

Acute Postobstructive Pulmonary Edema Following Laryngospasm in Elderly Patients: A Case Report

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Only a few cases regarding postobstructive pulmonary edema following laryngospasm in older patients aged more than 60 years have been reported; however, acute pulmonary edema or pulmonary hemorrhage would be more deadly to elderly patients who have cerebrovascular disease than young healthy adults. After review of the literature, we report an unusual case of a 67-year-old man with ischemic cerebrovascular disease, who underwent carotid angioplasty and stenting and experienced severe pulmonary edema and hemorrhage secondary to laryngospasm after general anesthesia with laryngeal mask airway. The patient required positive-pressure ventilation, supportive treatment, and active cerebroprotection in the intensive care setting for 3 days before the edema resolved, and subsequently made a complete recovery without new onset of neurologic sequelae. The possible pathophysiological mechanisms, precaution, and preventative strategy of postobstructive pulmonary edema in older patients are discussed.

Keywords: *postobstructive pulmonary edema, pulmonary hemorrhage, older patients, laryngospasm, general anesthesia, ischemic cerebrovascular disease.*

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OBJECTIVES—1. IDENTIFY PREVALENCE of postobstructive pulmonary edema (POPE) following laryngospasm in adults. 2. Discuss the possible pathophysiological mechanisms of POPE. 3. Describe precautions and preventative strategies for POPE in older patients.

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Postobstructive pulmonary edema (POPE), also referred to as negative pressure pulmonary edema or noncardiogenic pulmonary edema, which occurs in about 0.1% to 4% of general anesthesia cases in adults,¹⁻³ is a potentially fatal complication after surgery. POPE caused by an episode of laryngospasm is well known to be more prevalent in the young, male, muscular population, with presentation ranging from mild to severe.⁴⁻¹¹ However, few reports of POPE after general anesthesia in elderly patients aged more than 60 years have been documented,^{12,13} and thus, the incidence of POPE secondary to laryngospasm in older people remains unclear.

POPE accompanied by diffuse alveolar hemorrhage (DAH) is also a rare condition. Contou et al¹³ noted that the incidence of POPE-related DAH was about 0.1%, and the patients were

primarily young, male, and athletic. The predictors for POPE-related DAH have not been clearly established. Although POPE or DAH seldom occurs in older people aged more than 60 years, acute pulmonary edema with or without hemorrhage could be more deadly to elderly patients who have cardiovascular or cerebrovascular (CV) disease than young healthy adults. In this article, after review of the literature, we report a severe POPE and pulmonary hemorrhage following laryngospasm after general anesthesia in a 67-year-old male patient with ischemic CV disease undergoing carotid angioplasty and stenting. Furthermore, the possible pathophysiological mechanisms, precaution, and preventative strategies for POPE in older patients are discussed.

Review of the Literature

We searched the literature from databases including PubMed, MEDLINE, Ovid, ScienceDirect, Web of Science, and the Cochrane Library from 1975 to January 2018. The electronic search included the following medical subject heading terms: postobstructive pulmonary edema, negative pressure pulmonary edema, general anesthesia, laryngospasm, and older people. Only two cases of POPE occurring in older patients aged more than 60 years were found. Ikeda-Miyagawa et al¹² reported a 76-year-old female patient who experienced laryngospasm after surgery. About 5 minutes later, she started to breathe spontaneously, but a chest X-ray showed pulmonary edema. She had impaired cardiopulmonary function. In a recent retrospective study,¹³ only one of 15 patients was older patient in identified POPE cases. The 64-year-old male experienced pulmonary edema and hemoptysis (< 50 mL) after general anesthesia, and rapid, significant infiltrate clearance was seen in the chest X-ray at postoperative day 2 (POD2).

