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ORIGINAL COMMUNICATION

Logopenic syndrome in posterior cortical atrophy

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Abstract Few language disorders have been reported in posterior cortical atrophy (PCA). Furthermore, no study has focused on screening for them and described these language deficits. The goal of this work was to describe linguistic examination of PCA patients and the impact of language disorders on neuropsychological performances compared to patients with other neurodegenerative syndromes and control groups. Linguistic examination of 9 PCA patients was carried out. The neuropsychological performance of the PCA group (16 patients) in the RAPID battery tests was compared with performances of patients with a logopenic variant of primary progressive aphasia (LPPA), patients with Alzheimer's disease and patients with amnestic mild cognitive impairment, as well as the control group. A "logopenic syndrome" with anomia, fluency impairment, and length-dependent deficit was found in 8/9 PCA patients. A comparison with other neurodegenerative syndromes showed that not only visual disorders but also language and verbal short-term memory disorders, such as those found in LPPA, can explain neuropsychological performances. A "logopenic syndrome" is frequently found in PCA and may be associated with poor performance on other verbally mediated neuropsychological tasks (e.g., verbal memory). Specific logopedic rehabilitation should be offered to these patients.

Keywords Posterior cortical atrophy · Aphasia · Logopenic variant of primary progressive atrophia · Alzheimer's disease · Mild cognitive impairment · Neuropsycology

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Introduction

Posterior cortical atrophy (PCA) is a clinical syndrome characterized by a progressive decline in visuospatial and visuoperceptive function and sometimes other posterior functions (e.g., calculation, praxis and reading) [1, 2] in parallel with a posterior progressive atrophy including the parietal, temporo-occipital and occipital cortex [3].

In the initial description by Benson et al. [1], oral language disorders were reported in PCA and classified as transcortical sensory aphasia. However, since this first

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description, few studies have reported oral language disorders in PCA. Anomia is frequently described as a presenting syndrome (>80 %), indicating that language can be one of the first complaints in PCA [2]. McMonagle et al. [4] reported oral language disorders in >40 % of their series of PCA, while more than one-third of their patients had anomia. Of these, three cases were classified as Wernicke's aphasia and one case was classified as conduction aphasia. Migliaccio et al. [5] reported that one-third of their PCA patients had language complaints. They found no significant difference on naming and fluency when compared with the logopenic variant of progressive primary aphasia (LPPA). Semantic memory is reported to be relatively spared compared to Alzheimer's disease [6, 7]. Only one group has performed systematic oral linguistic examination. They reported language disorders in a series of 15 PCA patients compared to 7 LPPA patients and showed that the language profile of PCA patient resembled that of LPPA patients on auditory input processing, repetition, but was relatively stronger on tasks of comprehension and spontaneous speech [8].

As PCA frequently involves the left parietal and posterior temporal cortices, we hypothesized that our PCA patients would have an aphasic syndrome similar to the oral language pattern found in the logopenic variant of primary progressive aphasia. Left parieto-temporal abnormalities are a feature of the logopenic variant of primary progressive aphasia. This "logopenic syndrome" would include a reduction in speech rate, anomia, reduced fluency, and length-dependent deficit induced by a verbal short-term memory deficit [9, 10]. Language disorders and a verbal short-term memory deficit may also induce a deficit in neuropsychological assessments other than visuospatial and visuoperceptive assessments, such as verbal memory tests.

Our objective was to describe the linguistic examination of PCA patients and the impact on neuropsychological performances of the language disorders observed in a group of PCA patients. These observations were then compared to patients with logopenic variant of primary progressive aphasia (LPPA), Alzheimer's disease (AD) and amnestic mild cognitive impairment (MCI), as well as a control group.

Methods

A retrospective study was conducted with consecutive patients diagnosed with PCA. All patients were referred to the regional memory center from January 2006 to December 2010 and recruited through the database of the Regional Network for Diagnostic Aid and Management of Patients with Cognitive Impairment in the Franche-Comté region (RAPID-Fr network) [11, 12]. The diagnoses were re-evaluated according to the available criteria [2, 6]. For each inclusion, neurological, psychological, neuropsychological, linguistic and imaging examinations (MRI and sometimes 99m ECD cerebral perfusion SPECT) had to be performed in order to confirm the diagnosis and exclude other pathologies such as tumor or vascular disorders, with an evaluation of periventricular FLAIR hypersignals and/or deep white matter FLAIR hypersignals >1 on the Fazekas scale [13] Focal atrophy was evaluated using the 4-point rating scale for each hemisphere [14]. DAT-SPECT (I-123-FP-CIT) was also performed when melokinetic apraxia or isolated akinesia was found without parkinsonian syndrome (no tremor or hypertonia, which we considered as inconsistent with PCA and LPPA diagnosis) in the neurological examination (6 PCA patients and 5 LPPA patients).

