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Case Report / Olgu Sunumu

Neurogenic pulmonary edema accompanying subarachnoid hemorrhage: case report

Subaraknoid kanamaya eşlik eden nörojenik pulmoner ödem: olgu sunumu

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Abstract

Neurogenic pulmonary edema (NPE) is defined as an acute pulmonary edema occurring after a central neurologic insult. It is associated with head injury and seizures, intracranial hemorrhage and stroke are among the other causes. Pathologies increasing intracranial pressure may cause excessive sympathetic discharge and lead to pulmonary vascular pressure changes and transcapillary fluid leakage. Hereby we present an acute respiratory failure in a 50-year old woman with subarachnoid hemorrhage (SAH) due to aneurysmal rupture of anterior communicating artery. The patient had a history of bronchial asthma and respiratory failure was thought to have occurred by radiocontrast induced airway hyper-responsiveness during cerebral angiography. No response to bronchodilator therapy, pinkish foamy sputum from the trachea, diffusely dense infiltrates on both lungs demonstrated by chest X-ray, normal echocardiography, and negative tracheal culture disproving pneumonia were among the factors leading to the diagnosis of NPE. Consistent with the diagnosis, the patient rapidly improved with mechanical ventilation and subsequent removal of the intracranial hematoma. After evaluation of the clinical course of our case, we recommend that NPE should be considered in the differential diagnosis of acute respiratory failure accompanying SAH.

Keywords: Neurogenic pulmonary edema, subarachnoid hemorrhage

Özet

Nörojenik pulmoner ödem, santral nörojenik travma sonrası oluşan akut pulmoner ödem tablosudur. Kafa travması ile ilişkilidir ve nöbetler, intrakraniyal kanama ve inme diğer

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nedenleri arasındadır. İntrakraniyal basıncı arttıran patolojik durumlar aşırı sempatik uyarıma yol açabilir ve pulmoner vasküler basınç değişiklikleri ve transkapiller sıvı kaçağına neden olabilir. Bu yazıda anterior komünikan arterin anevrizmal rüptürüne bağlı subaraknoid kanaması olan 50 yaşında bir kadında gelişen akut respiratuvar yetmezlik olgusunu sunuyoruz. Hastanın bronşiyal astma öyküsü vardı ve respiratuvar yetmezliğin serebral anjiyografi sırasında radyokontrasta karşı gelişen aşırı havayolu duyarlılığı ile oluşmuş olabileceği düşünüldü. Bronkodilatör tedaviye yanıtsızlık, trakeadan pembe köpüklü balgam gelmesi, akciğer grafisinde her iki tarafta yoğun ve yaygın infiltrasyon olması, normal ekokardiyografi ve pnömoni ile uyuşmayan negatif trakeal kültür, nörojenik pulmoner ödem tanısına götüren faktörler arasında idi. Tanı ile uyumlu şekilde, hasta mekanik ventilasyon ve daha sonra intrakraniyal hematomun boşaltılması ile hızlıca iyileşti. Bu olgunun klinik seyrinden yola çıkarak, subaraknoid kanamaya eşlik eden akut respiratuvar yetmezliğin ayırıcı tanısında nörojenik pulmoner ödemin de düşünülmesini öneriyoruz.

Anahtar sözcükler: Nörojenik pulmoner ödem, subaraknoid kanama

Introduction

Neurogenic pulmonary edema (NPE) is defined by the presentation of acute pulmonary edema secondary to a central neurologic insult [1]. Cases have been reported in aneurysmal subarachnoid hemorrhage (SAH), traumatic brain injuries, cerebral thrombosis, cerebral gas embolism, intracerebral hemorrhage, intracranial tumors, epilepsy, postoperative intracranial surgery, enterovirus encephalitis, meningitis and multiple sclerosis [2]. The sympathetic system plays a crucial role in the pathogenesis of NPE by activating a rapid cascade of processes leading to interstitial and intraalveolar edema, together with hemorrhage [3].The clinical features can be mistaken for other causes of respiratory failure. Here we present a case of NPE caused by SAH, which was confused priorly with an asthmatic exacerbation due to radiocontrast induced airway hyperresponsiveness during cerebral angiography.

