Case Report

Sudden Migration of a Thalamic Hemorrhage into the Ventricles

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Spontaneous intracerebral hemorrhage (ICH) is a common condition that often leads to death or disability. Accurate prediction of the outcome and decisions regarding the treatment of ICH patients are important issues. We report a case of thalamic hemorrhage with an intraventricular hemorrhage that was suddenly migrated into the third and fourth ventricles in its entirety 8 hours after symptom onset. To our knowledge, this case is the first report of spontaneous migration of thalamic ICH into ventricles, and we suggest a possible mechanism for this case with a brief review of the literature.

KEY WORDS : Cerebral hemorrhage · Cerebral ventricles.

INTRODUCTION

The worldwide incidence of spontaneous intracerebral hemorrhage (ICH) ranges from 10 to 20 cases per 100,000 population, and increases with age[14,16]. Several studies have identified individual clinical and radiological variables that are correlated with mortality in ICH. These include the hematoma size or volume, Glasgow coma scale (GCS) score, presence of hydrocephalus, intraventricular extension, degree of midline shift, level of consciousness, age, fever, hyperglycemia, and pulse pressure[1,2,4-7,11,13,16,18,19].

It is difficult to compare the effectiveness of medical and surgical treatment in patients with ICH and it is frequently necessary to perform both forms of treatment, as they are often complementary[3].

We experienced a case of right thalamic ICH with intraventricular hemorrhage (IVH). The hematoma volume was about 12 mL. Initially, the patient was treated conservatively. However, 8 hours after symptom onset, the patient abruptly became semi-comatose, and computed tomography (CT) revealed that the entire thalamic ICH had been sud-

denly migrated into the third and fourth ventricles. Emergency external ventricular drainage (EVD) and bifrontal decompressive craniectomy were performed. However, he died five days later. We introduce this case with a brief review of the literature.

