eScientific Library

Volume 1 Issue 1
www.escientificlibrary.com

ES Journal of Case Reports

Neurogenic Pulmonary Edema: (TIA complicated with Acute Pulmonary Edema)

Case report

Getaw Worku Hassen[1]*, MD, PhD, Dennis Brown[2], MD,Golnar Pashmforoosh[1], MD, Frosso Adamakos[1], MD, Tracy Barry[1], DO, Rajnish Jaiswal[1], MD, Leidy Gonzalez[1], MD, Roger Chirurgi[1] and Hossein Kalantari[1]

[1]NYMC, Metropolitan Hospital Center, Department of Emergency Medicine, New York, NY

[2]Harlem Hospital Center, New York, NY

Received: Jan 30, 2020; **Accepted:** Feb 18, 2020; **Published:** Feb 19, 2020

***Corresponding author:** Getaw Worku Hassen, Professor of Emergency Medicine, Director of Research, NYMC, Metropolitan Hospital Center, 1901 First Avenue, New York

Abstract

Neurogenic pulmonary edema (NPE) is a rare clinical entity that is seen in the setting of epileptic seizure, head injury and cerebral hemorrhage. NPE in the setting of transient ischemic attack (TIA) has not been reported before. Here we report the case of a 67 year old female with TIA complicated with NPE. NPE has been reported to occur predominantly in the setting of epileptic seizure, head injury and cerebral hemorrhage. Other presentation may be rare, but may occur and require emergent diagnosis and treatment when they do.

Introduction

Neurogenic pulmonary edema is a rare clinical entity that can present within minutes to hours after a brain insult. The clinical picture of NPE is dyspnea with hemoptysis associated with tachypnea, tachycardia and on physical exam basilar rales similar to that observed in pulmonary edema of other causes [1]. Additional tests that aid the diagnosis or exclusion of NPE include chest x-ray with normal heart size with evidence of bilateral alveolar filling [2,3]. In lung sections pulmonary congestion, extravasations and intra-alveolar accumulation of edema fluid and intra-alveolar hemorrhage is visible. Swan-Ganz catheters measurements of hemodynamic parameters are usually normal [4,5]. An increase in pulmonary fluid occurs shortly after the central nervous insult [2]. The pathophysiologic mechanism for the development of NPE remains poorly understood. Various theories exist and different structures of the brain are attributed as important structures in the development of NPE [6-9]. The only structure for which experimental evidence exists in support of its role is the medulla oblongata [10,11]. Common causes of NPE are epileptic seizure, head injury and cerebral hemorrhage. Less common causes include non-hemorrhagic stroke, multiple sclerosis, brain tumor and bacterial meningitis [12-14]. NPE in the setting of TIA has not been reported to our best knowledge.

Case description

This is the case of a 67 year old female with past medical history of diabetes mellitus, hypertension and hyperlipidemia, who presented to our emergency department with sudden onset of left sided arm weakness and left facial weakness. Symptom started 45 min before presenting to the hospital. Patient reported that she was having conversation with family members and they noticed her face was asymmetrical and she spoke funny. Patient has not noticed these symptoms at all. Family members

alarmed 911 and patient was brought to the emergency department. On presentation to the emergency department patient has mild left facial droop and weakness of the left arm that was improving. The NIHSS at presentation was 3. The rest of the physical exam was unremarkable with clear lungs, no respiratory distress, no murmur and clear mental status. Initial vital signs were as follows: blood pressure 167/94; heart rate 76; oxygen saturation 100% on 2L nasal canula. Patient was sent for the initial CT scan. After the CT scan was done the arm weakness has resolved and mild facial droop was present. The initial CT scan was read as no acute pathology, no bleeding or obvious ischemic stroke (Figure 1A). The laboratory examination and the electrocardiogram did not suggest a myocardial infarction. After consultation with the stroke team, the decision was made not to use thrombolytic medication due to the low NIHSS and the rapid improvement of her symptoms. Shortly after patient started complaining of difficulty of breathing. She was found to have elevated blood pressure of 230/120. She was given oxygen per 100% nonrebreather mask. Repeat physical exam reveal bilateral rales in the basal aspects of the lungs. No jugular vein distention (JVD) appreciated and there were no bruits heard over 4 the carotids. Her oxygen saturation remained in low 80's despite 100% oxygen. She became tachypnic and was subsequently intubated. The patient was started on a nitroglycerin-drip, was give enalapril and lasix. The chest radiography revealed increases opacity of the lung fields consistent with pulmonary edema with a pacemaker in place (Figure 1B). Patient was admitted to the intensive care (ICU) unit for further management. Patients' neurological status worsened with complete hemiplegia. The repeat CT scan showed large right middle cerebral artery (MCA) infarct (Figure 1C). Transesophageal echocardiogram revealed vegetation vs. thrombus on the right ventricular pacer leads, estimated ejection fraction 25-30% (Figure 1).

