

Case Study

Gerstmann's syndrome associated with chronic subdural haematoma: a case report

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We report a patient who exhibited Gerstmann's syndrome in association with a chronic subdural haematoma. A 71-year-old right-handed woman presented with mild right arm and leg weakness that began 2 weeks prior to admission. Neurological examination on admission revealed a mild right hemiparesis. Neuropsychological examination revealed right-left disorientation, finger agnosia, agraphia, and acalculia, but no language disturbance. A computerized tomographic (CT) scan revealed a large left frontoparietal, extra-axial hypodense fluid collection containing scattered hypodense foci. A left parietal evacuation of the haematoma was performed. Following surgery the patient dramatically improved. We suggest that the direct compression by the chronic subdural haematoma or a hemispheric pressure difference caused Gerstmann's syndrome. This is an unusual report of a Gerstmann's syndrome following chronic subdural haematoma.

Introduction

Gerstmann's syndrome consists of right-left disorientation, finger agnosia, agraphia, and acalculia [1,2]. The lesion has been localized to the left parietal cortex including the angular and second occipital gyrus, supramarginal gyrus, and superior parietal lobule [3,4]. Constructional apraxia and mild aphasia can also occur in Gerstmann's syndrome.

Patients with a chronic subdural haematoma often have cognitive impairments, which may not be severe enough to meet the criteria for the clinical diagnosis of dementia. Rarely, aphasia can also be a presenting symptom of subdural haematomas [5,6]. However, to our knowledge, Gerstmann's syndrome has not been previously reported. We describe a patient with Gerstmann's syndrome due to a subdural haematoma, and then discuss its possible pathogenesis.

Case report

A 71-year-old right-handed woman presented with a 2-week history of right arm and leg weakness. She denied headache, nausea, and vomiting. She had 9 years of formal education. She had no history of hypertension, stroke, transient ischaemic attacks, seizures, or alcoholism.

Physical examination on admission revealed a blood pressure of 150/90 mmHg and a pulse of 66 per minute. No carotid bruits or cardiac murmur was present.

On neurological examination she was alert and cooperative. Her cranial nerve examination was normal. Examination of the extremities showed mild pronator drift of the right upper extremity but good strength on individual muscle group testing and normal tone. Sensory testing to pin, touch, vibration, position, graphaesthesia, and stereognosis was normal. Finger-to-nose and heel-shin testing were normal. Gait was grossly normal.

On neuropsychological examination her attention was relatively intact (digit span, seven forward and four backward) Language testing did not reveal any impairment in spontaneous speech such as word-finding difficulties or paraphasic errors. Prosody was preserved. Repetition was not impaired. Naming of objects in the room and elements of the examiner's clothing and body parts was performed with better than 90% accuracy. Comprehension was unimpaired, as demonstrated by appropriate verbal responses to questions and correct execution of simple and multiple step directives. Striking right-left confusion was noted. Any tasks that required right-left discrimination led to hesitation, frustration, and repeated errors in execution. She was unable to identify digits on either hand. She could not reliably name the digits and made frequent errors in attempting to follow commands instructing her to identify or utilize a specific digit on her own hand or on the hand of the examiner. She was aware of her difficulty and was frustrated by it. She also was unable to perform any calculations. Even very simple calculations, such as adding $12 + 13$ or subtracting $93 - 7$, could not be carried out. Her performance did not improve with the use of pencil and paper. Reading was not impaired. The patient could read sentences out loud correctly. She was able to write with either hand, but demonstrated spelling errors and omissions (Figure 1a).

	Aug 11		Aug 16
	Dictation	Copy	Dictation
鉛筆 エンピツ (えんぴつ)	エンピツ	鉛筆 エンピツ	鉛筆 えんぴつ
灰皿 ハイザラ (はいざら)	ハイザラ	灰皿 ハイザラ	灰皿 はいざら
新聞 シンブン (しんぶん)	新聞 しんぶん	新聞 シンブン	新聞 しんぶん
毛糸 ケイト (けいと)	毛糸 けいと	毛糸 ケイト	毛糸 けいと
		a	b

Figure 1. Dictation and copy in the patient. (a) The patient was able to write with either hand but had numerous spelling errors and omissions. (b) Three days following surgery she dramatically improved. She could write accurately without minor errors.

Scores on cognitive tests were as follows: the Mini-mental state, 23/30; Osaka Memory Scale (the Japanese version of the Wechsler Memory Scale), 45/100; word fluency test, 6/min; and Rave's CPM, 18/36. The GBS scale was 12 points, showing a deterioration of motor function and intellectual deficits (Table 1).

Computerized tomography (CT) of the head revealed a large left frontoparietal, extra-axial hypodense fluid collection containing scattered hyperdense foci (Figure 2).

Clinical course

The patient underwent a left parietal trepanation and evacuation of a haematoma on 11 August 1996.

Three days following surgery she improved clinically. She could write without minor errors (Figure 1b), and her hemiparesis was resolved. The results of neuropsychological examinations performed 7 days following surgery were also markedly improved. A follow-up CT on the 7th postoperative day demonstrated only mild residual fluid and little mass effect. Neuropsychological examinations performed 1 month following surgery demonstrated continued improvement.