CASE REPORT

A 31-year-old man was hospitalized because he became stuporous abruptly. He had no history of trauma, hypertension, tumor, infectious disease, or previous surgery. On physical examination, his systolic blood pressure was 130-140 mmHg. On neurological examination, he was stuporous, and his pupil size was 3/2 mm, with no light reflex. His GCS score was 9. Brain CT on admission showed a right thalamic ICH (3.5 × 2.3 × 3.0 cm), with intraventricular extension and a midline brain shift of about 0.8 cm. The estimated total volume of the ICH was about 12 mL (Fig. 1). Eight hours after symptom onset, he suddenly became semi-comatose, and his pupils were 5/5 mm, with no light reflex. Follow-up brain CT showed no evidence of rebleeding, but the entire right thalamic ICH had migrated into the ventricular system suddenly, with marked hematoma accumulation in the third and fourth ventricles. The suprasellar and prepontine cisterns were obliterated and severe brain swelling with pseudo-subarachnoid hemorrhages (SAH) was noted (Fig. 2). We performed emergency
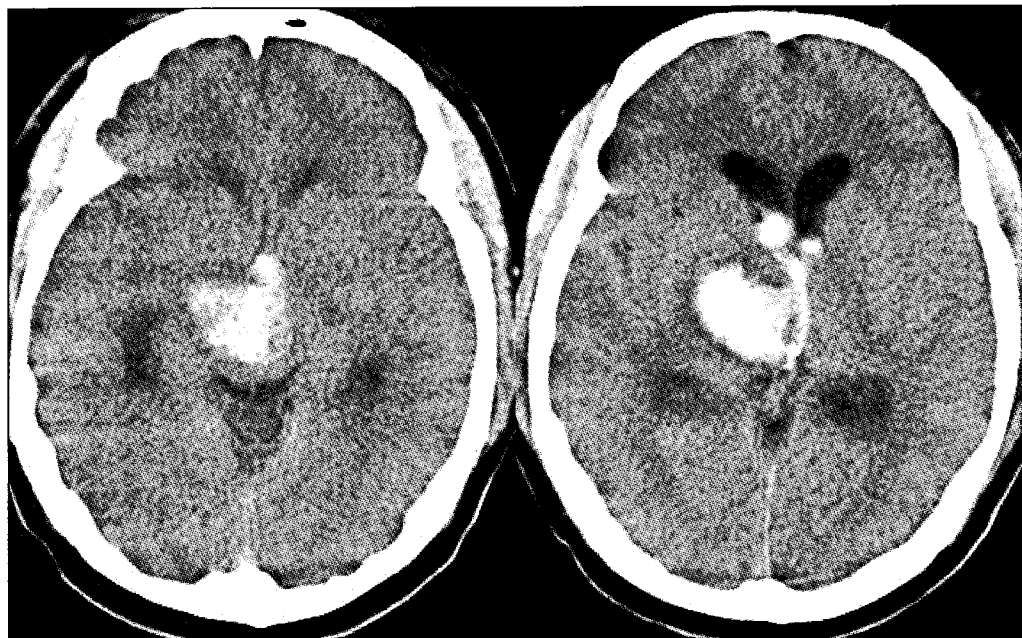
Fig. 1. Computed tomography scans on admission showing thalamic hemorrhage with ventricular extension. There is no evidence of non-communicating hydrocephalus.
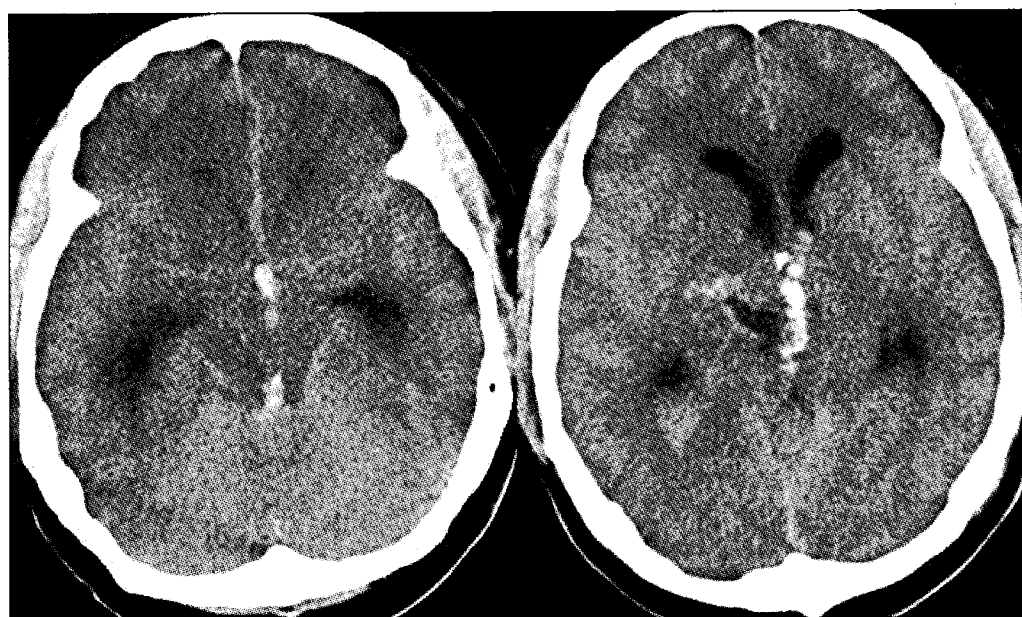

Fig. 2. Computed tomography scans were obtained 8 hours after the intracerebral hemorrhage (ICH). Note the entire thalamic ICH had been migrated into the ventricle abruptly and perimesencephalic cistern is obliterated. The bilateral temporal horns of lateral ventricle are slightly increased.

EVD on right Kocker's point for correction of non-communicating hydrocephalus. Follow-up brain CT after EVD showed decreased ventricle size, but remained intraventricular hemorrhage in both lateral ventricles, and ill defined low density lesions were seen on brain stem, medial frontal, temporal and occipital lobe. This lesion was suggested acute cerebral infarction due to increased intracranial pressure (ICP) (Fig. 3). Emergency bifrontal decompressive craniectomy was carried out in order to decrease ICP. However, his level of consciousness was deteriorated. He became comatose, and both pupils were fully dilated, with no light reflex. Magnetic resonance imaging (MRI) revealed acute infarction in posterior cerebral artery territory, including the pons and mid-brain (Fig. 4). He died five days after symptom onset.

