



Fig. 3 CT scan, 2 months after the injury, shows a wedge-shaped low density area in the left temporal lobe with its base extending along the inner table of the skull.

At follow-up 6 months after the trauma, his language ability was judged to be at the same level as before the accident. There was no change in personality and he had returned to his previous work as a truck driver.

DISCUSSION

Aphasias in patients with head injury, especially if there are no other neurological signs, can present a puzzling clinical picture. Fluent aphasia, in particular, may go unrecognized as such, and be interpreted as confusion, mental aberration, or even malingering, especially when no other neurological signs are found.⁵ Initially, our patient presented with such aphasic symptoms.

Heilman et al.¹ reported that post-traumatic aphasia was associated most commonly with right orbito-frontal trauma. A left temporo-parietal lesion is the second most common injury site. The lateral surface of the temporal lobe, noted for having a special relationship to aphasia, and to some extent, to psychiatric disability,^{5,6} has been known as a frequent site for contusions. Other regions of the speech area, including the frontal and parietal opercula, rarely cause confusion.¹ In our patient, a right fronto-temporal scalp contusion, bleeding from the right external auditory meatus, and the left temporal lobe contusion, would be compatible with a contre-coup mechanism. The presence of a fluent aphasia implied that Broca's area was intact.⁵

Several studies have been done on the relationship between closed head trauma and aphasia. Glaser and Shafer⁷ reported that 16 of 255 patients with a non-penetrating head trauma developed aphasia. Thomsen² reported that anomic aphasia and verbal paraphasia were the most frequent symptoms. Heilman et al.¹ also reported that approximately two thirds of the aphasias resulting from closed head injuries were of the anomic (amnestic) type, with Wernicke's aphasia being the next most common. While anomic aphasia is a fluent aphasia with essentially normal comprehension, repetition, and abnormal naming of objects and circumlocutions,⁵ a Wernicke's aphasia is characterized as a fluent aphasia with paraphasia, poor comprehension for spoken and written language, and poor repetition.¹ The aphasia in our patient was a fluent aphasia with some verbal and phonetic paraphasias, impaired word finding, and impaired verbal comprehension.

These aphasic symptoms are in agreement with the findings where patients with closed head injuries are concerned.

Alajouanine et al.⁸ reported that the prognosis of post-traumatic aphasia is rather good, being better than that of aphasia secondary to cerebrovascular diseases. Several studies have also pointed out that aphasia secondary to severe closed head injury tends to recover well,^{1,2} and that the greatest recovery occurs in the first 6 months⁹ though improvement is possible for as long as 3 years after the injury.¹⁰ Few, if any, could regain their premorbid language abilities,^{1,4,11} and the presence and degree of concomitant neuropsychological disorders may be the most important aspect of the final outcome.² However, the prognosis of post-traumatic aphasia for recovery appears good as far as functional communication is concerned,¹¹ and the same was true in our case.

REFERENCES

1. Heilman KM, Safran A, Geschwind N. Closed head trauma and aphasia. *J Neurol Neurosurg Psychiatry* 1971; 34: 265–269.
2. Thomsen IV. Evaluation and outcome of aphasia in patients with severe closed head trauma. *J Neurol Neurosurg Psychiatry* 1975; 38: 713–718.
3. Levin HS, Grossman RG, Kelly PJ. Aphasic disorder in patients with closed head injury. *J Neurol Neurosurg Psychiatry* 1976; 39: 1062–1070.
4. Sarno MT. The nature of verbal impairment after closed head injury. *J Nerv Ment Dis* 1980; 168: 685–692.
5. Stone JL, Lopes JR, Moody RA. Fluent aphasia after closed head injury. *Surg Neurol* 1978; 9: 27–29.
6. Lishman WA. Psychiatric disability after head injury: the significance of brain damage. *Proc Roy Soc Med* 1966; 59: 261–266.
7. Glaser MA, Shafer FP. Skull and brain traumas: their sequelae. *J Am Med Assoc* 1932; 98: 271–276.
8. Alajouanine T, Castaigne P, Lhermitte F, Escourolle R, de Ribaucourt B. Étude de 43 cas d'aphasie post-traumatique. *Encephale* 1957; 46: 1–45.
9. Bond MR, Brooks DN. Understanding the process of recovery as a basis for the investigation of rehabilitation for the brain injured. *Scand J Rehab Med* 1976; 8: 127–133.
10. Najenson T, Mendelson L, Schechter I, David C, Mintz N, Grosswasser Z. Rehabilitation after severe head injury. *Scand J Rehab Med* 1974; 6: 5–14.
11. Thomsen IV. Late outcome of very severe blunt head trauma: a 10–15 year second follow-up. *J Neurol Neurosurg Psychiatry* 1984; 47: 2160–2168.
12. Nirayama Conference. Standard Language Test of Aphasia. Manual of Directions, Homeido, Tokyo; 1975.

