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Case Study
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Neurogenic Pulmonary Edema Caused by Subarachnoid Hemorrhage: A Case Report

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Abstract

Neurogenic pulmonary edema is a serious and life-threatening complication caused by central nervous system diseases, excluding cardiogenic pulmonary edema; other causes of pulmonary edema, lung injury, etc. The lack of specific diagnostic criteria for NPE and the lack of awareness among clinicians often lead to under diagnosis and misdiagnosis.

Neurogenic pulmonary edema is not uncommon in neurosurgical patients with aneurysmal subarachnoid hemorrhage, which usually occurs on the same day of hemorrhage and subsides in the following days. However, if a re-rupture of the aneurysm occurs, the condition can worsen dramatically and progress rapidly, with a poor prognosis. Therefore, early detection and early treatment of neurogenic pulmonary edema disease are more effective, but there are no specific manifestations in the early stage, which makes the diagnosis more difficult. Therefore, clinical awareness of this disease needs to be improved.

Keywords: Neurogenic pulmonary edema; Anesthesiology; Subarachnoid hemorrhage

Introduction

Neurogenic pulmonary edema is a serious and life-threatening complication caused by central nervous system diseases, excluding cardiogenic pulmonary edema; other causes of pulmonary edema, lung injury, etc. The lack of specific diagnostic criteria for NPE and the lack of awareness among clinicians often lead to under diagnosis and misdiagnosis. The disease progresses rapidly with poor prognosis and high mortality [1]. In this paper, we report a patient with a ruptured right middle cerebral artery aneurysm causing subarachnoid hemorrhage, which developed neurogenic pulmonary edema and underwent elective aneurysm clamping under general anesthesia. The relevant perioperative management is reported as follows, and the anesthesia management of patients with neurogenic pulmonary edema is analyzed in the context of the relevant literature.

Case Study

Medical history

The patient was a 69-year-old male, with a height of 175 cm and a weight of 79 kg, who was admitted to the emergency department of our hospital due to "sudden disturbance of consciousness for more than 4 days". The patient was in shallow coma, with Blood Pressure (BP) of 163/117 mmHg, Heart Rate (HR) of 99 beats/min, Respiratory Rate (RR) of 20 breaths/min, and Oxygen Saturation (SPO_2) was 93% while he received 5 L/min oxygen through a nasal cannula and the GCS (Glasgow Coma Scale) was 10 points. External cranial CT of another hospital showed subarachnoid hemorrhage, and whole brain angiography revealed a right middle cerebral aneurysm. The diagnosis of "right middle cerebral artery aneurysm (near the insula), subarachnoid hemorrhage and hypertension" was considered, and the patient was admitted to the neurosurgery department, and an elective operation was planned. There was no previous history of hypertension, diabetes mellitus, or heart disease. Preoperative electrocardiogram showed sinus rhythm, with ST-segment T-wave changes, and external chest radiograph of other hospital showed increased texture in both lungs. He was admitted to the emergency department with routine blood: Hemoglobin (Hb) of 181 g/L, Hematocrit (Hct) of 0.53, Troponin-T of 17.4 ng/ml. Blood gas analysis: Pondus Hydrogenii (pH) of 7.46, Partial Pressure of Oxygen (PaO_2) of 62 mmHg, Partial Pressure of Carbon Dioxide ($PaCO_2$) of 22.5 mmHg, Base Excess (BE) of -5.34 mmol/L.

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Anesthesia and operation procedure

After admission, cranial hematoma removal was proposed as an elective procedure, and relevant investigations and preoperative preparations were completed. During the pre-anesthesia visit, the patient was in shallow coma, with BP of 126/84 mmHg, HR of 104 beats/min, RR of 27 to 30 breaths/min, SpO_2 93% (the patient was receiving 8 L/min to 9 L/min oxygen *via* mask), and Urapidil Hydrochloride was pumped to control blood pressure, and the GCS was 9 points. At 11:00 p.m. of the same night after admission, the patient suddenly developed deepening consciousness and decreased blood pressure (considering re-rupture of the aneurysm). The chief resident of the department of anesthesiology was contacted for emergency tracheal intubation and emergency craniotomy for hematoma removal was proposed. On examination: dopamine (8-10 $\mu g/kg^{-1}/min^{-1}$) to maintain blood pressure to 131/90 mmHg, HR of 105 beats/min, SpO_2 of 95% (Fraction of inspiration O_2 (FiO_2) 100%) and the GCS was 5 points.

