

Rapid Resolution of the Neurogenic Pulmonary Edema after Evacuation of the Cerebellar Hematoma: Case Report

*Serebellar Yerleşimli Hematomun Boşatılması Sonrası Hızlı İyileşen
Nörojenik Akciğer Ödemi: Olgu Sunumu*

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Neurogenic pulmonary edema is a well-known entity that may be seen after head trauma or any pathology leading to increase intracranial pressure. Any pressure effect on the solitary nucleus or hypothalamus or medulla oblongata may cause excessive sympathetic discharge and leads to changing of the pulmonary vascular pressure causing transcapillary fluid leak and resulting in pulmonary edema. However, in some cases no pathology is observed at the hypothalamus, medulla or the solitary nucleus, so the exact mechanism leading to neurogenic pulmonary edema could not be explained as yet. We presented this case to emphasize the importance of the evaluation of the respiratory function in patients with hemorrhage at the cerebellopontine angle due to arteriovenous malformation.

Key words: Cerebellar hematoma; neurogenic pulmonary edema; sympathetic discharge.

Nörojenik akciğer ödemi kafa içi basınç artışı yapabilecek herhangi bir patoloji sonrası görülebilir. Medulla oblongata, soliter nükleus veya hipotalamus direkt olarak etkileyen lezyonlar veya bu bölgelerde basınç artışı yapan patolojiler normalin üzerinde sempatik boşalmaya neden olur, pulmoner vasküler basınç artar; artmış pulmoner vasküler basınç pulmoner kapiller yataktan sıvı kaçışına neden olur ve pulmoner ödem meydana gelir. Bazı nörojenik pulmoner ödem olgularında ise yukarıda sayılan anatomik oluşumlardan hiçbirinde patoloji görülmez, bu nedenle nörojenik pulmoner ödeme yol açan mekanizmalar tam olarak anlaşılamamıştır. Serebellopontin köşe hematomu olan hastalar solunum fonksiyonları açısından yakından takip edilerek, gelişebilecek nörojenik pulmoner ödem açısından dikkatli olunmalıdır.

Anahtar sözcükler: Serebellar hematom; nörojenik pulmoner ödem; sempatik boşalma.

It is generally accepted that neurogenic pulmonary edema (NPE) is caused by an imbalance characterized by an excessive sympathetic outflow, but the precise cause of the NPE is still unclear. In different studies authors has proposed possible mechanisms to explain the cause

of NPE.^[1-6] Yabumoto et al.^[7] advocated that circulating catecholamines exert a direct effect on the pulmonary vascularization which being independent of any systemic hypertension will disrupt and damage the capillary endothelium. In the present report, we described a case of NPE

caused by right cerebellopontine angle hemorrhage due to arteriovenous malformation.

CASE REPORT

An 18-year-old young woman complaining of headache, nausea and dyspnea was admitted to the emergency room. On admission, her Glasgow coma scale score of 15. She was not able to walk due to right sided ataxia. She had dysmetria, dysdiadochokinesia, intentional tremor on the right side and nystagmus. She had tachycardia (138 bpm), with systolic-diastolic hypertension (160/110 mmHg) and respiratory failure and dyspnea, breathing about 28 times per minute. Her friends escorting her stated that she was fully cooperated, but she had headache and nausea, and then she developed respiratory failure after 20 minutes of her headache. Her arterial blood gas values were: PH = 7.24; PO₂ = 58 mmHg; PCO₂ = 52 mmHg; HCO₃ = 32 mEq/L. The patient was immediately intubated due to serious dyspnea and respiratory acidosis. A lung radiograph showed diffuse pulmonary edema, (Fig. 1) and a cranial CT and CT angiography demonstrated right cerebellar hemorrhage just beside the mesencephalon (Fig. 2). She had undergone surgery to drain a cerebellar hemorrhage. According to her relatives' information, she had undergone gamma knife surgery for AVM located at the brain stem two years ago and glue embolization for AVM

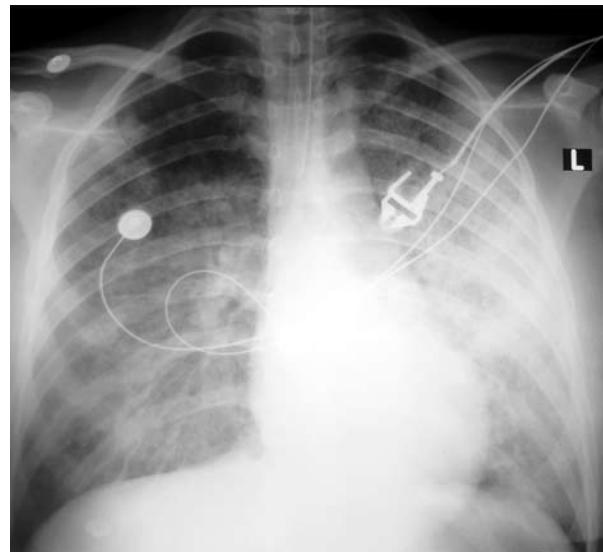


Fig. 1. Chest radiograph showing diffuse lung edema.

