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J. Servan
P. Verstichel
M. Catala
A. Yakovleff
G. Rancurel

Aphasia and infarction of the posterior cerebral artery territory

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J. Servan · M. Catala · A. Yakovleff
G. Rancurel
Urgences Cérébrovasculaires,
Hôpital de la Salpêtrière, Paris, France

P. Verstichel
Service de Neurologie (Pr M. Masson),
Hôpital Beaujon, Clichy, France

J. Servan (✉)
Service de Neurologie,
Hôpital de la Salpêtrière (Pr P. Brunet),
47 Bd de l'Hôpital, F-75013, Paris, France

Abstract Spoken language disorders are rarely mentioned in superficial infarction of the posterior cerebral (PCA) territory. Two clinical types have been reported: transcortical sensory and amnesic aphasia. Between 1979 and 1990, we studied retrospectively 76 patients suffering from an occipitotemporal infarction located in the superficial territory of the posterior cerebral artery, all well documented by CT. Aphasia was one of the first and prominent signs in 18 cases. Middle cerebral artery concomitant infarction could have been the cause of language impairment in 10. In 8 patients aphasia was only explained by a PCA territory infarct. Three patients showed features of transcortical sensory aphasia. CT localization showed internal lobe and thalamic involvement of the dominant hemisphere. Five patients exhibited word finding impairment with various degrees of amnestic syndrome. The dominant internal temporal lobe was always afffected. Dominant thalamus involvement was found in one case only. Some correlations between clinical features and anatomical support (vascular supply and anatomical structure) might be suggested in our 8 cases of aphasic disorders due to PCA infarcts. They are discussed and compared with data in the literature.

Key words Aphasia · Posterior cerebral artery · Stroke

Introduction

Infarction of the posterior cerebral artery (PCA) can induce language disorders. Amnesic aphasia is classically described with lesions of the left temporal lobe [1]. Among the few clinical series of PCA infarction published in the literature, aphasia is rarely mentioned. Kinkel et al. [2] reported 3 cases of aphasia out of 34 patients. In the series of Pessin et al. [3], none of the 35 patients seemed to be aphasic. More recently, Chambers et al. [4] reported 8 patients with language disorders similar to those described with infarctions of the middle cerebral artery (MCA) territory. From all these data, it seems difficult to identify a definable aphasic syndrome. The role played by lesions of the thalamus in aphasia is still a matter of debate. It is well known that lesions of the left thalamus can be responsible for speech disorders, whether they are due to haematoma [5–7], brain tumours [8, 9], thalamotomy [10, 11] or to electrical stimulation of the left thalamus [12–14]. In the case of infarction, thalamic softening is usually associated with posterior cortical lesions. In some cases [15–20], it has been possible to isolate dysphasic symptoms related to localized ischaemic lesions of the thalamus. In this paper, 8 cases of aphasia due to infarction of the PCA are reported. Their clinical presentations are different from one another depending on whether lesions of the PCA involve the thalamus.

Patients and methods

A retrospective study including 76 cases of patients suffering from PCA ischaemic infarction was made. All were admitted to the neurological department (Hôpital de la Salpêtrière, Paris) between 1974 and 1990. There were 14 females and 62 males, ranging from 24 to 99 years old (mean age: 62). Amongst these 76 patients, 10

patients (13%), all right-handed, exhibited oral aphasia. In all our cases, only the left PCA territory was involved, according to the CT findings. Patients presenting with associated infarction of the MCA or the MCA-PCA junctional territories were excluded. Two of these ten patients were excluded from this study because of insufficient documentation.

Investigations

Routine investigations including standard haematological and biochemical tests, ECG, chest radiograph and cerebral CT were performed in all patients. Cerebral angiography was performed in four cases. Topographical diagnoses of cortical and thalamic infarctions were determined on CT scans, by using templates from Matsui and Hirano's atlas [21] and Percheron's descriptions [22, 23].

Neuropsychological study

A comprehensive battery of tests was applied to all patients from 1 week to 3 weeks after the onset of the disease. Subsequently, only two patients had a complete neuropsychological follow-up. Neuropsychological tests included the following: orientation to time and space, WAIS, Wechsler's memory scale, Benton's visual retention test, Binet Simon's colour recognition test, Binois Pichot's vocabulary test, Rey's figure. The diagnosis of transcortical sensory aphasia (TSA) or amnesic aphasia (AA) was established according to the criteria of Benson and Geshwind [24].