All patients underwent the RAPID neuropsychological assessment battery [11, 12] by trained neuropsychologists. This battery included the Mini Mental State Examination (MMSE), memory impairment screen (MIS), Isaacs set test (IST), free and cued recall test (FCRT), visuoconstruction by copying geometric figures as part of the BEC 96 (VC), picture naming test 30 items (PNT30), and categorical matching (CM).

The linguistic tests consisted of the following: a sentence comprehension assessment with a Verbal Comprehension Test (VCT) and a word and sentence comprehension test (WSCT) [15], a picture naming test with 80 items (PNT80) [16], a naming to verbal description and definition task [17], word and sentence repetition, a complex picture description and a semantic test with a matching and judgment subscale [18], phonological and categorical fluency [19], and qualitative analysis of spontaneous speech.

Subsequently, cognitive scores on the RAPID battery of PCA patients were compared with those of groups of patients with LPPA [9], MCI [20], mild Alzheimer's disease according to NINCDS-ADRDA criteria for AD (Clinical Dementia Rating scale = 1) [21], and a control group. Groups were matched by age (except the LPPA group), sex and education level randomly extracted from the RAPID network database. The study was approved by the local ethics committee in accordance with the Declaration of Helsinki on Biomedical Studies involving human subjects.

Statistical analysis

The Gaussian distribution assumption was tested using the Shapiro–Wilk test. We compared the results of the different groups with the Student's t test or the Mann–Whitney Wilcoxon test for continuous variables and the Chi-squared (χ^2) test for categorical variables.

All statistical analyses were performed with Stata software (release 8.0, Statacorp, College Station, TX).

Results

We included 16 subjects in each group. Demographic data are shown in Table 1.

The comparison for the RAPID battery between the PCA and LPPA, AD, MCI and control groups are shown in Table 2. The PCA group performed poorly on all RAPID battery tests compared to the control group. The MCI group performed better than the PCA group in all RAPID battery performances apart from the memory test (TFR and TR of the FCRT), which was not significantly different. The PCA group performed better than the AD group in the verbal memory tests (TFR and TR of the FCRT), while the PCA group performed poorly in the MMSE test (especially calculation and copy subscale) and VC. The PCA group performed less well than the LPPA group in the MMSE test (calculation and copy subscale), action naming on the PNT30, COT and VC.

Nine PCA patients underwent an additional linguistic examination (Table 3). We observed anomia in spontaneous oral discourse in 7/9 (77 %). On PNT80 and a naming to verbal description and definition task, anomia was observed in 6 patients with frequent visual mistakes (6/9; 66 %) and sometimes paraphasia (3/9; 33 %) or anomia (1/9; 11 %). Although all patients had preserved semantic memory, 6/9 (66 %) patients presented a length-dependent difficulty understanding long sentences with no disorders of syntax. Only 1/9 (11 %) patients showed difficulties in word repetition, while 5/9 (55 %) had difficulties when the repetition involved sentences. Finally, 8/9 (88 %) PCA patients present oral language disorders such as those found in LPPA patients (Table 3). The one patient who had no language disorders had an isolated right posterior cortical abnormality (Fig. 1).

Neuroimaging findings of PCA and LPPA patients are shown in Table 4. No significant correlation with the side of atrophy or hypoperfusion was found in our study because most of our patients presented with bilateral atrophy or hypometabolism.

Discussion

We report here the language skills of 9 PCA patients and the neuropsychological performances of 16 PCA patients compared to 16 LPPA, 16 AD and 16 MCI patients, and 16 control subjects matched by age (apart from with the LPPA group), sex and level of education. Eight of 9 PCA patients demonstrated a "logopenic syndrome" with anomia, reduced fluency, and length-dependent deficit such as those found in LPPA patients.

Comparisons of the neuropsychological performances of our 16 PCA patients showed that not only visual skills were impaired. Verbal fluency was equally impaired in LPPA and AD groups in our study, as reported by Rogers et al. [7]. Mendez et al. [6] reported that semantic fluency is better in PCA than AD. However, the switching between different categories of IST is more difficult for the PCA patients because of their parietal lesions, which is a structure that plays a role in switching [22]. The performances of the PCA group are pathological in the non-visual tasks. The language disorders may influence their neuropsychological performance, especially in verbal memory (encoding difficulty in immediate recall of the FCRT) and in tasks that require verbal short-term or working memory (mental calculation, fluency). In PCA, verbal mnesic performances were pathological, but a free and total recall were better than AD. Severe encoding difficulties, which are secondary to the phonological disorders, may cause us to overestimate the long-term memory deficit. The severe mental calculation difficulty could be induced by two mechanisms: authentic acalculia, which is classically reported in PCA syndrome, and the verbal working memory deficit. Apart from their visual disorders and their greater calculation deficit, the neuropsychological profile of the PCA group is similar to that found in LPPA