Case Report

We experienced a case of severe pulmonary edema and hemorrhage following laryngospasm after general anesthesia in a 67-year-old man. The patient's consent was obtained before the medical data review. The patient was diagnosed with ischemic CV disease with severe left carotid artery stenosis. He had a history of hypertension and cerebellar infarction. The patient had a complex condition

demonstrated by CV radiography; moreover, he had poor cooperation because of his prior cerebral injury. Therefore, the patient was scheduled for carotid angioplasty and stenting under general anesthesia. Physical examination showed nonfluent speaking with no other significant findings. Electrocardiogram was within normal range and chest X-ray indicated clear lung fields. Transthoracic echocardiography data revealed 63% of left ventricular ejection fraction and 34% of left ventricular fractional shortening. Preoperative hemoglobin level was 157 g/L. The patient was categorized as American Society of Anesthesiologist status III.

After anesthesia induction, a laryngeal mask airway was placed and mechanical ventilation was initiated. The surgical duration was 110 minutes and computed tomography (CT)-guided carotid angioplasty and stenting was performed. The patient was hemodynamically stable during the procedure. Blood loss was 10 mL and infusion volume was 700 mL. After surgery, the patient was rapidly awakened. Laryngospasm occurred a few minutes after laryngeal mask removal. We tried to apply positive pressure ventilation via mask, but he became hypoxemic, with pulse oxygen saturation (SPO₂) falling to 67%. The heart rate and noninvasive blood pressure (BP) increased to 138 beats/min and 240/120 mm Hg, respectively. Urapidil was administered intravenously to decrease the BP level. From the time of nonresponse to noninvasive positive pressure ventilation, he was reintubated immediately after sedative and paralytics were administered. Blood-tinged fluid was suctioned from the endotracheal tube (about 150 mL). Auscultation disclosed diffuse bilateral crackles and the fiberoptic bronchoscopy detected fresh blood in bilateral bronchi. Positive-pressure ventilation with a PEEP of 5 to 8 cm H₂O and FiO₂ of 100% was applied. Intravenous injections of morphine and furosemide were administered to reduce pulmonary edema. Methylprednisolone and mannitol were used to prevent cerebral edema. Other cerebral protection measures included improving oxygenation, restrictive fluid infusion, avoiding hypertension and hypotension that can result in CV injuries, maintaining stable homeostasis, and using an ice cap to cool the head to decrease brain oxygen consumption during hypoxemia. At 20, 60, and 130 minutes after reintubation, arterial blood gas (ABG)

was analyzed using a blood gas analyzer (GEM 3000) (Table 1). ABG results demonstrated acute respiratory insufficiency. Chest CT scan revealed increases in diffuse density of the lungs, bilateral ground glass opacities, and dorsal consolidation of lungs because of pulmonary infiltrates at 2.5 hours after the procedure (Figure 1A). Meanwhile, brain CT scan disclosed encephalomalacia in left basal ganglia and radiological crown area as well as cerebral scattered ischemic foci, no new onset of cerebral hemorrhagic foci or infarction (Figure 2A).

The patient was transferred to the neurologic intensive care unit and repeated ABG was measured using an analyzer (ABL800 BASIC; Radiometer, Copenhagen, Denmark) (Table 1). Stable hemodynamics and homeostasis were maintained continuously. Methylprednisolone, human serum albumin, diuretics, and antiplatelet aggregation were used to reduce tissue edema and prevent CV thrombogenesis. Plasma biomarkers, such as pro-B-type natriuretic peptide, heart-type fatty acid binding protein, and troponin T, were measured for 4 days postoperatively and revealed no obvious myocardial injury (Table 2). The patient improved clinically with continuous

positive-pressure ventilation and supportive treatment. Chest CT scan showed rapid, significant infiltrate clearance at 24 hours after surgery (Figure 1B). The patient did not have clinical presentations of secondary cerebral hematoma, epilepsy, or infarction within 48 hours postoperatively. The tracheal tube was removed on POD3 and the patient was well enough to be discharged from the intensive care unit. Subsequently he made complete recovery without new onset of neurologic complications. On POD5, chest CT scan showed clear lung fields, indicating resolution of edema (Figure 1C), and brain CT scan showed encephalomalacia in paraventriculus of the left brain (Figure 2B). He was discharged from the hospital on POD6. In follow-up 6 months postoperatively by telephone interview, the patient did not report any new neurologic functional defects.