TSA is characterized by fluent aphasia, with numerous paraphasias. Oral comprehension and written comprehension are impaired, but repetition is remarkably preserved with a tendency to echolalia. Denomination is always impaired also. In AA, word finding impairment is predominant, and therefore interfers with naming and spontaneous verbal fluency. Comprehension and repetition are preserved.

Results

Neuropsychological results

Three patients (cases 1–3) had symptoms which were compatible with TSA. Spontaneous speech was fluent in

Table 1 Clinical characteristics of the aphasias in the eight patients (*F* fluent, *NF* non-fluent, + normal, ± occasionally impaired, – impaired, *P* paraphasias)

Cases	Spontaneous speech	Auditory comprehension	Repetition	Naming	Reading	Writing
1	F/P	–	+	–	–	–
2	F/P	–	+	–	–	–
3	F/P	±	+	–	–	–
4	F/P	+	+	–	+	+
5	F	+	+	–	+	+
6	F	+	+	–	+	+
7	F	+	+	–	+	+
8	F	+	+	–	+	+

Table 2 Clinical and radiological features of the eight patients (*M* male, *F* female, *L* left, *HA* hemianopia, *CA* calcarine artery, *PCA* posterior cerebral artery, *PICA* posterior and inferior cerebellar artery, *POA* parieto-occipital artery, *ATA* anterior temporal artery, *PTA* posterior temporal artery, *DM* dorsomedial, *VPL* ventro-postero-lateral, *VL* ventrolateral

Cases	Sex	Age (years)	Vascular risk	Side of deficits	Clinical signs	CT scan distribution of infarctions	Angiograms
1	M	76	Hypertension	Right	Headache, HA, hemiplegia, numbness of body	Left PTA, CA Left VL, VPL, nuclei	Not done
2	M	68	Smoker	Right	Confusion, HA, cerebellar and pyramidal signs, diplopia	Left PTA Left DM nucleus	Left vertebral artery hypoplasia
3	M	74	Smoker, dyslipidaemia, hypertension	Right	Confusion, HA, acalculia, amnesic disturbances	Left ATA, PTA and CA Left DM nucleus	Left vertebral artery occlusion Right vertebral artery finishing in PICA
4	M	57	Smoker, diabetes mellitus, hypertension	Right	HA, Parinaud's syndrome, cerebellar signs, amnesic syndrome, visual agnosia	Left ATA, PTA and CA Left DM nucleus	Left PCA occlusion
5	M	68	Migraine	Right	HA, amnesic syndrome, pyramidal signs	Left PTA	Not done
6	M	76	Smoker, hypertension	Right	HA, hemineglect, constructive apraxia, amnesic syndrome	Left ATA, PTA and CA	Not done
7	M	31	Migraine, dyslipidaemia	Right	Headache, HA, spatial disorientation	Left PTA and CA	Left CA occlusion
8	M	67	Smoker	Right	HA, cerebellar and pyramidal signs, facial weakness	Left PTA, POA and CA Right DM nucleus	Not done

Table 3 Results of neuropsychological tests of the eight patients (+ normal, – impaired, ± occasionally impaired, *IM* impossible, *ND* not done)

	1	2	3	4	5	6	7	8
Spatial and time orientation	–	+	–	–	+	+	±	–
WAIS (verbal/performance)	77/70	ND	IM	119/107	ND	ND	ND	IM
Wechsler	ND	ND	ND	95	88	ND	99	IM
Visual recognition test	10	ND	ND	5	8	ND	8	IM
Colour recognition test	–	ND	ND	–	ND	ND	+	IM
Vocabulary test	IM	ND	ND	117	111	ND	115	IM
Rey's figure	±	ND	IM	IM	36	ND	35	IM

patients 2 and 3. In patient number 1, oral fluency was transiently reduced and vocal volume vanished promptly along with alteration of alertness. Later on, his speech capacities improved progressively, and his language became fluent. Numerous paraphasias were observed in all three patients, and were close to semantic jargon in cases 2 and 3. All these patients had alexia with agraphia but no visual agnosia for objects. Repetition was always remarkably preserved. Denomination was altered with numerous paraphasias in patients 1 and 2. Oral comprehension and written comprehension were impaired in patients 1–3.

