Our patients followed up with a diagnosis of neurogenic pulmonary edema

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Abstract

Neurogenic pulmonary edema is a clinical situation which develops as a result of central nervous system injury. It is rare in the childhood. Neurogenic pulmonary edema is a clinical diagnosis. Although the pathogenesis is not elucidated well, there is increase in pulmonary interstitial and alveolar fluid. The main principle in treatment of neurogenic pulmonary edema is supportive treatment and decreasing intracranial pressure as in acute respiratory distress syndrome. In this article, clinical properties of our two patients diagnosed with neurogenic pulmonary edema developed as a result of central nervous system injury are presented. (Turk Pediatri Ars 2015; 50: 241-4)

Keywords: Child, head injury, neurogenic pulmonary edema

Introduction

Neurogenic pulmonary edema (NPE) may occur as a result of different central nervous system (CNS) diseases including brain malignancies, traumatic brain damage, infection and convulsions. Neurogenic pulmonary edema was described by Shanahan in 1908 as pulmonary edema which developed in the postictal period in 11 patients with epilepsy whose ages ranged between 9 and 36 years (1). In 1918, Moutier reported a patient who developed pulmonary edema following firearm injury. In 1939, Weismann mentioned cases of NPE accompanying spontaneous or trauma-related intracranial hemorrhage in a large patient series (2). Although the pathogenesis is not known fully, an increase in the interstitial and alveolar fluid occurs in the lung. In treatment of neurogenic pulmonary edema, the general principle is supportive treatment.

In this article, we present two patients who were diagnosed with NPE as a result of CNS injury.

Case 1

A previously healthy 16-year-old patient presented to our pediatric emergency department with complaints of fainting and severe headache. The assessment of the patient

revealed the following findings: body temperature: 37.7°C, respiratory rate: 17/min, arterial blood pressure (BP): 110/75 mmHg, apical heart beat (AHB): 63/min, peripheral oxygen saturation (SpO₂): 92%. Physical examination revealed the following findings: general status was poor, was unconscious, pupillae were anisocoric and mid-dilated, cardiac and lung sounds were normal, abdominal examination was normal without organomegaly, Glasgow coma score (GKS) was 5, pediatric risk of mortality score (PRISM II) was 31 (83.7%), pediatric index of mortality (PIM) score was 100%. The patient was intubated. The hemogram and biochemical tests were as follows: hemoglobin: 12.4 g/dL, white blood cells (WBC): 16 200/mm³, platelets: 116 000/mm³, glucose: 161 mg/dL, BUN: 28 mg/dL, Cr: 0.72 mg/dL, Na: 141 meq/L, K: 2.7 mEq/L, AST: 22 U/L, ALT: 15 U/L, LDH: 153 U/L, lung graphy: normal. Blood gas analysis was as follows: pH: 7.2 PaO₂: 86 mmHg, PaCO₂: 56 mmHg, HCO₃: 14.3 mmol/L, BE: -13.5. Cranial computerized tomography revealed a large area of hematoma in the right parietotemporal region, subfalcine herniation and advanced brain edema (Figure 1). After the patient was consulted with neurosurgery, he was operated immediately and internalized in the pediatric intensive care for postoperative follow-up. In the postoperative evaluation, his general status was found to be poor, GCS was found to be 3 and light reflexes were absent. Mechanical ventila-

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Figure 1. a-c. (a) Cranial computerized tomography: Large hematoma area in the right parietotemporal region and subfalcine herniation (b) consolidation areas compatible with diffuse edema on bilateral lung areas on lung graphy (c) lung graphy 36 hours after development of neurogenic pulmonary edema

