

An Unusual Presentation of Large Pneumoencephaly after Frequent Falls in an Elderly Patient

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Abstract- Pneumoencephaly is usually a complication of head trauma or craniotomy. We present a patient with large pneumoencephaly with acute neurological deficit after frequent falls, but without recent major trauma or surgery. Skull X-ray films show intracranial air in the frontal region and in the 2nd, 3rd, and 4th ventricles, reminiscent of an pneumoencephalogram. Computed tomography (CT) of the head shows air in the right frontal region and lateral ventricles. After craniotomy to relieve the pressure of the air, the clinical condition of the patient improved significantly after six months.

Key Words: Pneumoencephaly, Acute neurological deficit, Skull X-ray film, Computed tomography of the head

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INTRODUCTION

Pneumoencephaly (air inside the skull) is usually a complication of acute head trauma or neurosurgical procedures with craniotomy. There are also sporadic reports of iatrogenic causes such as pleurodural fistula after chest wall surgery⁽¹⁾ and dural puncture in an obstetric patient⁽²⁾. Pneumoencephaly happens because small volumes of air inside the skull communicate with either the subarachnoid space or the lateral ventricles when the dura is penetrated by trauma, and is typically asymptomatic with self-absorption of air within a few days. It is

unlikely to occur spontaneously without histories of major trauma or surgical procedures, or occur as a late complication of head trauma⁽³⁾. It is also uncommon to develop acute neurological deficits, such as sudden loss of consciousness, limb weakness or convulsion⁽¹⁻⁴⁾. We present a 76 years old man of large pneumoencephaly with acute neurological deficit after frequent falls, but without recent major head trauma. The patient received decompression craniotomy with improvement of the mental function and amelioration of the gait disturbance.

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CASE REPORT

A 76 year-old male farmer was taken to the emergency department after he was unable to be awakened by his families in the morning. General weakness and urine incontinence were also noted for several hours. According to his families, the patient had progressive forgetfulness, decreasing verbal output, and recurrent falls in the recent one year. He sometimes choked while drinking water, but he still went for farming everyday and he could perform self-care properly.

He has two episodes of head injuries 30 years and 6 months ago but did not seek for any medical attention. The families denied that he had stroke before. Also, he has no fever, nausea, vomiting, or headache at home, and he did not take any medication regularly. He has hypertension without medical control and he has smoked 2 packs of cigarettes per day for 30 years.

On examination, the patient was stuporous and unable to follow orders. There was no verbal communication. Test of motor strength in both upper and lower extremities revealed muscle power of 3/5 on the left and 4/5 on the right. The deep tendon reflexes were 1+ in all four extremities. The plantar responses were flexor. The blood pressure was 188/121 mmHg, and the body temperature was 37.2°C.

The patient was admitted to the hospital. The laboratory data, including complete blood count, serum electrolytes, albumin, tests of renal and hepatic function, CPK, CRP thyroid function, folic acid, and vitamin B12, were in general unremarkable, except that the glucose level was 210 mg per deciliter. Skull X-ray films (AP and lateral views) showed intracranial air collection in the frontal region and in the 2nd, 3rd, and 4th ventricles, reminiscent of an pneumoencephalogram (Fig. 1). Computed tomographic (CT) scanning of the head with bone windows showed air-densities in the right frontal region and lateral ventricles with mass effect on the remaining parts of the frontal lobe (Fig. 2A-B). Small patchy hypodensities in the right frontal and occipital lobes (Fig. 2C-D) and brainstem (Fig. 2E-F) suggested old lacunar infarctions and old fracture of the right temporal bone (Fig. 3). After admission, the patient received conservative treatment. On the 10th day of hospitalization, the patient remained drowsy, only roused briefly to voice and able to follow some simple commands. He also showed symptoms of frontal lobe dysfunction including apathy, perservation and scanty verbal output. The score of the Mini-Mental State Examination was 4/30. Motor strength was mildly impaired (4/5) in the arms and the right leg and 3/5 in the left leg. The patient was unable to stand unless given full assistance. The