DISCUSSION

ICH accounts for 10-15% of all strokes and is associated with a high mortality rate[3,7,10]. Indeed, the 30-day mortality for ICH is 34-53%[4,7,14]. Strong associations with poor out-
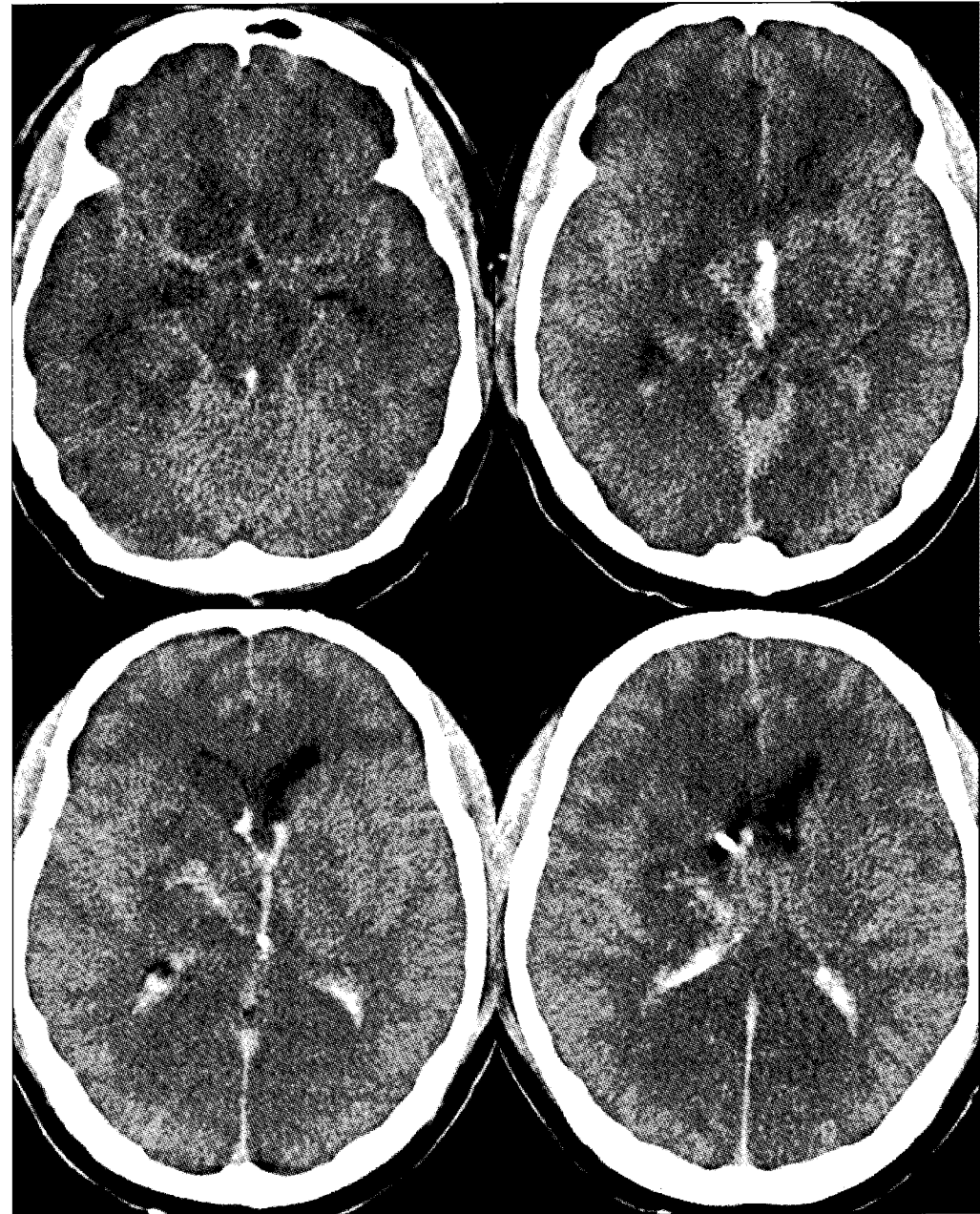
Fig. 3. Computed tomography scans after external ventricular drainage showing decreased ventricles size, both lateral and 3rd ventricle. Ill defined low density lesions are seen on brain stem, medial frontal, temporal and occipital lobe. Pseudo-subarachnoid hemorrhages are also seen in basal cistern.

come are known for mass effect and size, intraventricular extension, and decreased level of consciousness[1,2,4-7,9,10,14,16,18-20].

Early neurological deterioration (END) is common after an ICH; it occurs in 20-40% of patients and is associated with a poor prognosis[12]. The identification of predictors of END may help in designing therapeutic strategies for ICH. High body temperature, neutrophil count, and plasma fibrinogen on admission are independent predictors of END. Hyperthermia may be a result of the acute-phase reaction and inflammatory response. The fibrinogen levels may reflect the activation of inflammatory mechanisms responsible for tissue damage around the hematoma[12].