tion and treatment for brain edema was initiated. At the 12th hour of the follow-up, the patient's saturation values were reduced up to 70%. Simultaneous arterial blood gas analysis was as follows: pH 7.0, PaO₂: 56 mmHg, PaCO₂: 96 mmHg, HCO₃⁻: 115 mmol/L, BE: -12.4, PaO₂/FiO₂: 70. Treatment for acute respiratory distress syndrome (ARDS) was administered. Among mechanical ventilation variables, positive end expiratory pressure (PEEP) was increased gradually from 5 cm H₂O to 10 cm H₂O and peak inspiration pressure (PIP) was increased from 21 cm H₂O to 32 cm H₂O. On posteroanterior lung graphy, an appearance compatible with pulmonary edema was present. The posteroanterior lung graphy obtained at the 36th hour was observed to be normal and mechanical ventilation variable values were reduced. NPE was considered, because the patient had CNS hemorrhage, symptomatic and radiological improvement occurred rapidly, cardiac dysfunction was not observed and tracheal aspirate culture was negative. On echocardiogram, cardiac functions were evaluated to be normal. A diagnosis of brain death was made, because the brainstem reflexes became negative and the electroencephalogram findings were compatible with cerebral bioelectrical silence in the follow-up. The patient was lost on the 10th day of the follow-up. Written informed consent was obtained from the family.

Case 2

A previously healthy 2-year-old patient presented to pediatric emergency department because of in vehicle traffic accident. The assessment of the patient revealed the following findings: body temperature: 36.7°C, respiratory rate: 125/min, BP: 80/40 mmHg, AHB: 63/min, SpO₂: 80%. Physical examination revealed the following findings: general status was poor, was unconscious, pupillae were

anisocoric and midsized, normal cardiac sounds, lung sounds were reduced, GCS was 6, PRISM II score was 30 (80.7%), PIM score was 79.8%. The patient was intubated. The hemogram and biochemical tests were as follows: WBC: 27 060/mm³, platelets: 386 000/mm³, hemoglobin: 12.4 g/dL, glucose: 123 mg/dL, BUN: 13 mg/dL, Cr: 0.26 mg/dL, Na: 137 meq/L, K: 3.9 mEq/L, AST: 305 U/L, ALT: 105 U/L, LDH: 716 U/L, lung graphy: minimal pneumothorax and atelectatic areas. Blood gas analysis was as follows: pH: 7.23, PaO₂: 96 mmHg, PaCO₂: 46 mmHg, HCO₃⁻: 14.6 mmol/L, BE: -11.7. Subdural hematoma and brain edema were found on cranial computerized tomography and the patient was consulted with neurosurgery. Immediate operation was not considered (Figure 2). The patient was hospitalized in the pediatric intensive care unit with the diagnoses of in vehicle traffic accident, subdural hematoma, brain edema and pneumothorax. Mechanical ventilation and treatment for brain edema was initiated. Pneumothorax and atelectasies were observed to be improved on the lung graphy obtained on the fourth day. In the follow-up, his blood pressure values were regular and lung graphy was normal. A tracheostomy was performed on the 17th day of the follow-up because of failure in extubation and severe neurological damage. Surgical intervention was performed by the neurosurgery department because of presence of subdural effusion on the follow-up cranial computerized tomography. During the operation, massive hypoxemia occurred and his saturations ranged between 80% and 85%. The simultaneous blood gas analysis was as follows: pH: 7.12, PaO₂: 67 mmHg, PaCO₂: 87.9 mmHg, HCO₃⁻: 19.1 mmol/L, BE: -4.1, PaO₂/FiO₂: 98. Treatment for ARDS was administered. Among mechanical ventilation parameters, the PEEP pressure was gradually increased from 6 cm H₂O to 10 cm H₂O and the PIP pressure was increased from 23 cm H₂O to 35 cm H₂O.

Figure 2. a-c. (a) Subdural effusion containing air collections in bilateral frontoparietal regions (b) consolidation areas compatible with diffuse edema in bilateral lung areas on lung graphy (c) lung graphy 48 hours after development of neurogenic pulmonary edema

The lung graphy obtained revealed pulmonary edema. The patient was consulted with the cardiology department and his echocardiogram and electrocardiogram were found to be normal. The lung graphy obtained 48 hours later was observed to be normal. The $\text{PaO}_2/\text{FiO}_2$ value increased and the mechanical ventilation variables were reduced. NPE was considered because of presence of CNS injury and rapid symptomatic and radiological improvement. In the follow-up, hypoxemia regressed, the blood pressure values were regular and his mechanical ventilation variables were reduced. The tracheostomy was closed on the 30th day of the follow-up and the patient was discharged on the 35th day without sequela. Written informed consent was obtained from the family of the patient.