Kernohan's phenomenon associated with left ruptured occipital arteriovenous malformation

A. Fujimoto¹, H. Sato¹, W. Katayama¹, K. Nakai¹, T. Tsunoda¹, E. Kobayashi¹, T. Nose²

¹Department of Neurosurgery, Tsukuba Memorial Hospital, Ibaraki, Japan,

²Department of Neurosurgery, Institute of Clinical Medicine, University of Tsukuba, Ibaraki, Japan

Summary A 23-year-old woman presented with ipsilateral hemiparesis due to rupture of a left occipital arteriovenous malformation (AVM). Emergency decompression (the onset-operation interval was 46 minutes,) was carried out and the patient could leave the hospital upon recovery without neurological deficits. In general, Kernohan's phenomenon is caused by the gradual displacement of the cerebral peduncle against the tentorial edge caused by compression by the contralateral mass. This phenomenon is very rare among the cases

with spontaneous intracranial hemorrhage and only three cases including the present one have been reported in the literature. In all cases the onset-operation intervals of were very short.

Kernohan's phenomenon associated with a ruptured AVM is a rare condition and emergency decompression is required.

© 2004 Published by Elsevier Ltd.

Journal of Clinical Neuroscience (2004) 11(4), 444–446
0967-5868/\$ - see front matter © 2004 Published by Elsevier Ltd.
doi:10.1016/j.jocn.2002.09.003

Keywords: Kernohan's phenomenon, Ipsilateral hemiparesis, Arteriovt malformation (AVM)

Received 30 July 2002

Accepted 12 September 2002

Correspondence to: Ayataka Fujimoto, Department of Neurosurgery, Institute of Clinical Medicine, University of Tsukuba, 1-1-1 Tennodai Tsukubashi, Ibaraki, Japan. Tel.: +81-298-53-3525; Fax: +81-298-53-3039/3214; E-mail: hao36870@rio.odn.ne.jp

INTRODUCTION

Kernohan's phenomenon consists of ipsilateral hemiplegia and ipsilateral oculomotor nerve palsy due to compression of the cerebral peduncle against the tentorial edge by the contralateral mass. In general, this phenomenon occurs in patients with advanced brain tumor or severe head injury.¹ Kernohan's phenomenon caused by ruptured parenchymal arteriovenous malformation (AVM) is infrequent.



Fig. 1 CT scan showing that the brain was shifted from left to right by the acute subdural hematoma (ASDH) and the temporo-occipital hematoma.

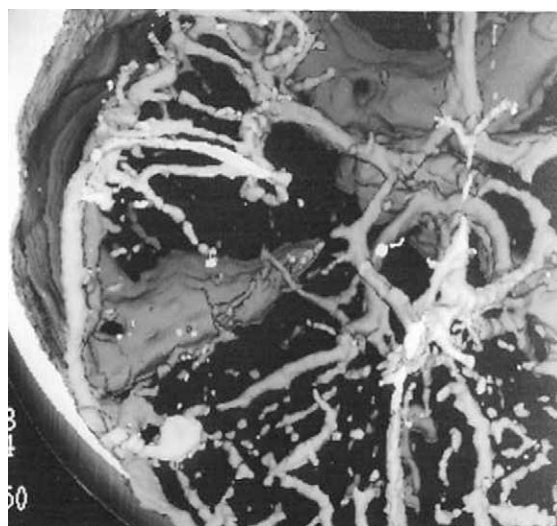


Fig. 2 3DCTA showing a small lesion in the occipital area.