The mass effect, which results from the volume of the hematoma, the edematous tissue surrounding the hematoma, and obstructive hydrocephalus with subsequent herniation, remains the chief secondary cause of death in the first few days after ICH. Progressive elevation of intracranial pressure is seen in patients with massive ICH[3,15,16]. Extension of the ICH into the ventricles occurs in association with large, deep hematomas[6,16]. The presence of blood in the ventricle may be related to the development of obstructive hydrocephalus or a direct mass effect if the ventricular blood is located on a periventricular structure, which is associated with global hypoperfusion of the overlying

Fig. 4. After bifrontal decompressive craniectomy, fluid attenuated inversion recovery imaging magnetic resonance imaging demonstrates hyperintense infarcted regions in the posterior cerebral artery territories bilaterally.

cortex[6,9,15,16]. In our case, intraventricular extension of the hematoma was seen, but there was no obstructive hydrocephalus initially. In one quarter of patients with ICH, secondary deterioration of consciousness occurs within the first 24 hours, and worsening cerebral edema is implicated in the neurological deterioration that typically occurs within 24-48 hours after the onset of hemorrhage[8,9,16].

A comparison of the effectiveness of medical and surgical treatment in patients with ICH is difficult and both forms of treatment frequently complement each other[1,3]. A recent study suggested that aggressive, timely reversal of transtentorial herniation, through the use of hyperventilation and osmotic agents improved the long-term outcome[16]. The surgical removal of any hemorrhage is beneficial because it reduces the hematoma volume, lowers ICP, improves perfusion in the affected hemisphere, prevents secondary enlargement of the hematoma, and reduces neurotoxic edema[3,8,9,14]. The surgical results may be better if patients are selected for surgery earlier, before they deteriorate[3,7,20]. This is associated with decreased 30-day mortality, although the overall morbidity and mortality did not differ significantly in patients who underwent surgery compared with those who did not[4,14].

ICP may not respond to conservative management strategies, necessitating a decompressive craniectomy. Positive effects of bifrontal craniectomy on both ICP and the clinical

outcome of head-injury patients have recently been reported[20]. The effects of craniectomy include regression of blood congestion, reduced edema formation, and increased edema absorption. The ideal time when a craniectomy should be performed and the exact effects of a decompressive craniectomy on ICP and cerebral tissue oxygenation remain unclear[17]. The indications for a craniectomy are a decrease in local brain tissue oxygen pressure (ptiO$_2$) below 10 mmHg, coupled with ICP exceeding 20 mmHg, or a pro-gradient increase in ICP of at least 25 mmHg, resistant to conservative treatment[3,17].

In our case, follow up CT scans revealed that the entire hematoma was migrated into the ventricular system abruptly, and pseudo-SAH was newly developed in basal cisterns at 8 hours after onset. The mechanism for the development of a pseudo-SAH has not been elucidated fully. Severe brain edema compresses the dural sinuses, compromising the venous drainage from the brain and resulting in engorgement of the superficial veins, which stand out against the edematous low-attenuated brain parenchyma, mimicking an SAH and narrowing or disappearance of hypo-attenuated CSF space as an additional factor generating a pseudo-SAH[20]. A possible cause for this sudden migration of the hematoma into the ventricles may be that the hemispheric ICP was increased suddenly and pushed it into the ventricle. The causes of increased hemispheric ICP are unknown, but we postulate that a gradual increase in cerebral edema around the hematoma and compromising the venous drainage from the brain may be causes.

CONCLUSION

We report an extremely rare case involving the sudden migration of a thalamic ICH into the 3rd and 4th ventricles. A sudden migration of an ICH with pseudo-SAH may indicate increased ICP. Immediate ICP control, with medical and surgical treatment, is necessary in such patients.