Table 1 Demographic data of our five groups

	PCA	LPPA	AD	MCI	Contol
Number of subjects	16	16	16	16	16
Age (SD)	61.7 (5.1)*	66.5 (7.2)	62.1 (4.5)	61.9 (5.6)	61.7 (5.1)
Sex (women/men)	11/5	6/10	11/5	10/6	11/5
Level of education	8/6/2	6/4/6	8/6/2	9/5/2	8/6/2
Laterality (right/left)	16/0	16/0	16/0	16/0	16/0

PCA posterior cortical atrophy, *LPPA* logopenic variant of primary progressive aphasia, *AD* Alzheimer disease, *MCI* mild cognitive impairment

* Significant difference between PCA and LPPA

Table 2 Neuropsychological results on the RAPID battery for PCA, LPPA, AD and MCI and control group

	N	Age	MMSE						FCRT		
			Total/30	TO/5	SO/5	Calcul/5	Copy/1	DR/3	IR/16	TFR/48	TR/48
PCA	16	61.7[a] (5.1)	19.6[a,b,c,d] (3.8)	4[d] (1.5)	4.4[c,d] (0.9)	1.1[a,b,c,d] (1.2)	0.4[a,b,c,d] (0.5)	0.9[c,d] (1.2)	12.8[c,d] (3.4)	18.1[b,d] (11.9)	37.6[b,d] (8.8)
LPPA	16	66.5 (7.2)	22.9 (3.9)	4.3 (0.7)	4.2 (0.8)	2.5 (1.9)	1 (0)	1.1 (1.3)	11.6 (3.7)	17.9 (9.1)	34.4 (11.5)
AD	16	62.1 (4.5)	22.2 (3)	3.5 (1.3)	4.3 (1.1)	2.8 (1.8)	0.7 (0.4)	0.5 (0.6)	12.4 (2.2)	8.2 (5.8)	24.6 (7.7)
MCI	16	61.9 (5.6)	27.2 (1.9)	4.5 (0.8)	5 (0)	4.3 (1.3)	1 (0)	1.7 (0.9)	15.2 (1.2)	18.2 (7.4)	40.3 (43.9)
Control	16	61.7 (5.1)	28.9 (1.1)	5 (0)	5 (0)	4.7 (0.6)	1 (0)	2.5 (0.9)	15.5 (0.6)	30.9 (5.1)	46.6 (1.5)

	MIS/8	IST	PNT30				CM/10	COT	VC/6
			Total/30	Animal/10	Object/10	Action/10			
PCA	5.4[c,d] (2.3)	23.6[c,d] (7.6)	27.2[c,d] (2.2)	8.9[c,d] (1.4)	9.3[c,d] (0.8)	9[a,c,d] (1.2)	9.6[d] (0.8)	136[a,c,d] (60.5)	2.8[a,b,c,d] (2.9)
LPPA	4.4 (2.6)	21.1 (7.8)	24.9 (6)	7.2 (3.2)	7.9 (2.8)	9.8 (0.4)	9.9 (0.3)	184.1 (45.4)	5.9 (0.3)
AD	3.8 (2.3)	24.9 (6.1)	27.7 (3.2)	9.2 (1.3)	8.9 (1.7)	9.6 (0.7)	9.8 (0.4)	142 (41.3)	5.2 (1.5)
MCI	7.2 (1.3)	36.3 (5.7)	29.9 (0.3)	10 (0)	9.9 (0.3)	10 (0)	9.9 (0.3)	228.1 (34.3)	6 (0)
Control	7.7 (0.4)	37.7 (5.8)	29.9 (0.3)	9.9 (0.3)	9.9 (0.3)	10 (0)	10 (0)	218.7 (36.9)	6 (0)

PCA posterior cortical atrophy, *LPPA* logopenic variant of primary progressive aphasia, *AD* Alzheimer disease, *MCI* mild cognitive impairment, *N* number of subjects, *MMSE* mini mental state examination, *TO* temporal orientation, *SO* spatial orientation, *DR* delayed recall, *FCRT* free and cued recall test, *IR* immediate recall, *TFR* total free recall, *TR* total recall, *MIS* memory impairment screen, *IST* Isaacs set test, *PNT30* picture naming test 30 items, *CM* categorical matching, *COT* crossing-off test, *VC* visuo-construction with copy of the triangles of the BEC96

[a] Significant difference between PCA and LPPA ($p < 0.05$)