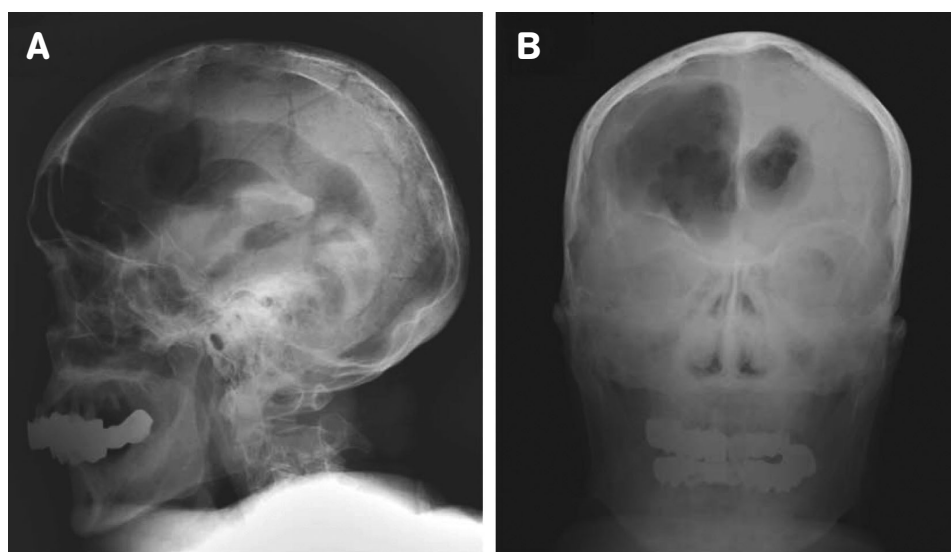


Figure 1. The lateral view (A) and AP view (B) of skull X-ray films show intracranial air collection in the frontal region and the 2nd, 3rd, and 4th ventricles, reminiscent of a pneumoencephalogram.

