



Symptomatic subacute subdural hematoma associated with cerebral hemispheric swelling and ischemia

Nobuhiko Aoki, Akihiro Oikawa & Tatsuo Sakai

To cite this article: Nobuhiko Aoki, Akihiro Oikawa & Tatsuo Sakai (1996) Symptomatic subacute subdural hematoma associated with cerebral hemispheric swelling and ischemia, Neurological Research, 18:2, 145-149, DOI: [10.1080/01616412.1996.11740393](https://doi.org/10.1080/01616412.1996.11740393)

To link to this article: <http://dx.doi.org/10.1080/01616412.1996.11740393>



Published online: 21 Jul 2016.



Submit your article to this journal [↗](#)



View related articles [↗](#)



Citing articles: 4 View citing articles [↗](#)

Symptomatic subacute subdural hematoma associated with cerebral hemispheric swelling and ischemia

Nobuhiko Aoki, Akihiro Oikawa and Tatsuo Sakai

Department of Neurosurgery, Tokyo Metropolitan Ohkubo Hospital Tokyo, Japan

A 55-year-old man with traumatic acute subdural hematoma (SDH) was managed nonsurgically because of having isolated mild headache that resolved within a week. However, the patient developed an acute onset of amnesic aphasia 12 days post-trauma. Although the SDH itself did not increase in volume, remarkable swelling of the ipsilateral cerebral hemisphere was observed on CT scanning. Craniotomy was required since the main component of the SDH was solid and partially organized. Post-operative recovery from his speech disturbance was delayed, necessitating speech therapy for more than two months. Additionally, decreased cerebral blood flow on the affected side was observed even 3 months after injury. Symptomatic subacute SDH exclusively presenting as focal neurological deficits, though very rare, is worthwhile to be kept in mind as a pitfall in nonsurgical management of acute SDH. [Neurol Res 1996; 18: 145–149]

Keywords: Acute subdural hematoma; amnesic aphasia; head trauma; subacute subdural hematoma

INTRODUCTION

Most neurosurgeons seem to hesitate to operate on patients with acute subdural hematoma (SDH) which is thin and represents minimal mass effect on computed tomography (CT) scans. This is a particularly practical dilemma in patients complaining of isolated mild headache without neurological findings. When acute SDH is managed neurosurgically, however, the patients may carry significant risks during the clinical course; i.e., rapid increase of the hematoma volume in the acute stage¹, symptomatic subacute SDH^{2–5}, and evolution to chronic SDH^{6,7}. Among these pathological processes, subacute SDH has received less attention, and the mechanism of its symptom onset has been considered to be secondary to the increase in the volume of hematoma^{2–4}. Recently, the authors encountered a patient with acute SDH which caused rapid onset of a focal neurological deficit in the subacute stage. In addition, the mechanism of the pathophysiological process in this patient was considered to be different from that reported previously. Details of the clinical course in this patient are described, and the pitfall of nonsurgically managed acute SDH in the subacute stage is highlighted.

CASE REPORT

A 55-year-old right-hand male journalist was referred to us for further management of acute SDH on November

29, 1994. He struck the left parietal region under a heavily drunken state at midnight on November 27. He ignored his symptom until 2 days later when he complained of headache, and presented to a nearby hospital, where CT scans revealed acute SDH on the left side. His medical history was significant for hypertension, diabetes mellitus and duodenal ulcer without treatment.

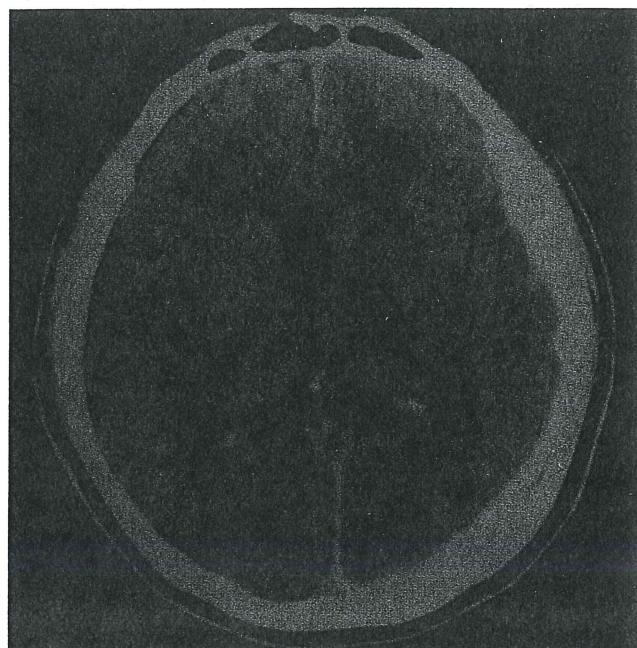
Examination

At the time of transfer to our hospital the patient complained of mild headache, but was alert and cooperative. His neurological examination did not show any abnormalities. A bruise was found on the left parietal region. A skull x-ray series failed to reveal fractures. CT scans of the head performed on admission demonstrated high-density SDH on the left side together with mild compression of the left lateral ventricle (*Figure 1*). Results of laboratory studies including coagulation tests were all within normal range.