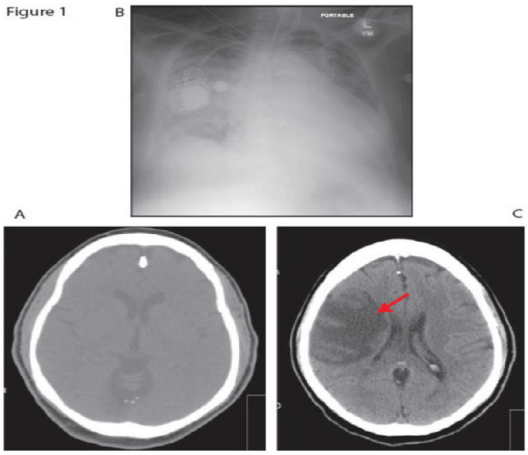

Figure 1: 1A. Chest X-ray findings. CXR with increased opacity of both lung fields consistent with pulmonary edema 1B. CT sacn of the head Initial CT scan of the head with no acute abnormal finding. 1C. Repeat CT sacn of the head Repeat CT scan of the head with massive acute infarction in the right middle cerebral artery area (red arrow).

Discussion

Neurogenic pulmonary edema is a rare clinical picture that can develop within minutes. The pathophysiology is not quite known, but the presumed mechanism for the development of CNS event stimulates the hypothalamic and vasomotor centers of the medulla. The result is believed to be massive autonomic discharge causing a dramatic change in starling forces which ends in shift of fluid between the capillaries and interstitium. Two proposed mechanism for the development of NPE are hemodynamic and inflammatory processes. A sudden increase in intracranial pressure (ICP) causes increased alpha-adrenergic response that results in increased systemic and pulmonary vascular resistant. The increased pulmonary vascular pressure causes an alteration of the Frank Starling forces with subsequent fluid shift to the alveolar space and interstitium [15]. The increased hydrostatic pressure results in injury to the capillary bed leading to increased capillary permeability with leakage of protein-rich material in to the alveolar space. In addition, local and systemic inflammatory responses further contribute to the clinical picture of the NPE. Acute brain insult causes local and systemic production of cytokines. Cytokines from the astrocytes and microglia gain access to the systemic circulation and stimulate target organs such as the lung. One such cytokines is substance P. Experimentally induced brain insult caused increased production of substance P resulting in bronchoconstriction, and increased capillary pressure with pulmonary edema [16]. Our patient presented with minor neurological deficit that improved within short period of time. Shortly after patient started complaining of difficulty of breathing. Her 6 blood pressure increased, she became tahycardic and tachypnic. Her lung exam changed with rales half way her chest, decreased oxygen saturation and fatigue. She did not have JVD. The chest radiograph showed changes consistent with pulmonary edema. Patient was intubated and transferred to the intensive care unit. A repeat CT scan showed massive right sided infarct with worsening of her neurological status. Consistent with the proposed mechanism of increased sympathetic activity

the elevated blood pressure, followed by respiratory distress made us believe that her pulmonary edema is caused by the neurogenic event. The change in her lung exam within minutes without proceeding respiratory complaint and changes in her chest radiography indicated the acute nature of her pulmonary edema in the setting of a neurologic insult. The subsequent CT scan of the brain showed a big infarct zone in the right middle cerebral artery region. This patient may have a herald TIA as a stroke in progress that worsened over the course her emergency department stay. The progression of her stroke over a short period of time is likely the cause of her acute pulmonary event with increased autonomic activity and increased blood pressure. An explanation for her condition is probably a minor stroke with the initial symptom and an additional event that led to worsening of her condition with pulmonary edema and complete left hemiplegia. The worsening of her neurological status could also be contributed by the associated hypoxic event that resulted from her respiratory status or from worsening of the neurological status. In addition, aggressive management of the pulmonary edema with subsequent drop in blood pressure might have also contributed to the worsening of the neurological status.