Discussion

In our patient, finger agnosia and right-left disorientation were prominent symptoms. Her agraphia, characterized by paraphasia with misspellings and syntax errors, and spatial dysgraphia (poor word alignment across a page) were mild. Her acalculia also was severe. These features are commonly associated with Gerstmann's syndrome.

Table 1. Neuropsychological examination results and GBS scale scores

	11 August	16 August	5 September	4 December
Mini-mental state (/30)	23	28	28	28
Word fluency (/min)	6	8	8	11
Raven's CPM (/36)	18	21	21	22
Osaka Memory Scale (/100)	45	59	65	57
Information	7	9	9	9
Mental control	6	6	9	9
Story recall	6	10	10	10
Digit span	10	9	10	11
Visual reproduction	5	5	10	0
Associated learning	9	17	12	13
Delayed recall	2	3	5	5
GBS scale	12	4	2	2
Motor	8	1	0	0
Intellectual	4	3	2	2
Emotion	0	0	0	0
Others	0	0	0	0

Mean (SD) compared with 20 age-matched controls

Mini-mental state: 28.4 (2.0)

Osaka Memory Scale: 71.9 (8.0)

Word fluency: 12.0 (1.9)

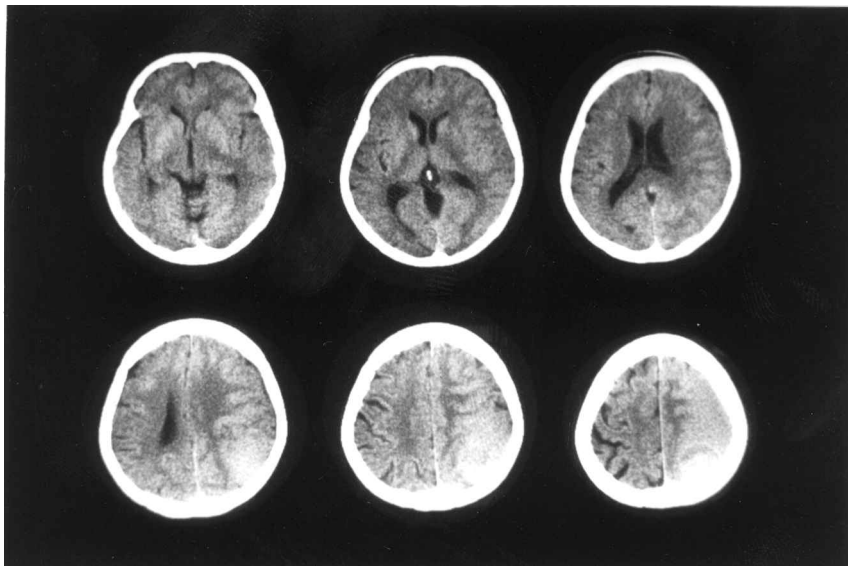


Figure 2. Computerized tomography (CT) of the head revealed a large left frontoparietal, extra-axial hypodense fluid collection containing scattered hyperdense foci.

Subdural haematomas, caused by haemorrhage from bridging veins between the dura and arachnoid, are usually associated with head trauma. The blood typically lies near the frontal and parietal convexities, and in some cases is bilateral. Patients usually complain of a headache, and they may have cognitive impairment, aphasia and a hemiparesis contralateral to the haematoma. Gerstmann's syndrome is uncommon. Patients with Gerstmann's syndrome typically have a lesion which involves either the angular gyrus or the subcorical white matter posterolateral to the posterior horn of the left lateral ventricle. The resolution of our patients' Gerstmann's syndrome following haematoma evacuation suggests a causal relationship. The post-operative CT scan showed no other responsible cerebral infarction or mass lesion.

Daly *et al.* [7] have suggested that mass lesions can cause neurological symptoms by mechanical stimulation of the cortex followed by a spreading depression of cerebral electrical activity [8]. However, this mechanism would be too diffuse to cause focal symptoms. Direct compression, or a hemispheric pressure difference created by the chronic subdural haematoma [9], would also be too widespread. We cannot exclude the possibility that these diffuse processes may have induced the expression of an otherwise subclinical focal lesion. Otherwise our patient would have had a mild cognitive impairment due to a diffuse brain lesion.

McLaurin [10] has recognized that subdural haematomas may compromise regional cerebral blood flow by indirectly shifting the anterior and posterior cerebral arteries. The size of the haematoma, however, does not necessarily determine symptoms, nor have most of the reported patients had significant midline shift or syndromes suggesting anterior or posterior cerebral artery ischaemia. The haematoma in our patient would not be expected to cause a shift of intracranial structures leading to contralateral compression of the left frontoparietal lobe.

Since patients can respond dramatically to surgical evacuation of a subdural haematoma, physicians should be aware that, in addition to strokes and tumours, chronic subdural haematoma may lead to Gerstmann's syndrome, prior to progressing to dementia.

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