Clinical course

Because of having tolerable but persistent headache coupled with the possibility of evolution to symptomatic subacute SDH, he was scheduled for inpatient observation. His headache resolved one week after admission without treatment. CT scans obtained on December 5 (8 days post-trauma) showed seemingly minimal reduction of the hematoma volume without decrease of mass effect. He was confirmed to remain neurologically intact and fluently talked to his primary nurse at the time of lunch on December 9. At 1:30 p.m., (12 days post-

Correspondence and reprint requests to: Nobuhiko Aoki, MD, Department of Neurosurgery, Tokyo Metropolitan Ohkubo Hospital, 44-1, Kabukicho 2-chome, Shinjuku-ku, Tokyo 160, Japan. Accepted for publication December 1995.



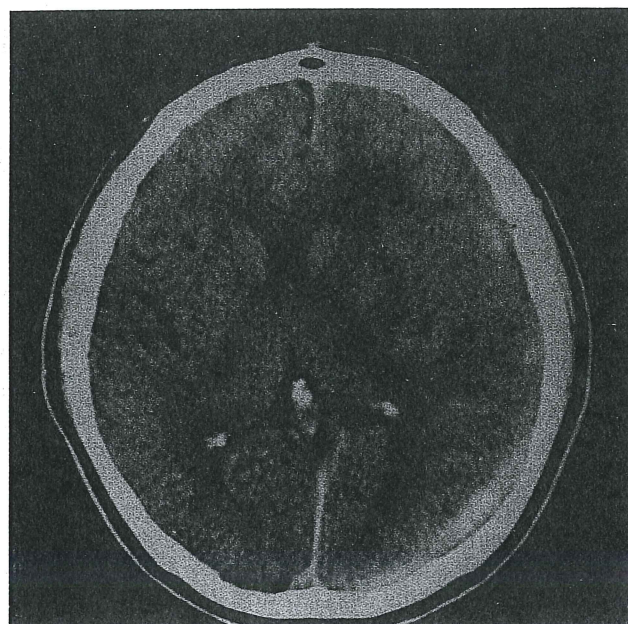
(A)



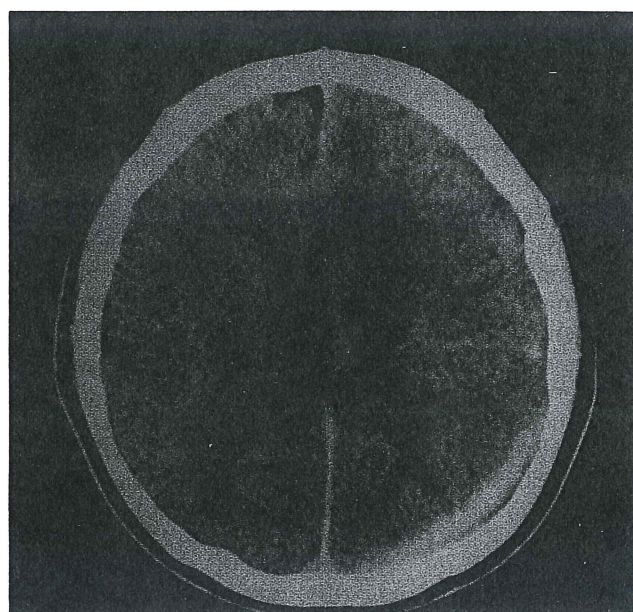
(B)

Figure 1: Plain computed tomography scans on admission show high-density subdural hematoma on the left side, associated with mild compression of the left lateral ventricle

trauma), however, the nurse noted abnormality in his speech. An emergency neurological examination revealed that he was alert, but was unable to name any objects. He could repeat words and follow simple one-step commands. No other abnormalities in the motor, sensory and cranial nerve functions were observed. His neurological symptomatology was evaluated as amnesic aphasia by the neuropsychologist. Emergency CT scans showed rather slightly reduced volume of the