CASE REPORT

The patient, a 23-year-old woman, visited our outpatient clinic with a chief complaint of severe headache, nausea and vomiting. During brain computed tomographic scanning (CT scan), she became comatose and exhibited decerebral posturing. Her Glasgow Coma Scale (GCS) was scored of 4 (E1V1M2). Her pupils did not react to light and her left pupil was mydriatic. The CT scan revealed a left occipital intracerebral hematoma with acute subdural hematoma (ASDH) (Fig. 1). Three dimensional CT angiography (3DCTA) confirmed the presence of an abnormality in the corresponding area (Fig. 2). The patient was immediately operated on to decompress the brain and remove the subdural hematoma. The onset-operation interval was 46 min. After the first operation, her conscious level ameliorated to GCS 10 (E4V1M6). We performed digital subtraction angiography postoperatively (DSA) to identify the origin of bleeding, but only a slight hypervascularity was detected in the corresponding area. Two days after the onset, the patient was operated on again to remove the occipital intracerebral hematoma. Intraoperatively, a parenchymal AVM was found in the occipital area, which was removed. On day 4 part onset, we noticed left hemiparesis with a partial left oculomotor nerve palsy, the so called Kernohan's phenomenon. The voltage of the left medial nerve stimulation/right scalp recording, somato sensory evoked potential (SEP) was less than the right stimulation/left recording one.

One month after the onset, the patient had no significant neurological deficit.

DISCUSSION

Kernohan's phenomenon is due to cerebral herniation. It is mainly seen in patients with an advanced brain tumor or those who have suffered a severe head injury.^{1,2} Although a lesion above the py-

Table 1 Cases of Kernohan's phenomenon by ICH

No.	age/sex	Hematoma	origine of hematoma	interval to first ade	outcome	authors/year of issue
1	25/M	Rt.ASDH	unkown	unknown	good recovery	Jone et al. ¹ /1991
2	39/M	Lt.ASDH	ruptured aneurysm	2 h	good recovery	Iwama et al. ⁶ /1992
3	23/F	Lt.ASDH	AVM	46 min	good recovery	our case.

ICH; intra cerebral hemorrhage. ASDH;acute subdural hementia. AVM;arteriovenous malformation.

ramidal decussation usually causes contralateral symptoms on the body, our patient presented with ipsilateral hemiparesis. This is typical of Kernohan's phenomenon. Non-tumoral, non-traumatic intracranial haemorrhage rarely causes this phenomenon. According to our review of the literature, our patient is the third case of Kernohan's phenomenon caused by intracranial haemorrhage (Table 1). The age of the three patients ranged from 23 to 39. The ASDH was located on the right side in one case and on the left side in two cases. Our patient also had an intracerebral hematoma located in the occipital region. Case 1 was a graduate student of the university and was taken to the regional hospital. Case 2 was taken to hospital and underwent surgery within 2 h. In our patient, intracranial haemorrhage occurred during a CT scan and she was taken to the operation room with 46 min.

Kernohan's phenomenon has not been reported in a patient with an AVM until now. In general, the typical location of Kernohan's notch is around the midbrain.³ Patients with hematomas in the temporal or temporo-occipital region are more likely to have signs of tentorial herniation, according to Andrews.⁴ In our patient, we speculated that the occipital hematoma was additionally compressed by the ASDH. This force compressed the contralateral cerebral peduncle against the tentorial edge and caused Kernohan's phenomenon, finally the patient presented ipsilateral hemiparesis.

Kernohan's phenomenon is due to uncal herniation, which is a neurosurgical emergency. It is obvious that the sooner the patient is operated on, the better the outcome will be.⁵

We believe that her good outcome with little neurological deficit was due to the short interval from the onset to the first operation.

REFERENCES

1. Jones KM, Seeger JF, Yoshino MT. Ipsilateral motor deficit resulting from a subdural hematoma and Kernohan notch. *AJNR Am J Neuroradiol* 1991; 12: 1238–1239.
2. Itoyama Y, Fujioka S, Ushio Y. Kernohan's notch in chronic subdural hematoma: findings on magnetic resonance imaging. *J Neurosurg* 1995; 82: 645–646.
3. Ozaki H. *Fundamentals of neuropathology*. Igaku Shoin, New York 1983. p. 81–5.
4. Andrews BT, Chiles BW, Walter La, Pitts LH. The effect of intracerebral hematoma location on the risk of brain-stem compression and on clinical outcome. *J Neurosurg* 1988; 69: 518–522.
5. Andrews BT, Pitts LH. Functional recovery after traumatic transtentorial herniation. *Neurosurgery* 1991; 29: 227–231.
6. Iwama T, Kuroda T, Sugimoto S, Miwa Y, Ohkuma A. MRI demonstration of Kernohan's notch: case report. *Neuroradiology* 1992; 34(3): 225–226.