Discussion

Laryngospasm is the most common cause of type I POPE in the adult population in the acute postoperative setting.^{14,15} A study by Contou et al¹³ showed that POPE-related pulmonary hemorrhage was rarely massive (greater than 200 mL) and

Table 1. Arterial Blood Gas Analysis of the Patient After Reintubation

Arterial Blood Gas Analysis	20 min After Reintubation	60 min After Reintubation	130 min After Reintubation	10 min After Shifting to ICU
pH	7.18	7.24	7.30	7.403
PCO ₂ (mm Hg)	79	60	54	37.8
PO ₂ (mm Hg)	16	45	78	191
Na ⁺ (mmol/L)	135	132	136	138
K ⁺ (mmol/L)	4.5	4.5	5.0	4.4
Ca ²⁺ (mmol/L)	1.2	1.10	1.19	1.16
Cl ⁻ (mmol/L)	N	N	N	112
Glu (mmol/L)	5.2	5.9	5.3	N
Lac (mmol/L)	1.5	1.2	1.1	1.0
Hct (%)	56	57	49	N
HCO ₃ ⁻ (mmol/L)	29.5	25.7	26.6	23.8
HCO ₃ std (mmol/L)	19.4	21.0	23.8	N
TCO ₂ (mmol/L)	31.9	27.5	28.3	N
BE _{ecf} (mmol/L)	1.1	-1.7	0.2	-1.0
BE(B) (mmol/L)	-2.5	-3.6	-1.2	N
SO _{2c} (%)	13	72	94	98.7
THbc (g/dL)	20.7	21.1	18.1	16.4
ctO _{2, e} (mmol/L)	N	N	N	10.2
P50, e (mm Hg)	N	N	N	26.42

ICU, intensive care unit; N, none.