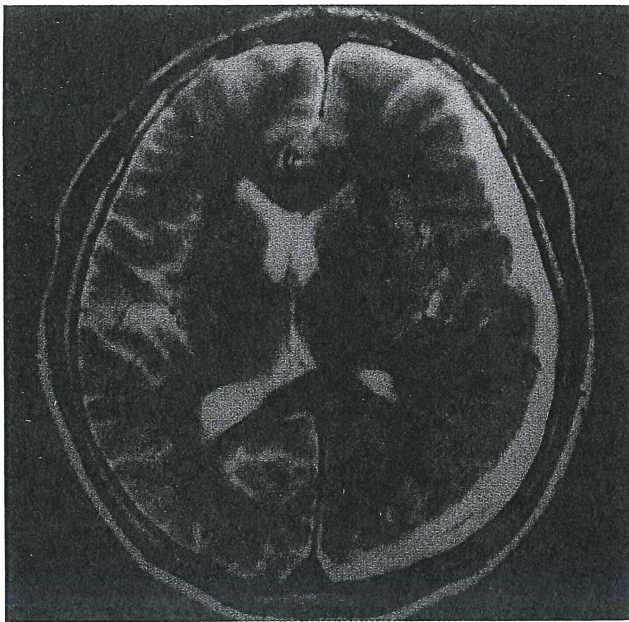
(A)



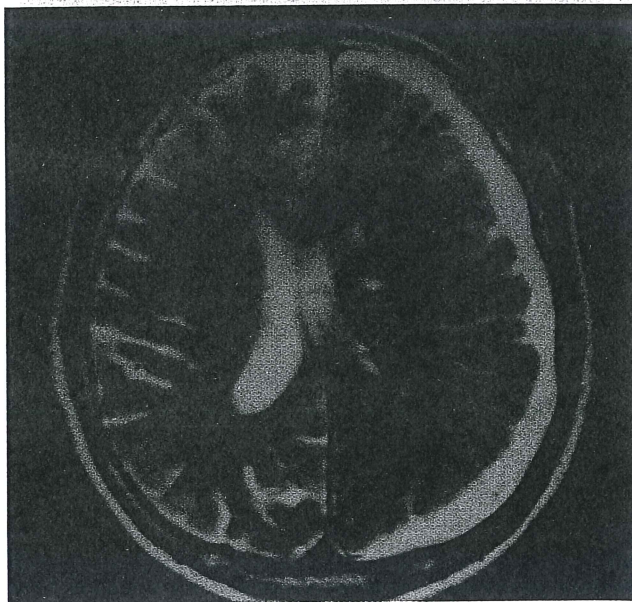
(B)

Figure 2: Plain computed tomography scans 12 day post-trauma reveal rather slight reduction of the subdural hematoma with decreased density, but demonstrate definite increase in the shift of the midline structures to the right side

hematoma with decreased density, but revealed definite increase in the shift of the midline structures to the right side. (Figure 2) T2-weighted magnetic resonance (MR) images showed SDH, shift of the midline structures and multiple lacunar infarctions in the left basal ganglia, corona radiata and pons; however, no pathological features producing swelling of the left cerebral hemispheric parenchyma were noted. (Figure 3) Arterial occlusion or veno-sinus thrombosis were not exhibited on MR angiography.



(A)



(B)

Figure 3: T2-weighted magnetic resonance images 12 days after injury show subdural hematoma, shift of the midline structures and multiple lacunar infarctions, but fail to exhibit other pathological features producing swelling of the left cerebral hemispheric parenchyma

Under a diagnosis of symptomatic subacute subdural hematoma, percutaneous subdural tapping was performed at the bedside on an emergency basis². Although this procedure resulted in drainage of only 15 ml of liquid hematoma, his speech function improved to the level of being able to name simple objects. On the following morning, however, rapid deterioration of his

speech function was evidenced by global aphasia showing complete loss of auditory comprehension. Because of his neurological worsening associated with persistent mass effect on CT scans despite administration of steroids and osmotic agents, an emergency craniotomy was performed.

Surgery

A left parieto-occipital craniotomy revealed partially organized, clots. Removal of these clots allowed drainage of liquid hematoma from the frontal region. The outer membrane of the hematoma was present, but the inner membrane or other abnormal findings were not seen on the exposed cortical surface. Post-operative CT scans did not show residual hematoma or cerebral parenchymal abnormalities. Recovery of his speech deficits was unexpectedly delayed; nominating dysphasia was observed at the time of discharge from this hospital two months after surgery. In addition, one month after discharge, although his speech function was completely normalized, and MR images failed to exhibit abnormalities apart from preexisting lacunar infarctions. Single photon emission computed tomography (SPECT) images revealed definite reduction of cerebral blood flow (CBF) on the left fronto-temporo-parietal regions. (Figure 4)

