SHORT REPORT

Subcortical aphasia from a thalamic abscess

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Abstract

A patient is reported who was treated successfully for a left thalamic abscess that resulted in subcortical aphasia. A SPECT scan showed large areas of hypoperfusion in the cortex of the left hemisphere. At follow up after seven months there was marked improvement in the language disorder and the cortical hypoperfusion. It is suggested that aphasia in patients with subcortical lesions results from secondary cortical dysfunctions. The evidence is confined to patients with stroke lesions. The possible implications of this case on current theories of pathophysiological mechanisms, in particular the ischaemic penumbra theory and the cortical diaschisis theory, are briefly discussed. Cortical diaschisis may be the appropriate explanation in this patient.

Aphasia may occur after lesions of deep subcortical structures. Patients with haemorrhagic and ischaemic strokes in the region of the basal ganglia of the dominant hemisphere have been extensively studied.¹⁻¹¹ A relatively consistent picture has emerged, thus the concept of "subcortical aphasia".¹² Many issues, however, remain controversial. To date studies on subcortical aphasia have focused on stroke patients, thereby neglecting patients with non-vascular lesions. This omission might have influenced current theories about the mechanisms involved.

Language deficits following thalamotomy for Parkinsonism and related disorders have been well documented.¹³⁻¹⁷ Aphasia as a sign of an intrathalamic tumour has been mentioned infrequently.¹⁸⁻²⁰ We describe a patient with subcortical aphasia from a thalamic abscess.

Case report

A 33 year old, right handed, physician was in good health until he developed a left retro-orbital throbbing headache, associated with vomiting and mild fever. A few days later the headache improved, but he complained of double vision. His wife noticed a difficulty with his speech and a clumsiness in his right hand. Six days after the onset of the headache he was admitted to hospital.

Neurological examination showed the patient to be lethargic, but fully oriented and cooperative. His temperature was 37.7°C and

there was no neck stiffness. Spontaneous speech was not fluent with grammatical errors and some paraphasias. The volume of his voice was considerably reduced. Object naming, repetition and reading were all impaired. In contrast, comprehension was well preserved. A mild right hemiparesis caused difficulty in writing. Calculations, finger naming and right-left orientation were normal. The remainder of the neurological examination showed a vertical gaze paresis.

A CT scan of the head with intravenous iodamide showed a mass lesion in the region of the left thalamus (fig 1a). There was marked ring enhancement, perifocal oedema and a slightly deviated third ventricle, but no enlargement of the ventricular system. Left carotid and vertebral angiography confirmed the presence of a mass lesion in the left thalamic region. There were no other angiographic abnormalities.

A CT guided stereotactic puncture of the lesion allowed aspiration of pus. *Streptococcus milleri* was identified as the infecting organism and the patient was treated with high doses of intravenous penicillin and metronidazole. Infected dental caries appeared as a probable source.

During the following weeks the patient's condition improved considerably, together with a progressive shrinkage of the abscess on CT and MRI (fig 1b). Four weeks after admission the patient had an extensive neuropsychological assessment, including detailed tests of language and cognitive functioning: Porch Index of Communicative Ability (subtest 1: P95-99; subtest 4: P99; subtest 5: P99; subtest A: P85-90; subtest B: P90), Token-test (59/61), Minnesota-test (problems with giving definitions), WAIS (verbal IQ 100; performance IQ 102), ADM (I: 30; while 37 is a minimum for his age; II: 19, while 27 is a minimum for his age), AVLT (third trial 9, fifth trial 12, delayed recall trial 10, recognition trial 14), WMS [logical memory 13.5 (normal), paired associate learning 14 (normal)], BVRT [correct: 6 (normal=9); errors: 5 (normal=1)], Word Fluency (below P20), PRM (subtest 2: 3.3 SD -; subtest 6: 2 SD -; subtest 7: 4.3 SD all below that expected for his age and intelligence.^{22 23} There was a mild residual aphasia, consisting mainly of a hesitant speech and word-finding impairments. In addition general intellectual functioning, memory, attention and visuospatial abilities were all affected to some degree. Neurological examination at that

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Figure 1 a) CT scan showing mass lesion in the region of the left thalamus; b) CT scan four weeks after admission showing regression of the lesion; c) SPECT scan four weeks after admission, showing areas of hypoperfusion in the cortex of the left hemisphere (difference of more than 10% in counts per square mm between left and right); d) SPECT scan seven months later, showing improvement of the perfusion in the left cerebral cortex (no significant difference in counts per square mm between left and right).

time revealed only a slight difficulty with fine movements of the right hand.