The patient was admitted to the operating room at 11:30 p.m. with routine Electrocardiogram (ECG) monitoring, BP 92/75 mmHg (no vasoactive drugs), HR 99 beats/min, SpO_2 94% (the patient was receiving oxygen through invasive ventilator with FiO_2 60%), RR 12 of beats/min, and the GCS was 5 points, End-Tidal Carbon Dioxide ($ETCO_2$) was 36 mmHg and airway pressure was 23 cmH_2O. Anesthesia induction: intravenous injection of Propofol 50 mg, Sufentanil 20 μg, Cis-atracurium 14 mg. Anesthesia maintenance: Propofol TCI mode 3 μg/ml and Remifentanil 0.1 $\mu g/kg^{-1}/min^{-1}$ continuous pumping. A few wet rales were heard in both lungs, and sputum was aspirated. The left radial artery was punctured and cannulated, invasive blood pressure monitoring was continued, an arterial blood gas reveals a pH of 7.25, PaO_2 of 104.2 mmHg (the patient was receiving oxygen through invasive ventilator with FiO_2 60%), $PaCO_2$ of 45.6 mmHg, BE of -7.74 mmol/L, Lactic Acid (Lac) of 1.6 mmol/L. The operation started at 00:05 a.m. and 250 ml of mannitol and 80 mg of methylprednisolone were given intravenously before craniotomy. Half an hour later SpO_2 gradually decreased to 81% and airway pressure rose to 34 cmH_2O. (Ventilator mode: Pressure Controled Ventilation-Volume Guarantee (PCV-VG) mode, with FiO_2 of 60%, Tidal Volume (VT) of 550 ml, RR of 12 breaths/min, $ETCO_2$ of 239 mmHg). Immediately rechecked blood gas analysis with pH of 7.23, PaO_2 of 57.6 mmHg (the patient was receiving oxygen through invasive ventilator with FiO_2 60%), $PaCO_2$ of 50.3 mmHg, BE of -7.27 mmol/L, and Lac of 1.8 mmol/L. Confirming normal tracheal tube depth and no discounting, adjusted inhaled oxygen concentration to 100%, auscultated obvious wet rales in both lungs, aspirated pink secretions, SpO_2 rose to 93%, airway pressure of 24 cmH_2O. And total liquid intake was 900 ml, bleeding was 100 ml and urine volume was 250 ml. The respiratory parameters were adjusted (PCV-VG mode, FiO_2 of 100%, VT of 400 ml, RR of 16 breaths/min, $ETCO_2$ of 40 mmHg, Positive End Expiratory Pressure (PEEP) of 8 cmH_2O, SpO_2 gradually increased to 96%, and Furosemide 10 mg was added. At 02:15 when the main surgeon performed intracranial hematoma removal, the patient had a large bleeding volume and at the same time, blood pressure dropped to 72/39 mmHg and heart rate increased to 138 beats/min. Immediately, the infusion rate was accelerated, red blood cell suspension was infused, while 7F double-lumen central venous placement was performed through the left femoral vein. Then norepinephrine and epinephrine were continuously pumped to maintain blood pressure, which was maintained at 110~116/79~84 mmHg, heart rate was controlled at 80~100 beats/min and SpO_2 was maintained at 95%. And then the vital signs were stable. The operation ended at 05:20 a.m. and the patient was transferred to the Neuroconscious Intensive Care Unit (NICU) with a tracheal tube at 05:35 a.m. The duration of the operation was 5 h and 15 min, and the anesthesia time was 6 h and 5 min. The total intraoperative intake was 6100 ml, red blood cell suspension 4 μ, fresh frozen plasma 400 ml, and autologous recovered blood 600 ml. And bleeding was about 1800 ml, and urine volume was 1900 ml.

At the time of admission to the NICU, norepinephrine (0.5 μg/kg.min to 1 μg/kg.min) and epinephrine (0.05 μg/kg.min to 0.1 μg/kg.min) were pumped, initial vital signs include: BP 110/70 mmHg, HR 95 beats/min, R 14 breaths/min, SPO_2 78% (the patient was receiving oxygen through invasive ventilator with FiO_2 60%), and the GCS was 3 points, sputum was administered, and SPO_2 rose to 92% after adjusting the inhaled oxygen concentration to 100%. After the patient was admitted to the NICU, he was treated with sputum aspiration, diuresis, and reduction of intracranial pressure, cardiac strengthening, maintenance of circulation, and maintenance of electrolyte balance. Postoperative review of chest radiograph: Reduced translucency of lungs, right lung predominant, increased and blurred texture of both lungs, possible scattered infection in both lungs. Bedside fibrinoscopy: Large amount of pale red thin sputum was seen in the left and right main airways and lower lobe airways of both lungs, diffuse mucosal edema was seen in both lungs, and a total of 20 ml of pale red thin sputum was aspirated from both lungs. Myocardial markers were rechecked, uromodulin was 2015 pg/ml and troponin T was 30. 9 ng/ml. At 5:00 p.m. on the day of surgery, the patient again showed brain herniation manifestations such as decreased heart rate and blood pressure, and was immediately given mannitol 250 ml intravenous drip and rechecked cranial CT, the examination results showed that the patient had intracranial hemorrhage again after the operation, and explained to the patient's family that the condition was heavy and the prognosis was poor, the patient's family asked to go back to the local hospital to continue treatment, and signed for automatic discharge at 05:30 p.m.