Case report

A 50-year-old woman was admitted to the emergency room with sudden loss of consciousness. On arrival at the hospital her Glasgow coma scale (GCS) score was 9, with motor response to painful stimuli. Her blood pressure (BP) was 110/50 mmHg, pulse rate (PR) 130/min, respiratory rate (RR) 35/min and SpO2 95%. Diffuse bilateral ronchi and rales were detected on the auscultation of the lungs. The patient had a prior history of asthma bronchiale and smoking (2 packs/day for 30 years). During the cerebral CT angiography which revealed subarachnoid hemorrhage due to ruptured aneurysm of anterior communicating artery (ACoA) (Figures 1a and 1b), the patient became dyspneic with a RR of 42 and SpO2 of 90% with nasal O2 6 L/min. Respiratory distress was assumed to have occurred as a result of radiocontrast-induced airway hyper-responsiveness. The patient was transferred to the intensive care unit where bronchodilator therapy and methylprednisolone 80mg was administered.intravenously. The patient's blood pressure was within normal ranges (115/50 mmHg), while she had tachycardia (140 bpm), and signs of respiratory failure with respiratory rate of 45/min, and arterial blood gas values of pH

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7.3, PCO2 37 mmHg, PO2 66.9 mmHg, HCO3 18.2 mEq/L. The patient, with no response to bronchodilator therapy but worsening hypoxemia was intubated and mechanical ventilation with positive end-expiratory pressure (PEEP) was initiated. A pinkish foamy sputum was observed during the suctioning of the trachea and the chest X-ray revealed diffuse bilateral infiltrates (Figure. 2). Cardiac enzymes, WBC values were slightly elevated (Table 1). ECG revealed ST elevation on D2-3, aVF and V2-3 derivations, which became normal a few hours later. Echocardiographic findings were normal. Controlled ventilation with 5cmH2O PEEP and FiO2 0.6 (fraction of inspired oxygen 60%) was maintained for two days. During her follow-up she became fully conscious and cooperative, but slightly disoriented. After gradual weaning, the patient was disconnected from the ventilator on the third day of her admission when her chest X-ray revealed improvement of pulmonary edema (Figure 3) and her PaO2 was 80 mmHg with FiO2 0.3. Until the operation she frequently needed noninvasive ventilation with FiO2 0.4, PEEP 5 mbar. She underwent surgical clipping of ACoA aneurysm on her fifth hospital day. Postoperative period was uneventful and cerebral angiography confirmed the total removal of the aneurysm. She was discharged from the hospital with full recovery 12 days after the surgery.

Table 1. Cardiac markers and white blood cell count during the first two days of hospitalization.

	At admission	Postadmission day 2	Normal reference values
Troponin T	1.35	0.08	<0.01 ng/ml
CK-MB	39	27	0-25 U/L
CPK	188	150	20-180 IU/ml
WBC	16	12.69	4.1-10.9 x 10/uL
WBC, white blood cell; CK-MB, creatine kinase muscle & brain; CPK, creatine phosphokinase.			

Discussion

NPE, leading to cardiopulmonary dysfunction, is a potentially life threatening complication in patients with increased intracranial pressure of any cause. Fontes et al. [4] noted that the most frequent underlying factor was subarachnoid hemorrhage (42.9%) for the development of NPE. Son et al. [5] found the NPE incidence as 1.3% amongst their 546 SH patients. Muroi et al. [6] reported, evaluating their data of 477 patients with SAH that the incidence of NPE in SAH was 8%, being lower compared with previous studies with an incidence ranging from 10% to 29%. They also found that the patients with clinically and radiologically severe bleeding have a higher risk for developing NPE. The outcome in those patients was poor due to grade of the bleeding at the time of admission, rather than cardiopulmonary failure. A hyperdynamic state similar to sympathetic storm is observed with an increase of cardiac output, blood pressure, heart rate and systemic vascular resistance leading to the pulmonary edema [7]. Bindra et al. [8] suggested in their case report that the significant cardiovascular instability in their patient was possibly due to stimulation of hypothalamus adjacent to the site of aneurysm. EKG changes, elevation of cardiac markers may occur secondary to SAH which can confuse the diagnosis with cardiac ischemia. In addition to cardiogenic pulmonary edema, the differential diagnosis includes

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aspiration pneumonitis and pneumonia. In a postmortem study of a patient with progressive multiple sclerosis accompanied with fatal pulmonary edema, no explanatory findings such as tracheobronchial foreign bodies, pneumonia, sarcoidosis or cardiomyopathy were found indicating that the pulmonary edema could have been neurogenic [9].