Discussion

Neurogenic pulmonary edema is a condition which develops following central nervous system injury and leads to increased mortality by causing to severe hypoxemia (3, 4). Beginning from the early years of the 20th century, the first cases of neurogenic pulmonary edema were reported after epileptic seizures and head traumas (4). The most known causes include subarachnoid hemorrhage and seizures (4, 5). However, neurogenic pulmonary edema has also been reported during ventriculoperitoneal shunt operation and in cases of acute hydrocephaly related with tumor metastasis, cerebellar hematoma occurring as a result of spontaneous rupture of arteriovenous malformation and massive intracranial hemorrhage (3, 6).

It may not always be easy to make a diagnosis of NPE because of absence of specific diagnostic criteria. Sudden

onset respiratory distress, tachypnea, hypoxemia, reduction in the peripheral oxygen saturation and pink frothy secretion develop with regression of the neurological picture. The partial oxygen pressure is reduced in blood gas analysis and bilateral diffuse infiltration on lung graphy is typical. Since there are no specific criteria for neurogenic pulmonary edema, other pathologies which may lead to the picture of pulmonary edema should be excluded (3-5). In cases of NPE, rapid improvement of the clinical and radiological findings in 48-72 hours and absence of findings belonging to gastric content in the mouth after extubation are among the criteria which show the accuracy of the diagnosis. Our patients also improved clinically and radiologically in 48-72 hours.

Neurogenic pulmonary edema frequently occurs as a result of severe head trauma. It is difficult to determine the actual frequency of NPE developing after acute head trauma, because most information has been reported in relation with autopsy series and isolated cases. Rogers et al. (7) reported the frequency of NPE in association with isolated head trauma to be 32% in the patients who died at the scene of accident and 50% in the patients who died in 96 hours. In the study of Akıncı et al. (8), it was reported that NPE developed in 5 of 223 patients who were hospitalized in neurosurgery intensive care department.

Increased intracranial pressure developing after head trauma may lead to NPE (9). Many causes have been proposed to explain the relation between central nervous system injury and pulmonary edema. Many anatomic locations including especially medulla oblongata and hypothalamus have been blamed in occurrence of NPE (10). In-

creased intracranial pressure which develops after head trauma causes an increase in the activity of the sympathetic nervous system and an increase in serum catecholamine levels by directly or indirectly stimulating the hypothalamic or subthalamic nuclei. Increased activity of the sympathetic nervous system leads to systemic and pulmonary vasoconstriction, increased systemic blood pressure and increased pulmonary venous pressure. In addition, left ventricular compliance and left atrial pressure increase. All these changes lead to an increase in the pulmonary blood flow and pulmonary capillary pressure and development of acute pulmonary edema (11). Independent of the underlying cause, the mutual specific picture is prominent fluid collection in the alveolar areas and interstitium in relation with alveolar capillary membrane injury (12). In many cases, it has been reported that cardiac enzymes may be increased without cardiopulmonary disease and echocardiographic and electrocardiographic findings may be normal (5). In our cases, cardiac dysfunction was not found on echocardiogram and electrocardiogram.

The principal treatment in neurogenic pulmonary edema is supportive treatment. Many patients with neurogenic pulmonary edema improve only with oxygen treatment and close clinical follow-up. Patients with prominent hypoxemia should be treated as ARDS (13). In experimental studies, it has been shown that alpha-adrenergic blockers may be beneficial by blocking excessive sympathetic response and dobutamine may be beneficial by increasing the cardiac output and decreasing peripheral vasoconstriction in treatment of NPE. The prognoses of the patients are generally related with the neurological picture and the mortality has been reported to range between 7% and 10% (4). Regression and improvement of the picture in 48-72 hours is a criterion which shows the accuracy of the diagnosis.

In conclusion, it should be kept in mind that NPE may develop following central nervous system injury.

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