Regional cerebral blood flow was also investigated by SPECT, using ^{99m}Tc Technetium HM PAO.²¹ Apart from the abnormalities in the region of the left thalamus and a diminished flow in the right cerebellum, there were large areas of hypoperfusion in the cortex of the left hemisphere. (fig 1c).

A follow up examination after seven months revealed no clinically detectable language and intellectual impairments and the patient returned to working as a physician without problems. SPECT scan at that moment showed a marked improvement of the hypoperfusion in the left brain (fig 1d).

Discussion

Thalamic lesions may induce ipsilateral cortical hypoperfusion and hypometabolism.²² This phenomenon has been demonstrated several times in patients with subcortical aphasia.^{23–27} It has therefore been proposed that the language disorder results from secondary cortical alterations where functional deficits are potentially reversible, as opposed to the structural damage caused by the lesion itself. Subcortical aphasia may have a strong tendency for improvement, which might be explained by the reversible nature of the corresponding deficits.¹²

The cortical dysfunction hypothesis may be criticised on several grounds. So far the evidence is rather scarce and confined to patients with haemorrhagic or ischaemic insults. It could be argued that studies on regional brain perfusion and metabolism in patients with cerebrovascular disease are flawed by confounding variables, such as previous cerebral infarctions and small vessel disease from chronic hypertension. Conversely in this patient with restricted space-occupying lesion, oedema and pressure effects have to be taken into account. It seems unlikely, however, that these factors induce a pattern of hypoperfusion at a distance from the lesion. In this patient large areas of hypoperfusion were found over the left hemisphere and the right cerebellum.

In general two different theories make an attempt to interpret the cortical disturbances in terms of underlying pathophysiological processes. Olsen *et al*²⁵ suggest that the cortical dysfunctions result from an ischaemic penumbra. The latter refers to an area of hypoperfusion located adjacent to a cerebral infarct.^{28–29} Blood flow in this area is sufficient for tissue viability, but not sufficient for normal function. In our opinion the cortical hypoperfusion and aphasia in this patient with a non-ischaemic lesion and an otherwise normal arteriography argues against a purely vascular explanation and requires at least an extension of this theory.

Perhaps another more satisfying explanation is the cortical diaschisis theory.³⁰ Diaschisis is defined as a transient depression of the function of intact brain regions, remote from, but connected to a focal lesion. According to this theory cortical neuronal activity is critically decreased due to the loss of essential input from thalamocortical projections. Hypoperfusion is thus secondary to hypometabolism and loss of function. There is some support for this theory from animal experiments.^{31–32} The exact anatomical basis for this process, however, is not entirely clear.

A wide range of subcortical lesions may produce language disturbances, including lesions of the ventrolateral thalamus, striatum, portions of the internal capsule and periventricular white matter. There have been attempts to correlate lesion sites to specific aphasia profiles.^{33–35} This undertaking, already a difficult one in this region with many different grey matter structures and fibre tracts tightly packed together, seems further complicated, if diaschisis effects are to be taken into account.

If the language disturbance is due solely to cortical dysfunctions, the specificity of the clinical picture of subcortical aphasia might be questioned. Some symptoms, however, are thought to be characteristic. A striking feature in many patients is the association with speech abnormalities, such as hypophonia.¹² This may reflect interruption of extrapyramidal connections.⁸ Another common finding is the "transcortical" nature of the aphasia, that is spared repetition abilities.³

Finally the language disorder in this previously healthy highly intelligent young man,

may be part of a more general disturbance of cognitive functions. It is tempting to correlate this with the demonstration of multiple areas of hypoperfusion in the cortex of the left hemisphere. Recently neurobehavioural deficits following lesions of subcortical structures have been described in detail.^{9 10 36} Further studies are needed to investigate the role of cortical diaschisis as the underlying cause in these situations.