Discussion

Neurogenic pulmonary edema has a rapid onset, rapid progression, and high mortality rate, with acute respiratory distress and hypoxemia as the main clinical manifestations. The common causes of NPE include EV71 virus infection, subarachnoid hemorrhage, traumatic cranial injury, and brain tumor. The mechanism of neurogenic pulmonary edema is currently considered to be the excessive activation of sympathetic nerves after hypothalamic or medulla oblongata injury, which leads to the massive release of catecholamines (catecholamine storm), resulting in systemic vasoconstriction, and elevated vascular resistance of the body circulation, elevated left atrial pressure, and increased pulmonary capillary hydrostatic pressure. In addition, dramatic changes in hemodynamics can also directly damage the pulmonary capillary endothelium causing fluid leakage and resulting in pulmonary edema. A study found that children in the group with intracranial pressure >15 mmHg had increased extra vascular pulmonary water index and lung permeability, and decreased systemic vascular resistance, cerebral perfusion, and oxygenation index [2].

Neurogenic pulmonary edema lacks specific diagnostic criteria, and the diagnosis of neurogenic pulmonary edema is considered in patients with clinical presence of central nervous damage, pink foamy sputum, bilateral pulmonary infiltrative shadow on chest radiograph,

and oxygenation index <200 mmHg, after excluding other causes such as cardiopulmonary [3]. In this case, the patient had persistent intraoperative aspiration of pink foamy sputum, wet rales in both lungs, oxygenation index of 173 mmHg with progressive decrease and persistent uncorrectable hypoxemia, no previous history of cardiopulmonary and other diseases, any massive infusion, allergy, etc. The diagnosis of NPE was clear. In this case, the patient abandoned treatment on the first postoperative day due to severe disease. Some studies have shown that early diagnosis can improve patient prognosis [4], however, there are no specific clinical manifestations in the early stage of NPE, and the diagnosis is relatively difficult. Our patient was found to have increased texture in both lungs at the time of consultation at our hospital 4 days after the onset of the disease and had a poor prognosis.

The patient in this case had a preoperative Hunt-Hess classification of grade III, and the preoperative chest radiograph only indicated increased pulmonary texture in both lungs, and the electrocardiogram was sinus rhythm with ST-T changes. The patient was preoperatively comatose, on high concentration mask oxygen, with SPO_2 fluctuating at 90%, coarse breath sounds in both lungs, scattered wet rales, and no obvious specific clinical manifestations, making early diagnosis of NPE difficult. In a prospective, observational study, an APACHE II score ≥ 20, IL-6>40 pg/mL was found to be an independent risk factor for the development of NPE [5], and our patient had an APACHE II score of 31 and an increased troponin. The literature reports that in patients with subarachnoid hemorrhage, excessive sympathetic excitation, catecholamines can directly damage the myocardium and elevated troponin is seen in approximately 20% to 68% of patients. In studies of adults with subarachnoid hemorrhage, abnormal ECG was found to predict the development of NPE [6] and was associated with risk such as poor prognosis [7].

Although the exacerbation of the patient's condition in this case was due to postoperative rebleeding, symptoms related to neurogenic pulmonary edema appeared right after the start of the first operation. After the diagnosis was clarified intraoperatively, tachypnea was added immediately and respiratory parameters were adjusted to improve oxygenation, including low tidal volume ventilation and PEEP to maintain normal oxygen supply and carbon dioxide levels. Methylprednisolone was also given to improve vascular permeability, but the use of glucocorticoids is still controversial [8]. Katharina et al. [9] concluded that the main treatment options for NPE are adequate sedation and analgesia, reduction of sympathetic excitability, reduction of intracranial pressure, and supportive treatment for pulmonary edema, including volume management, ventilation strategies, and optimal oxygen supply. Reducing intracranial pressure while ensuring adequate circulating blood volume is a perioperative challenge, and ultrasound and PICCO monitoring are useful as a guide. The presence or absence of pulmonary edema can be determined by ultrasound detection of B-line and guides intraoperative fluid management [10].

Other measures have been found in previous experiments to reduce the occurrence of NPE. In animal experiments, intrathecal lidocaine decreased sympathetic excitability, lowered intracranial pressure, and reduced cardiopulmonary complications, neurogenic pulmonary edema [11]. It has also been found that nitric oxide [12] and cytokines [13] reduce pulmonary capillary permeability, thereby reducing the occurrence of NPE.

Neurogenic pulmonary edema is not uncommon in neurosurgical patients with aneurysmal subarachnoid hemorrhage, which usually occurs on the same day of hemorrhage and subsides in the following days. However, if a re-rupture of the aneurysm occurs, the condition can worsen dramatically and progress rapidly, with a poor prognosis. Therefore, early detection and early treatment of neurogenic pulmonary edema disease are more effective, but there are no specific manifestations in the early stage, which makes the diagnosis more difficult. Therefore, clinical awareness of this disease needs to be improved.

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