DISCUSSION

Unlike acute or chronic SDH, there have been no large series of subacute SDH⁸, necessitating accumulation of detailed documentation of sporadic cases with subacute SDH. When summarizing previously reported cases, nonsurgically managed acute SDH, though rarely encountered, becomes symptomatic most frequently eleven or twelve days after injury²⁻⁴. The mechanism of symptomatic subacute SDH has been reported to be secondary to influx of cerebrospinal fluid (CSF) into the subdural space either by colloid-osmotic pressure difference between the liquid hematoma and CSF², or by tearing of the arachnoid membrane^{3,4}. Thus, the pathogenesis of symptomatic subacute SDH has been considered exclusively attributable to the progressive volume expansion of the hematoma with corresponding increase in the intracranial pressure. For this reason, it has not been difficult to predict possible development of symptomatic subacute SDH on serial CT studies and neurological observation. Additionally, the liquid hematoma has consisted of the main component in the pathological process, facilitating evacuation of the hematoma via percutaneous subdural tapping² or burr-hole craniostomy in most cases. Based on our experience and the previously reported series^{3,4}, the authors have closely observed this patient at the neurosurgical ward. Unexpectedly, however, the patient developed rapid onset of isolated speech disturbance without association of increase in the volume of the hematoma.

The pathophysiological basis for his focal neurological deficit characterized by aphasia seems to be produced by relatively long-standing compression of the cortical surface, similar to transient ischemic attack

