Neurogenic pulmonary edema associated with a colloid cyst in the third ventricle

Case report

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Neurogenic pulmonary edema (NPE) is usually the result of head trauma. The authors describe the case of a 13-year-old girl, in whom NPE was associated with a colloid cyst of the third ventricle causing acute hydrocephalus. The mechanisms involved in the development of NPE are briefly discussed. The possible role of the colloid cyst in the distortion of the anatomical relationships in the vicinity of the hypothalamic nuclei is considered.

Key Words • colloid cyst • increased intracranial pressure • acute hydrocephalus • neurogenic pulmonary edema

The symptoms most frequently produced by a colloid cyst in the third ventricle are generally attributed to intermittent obstruction of the foramina of Monro, with acute increase in intracranial pressure (ICP). The symptoms consist mainly of severe headache, sudden bouts of vomiting, blurred vision, drop attacks, and even sudden death. Neurogenic pulmonary edema can complicate increased ICP, especially when the latter is due to head trauma.

This paper describes a patient in whom acute pulmonary edema was one of the presenting signs of a colloid cyst in the third ventricle. The pulmonary edema resolved rapidly following drainage of the obstructed ventricles. To our knowledge, such a course of events has not been documented before.

Case Report

This previously healthy 13-year-old girl suffered a sudden attack of headache, vomiting, and blurred vision a few hours before her admission. Upon arrival in the emergency room she was stuporous, and became deeply comatose over the next 2 hours. There was no history of trauma.

Examination. She responded to painful stimuli only, without lateralizing signs. Pupils were equal and reacted sluggishly to light. There was slight blurring of both disc margins, but no frank papilledema. Tendon reflexes were hyperactive. Blood pressure was 130/100 mm Hg, and the pulse 62/min. She was afebrile. There were no signs of meningeal irritation.

Routine blood and urine examinations were normal. A supine chest film and skull films did not disclose any pathology. A computerized tomography (CT) scan demonstrated marked dilation of both lateral ventricles, and of the anterior part of the third ventricle (Fig. 1 left). The child was immediately transferred to the operating room, with the diagnosis of acute obstructive hydrocephalus.

Operations. Under general anesthesia induced with Pavulon (pancuronium bromide) and Pentothal (sodium thiopental), she was intubated without complications. Through a right frontal burr hole, a cannula was placed in the frontal horn of the right lateral ventricle. The cerebrospinal fluid (CSF) was clear, but had an opening pressure of 600 mm H2O. At the end of the procedure, the patient was extubated. Although she was still unconscious, spontaneous breathing was
FIG. 1. Computerized tomography scans. Left: Scan showing symmetrical dilation of both lateral ventricles. Right: Scan performed 6 hours after initial surgery. The left lateral ventricle is still dilated, indicating pressure of obstruction in the third ventricle. Note the midline shift to the right. The white spot in the center of the brain is the cannula.

The CSF drainage rate was 10 cc/hr. The ICP was 12 mm Hg. Two hours later, however, increasing respiratory distress was noted, with tachypnea of 36 breaths/min and intercostal retractions. Repeat chest film revealed bilateral pulmonary edema without enlargement of the cardiac shadow (Fig. 2). The ICP at that time had increased to 35 mm Hg.

The patient was reintubated and abundant frothy pink fluid appeared in the airway. She was placed on a volume ventilator with 50% inspired oxygen concentration ($F_\text{IO}_2 = 0.5$) and positive end-expiratory pressure (PEEP) of 5 cm H$_2$O. Arterial blood gases under those circumstances still indicated severe hypoxemia ($PaO_2$ 37 mm Hg) and mild respiratory acidosis ($PaCO_2$ 46 mm Hg). There was no evidence of over-hydration (central venous pressure 6 cm H$_2$O, blood pressure 110/70 mm Hg) or electrolyte imbalance. Mechanical ventilatory rate was increased and $PaCO_2$ reduced to 30 mm Hg. The patient was rapidly given digitalis, and treated with diuretics; however, arterial oxygenation improved only moderately ($PaO_2$ 67 mm Hg). The CT scan was repeated and revealed a collapsed right ventricle, while the left ventricle was still markedly dilated with midline shift to the right (Fig. 1 right).