Conclusion

Different clinical scenarios may be present at the same time or one event may lead to the other. Heightened awareness, quick diagnosis and treatment are essential for a better out come. Early and appropriate treatment of both the underlying neurological cause and the pulmonary edema is the cornerstone of NPE.

References

1. Brambrink AM, Dick WF. Neurogenic pulmonary edema. Pathogenesis, clinical picture and therapy. Anaesthesist. Nov 1997;46(11):953-963.
2. Baumann A, Audibert G, McDonnell J, et al. Neurogenic pulmonary edema. ActaAnaesthesiol Scand. Apr 2007;51(4):447-455.
3. Sedy J, Zicha J, Kunes J, et al. Mechanisms of neurogenic pulmonary edema development. Physiol Res. 2008;57(4):499-506.
4. Leal Filho MB, Morandin RC, de Almeida AR, et al. Hemodynamic parameters and neurogenic pulmonary edema following spinal cord injury: an experimental model. ArqNeuropsiquiatr. Dec 2005;63(4):990-996.
5. Urdaneta F, Layon AJ. Respiratory complications in patients with traumatic cervical spine injuries: case report and review of the literature. J ClinAnesth. Aug 2003;15(5):398-405.
6. McClellan MD, Dauber IM, Weil JV. Elevated intracranial pressure increases pulmonary vascular permeability to protein. J Appl Physiol. Sep 1989;67(3):1185- 1191.
7. Ott L, McClain CJ, Gillespie M, et al. Cytokines and metabolic dysfunction after severe head injury. J Neurotrauma. Oct 1994;11(5):447-472.
8. Skrabal CA, Thompson LO, Potapov EV, et al. Organ-specific regulation of proinflammatory molecules in heart, lung, and kidney following brain death. J Surg Res. Jan 2005;123(1):118-125.
9. Minnear FL, Connell RS. Increased permeability of the capillary-alveolar barriers in neurogenic pulmonary edema (NPE). Microvasc Res. Nov 1981;22(3):345-366.
10. Sedy J. The rapidity of intracranial pressure increase reflects the grade of neurogenic pulmonary edema. J ClinAnesth. Sep 2008;20(6):479; author reply 480.
11. Ochiai H, Yamakawa Y, Kubota E. Deformation of the ventrolateral medulla oblongata by subarachnoid hemorrhage from ruptured vertebral artery aneurysms causes neurogenic pulmonary edema. Neurol Med Chir (Tokyo). Nov 2001;41(11):529-534; discussion 534-525. 12. Ahrens J, Capelle HH, Przemeck M. Neurogenic pulmonary edema in a fatal case of subarachnoid hemorrhage. J ClinAnesth. Mar 2008;20(2):129-132.
12. Prager P, Nolan M, Andrews IP, et al. Neurogenic pulmonary edema in enterovirus 71 encephalitis is not uniformly fatal but causes severe morbidity in survivors. PediatrCrit Care Med. Jul 2003;4(3):377-381.
13. Summerfield R, Tubridy N, Sirker A, et al. Pulmonary oedema with multiple sclerosis. J R Soc Med. Aug 2002;95(8):401-402.
14. Sarnoff SJ, Berglund E, Sarnoff LC. Neurohemodynamics of pulmonary edema. III. Estimated changes in pulmonary blood volume accompanying systemic vasoconstriction and vasodilation. J Appl Physiol. Jan 1953;5(7):367-374.
15. Campos MM, Calixto JB. Neurokinin mediation of edema and inflammation. Neuropeptides. Oct 2000;34(5):314-322.

Citation: Hassen GW, Brown D, Pashmforoosh G, Adamakos D, Barry T, Jaiswal R, Gonzalez L, Chirurgi R and Kalantari H. Neurogenic Pulmonary Edema: (TIA complicated with Acute Pulmonary Edema). ES J Case Rep. 2020; 1(1): 1006.