SHORT REPORT

Uncoupling between CBF and oxygen metabolism in a patient with chronic subdural haematoma: case report

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Abstract

The regional cerebral blood flow (rCBF) and oxygen metabolism of a patient with a chronic subdural haematoma were examined quantitatively, using positron emission tomography (PET). Before operation, the rCBF was decreased slightly throughout the brain, whereas the regional oxygen extraction fraction (rOEF) was increased throughout the brain, with values ranging from 0.36 to 0.60. One month after operation, the rCBF had recovered remarkably in almost all regions and rOEF had decreased to within the normal range.

It is unclear how a chronic subdural haematoma causes characteristic symptoms such as hemiparesis, aphasia, dementia, and impaired consciousness. Impairment of cerebral blood flow and metabolism may have been involved¹⁻⁵ but so far without evidence. This study is the first report of the use of positron emission tomography (PET) to measure the cerebral blood flow and oxygen metabolism of a patient with a chronic subdural haematoma.

Case report

The patient was a 56 year old hypertensive

drinker. He had no previous head injury and was admitted to a local hospital complaining of both a gradual loss of activity during a two month period, and a weakness in his right hand and leg. The following day he had two grand-mal seizure attacks. Ten days later, a large extra-axial mass lesion was detected by CT. He was then transferred to our hospital. When admitted he was disorientated and complained of frontal headache. Neurological examination revealed mild dementia and mild hemiparesis of the right side. The CT scan showed a massive subdural mass of mixed density, measuring 154 ml, with a shift of the mid-line structures (fig 1a). Regional cerebral blood flow (rCBF-ml/100 ml/min), cerebral blood volume (CBV-ml/100 ml/min), oxygen utilization (CMRO₂-ml/100 ml/min), and oxygen extraction fraction (OEF), were measured using a PET (HEADTOME III) and arterial blood sampling, during the sequential constant inhalation of C¹⁵O₂ and ¹⁵O₂ and the single inhalation of C¹⁵O.

The results in the regions of interest, were identified from the CT scan and are listed in the table. (Age-matched normal values for means (SD) measured by the HEADTOME III of our institute are as follows; CBF: 44.2 (7.3), CBV: 4.31 (0.48), CMRO₂: 3.04 (0.44), OEF: 0.41 (0.06), age: 56.7 (5.6) years old.) The rCBF and rCMRO₂ were decreased slightly throughout the brain. In the left fronto-temporo-parietal cortex just under the haematoma, both rCBF and rCMRO₂ had slightly higher values than in the corresponding region of the non-affected side. Only in the paraventricular deep white matter (corona radiata) were both rCBF and rCMRO₂ lower in the affected side than in the non-affected side. The rCBV value was within the normal range, and there was not a focal increase in the region just under the haematoma. The rOEF throughout the brain was increased, with values over 0.45 in almost all the region. In the left cerebral hemisphere the OEF was slightly higher than on the right side (fig 2a).

Evacuation, irrigation, and drainage of the haematoma, through a single burr hole, were performed immediately after the PET study. The haematoma found was dark-red in colour and muddy. One week after the operation, the patient was alert and had no neurological deficit. The complete evacuation of the haematoma and recovery from the shift of the midline structures were verified by a CT scan (fig 1b).

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Figure 1 A) The CT scan on admission showed a massive subdural mass of mixed density, with a shift of mid-line structures; B) The CT scan, taken at six weeks after the operation, shows complete evacuation of the haematoma and resolution of the shift of the midline structures.

Table Quantitative evaluation of rCBF, rCBV, rOEF, and rCMRO₂

	right (non-affected side)				left (affected side)				
	rCBF	rCBV	rOEF	rCMRO ₂	rCBF	rCBV	rOEF	rCMRO ₂	
Before operation									
Cerebellum	43.6	3.37	0.53	3.15	46.3	3.69	0.50	3.17	
Frontal cortex	28.5	3.43	0.48	1.91	31.8	2.24	0.55	2.39	
Temporal cortex	22.5	2.80	0.36	1.23	32.7	3.17	0.57	2.57	
Head of caudate	25.3	2.80	0.60	2.04	31.5	3.16	0.55	2.32	
Thalamus	33.9	3.21	0.49	2.26	36.2	6.28	0.46	2.28	Hb: 10.0
Paraventricule*	24.4	2.76	0.57	1.85	19.9	2.98	0.49	1.31	PH: 7.484
Parietal cortex	33.1	4.54	0.52	2.34	36.1	2.15	0.47	2.34	PaCO ₂ : 34.7
Occipital cortex	26.4	1.98	0.42	1.55	43.5	9.14	0.51	3.06	PaO ₂ : 91.7
After operation									
Cerebellum	75.5	3.70	0.38	4.50	76.3	4.50	0.35	4.28	
Frontal cortex	47.3	3.94	0.34	2.06	35.3	3.13	0.34	1.75	
Temporal cortex	40.0	4.07	0.36	2.32	36.8	1.92	0.39	2.15	
Head of caudate	70.0	3.91	0.34	3.70	61.5	4.14	0.36	3.35	
Thalamus	51.4	2.51	0.35	2.76	58.7	4.96	0.33	3.04	Hb: 11.7
Paraventricle*	26.8	1.28	0.42	1.74	17.2	3.50	0.45	1.12	PH: 7.422
Parietal cortex	51.8	3.51	0.36	2.92	54.5	3.55	0.32	2.70	PaCO ₂ : 34.3
Occipital cortex	43.3	4.05	0.36	2.40	54.7	6.98	0.42	3.60	PaO ₂ : 85.9