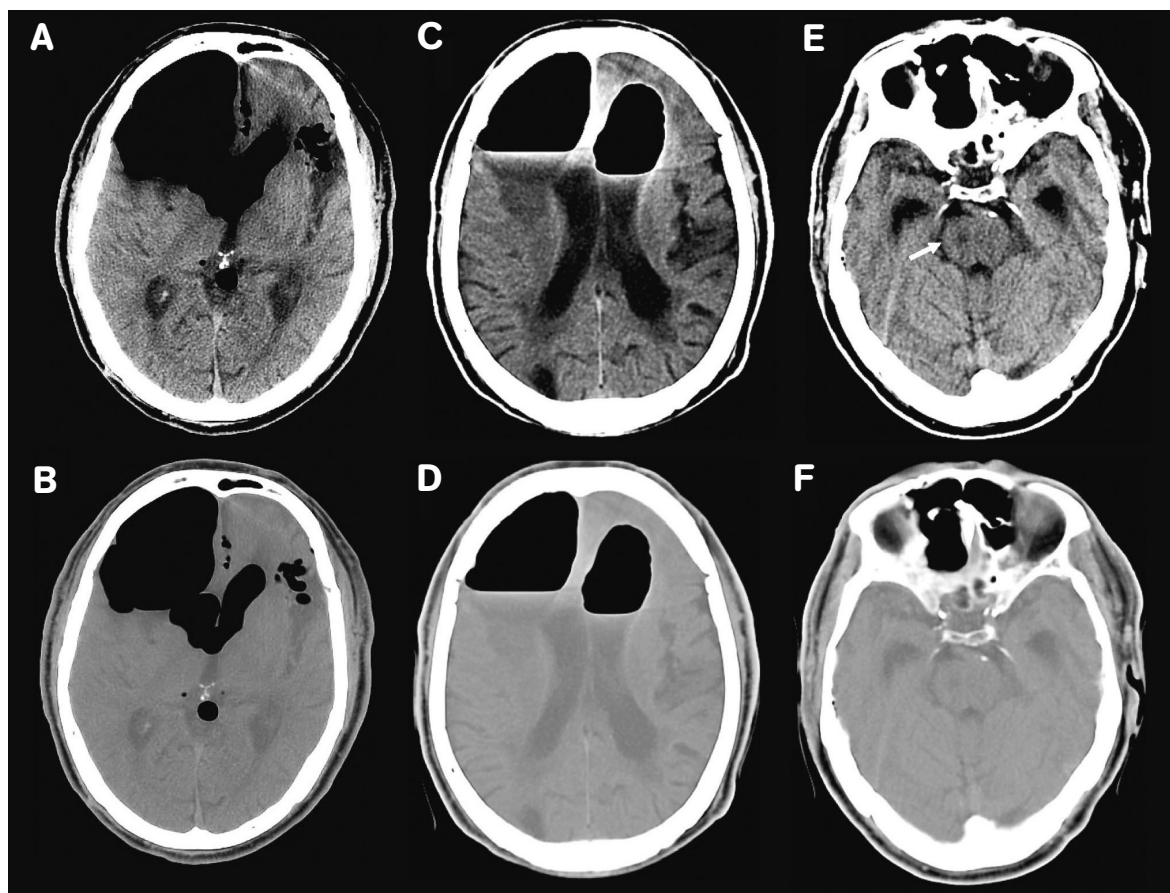


Figure 2. Routine noncontrast enhanced CT (A,C,E) and bone-window CT (B,D,F) show extensive pneumocephalus in the right frontal subdura, the left anterior parafalcine (A,B), both lateral ventricles, the supracerebellar cistern, and the left sylvian fissure, with flattening of the right frontal lobe. Small patchy hypodensities in the right frontal and occipital lobes (C,D) and brainstem (E,F) suggest old lacunar infarctions (arrow).

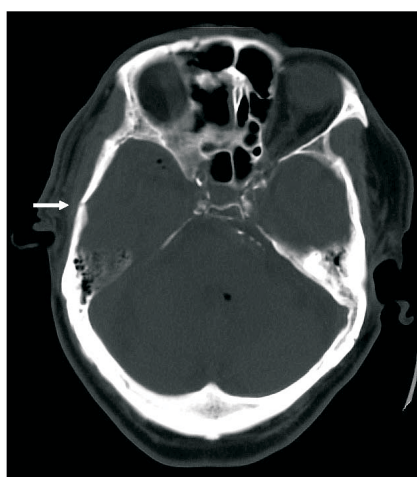


Figure 3. Bone-window CT shows mild deformity of the right temporal bone (arrow).

cerebellar function, pinprick as well as vibration sensations, and proprioception were intact. Neurosurgical consultation and decompression craniotomy was performed. Isotonic saline 200 ml was infused inside the burr-hole. After craniotomy, the patient became awake but showed symptoms of frontal lobe dysfunction such as uncontrollable crying occasionally. He could then walk with support and was discharged one week later. Six months after discharge, the patient has significant improvement in both mental and motor functions. His score of Mini-Mental State Examination was 11/30. Follow-up head CT of bone windows revealed right frontal subdural effusion in replacement of the previous air-densities (Fig. 4).