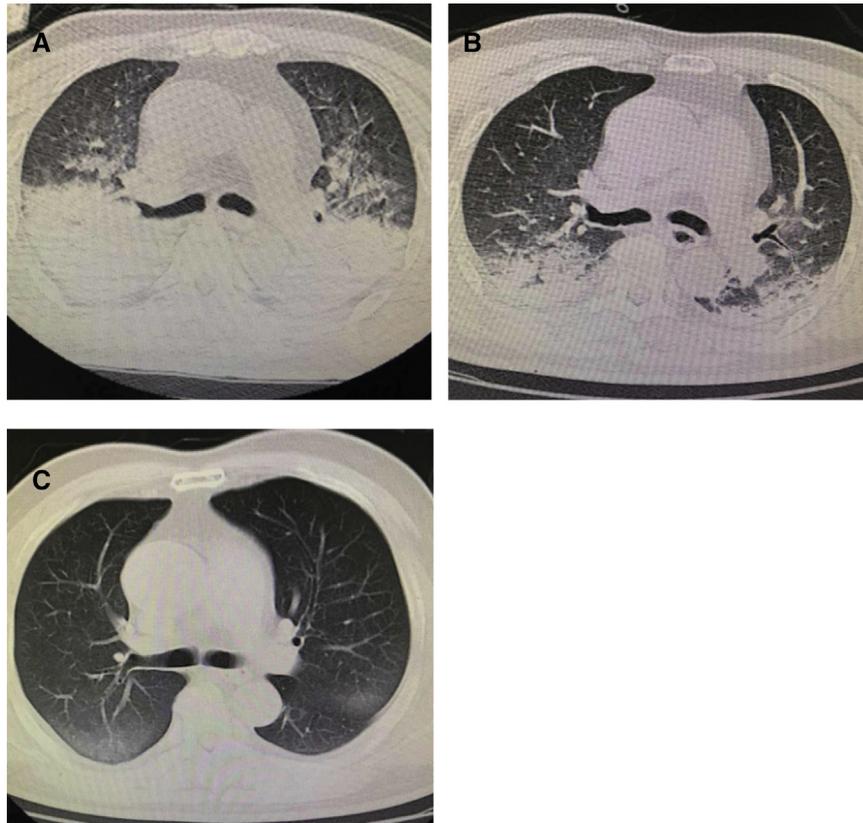


Figure 1. Typical changes in chest computerized tomography (CT) scans in an axial plane: (A) Chest CT scan depicting increase in diffuse density of the lungs, bilateral ground glass opacities, and dorsal consolidation of lungs because of pulmonary infiltrates, supporting acute pulmonary edema and hemorrhage at 2.5 hours after surgery. (B) Chest CT scan depicting most of the lung infiltrates resolved at 24 hours after surgery. (C) Chest CT scan depicting clear lung fields, indicating normal performance of the lungs at 5 days after surgery. Copyright of the images is reserved by Chinese People's Liberation Army general hospital. This figure is available in color online at www.jopan.org.

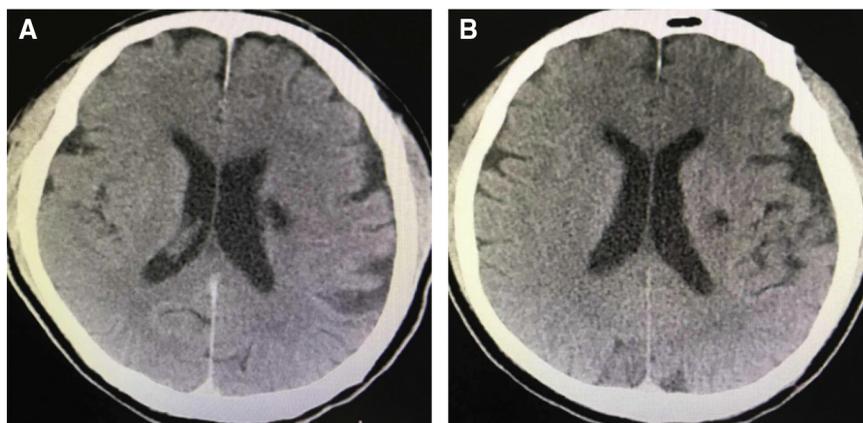


Figure 2. Typical changes in brain computerized tomography (CT) scans in an axial plane: (A) Brain CT scan depicting encephalomalacia in left basal ganglia and cerebral scattered ischemic foci at 2.5 hours after surgery. (B) Brain CT scan depicting encephalomalacia in paraventriculus of the left brain at 5 days after surgery. Copyright of the images is reserved by Chinese People's Liberation Army general hospital. This figure is available in color online at www.jopan.org.

Table 2. Postoperative Plasma Biomarkers for Myocardial Injury

Plasma Biomarkers	POD1	POD2	POD3	POD4
Pro-B-type natriuretic peptide (pg/mL)	91.9	227.9	712.7	382.8
Heart-type fatty acid binding protein (mcg/L)	7.8	3.6	N	1.0
Troponin T (ng/mL)	N	N	0.013	0.006

N, none; POD, postoperative day.

mostly less than or equal to 100 mL. In our case, the patient had a moderate hemoptysis (150 mL). Although we did not prove its alveolar origin using bronchoalveolar lavage, the bronchoscopy detected bilateral bronchial hemorrhage and blood-tinged fluid was continuously suctioned from the tube. Further chest CT scan showed that a “ground glass” appearance and decreased hemoglobin level were consistent with alveolar hemorrhage. Although the 67-year-old man who had a history of CV disease presented with a severe POPE and prolonged hypoxemia, he did not have CV disease following his laryngospasm. Rapid relief of laryngospasm, early recognition, and prompt treatment of POPE as well as active cerebroprotection were essential in preventing neurologic complications in this patient.

Pathophysiological Mechanisms of POPE

The exact mechanism of POPE remains unclear; however, the generally accepted principal pathophysiological mechanisms involve hemodynamic changes occurring in the lung and the heart after the generation of excessive negative intrathoracic pressure because of forceful inspiration after airway obstruction. A hydrostatic mechanism plays an important role in edema fluid formation, because of increased transmural capillary pressure (capillary P_{TM}) favoring transudation of fluid into the pulmonary interstitium and alveolar space.¹⁴ Young healthy people are able to generate highly negative intrathoracic pressure (up to as extreme as -140 cm H_2O) to induce the pulmonary edema when the airway is closed,^{3,13,16,17} accounting for most cases of POPE occurring in young adults. Our patient was of a relatively strong build, so he could generate strong enough negative pressure to cause the pathophysiological changes associated with POPE.