References

1. Ahn CS, Lee SK, Kim HS, Kang MH, Song MY, Kang DS : Surgical outcome of spontaneous intracerebral hemorrhage in less than stuporous status. J Korean Neurosurg Soc 35 : 290-296, 2004
2. Broderick JP, Adams HP Jr, Barsan W, Feinberg W, Feldmann E, Grotta J, et al : Guidelines for the management of spontaneous intracerebral hemorrhage : a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke 30 : 905-915, 1999
3. Broderick JP, Brott TG, Duldner JE, Tomsick T, Huster G : Volume of intracerebral hemorrhage. A powerful and easy-to-use predictor of 30-day mortality. Stroke 24 : 987-993, 1993
4. Daverat P, Castel JP, Dartigues JF, Orgogozo JM : Death and functional outcome after spontaneous intracerebral hemorrhage. A prospective study of 166 cases using multivariate analysis. Stroke 22 :

1-6, 1991

5. Diringer MN, Edwards DF, Zazulia AR : Hydrocephalus : a previously unrecognized predictor of poor outcome from supratentorial intracerebral hemorrhage. **Stroke 29** : 1352-1357, 1998

6. Franke CL, van Swieten JC, Algra A, van Gijn J : Prognostic factors in patients with intracerebral haematoma. **J Neurol Neurosurg Psychiatry 55** : 653-657, 1992

7. Gebel JM Jr, Jauch EC, Brott TG, Khoury J, Sauerbeck L, Salisbury S, et al. : Natural history of perihematomal edema in patients with hyperacute spontaneous intracerebral hemorrhage. **Stroke 33** : 2631-2635, 2002

8. Gebel JM Jr, Jauch EC, Brott TG, Khoury J, Sauerbeck L, Salisbury S, et al. : Relative edema volume is a predictor of outcome in patients with hyperacute spontaneous intracerebral hemorrhage. **Stroke 33** : 2636-2641, 2002

9. Hallevy C, Ifergane G, Kordysh E, Herishanu Y : Spontaneous supratentorial intracerebral hemorrhage. Criteria for short-term functional outcome prediction. **J Neurol 249** : 1704-1709, 2002

10. Hayashi M, Handa Y, Kobayashi H, Kawano H, Nozaki J, Hirose S : Prognosis of intraventricular hemorrhage due to hypertensive hemorrhagic cerebrovascular disease. **Zentralbl Neurochir 49** : 101-108, 1988

11. Leira R, Dávalos A, Silva Y, Gil-Peralta A, Tejada J, Garcia M, et al. : Early neurologic deterioration in intracerebral hemorrhage : predictors and associated factors. **Neurology 63** : 461-467, 2004

12. Lisk DR, Pasteur W, Rhoades H, Putnam RD, Grotta JC : Early presentation of hemispheric intracerebral hemorrhage : prediction of outcome and guidelines for treatment allocation. **Neurology 44** : 133-139, 1994

13. Mendelow AD, Gregson BA, Fernandes HM, Murray GD, Teasdale GM, Hope DT, et al. : Early surgery versus initial conservative treatment in patients with spontaneous supratentorial intracerebral haematomas in the International Surgical Trial in Intracerebral Haemorrhage (STICH) : a randomised trial. **Lancet 365** : 387-397, 2005

14. Nieuwkamp DJ, de Gans K, Rinkel GJ, Algra A : Treatment and outcome of severe intraventricular extension in patients with subarachnoid or intracerebral hemorrhage : a systematic review of the literature. **J Neurol 247** : 117-121, 2000

15. Qureshi AI, Tuhrim S, Broderick JP, Batjer HH, Hondo H, Hanley DF : Spontaneous intracerebral hemorrhage. **N Engl J Med 344** : 1450-1460, 2001

16. Reithmeier T, Löhr M, Pakos P, Ketter G, Ernestus RI : Relevance of ICP and ptiO2 for indication and timing of decompressive craniectomy in patients with malignant brain edema. **Acta Neurochir (Wien) 147** : 947-951; discussion 952, 2005

17. Schwarz S, Häfner K, Aschoff A, Schwab S : Incidence and prognostic significance of fever following intracerebral hemorrhage. **Neurology 54** : 354-361, 2000

18. Tuhrim S, Dambrosia JM, Price TR, Mohr JP, Wolf PA, Heyman A, et al. : Prediction of intracerebral hemorrhage survival. **Ann Neurol 24** : 258-263, 1988

19. Yuzawa H, Higano S, Mugikura S, Umetsu A, Murata T, Nakagawa A, et al. : Pseudo-subarachnoid hemorrhage found in patients with postresuscitation encephalopathy : characteristics of CT findings and clinical importance. **AJNR Am J Neuroradiol 29** : 1544-1549, 2008

20. Zuccarello M, Brott T, Derex L, Kothari R, Sauerbeck L, Tew J, et al. : Early surgical treatment for supratentorial intracerebral hemorrhage : a randomized feasibility study. **Stroke 30** : 1833-1839, 1999