located at the right maxillomandibular region one and a half year ago. The patient was operated immediately. In the operation, the region of the suboccipital craniectomy was enlarged to the margin of the sigmoid sinus. Cerebellum was minimally retracted and hemorrhage was seen and evacuated, but we did not see any vascular malformation around the hemorrhagic area. After the operation the patient was transferred to the intensive care unit. Her lung radiography revealed improvement of pulmonary edema (Fig. 3), and respiratory acidosis, tachycardia and blood pressure returned within physiologic

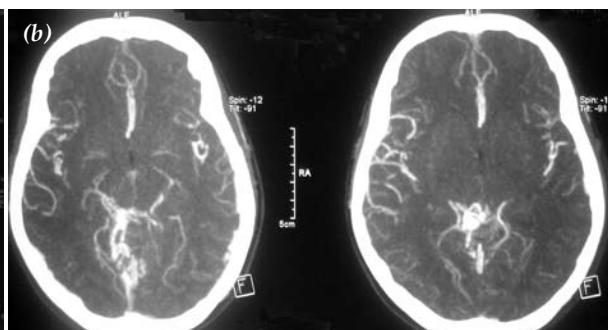
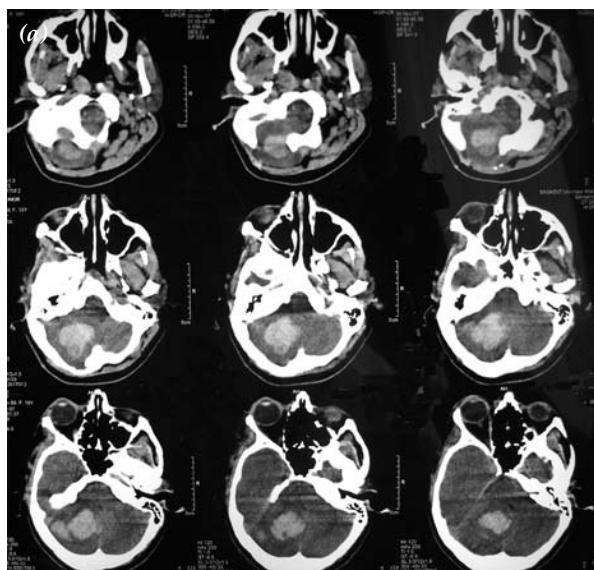


Fig. 2. (a) Preoperative CT brain scan without contrast showing the suboccipital craniectomy defect and cerebellar hemorrhage (top three) and left paravermian cerebellar hemorrhage (middle three). Vermian hemorrhage at the level of the mesencephalon and fourth ventricle shifted to the right (bottom three). (b) Preoperative CT angiography showing right arteriovenous malformation located at the vermis and tectum and no enlargement at the third ventricle.

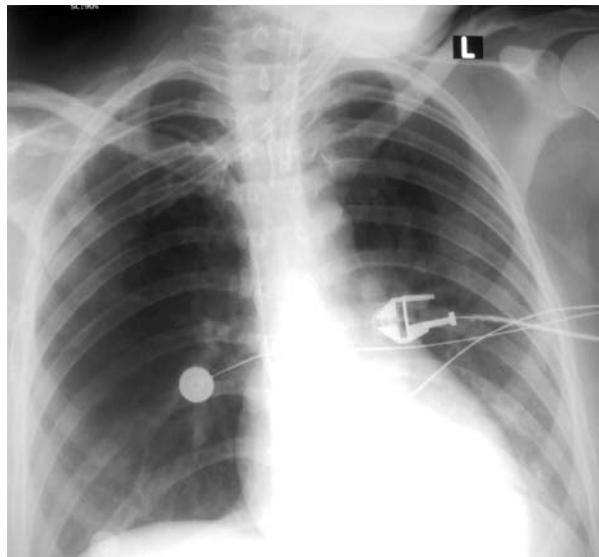


Fig. 3. Chest radiograph showing no pathology at the post-operative period.

limits 12 hours after the operation. Twenty four hours after the operation, the patient became mobilized but was unable to walk independently. She had right-sided ataxia. She recovered and started to walk independently, but had mild ataxia. Ten days after the operation, conventional angiography showed multiple small feeders of the arteriovenous malformation around the brain stem region. It was found inappropriate for glue embolization and for surgical intervention (Fig. 4). When we compared this recent angiography with the other one performed, after the Gamma Knife surgery and glue embolization,

about one year before, it showed no change in the pattern of the arteriovenous malformation.