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- 1 Ciemins VA. Localized thalamic hemorrhage. A cause of aphasia. *Neurology* 1970;20:776-82.
- 2 Demeurisse G, Derouck M, Coekaerts MJ, et al. Study of two cases of aphasia by infarction of the left thalamus without cortical lesion. *Acta Neurol Belg* 1979;79:450-9.
- 3 McFarling D, Rothi LJ, Heilman KM. Transcortical aphasia from ischaemic infarcts of the thalamus: a report of two cases. *J Neurol Neurosurg Psychiatry* 1982;45:107-12.
- 4 Damasio AR, Damasio H, Rizzo M, Varney N, Gersh F. Aphasia with nonhemorrhagic lesions in the basal ganglia and internal capsule. *Arch Neurol* 1982;39:15-20.
- 5 Kirshner HS, Kistler KH. Aphasia after right thalamic hemorrhage. *Arch Neurol* 1982;39:667-9.
- 6 Papagno C, Guidotti M. A case of aphasia following left thalamic hemorrhage. *Europ Neurol* 1983;22:93-5.
- 7 Davous P, Bianco C, Duval-Lota AM, de Recondo J, Vedrenne C, Rondot P. Aphasie par infarctus thalamique paramédian gauche. Observation anatomo-clinique. *Rev Neurol* 1984;140:711-9.
- 8 Gorelick PB, Hier DB, Benevento L, Levitt S, Tan W. Aphasia after left thalamic infarction. *Arch Neurol* 1984;41:1296-8.
- 9 Graff-Radford NR, Damasio H, Yamada T, Eslinger PJ, Damasio AR. Nonhaemorrhagic thalamic infarction. Clinical, neuropsychological and electrophysiological findings in four anatomical groups defined by computerized tomography. *Brain* 1985;108:485-516.
- 10 Fromm D, Holland AL, Swindell CS, Reinmuth OM. Various consequences of subcortical stroke. Prospective study of 16 consecutive cases. *Arch Neurol* 1985;42:943-50.
- 11 Puel M, Cardebat D, Demonet JF, et al. Le rôle du thalamus dans les aphasies sous-corticales. *Rev Neurol* 1986;142:431-40.
- 12 Benson DF, Geschwind N. Aphasia and related disorders: a clinical approach. In: Mesulam MM, ed. *Principles of Behavioral Neurology*. Philadelphia: Davis, 1985: 193-238.
- 13 Bell DS. Speech functions of the thalamus inferred from the effects of thalamotomy. *Brain* 1968;91:619-38.
- 14 Samara K, Riklan M, Levita E, et al. Language and speech correlates of anatomically verified lesions in thalamic surgery for Parkinsonism. *J Speech Hearing Res* 1969;12:510-40.
- 15 Riklan M, Levita E. Psychological studies of thalamic lesions in humans. *J Nerv Ment Dis* 1970;50:251-65.
- 16 Villki M, Laitinen LV. Differential effects of left and right ventrolateral thalamotomy on receptive and expressive verbal performances and face-matching. *Neuropsychologia* 1974;12:11-9.
- 17 Darley FL, Brown JR, Swenson WM. Language changes after neurosurgery for Parkinsonism. *Brain and Language* 1975;2:65-9.
- 18 Smyth GE, Stern K. Tumours of the thalamus. A clinicopathological study. *Brain* 1938;61:339-74.
- 19 Chandler WF, Farhat SM, Pauli FJ. Intrathalamic epidermoid tumor. Case report. *J Neurosurg* 1975;43:614-7.
- 20 Hirose G, Lombroso CT, Eisenberg H. Thalamic tumors in childhood. Clinical, laboratory, and therapeutic considerations. *Arch Neurol* 1975;32:740-4.