The five other patients (cases 4–8) exhibited signs of AA. Speech fluency was always normal, but verbal output was qualitatively reduced along with word finding impairment. Only one patient (case 4) showed paraphasias (mainly semantic). Repetition and comprehension was always preserved. Reading and writing capacities were preserved in all cases except for patient 6, who exhibited alexia without agraphia. Denomination was always impaired. The five patients suffered from anterograde memory loss especially for verbal material. The characteristics of each language disorder, for each of our eight patients, are shown in Table 1. The other clinical data and the results of the other neuropsychological tests are shown in Tables 2 and 3 respectively.

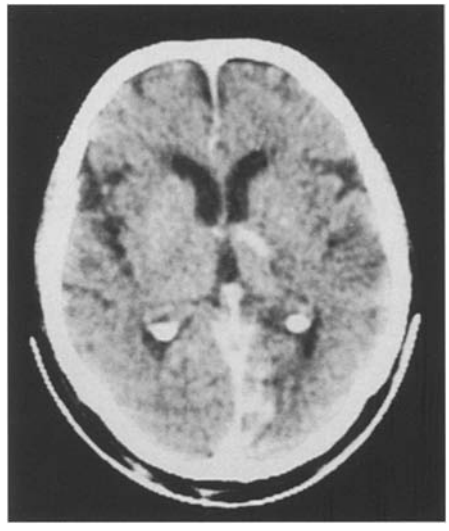

Fig. 1 Patient 1. Enhanced CT scan: left calcarine infarction with ipsilateral tuberothalamic territory involvement

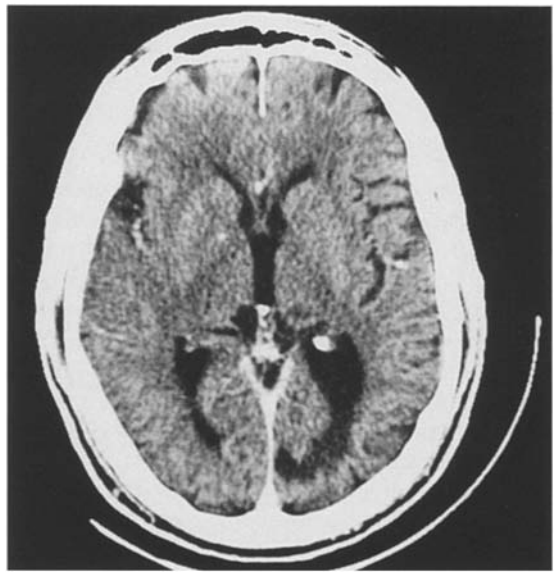

Fig. 2 Patient 2. CT scan without contrast: limited left posterior temporal infarction associated with paramedian territory infarction

Radiological results

In all cases, CT revealed PCA infarction in the left hemisphere. The MCA territory was never involved. Broca's and Wernicke's areas were intact.

In the three cases of TSA, the lesions involved the inferomedial portion of the left temporal lobe, and the occipital lobe in cases 1 and 3. Cortical infarct was always associated with ipsilateral thalamic lesions including the paramedial nuclear group in cases 2 and 3 or the tuberothalamic nuclear group in case 1 (Figs. 1–3).

In the five cases of AA, the cortical lesions were fairly similar. However, the dominant thalamus (in its paramedial nuclear group) was involved in case 4 only.

Discussion

In these eight patients, language disorders were separated into two groups, corresponding to either TSA or AA.

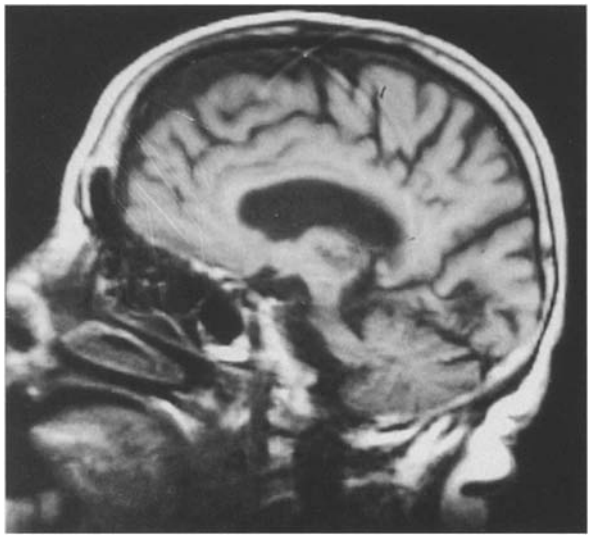

Fig. 3 Patient 3. MRI, sagittal sections: left dorsomedial nucleus involvement, associated with ipsilateral calcarine infarct

Transcortical sensory aphasia

In TSA, speech remains fluent, with marked paraphasias, anomia, empty circumlocutations and altered comprehension, but repetition is preserved. TSA has been described with diffuse lesions such as cortical degeneration [25], or with more focal lesions of vascular origin involving either the left temporo-parieto-occipital junction, posterior to Wernicke's area [26], the thalamus [7, 17, 27], or the MCA territory [28].

