were detected, indicating the presence of POPE. However, questions remain as to why the shadow appeared only in the right upper lobe, although the foreign body was present in the right second carina, and why the duration of the presence of the foreign body in the right main bronchus was short, while type II POPE is considered to be caused by chronic airway stenosis.

The possibility of reperfusion pulmonary edema was also investigated in the current case. Reperfusion pulmonary edema occurs due to cytokine release into ischemic regions following pulmonary reperfusion, which causes tissue disorders and enhances peripheral vascular permeability (7). There is no basis to conclude that the patient’s ischemia and reperfusion in the right upper lobe were induced by the removal of the foreign body, and, to our knowledge, no cases of airway foreign body-induced reperfusion edema have been reported. Although it is assumed that hypoxia and hyperinflation caused the local pulmonary arterial vasoconstric-
Figure 5. Bronchoscopic findings obtained 16 minutes after foreign body removal. Sixteen minutes after foreign body removal, bubbly secretions originating from the right upper lobe were noted on bronchoscopy (left). The segmental bronchi in the upper lobe were patent (center, right).

...tion observed in this case, while the procedure to remove the foreign body improved these findings, with subsequent reperfusion due to postobstructive changes (such observations may be included as postobstructive changes due to POPE in a wide sense, although the details differ from those of previous reports in addition to classification and mechanisms), the time to improvement after the onset of the shadow may have been too short to support this speculation.

Steroid and adrenaline were administered in this case because we initially considered the possibility of anaphylaxis, although the effects of steroids on POPE have not been established, and it takes time for the treatment effect to be realized. Furthermore, POPE develops via an adrenergic pathology (8), and both blood pressure elevation and tachycardia were observed in this patient. The severity of POPE varies; some patients spontaneously improve, while artificial respiratory management is required in severe cases. Therefore, the current patient’s condition may have spontaneously improved because he was under artificial respiratory management, not as a result of the effects of steroids or adrenaline.

The incidence of type I and II POPE complicating upper airway obstruction early after surgery has been reported to be 9.6-12% and 44%, respectively (9). The cause of POPE is laryngeal convulsions in many patients, accounting for approximately half of all cases (10); however, to our knowledge, no cases in which type II POPE has developed after endobronchial foreign body removal have been reported. In patients with POPE, the clinical symptoms usually include a decrease in oxygen saturation with the production of pink bubbly sputum and abnormalities on chest radiography starting in the early stage (9) and generally improving within 24 hours following an appropriate diagnosis and treatment, although the mortality rate rises from 11% to 40% if the diagnosis is delayed (11). Therefore, when removing a foreign body under positive pressure ventilation, attention must be paid to the potential for the development of type II POPE immediately after the procedure.

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

References