- 21 Sharp PF, Smith FW, Gemmel HG. Technetium-^{99m} HM-PAO stereoisomers as potential agents for imaging regional cerebral blood flow: Human volunteer studies. *J Nuclear Med* 1986;27:171-7.
- 22 Eisenberg J. *Adult aphasia. Assessment and treatment*. Englewood Cliffs: Prentice Hall, 1984.
- 23 Lezak MD. *Neuropsychological assessment*. New York: Oxford University Press, 1983.
- 24 Franck G, Salmon E, Sadzot B, Van Der Linden M. Etude hémodynamique et métabolique par tomographie à émission de positons d'un cas d'atteinte ischémique thalamo-capsulaire droite. *Rev Neurol* 1986;142:475-9.
- 25 Metter EJ, Wasterlain CG, Kuhl DE, Hanson WR, Phelps ME. ¹⁸F-DG Positron emission computed tomography in a study of aphasia. *Ann Neurol* 1981;10:173-83.
- 26 Baron JC, D'Antona R, Serdaru M, Pantano P, Bousser MG, Samson Y. Hypométabolisme cortical après lésion thalamique chez l'homme: étude par la tomographie à positons. *Rev Neurol* 1986;142:465-74.
- 27 Olsen TS, Bruhn P, Oberg RGE. Cortical hypoperfusion as a possible cause of "subcortical aphasia". *Brain* 1986;109:393-410.
- 28 Fasanaro AM, Spitaleri DLA, Valiani R, Postiglione A, Soricelli A, Mansi L, et al. Cerebral blood flow in thalamic aphasia. *J Neurol* 1987;234:421-3.
- 29 Perani D, Vallar G, Cappa S, Messa C, Fazio F. Aphasia and neglect after subcortical stroke. A clinical/cerebral perfusion correlation study. *Brain* 1987;110:1211-29.
- 30 Astrup J, Siesjö BK, Symon L. Thresholds in cerebral ischemia-The ischemic penumbra. *Stroke* 1981;12:723-5.
- 31 Olsen TS, Larsen B, Herning M, Skriver EB, Lassen NA. Blood flow and vascular reactivity in collaterally perfused brain tissue. Evidence of an ischemic penumbra in patients with acute stroke. *Stroke* 1983;14:332-41.
- 32 Feeney DM, Baron JC. Diaschisis. *Stroke* 1986;17:817-30.
- 33 Girault JA, Savaki HE, Desban M, Glowinski J, Besson MJ. Bilateral cerebral metabolic alterations following lesion of the ventromedial thalamic nucleus: mapping by the ¹⁴C-deoxyglucose method in conscious rats. *J Comp Neurol* 1985;231:137-49.
- 34 Girault JA, Savaki HE, Desban M, Glowinski J, Besson MJ. Conséquences de la lésion du noyau ventro-médian du thalamus sur le métabolisme cérébral: une étude expérimentale chez le rat par la méthode du ¹⁴C-desoxyglucose. *Rev Neurol* 1986;142:456-64.
- 35 Archer CR, Ilinsky IA, Goldfader PR, Smith KR. Aphasia in thalamic stroke: CT stereotactic localization. *J Comp Assist Tomogr* 1981;5:427-32.
- 36 Naeser MA, Alexander MP, Helm-Estabrooks N, Levine HL, Laughlin SA, Geschwind N. Aphasia with predominantly subcortical lesion sites. Description of three capsular/putaminal aphasia syndromes. *Arch Neurol* 1982;39:2-14.
- 37 Alexander MP, Naeser MA, Palumbo CL. Correlations of subcortical CT lesion sites and aphasia profiles. *Brain* 1987;110:961-91.
- 38 Katz DI, Alexander MP, Mandell AM. Dementia following strokes in the mesencephalon and diencephalon. *Arch Neurol* 1987;44:1127-33.
- 39 Mesulam MM. Patterns in behavioral neuroanatomy: association areas, the limbic system, and hemispheric specialization. In: Mesulam MM, ed. *Principles of behavioral neurology*. Philadelphia: Davis Company 1985:1-70.