Cortical localizations in TSA

In 1885, Lichteim [29] established a relationship between TSA and cortical lesions involving structures which surround the language areas. In his hypothesis, these damaged structures could disconnect language areas from cerebral conceptualizing and memorizing centres. Right from the beginning, this entity has been much debated. Wernicke [30] did not use this expression in his initial publication on sensory aphasia, but he adopted it later on. Henschen [31] distinguished two groups of aphasic syndromes: one associated with extensive cerebral atrophy, and one with lesions of the temporal lobe. TSA has often been considered as a stage of recovery from Wernicke's aphasia or from complete word deafness in focal ischaemic lesions [32]. More recently, some authors have tried to establish a relationship betwen TSA and rather diffuse lesions [33, 34]. Unfortunately, the neuropsychological and anatomical data were incomplete. In a study carried out in 15 patients, Kertesz et al. [26] suggested that TSA could be associated with either left temporo-occipital infarctions or with junctional infarctions between PCA and MCA territories. In these patients, lesions were diagnosed 6 times with isotopic brain scans, and only 9 times with CT. Thalamic lesions were not mentioned.

Thalamic localizations in TSA

In our study, the ischaemic lesions reported in the three patients who exhibited TSA were not limited to the posterior cortical region, but they also involved the left thalamus. Several series of the literature have emphasized the frequency of aphasia after infarction of the left thalamus [15, 17, 19, 20, 35, 36]. In comparison with our patients, similar localizations have been reported by Graff-Radford et al. [35] and by Bougosslavsky et al. [20]. In these series, language disorders were reported only with infarction of the left paramedial and tubero-thalamic regions. Aphasia was characterized by hypophonia, reduced verbal fluency, verbal paraphasias, moderate comprehension impairment and normal repetition. However, the precise site of the lesions within the thalamus is difficult to establish on CT scans which lack fine definition. In our series, TSA was not present in four other patients (cases 5–8), in whom the cortical lesions were not associated with lesions of the dominant thalamus.

Speech disorders associated with lesions of the thalamus have brought some authors to introduce the concept of "thalamic aphasia" [37, 38]. Here, verbal fluency is reduced and verbal motivation is constantly low. Denomination and reciting serials are always impaired and disturbed by numerous paraphasias (mostly semantic) and by perseverations. Comprehension is satisfactory and repetition is preserved. Hypophonia is often noted. According to several authors, thalamic aphasia and TSA are quite similar in terms of presentation, whether these clinical features are associated with haematoma [7, 27] or with infarction [4, 17, 19]. Following these descriptions, three different types of transcortical aphasias (motor, sensory or mixed) have been described. Some of the functional perturbations implicated in thalamic aphasia could possibly explain some of the signs of TSA reported in our cases.

Spontaneous speech in TSA is constantly associated with the inability to make a precise lexical selection with semantic fields [39]. For some authors [37, 40], the thalamo-cortical circuitry which associates posterior cortical language areas and the posterior thalamic nuclei plays a significant functional role in speech control. Within this circuitry, it has been proposed [41] that the thalamus could act at the semantic level and therefore control messages before they are to be expressed orally. This lack of semantico-lexical regulation could explain verbal paraphasias (mostly semantic) in thalamic or posterior cortical lesions. Two thalamic functions could be implicated: access to vocabulary itself, and a kind of language self-regulation, which is referred to as "alertness" by Mohr [42]. Impairment of this last function could be responsible for

lack of suppression of incorrect words, production of neologisms, jargon and perseverations. Thalamo-frontal projections also seem to regulate this semantico-lexical function by controlling the sequencing of speech, for instance. Gorelick et al. [19] consider that the anterior-ventral nucleus plays a major role in this regulation. Our observations tend to show that lesions of the dorsomedial and ventrolateral nuclei could induce similar disorders.