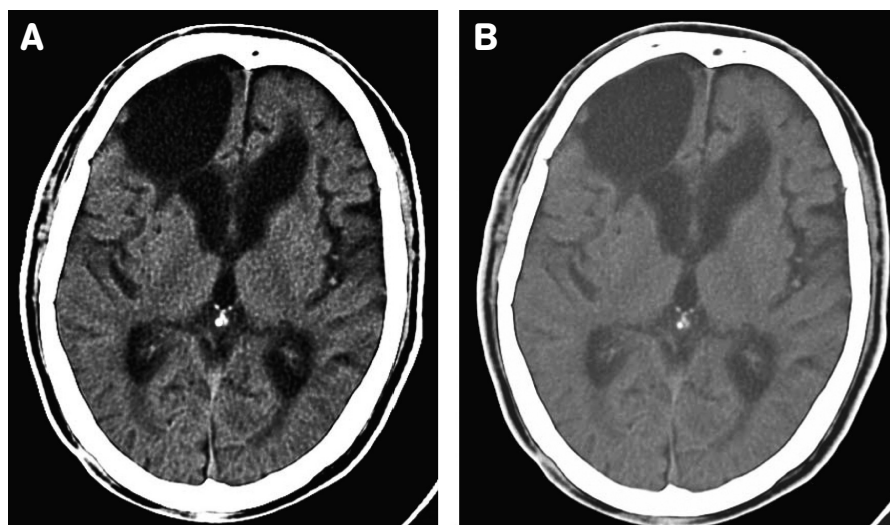


Figure 4. Six months after craniotomy, routine follow-up CT (A) and that with bone windows (B) reveal that the pneumocephalus is replaced by right frontal subdural effusion.

DISCUSSION

The causes of pneumoencephaly are (1) disruptions of the arachnoid and dura mater with (2) a skull bone defect, and (3) a CSF pressure gradient that is continuously or intermittently smaller than the tensile force of the disrupted tissue. We may call it “tension pneumoencephaly”. This patient presented with old temporal bony fracture and with tissue loss in the right frontal region and lateral ventricles with mass effect on the remaining parts of the frontal lobe prior to the pneumoencephaly. Although the patient denied any recent major head trauma, pneumoencephaly with acute neurological deficit may result from recent dura tear with old temporal bony fracture. It may happen as a late complication of head trauma⁽³⁾ after frequent falls.

In the normal state, the dural sac, CSF, brain, and blood constitute part of a closed system. The dura stretch and tear in areas of bony floor defect⁽⁴⁾ is subject to wide variations in CSF pressure because of the following factors: (a) arterial pressure fluctuations (b) respiratory fluctuations and (c) Valsalva-like actions during nose blowing⁽⁵⁾. Patients with a habit of forceful nose-blowing may result in dural injury with chronic CSF leaks⁽⁶⁾ or pneumocephalus⁽⁷⁾, especially for those who have congenital skull base dehiscences. This patient has hyperten-

sion without medical control, the fluctuation of arterial pressure may therefore contribute to dural stress in areas of bony fracture⁽⁸⁾. On the other hand, he did not have breathing difficulty and nose-blowing habit.

The “large tension pneumoencephaly” with partial reabsorption in this patient is very rare clinically. According to the Monro-Kellie hypothesis⁽⁹⁾, the combined CSF and blood volumes contribute to the buoyancy supporting the brain when the head is upright. Normally the intracranial pressure is -5 to +5 cm H₂O with reference to the foramen of Monro in upright position. After craniotomy or skull bone fracture, the closed system of CSF is to the atmosphere, and thus the Monro-Kellie hypothesis no longer applies. The CSF pressure gradient greater than the tensile strength of the disrupted tissue then results in CSF leaks. However, if the CSF production from choroids plexus is not sufficient to compensate for the loss of CSF, the intracranial pressure decreases. The intracranial pressure becomes more negative and air may enter the subarachnoid space⁽¹⁰⁾ to make an air-fluid level. It may explain the conditions in our patient, who has the air trapped inside the right frontal cavity which communicates with the lateral ventricle. The “tension pneumoencephaly” is dissimilar to “porencephaly”, which filled with CSF to prevent free communication between the fluid and the atmosphere.

Decompression craniotomy performed in this patient relieved the mass effect on the remaining parts of the frontal lobe and improved the clinical condition. Because the closed system is opened to the atmosphere, isotonic saline were infused to the burr-hole to prevent negative pressure and air trapping during craniotomy⁽¹¹⁾ and to compensate for CSF hypovolemia⁽¹²⁻¹³⁾. It should be noted that craniotomies performed in patients with CSF hypovolemia are subject to increased risk of brain herniation⁽¹⁴⁾ because of the decreased volume of CSF and consequent decreased brain buoyancy. The follow-up head CT revealed that the pneumocephalus is replaced by right frontal subdural effusion.

Long-term care for the disabled patients to prevent accidental head trauma are also important. In this patient, the families managed to provide a live-in caregiver at home, and both mental and motor functions significantly improved after six months.

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