It was obvious, at that time, that a third ventricle space-occupying lesion, missed on the first CT scan for technical reasons, was obstructing both foramina of Monro, preventing drainage of the left ventricle. The patient was reoperated on 8 hours after the initial procedure, and the left ventricle was similarly decompressed. Both ventricular cannulas were left in place to obtain adequate drainage and to monitor CSF pressure. The patient was maintained on hyper-ventilation for the following 3 days, and ICP was continuously monitored, ranging between 7 and 20 mm Hg. Pulmonary edema resolved within hours following the left ventricular decompression, arterial oxygenation improved ($PaO_2$ 127 mm Hg on $F_\text{IO}_2 = 0.3$, PEEP of 6 cm H$_2$O), and the chest x-ray film showed clearing of the lung fields. The patient was extubated 4 days after surgery.

FIG. 2. Plain chest film, 2 hours after placement of the cannula into the right lateral ventricle. Bilateral pulmonary edema is evident.
A pneumoencephalogram revealed a tumor of the third ventricle (Fig. 3). The patient underwent definitive surgery 9 days following admission, and total removal of a colloid cyst was performed via a transcallosal approach. She made a complete, uneventful recovery.

**Discussion**

Neurogenic pulmonary edema (NPE) as a complication of increased ICP resulting from head trauma has been amply described, both clinically and experimentally. Experimental models of NPE were also reported after the introduction of blood and fibrin into the subarachnoid space, and with cerebral hypoxic anoxia, achieved either by vascular occlusion or in association with convulsions. Reversible NPE, complicating hydrocephalus secondary to a tumor in the third ventricle, has not been reported previously.

The pathogenic mechanisms involved in NPE, as well as clinical and experimental evidence to support these mechanisms, have been excellently reviewed by Theodore and Robin. According to the accepted theory, NPE results from acute stimulation of the autonomic centers in the hypothalamus. A subsequent massive sympathetic discharge causes a sudden shift of blood from the peripheral circulation to the low-resistance pulmonary vasculature. There is a marked increase in pulmonary hydrostatic pressure, which leads to acute pulmonary edema. Damage to the endothelium of the pulmonary capillaries and a reduction in pulmonary surfactant activity have also been implicated.

A colloid cyst of the third ventricle may theoretically lead to NPE by direct pressure on the hypothalamic nuclei, or by interrupting the venous drainage of the hypothalamic structures. Stookey suggested the possibility of obstruction of both veins of Galen by a tumor. Sackett, et al., found compression of the internal cerebral veins and the thalamostriate veins, as well as closure of the venous angle, to be the most specific angiographic sign of a colloid cyst. This disturbed venous drainage of the hypothalamus may be the result of either direct pressure by the cyst, or of the acute increase of ICP following the obstruction of both foramina of Monro.

We could find only a very few reports of patients with a colloid cyst in the third ventricle who demonstrated any kind of respiratory distress, although Rinder and Cannon happened to mention the findings of pulmonary edema at autopsy in a patient who suddenly died of obstruction to CSF flow by a colloid cyst.

It is interesting to note that our patient did not show respiratory distress while suffering bilateral symmetrical hydrocephalus with intraventricular pressure as high as 600 mm H$_2$O. The NPE occurred only in association with midline shift due to insufficient drainage of one obstructed ventricle, and resolved completely following decompression of both ventricles. It is conceivable that NPE due to a colloid cyst could occur when elevated ICP is associated with anatomical distortion of the hypothalamic nuclei. This may result in the compression of these nuclei or in interference with their venous drainage. This hypothesis is consistent with most reports on NPE following head injury, where normal anatomical relationships are more likely to be disturbed.

**References**


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