In our three cases, comprehension capacities varied from one patient to another. They were severely impaired in patients 1 and 3, as classically described in TSA, and mildly in patient 2. All the other characteristics of their aphasia were identical in the three patients. Therefore, when the infarction of the PCA territory involves the thalamus, it seems possible to observe an intermediate language disorder between TSA and thalamic aphasia. In certain studies [20, 35], depending on the length of the verbal material to be used, both oral and written comprehension may be only moderately impaired after an isolated left thalamic lesion. These mild disorders could be the result of partial posterior cortical inactivation with deficient memory and deficient verbal attention. Amongst the two cases described by MacFarling et al. [17], one had an isolated thalamic lesion with preserved comprehension, whereas the other patient, who exhibited lesions involving both the left thalamus and the left occipital cortex, suffered from impaired comprehension. This discrepancy could be explained by the constant association of a posterior cortical lesion with thalamic infarction. However, it is difficult to implicate a specific neuronal circuitry since these cortical lesions do not involve the classical language areas. After correct phonological analysis, verbal comprehension requires access to semantic representations from lexical representations. This is mildly impaired in aphasia due to isolated thalamic lesions, but usually is very much altered in TSA. As opposed to the control of verbal expression, the role played by the thalamus does not seem to be so critical in comprehension processes. In thalamic lesions, the integrity of left temporal and parietal structures could allow rather good acoustico-semantic analysis, depending on the amount of verbal memory available.

In all our cases, repetition was preserved. In TSA, this could be due to the isolation of language areas [29], which means that the preservation of Broca's area, Wernicke's area and of the superior longitudinal fasciculus could act as a kind of "reflex arc" for repetition [30]. Nissl von Mayendorf [43] suggested that repetition could be taken over by the right hemisphere. This mechanism has been shown in a case of TSA after a cortico-subcortical lesion which spared to thalamus [44]. It the thalamus is involved, both mechanisms seem to be plausible. Either the lesion does not interfere with repetition within the left hemisphere, or the lesion induces sufficient deafferentation of the left hemisphere to let the right hemisphere take over repetition. Whichever mechanism is implicated, it explains why neither TSA with cortical lesions nor aphasia with thalamic lesions include repetition impairment.

Hypophonia is one of the characteristics of subcortical aphasia. However, this is an unusual feature in TSA. In our study, patient 1 presented with marked vanishing vocal volume especially during long sentences. This symptom and disorders of alertness appeared together and disappeared together rapidly. Hypophonia has been related to lesions involving the anterior area of the thalamus, the peri-aqueductal white matter and the motor supplementary area [40]. The interruption of extrapyramidal afferents which project from the pallidum and the pars reticulata of the substantia nigra towards the ventro-anterior nucleus and the rostral portion of the ventrolateral nucleus of the thalamus has also been proposed [19].

Amnesic aphasia

Difficulty in findings words spontaneously or in naming objects is one of the most common impairments reported in aphasia. Numerous cortical or subcortical lesions have been reported to be responsible for AA. According to Benson and Geschwind [24], when AA is severe and acute, the lesion seems to involve more often the temporo-occipital junction. It is more difficult to point out a precise anatomical lesion with moderate AA. Goldstein [45] suggested it could involve the deep inferior part of the temporal lobe. In many cases, AA is observed during the recuperating phase of other types of aphasia, which all rely on very different localizations of the dominant hemisphere (frontal, parietal, temporal, subcortical). Similar impairments have also been reported with lesions of the non-dominant hemisphere [14].

In our study, five patients had AA. Lesions involving the territory of the posterior temporal artery were constant. In case 4, there was also an infarction of the dorsomedial nucleus of the thalamus. This patient was the only one to exhibit paraphasias. However, his comprehension abilities were good, which is usually not be case with TSA. In patient 8, the lesion of the right thalamus did not influence dysphasic symptoms.

Conclusion

In infarctions involving the territory of the PCA, aphasia is a rare symptom. Two different types of aphasia have been described in this study. Besides the well-known AA, due to softening in the left postero-interior temporal lobe, several cases of TSA are reported. These could be explained by lesions involving the thalamus, possibly its dorsomedial and ventrolateral nuclei. Therefore, TSA could be considered a particular form of thalamic aphasia, in which dramatic comprehension impairment would depend more on the association of thalamic and posterior cortical lesions.

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