*Paraventricle deep white matter (corona radiata).

Cerebral angiography did not show either stenosis or occlusion of cerebral vessels. Six weeks after the operation, a PET study was repeated. The rCBF had increased to normal values in almost all regions. The relatively high level of rCBF and CMRO₂ in the left fronto-temporo-parietal cortex had fallen to normal. The rCMRO₂ had also recovered, but was still below the normal range in some regions (for example, bilateral frontal cortex). The rOEF had decreased to within the normal range, measuring 0.32 to 0.45 in all regions (fig 2b).

Discussion

A few reports describe the cerebral blood flow and oxygen metabolism of patients with a chronic subdural haematoma.¹⁻⁵ Kuhl *et al*² examined the cerebral blood volume of head-injured patients, and reported that the CBV of patients with a subdural haematoma was increased in the cortex just under the haematoma, and returned to normal after surgical

evacuation of the haematoma. They considered that the increase of CBV was due to the dilatation of the cortical vessels. They also believed that circulatory disturbance was responsible for the neurological deficits. However, only two of Kuhl's cases had a chronic subdural haematoma. Brodersen *et al*¹ used the intra-arterial ¹³³Xe method to measure the hemispheric CBF of seven patients with chronic subdural haematoma before and after surgery and found that the hemispheric CBF on the affected side was below normal before the operation, but recovered after surgical evacuation of the haematoma. CBF and CMRO₂ were measured in one patient whose CMRO₂ was below normal, to 1.1 ml/100 g/min, and was accompanied by a CBF thought to be caused by a reduced metabolic demand. Ikeda *et al*³ used the ¹³³Xe inhalation method to measure rCBF. Each of their 21 cases had a bilateral decrease in the mean hemispheric CBF before operation with increases following surgery. In patients with a hemiparesis, rCBF at the Rolandic motor and

Figure 2 The regional blood flow (rCBF), the regional blood volume (rCBV), the regional oxygen extraction fraction (rOEF), and the oxygen utilisation (rCMRO₂) were measured in the preoperative (A) and the postoperative period (B). (A): In the preoperative period, rCBF and rCMRO₂ slightly decreased throughout the brain. In the left fronto-temporo-parietal cortex just under the haematoma, both rCBF and rCMRO₂ had slightly higher values than in the corresponding region of the right side. The rOEF increased throughout the entire brain. (B) Six weeks after the operation, the rCBF and rCMRO₂ recovered in almost all regions. The rOEF decreased to within the normal range.

sensory area were decreased significantly, but a focal decrease was not seen in patients with only a headache.

In our patient, a decrease in rCBF was found throughout the entire brain. We believe that this was the result of cerebral compression by the massive haematoma. The metabolic rate of oxygen was also moderately below normal and the rOEF value was high throughout the brain. This contrasts with the findings in cerebral ischaemia in which a haemodynamic reserve is initially produced by vascular dilatation.² In our patient, vascular dilatation did not occur, oxygen transport reserve was called on, and consequently the OEF increased without an increase in CBV. Our findings also differ from those of Brodersen¹ who found CMRO₂ significantly decreased, along with a severe decrease of CBF, perhaps a reflection of the differences in the clinical severity of the two cases.

Ikeda *et al*^{3,4} noted hypoperfusion in the Rolandic cortex of patients with a hemiparesis. In our case, however, even with the higher spatial resolution of PET we could not detect a low perfusion area corresponding with his hemiparesis and aphasia. Indeed, the CBF value in the affected hemisphere was higher than in the non-affected hemisphere. It is possible that this seemingly paradoxically low level of CBF could be caused by an increase of cerebral tissue volume in each pixel, as a

consequence of compression of cortical sulci by the haematoma. On the other hand, the CBF was also depressed in the contralateral hemisphere. This general reduction of CBF is thought to be responsible for dementia and impairment of consciousness. A relatively preserved oxygen metabolism and a post-operative increase in CBF were related to the favourable response to surgery. However, in our patient, for reasons that are not clear, rCMRO₂ did not return to the normal range in the bilateral frontal and temporal cortex.

Although our experience is based on only one patient, we believe that uncoupling of the CBF and oxygen metabolism throughout the brain occurred, and caused the neurological deficits.

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