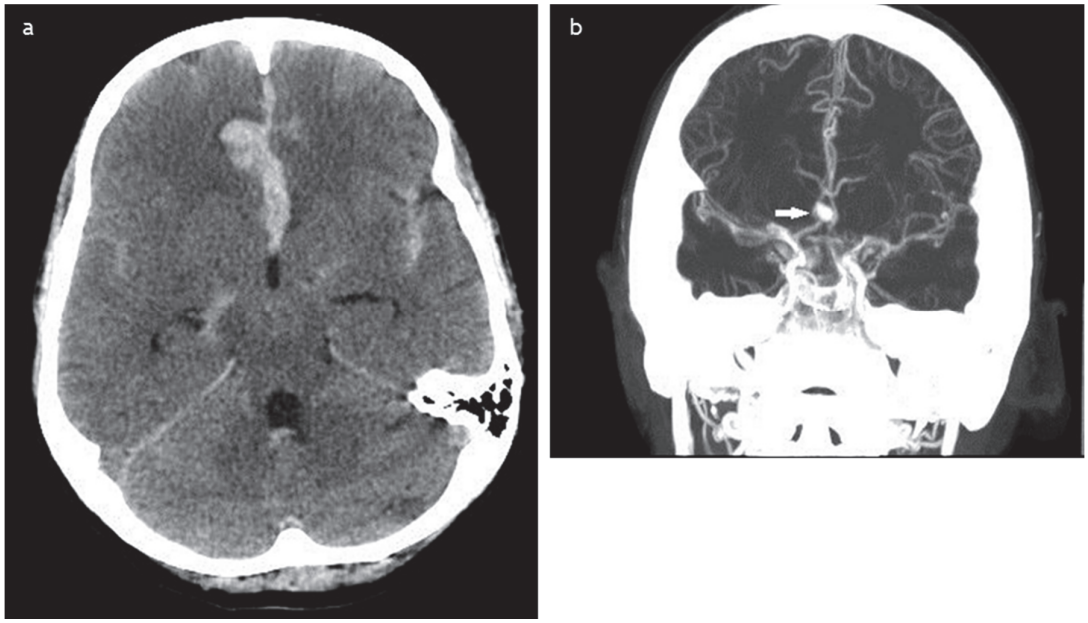

Figure 1. a. Cerebral CT showing subarachnoid hemorrhage. b. Cerebral CT angiography showing aneurysm of anterior communicating artery (ACoA).

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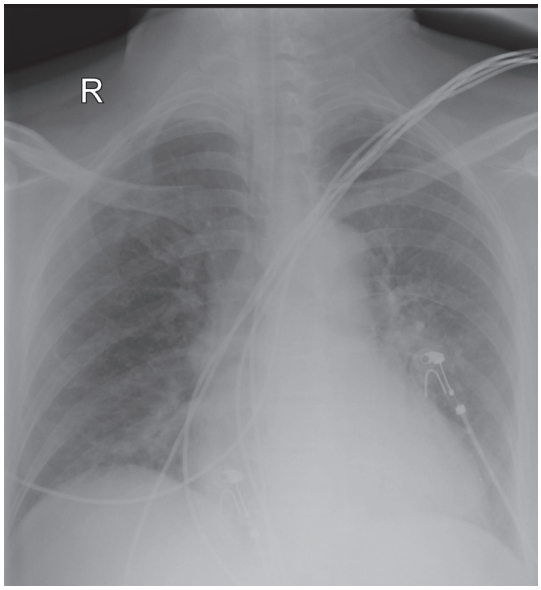

Figure 2. Chest X-ray showing diffuse bilateral infiltrates.

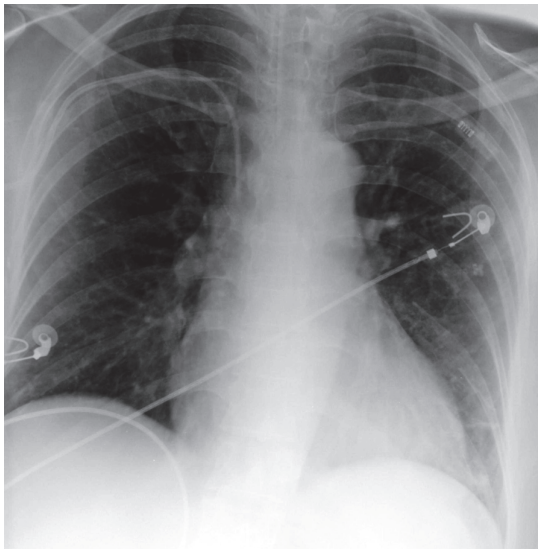

Figure 3. Chest X-ray showing improvement of pulmonary edema.

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Our patient had a prior history of bronchial asthma and the auscultation of lungs revealed rales and ronchi on admission. When the patient became dyspneic during the cerebral angiography, respiratory failure was attributed to radiocontrast-induced airway hyper-responsiveness. In the clinical course, no response to bronchodilator therapy, pinkish foamy sputum from the trachea, diffusely dense infiltrates on both lungs demonstrated by chest X-ray, normal echocardiography, negative tracheal culture disproving pneumonia, were among the factors leading to the diagnosis of NPE. Consistent with this diagnosis, the patient rapidly improved with mechanical ventilation and subsequent removal of the intracranial hematoma.

NPE is defined by the presentation of acute pulmonary edema secondary to a central neurologic insult. Its clinical features can be mistaken for other causes of pulmonary edema and may lead to confusion in the diagnosis. After evaluation of the clinical course of our case, we recommend that NPE should be considered in the differential diagnosis of acute respiratory failure accompanying SAH.

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