The effects of hypoxia and a hyperadrenergic state on cardiac function and pulmonary capillary

permeability may contribute to the development of pulmonary edema.¹⁸ Increased afterload resulting from stress-induced catecholamine release can lead to systolic left ventricular dysfunction. Interventricular septal shift along with tachycardia can result in diastolic left ventricular dysfunction. Both systolic and diastolic left ventricular dysfunction may further enhance the increased capillary P_{TM} to promote the pulmonary infiltrate, and severe increased P_{TM} even causes a capillary membrane rupture with increased permeability.¹³ The alveolar capillary membrane damage can vary in intensity, supporting POPE's clinical presentation from mild to severe. Extremely high capillary P_{TM} or increased capillary permeability may result in severe pulmonary microvasculature disruption with alveolar hemorrhage, as in our case.

In addition, it is a susceptible time for the development of pulmonary edema in the early postoperative period because of myocardial depression caused by residual anesthetic agents or other underlying risk factors.⁷ Given the potential for cardiac-associated pulmonary edema in this particular patient, an echocardiography would have been appropriate. Regrettably, we did not perform the postoperative transthoracic echocardiography examination. Fortunately, biomarkers for myocardial injury were within acceptable ranges and cardiogenic pulmonary edema was ruled out.

Precaution and Preventative Strategy

The development and resolved course of POPE in our patient were similar to that in young adults described in previous documents, but the acute pulmonary edema or hemorrhage could be more life-threatening to older adults who have CV disease. In our case, marked fluctuations of hemodynamics and prolonged hypoxemia were extremely harmful to an elderly patient with ischemic CV disease, because the new onset of cerebral hemorrhage or infarction would lead to

poor prognosis and extend the length of hospital stay and associated costs. In addition, the BP and heart rate were adversely affected because of pressure on the carotid artery, BP control should be very tight to prevent vascular damage or rupture for at least 24 hours after the procedure. The patient was hypoxic for 2 hours because of severe pulmonary edema and hemorrhage. We had taken measures to prevent the prolonged hypoxia, such as positive-pressure ventilation, promoting diuresis, elevation of plasma colloid osmotic pressure, and so on. Except for treatment of pulmonary edema, we also took active measures to prevent neurologic morbidities, including restrictive fluid infusion, maintaining hemodynamic stability, avoiding hypertension, hypotension and bradycardia, pharmaceutical prevention of cerebral edema, improving oxygenation, and decreasing brain oxygen consumption. Fortunately, our patient did not have secondary cerebral hematoma, epilepsy, or infarction during the postoperative period, and had no new neurologic functional defects in follow-up 6 months postoperatively by telephone interview.

Many factors such as undiagnosed or untreated obstructive sleep apnea, vocal cord palsy, and Parkinson's disease contribute to the development of

laryngospasm in the aging population.^{17,19} Identifying risk factors in regard to the elderly population is important as we are performing procedures on an aging population. We should carefully manage older patients who have risk factors of upper airway obstruction to prevent POPE. In addition, clearance of excessive secretion after laryngeal mask removal in older patients is very important to prevent laryngospasm.

Conclusions

The etiology of POPE in our patient may be multifactorial. Although POPE seldom occurs in older patients, we should know it can be associated with a higher morbidity in older adults with multiple comorbidities than in young healthy patients. A much more intense and complex postoperative assessment, prevention of laryngospasm, early recognition, and timely treatment as well as a cardiac or cerebral protection strategy should be emphasized in older patients who have risk factors for upper airway obstruction when undergoing general anesthesia. This case illustrates the importance of airway management during postanesthesia recovery in high-risk elderly patients.

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3. Describe precaution and preventative strategy of POPE in older patients.

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