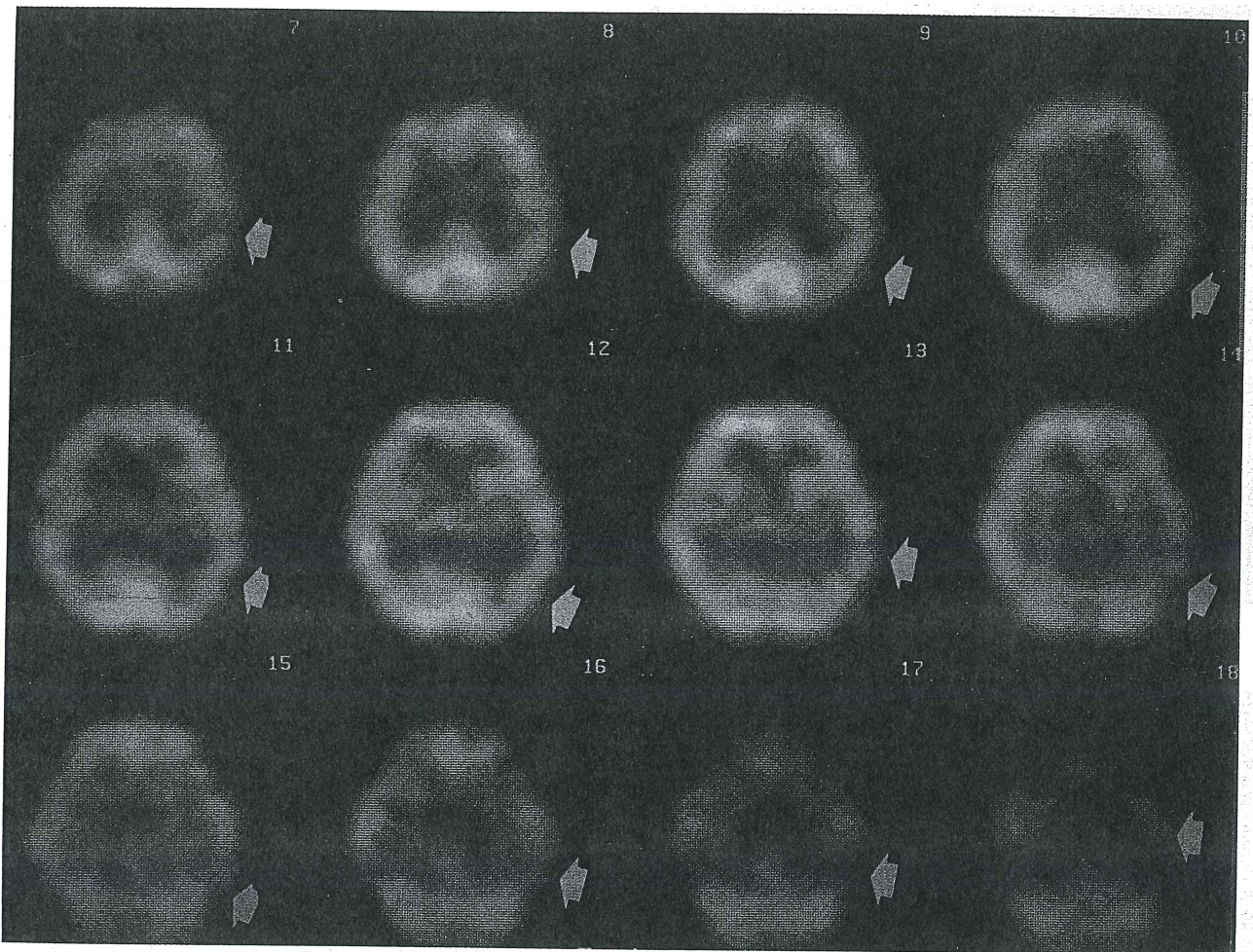


Figure 4: Single photon emission computed tomography images three months after injury demonstrate definite reduction of cerebral blood flow (arrows) on the left hemisphere

in the chronic SDH⁹, resulting in cerebrovascular hypoperfusion¹⁰. This hypothesis is supported by the results of SPECT study which revealed definite reduction of CBF at least until three months after injury despite evacuation of the hematoma. Moreover, since it has been reported that SDH is usually associated with cerebrovascular hypoperfusion^{11,12}, surgical consideration may be usefully assessed by serial CBF studies in patients with minimally or mildly symptomatic acute SDH.

Of particular note in the present patient is that remarkable increase of the hemispheric swelling is not associated with volume expansion of the hematoma itself, indicating the importance of repeat CT follow-up irrespective of neurological symptomatology. Although no evidence could be obtained by MR angiography on the venous phase, the above-described phenomenon may be related to altered venous hemodynamics due to mild but persistent compression of the cortex.

To summarize, in patients with nonsurgically mana-

ged acute SDH which is delayed in resolution, it is essential to be aware of rapid development of focal neurological deficits with delayed recovery even after emergency craniotomy.

REFERENCES

- 1 Cooper PR, Maravilla K, Moody S, Clark WK. Serial computerized tomographic scanning and the prognosis of severe head injury. *Neurosurgery* 1979; **5**: 566–569
- 2 Aoki N, Tsutsumi K. Symptomatic subacute subdural haematoma following spontaneous acute subdural haematoma. *Acta Neurochir (Wien)* 1990; **102**: 149–151
- 3 Karasawa H, Naito H, Sugiyama K, Ueno J, Kin H, Oketa R. Traumatic subacute subdural hematoma: possible mechanisms that cause their development. *Jap J Neurosurg* 1995; **4**: 121–127
- 4 Morinaga K, Matsumoto Y, Hayashi S, Omiya N, Mikami J, Sata H, Inoue Y, Okawara S. Subacute subdural hematoma: Findings on CT, MRI and operations and review of onset mechanism. *No Shinkei Geka* 1995; **23**: 213–216 (In Jap)
- 5 Ohaegbulam SC. Surgically treated traumatic subacute and chronic subdural hematomas: A review of 132 cases. *Injury* 1981; **13**: 23–26

- 6 Watanabe T, Watanabe T, Sasaki T, Murata S, Yamada H. Chronic subdural hematoma formed from acute subdural clot. In comparison with the type occurring subsequent to the traumatic subdural hygroma. *Neurotraumatology* 1980; **3**: 552-561 (In Jap)
- 7 Yoshimasu N, Tamura A, Wakai S, Yoshida T. Evolution from acute subdural hemorrhage to chronic subdural hematoma. Observation of subdural hematoma by computerized tomography. *No Shinkei Geka* 1981; **9**: 1025-1031 (In Jap)
- 8 Cooper PR. Traumatic intradural hematoma. In: Wilkins RH, Rengachary SS, eds. *Neurosurgery, Vol 2*. New York: McGraw-Hill Book Company, 1985; pp 1659
- 9 Moster ML, Johnston DE, Reinmuth OM. Chronic subdural hematoma with transient neurological deficits: A review of 15 cases. *Ann Neurol* 1983; **14**: 539-542
- 10 Ikeda K, Ito H, Yamashita J. Relation of regional cerebral blood flow to hemiparesis in chronic subdural hematoma. *Surg Neurol* 1990; **33**: 87-95
- 11 Salvant JB Jr, Muizelaar JP. Changes in cerebral blood flow and metabolism related to the presence of subdural hematoma. *Neurosurgery* 1993; **33**: 387-393
- 12 Schroder ML, Muizelaar Jp, Kuta AJ. Documented reversal of global ischemia immediately after removal of an acute subdural hematoma. *J Neurosurg* 1994; **80**: 324-327