DISCUSSION

A head trauma or the pathology related to the intracranial region may result in NPE, but the case of NPE with posterior fossa hemorrhage is not as common as NPE due to cerebral hemorrhage or another cerebral pathology such as trauma, tumor.^[5,8] Brain stem lesions, in different regions including ventrolateral medulla oblongata, the solitary nucleus in the dorsal medulla, dorsal motor nucleus of the vagus, A1 region and medial reticular nuclei in the medulla, sublobule IX-b which is located at the cerebellar uvula may cause NPE and sympathetic discharge.^[3,4,9] These structures are integrated functionally and anatomically for the regulation of the sympathetic discharge. For instance, destruction of the ventrolateral medulla oblongata including the solitary nucleus may cause a catecholamine surge and sympathetic vasoconstriction. Any lesion or stimulus such as chemical or electrical to the sub-lobule IX-b of the uvula could result in several changes in cardiovascular and respiratory function.^[3,9] It is shown that cerebellar uvula has interconnections with the pons, medulla, so the lesion affecting the cerebellar uvula could lead to increased sympathetic activity manifested by tachycardia, hypertension, respiratory irregularities and the damage of the pulmonary capillary vessels. However, some lesions could

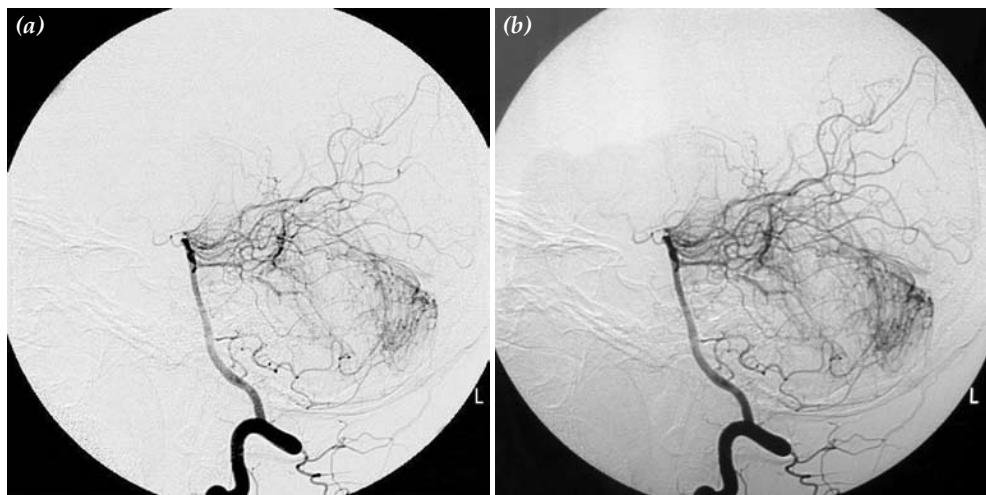


Fig. 4. (a) Lateral angiography performed one year before. (b) Postoperative lateral angiography. Arteriovenous malformation was still present at the postoperative angiography.

not cause sympathetic discharge, but could lead to NPE that is demonstrated in bilateral solitary nucleus lesions. This lesion results in a marked rise in pulmonary arterial pressure without changes in systemic or left atrial pressure.^[1,3,4,9,10] Our case showed increased sympathetic activity signs and pulmonary edema which possibly resulted in hematoma located in the cerebellopontine angle. The previous cerebellopontine angle incision site was used during surgical evacuation of the hematoma; when we reached at the bottom of the hematoma, we identified the base of the fourth ventricle laterally. We did not find any active bleeding area. We thought that the hematoma mostly affected the uvula, pyramis, tonsil, biventer lobule, gracile lobule and medulla according to the cranial CT image and intraoperative appearance. After the operation, our patient had no sympathetic activity signs and also her lung radiography showed rapid resorption of the pulmonary edema (Fig. 3). Preoperative cranial CT showed that the uvula, pyramis, tonsil, biventer lobule, gracile lobule and medulla was under pressure because of the cerebellar hemorrhage. This pressure, especially around the uvula might have caused interruption between the uvula and pontomedullary region, and the patient developed acute sympathetic discharge due to this interruption.

We infer that patients with cerebellar hemorrhage around the vermicular region and the signs of respiratory difficulty, hypertension and tachycardia should be promptly operated to evacuate the hemorrhage for alleviating the pressure. This helps decrease the pressure on the paravermian region and brainstem. It will prevent further

complications such as hypoxic effect of diffuse lung edema and further bleeding risk due to sympathetic discharge.

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