[b] Significant difference between PCA and AD ($p < 0.05$)

[c] Significant difference between PCA and MCI ($p < 0.05$)

[d] Significant difference between PCA and control ($p < 0.05$)

Table 3 Logopedic examination of posterior cortical atrophy (PCA) and logopenic variant of primary progressive aphasia (LPPA)

	PCA ($n = 9$)	LPPA ($n = 16$)
Picture naming test 30 items[a]		
Total (/30)	27.2 (2.2)	24.9 (6)
Animal (/10)	8.9 (1.4)	7.2 (3.2)
Object (/10)	9.3 (0.8)	7.9 (2.8)
Action (/10)	9 (1.2)*	9.8 (0.4)
Picture naming test 80 items (/80)	71.6 (7.4)	64.9 (10.1)
Naming to verbal description and definition task (/15)	12.2 (1.6)	10.3/15 (3.3)
Categorical fluency	19.7 (9.5)	18.7 (12.1)
Phonological fluency	15.8 (9.5)	13.9 (9.6)
Isaacs set test[a]	23.6 (7.6)	21.1 (7.8)
Word and sentence comprehension test		
Word (/9)	8.8 (0.5)	8.3 (0.6)
Sentence (/24)	19.3 (6.1)	14.7 (4.6)
Verbal comprehension test (/24)	21.8 (2.6)	20.9 (2.9)
Semantic test		
Matching subscale (/40)	38.2 (1.5)	37.5 (3.3)
Judgment subscale (/24)	22.8 (0.8)	23.6 (0.6)
Repetition		
Word (/25)	24.5 (1.4)	22.8 (2.6)
Sentence (/3)	1.5 (0.8)	1.3 (0.8)

n number of patients

* Significantly difference with *PCA* and *LPPA* ($p < 0.05$) explained by visuoperceptual impairment when viewing complex pictures of action

[a] Test included in the RAPID battery, $n = 16$ *PCA* patients

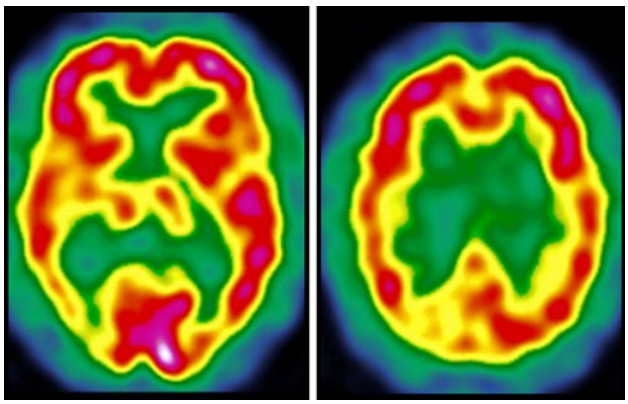

Fig. 1 Cerebral perfusion SPECT (ECD-Tc 99m) showing a pure right parieto-temporo-occipital hypoperfusion in a PCA patient without "logopenic syndrome"

groups (encoding impairment, verbal working memory deficit, impaired fluency performances) as described in the study by Migliaccio et al. [5].

No significant correlation with the side of atrophy was found in our study because most of our patients presented bilateral atrophy or hypometabolism. Neuroimaging of PCA and LPPA patients showed common abnormalities especially on the left temporo-parietal junction and the biparietal cortex: 15/16 (93 %) PCA patients had left parietal abnormalities and 12/16 (75 %) had an hypometabolism of the left temporo-parietal junction. Moreover, one patient presenting with pure right PCA did not have a logopenic syndrome. It suggests that the left parieto-temporal junction plays a key role in "logopenic" syndrome of PCA patients such as anatomical and functional abnormalities found in LPPA patients [8, 9]. Dopaminergic depletion was found in the DAT-SPECT examination of some PCA (3/16; 18 %) and LPPA patients (3/16; 18 %). Dopaminergic depletion associated with parietal abnormalities suggest the possibility of a continuum between some PCA patients, some LPPA patients and corticobasal degeneration (CBD) syndrome. CBD syndrome, such as PCA and LPPA, can induce language disorders and frequently involved neuropathological lesions of Alzheimer's disease [23, 24].

Our study highlights the difficulties we have in characterizing the neuropsychological profile in these patients with a visual and verbal deficit. The available PCA criteria do not consider aphasia and amnesia as exclusion criteria, [2, 6, 25] but the differential diagnosis with AD is sometimes difficult because of their performances in visual and verbal memory assessement. PCA is less likely to be mistaken for AD if ecological hetero-evaluations of memory are perform in clinical routine to exclude amnestic syndrome.

Our results suggest that a systematic linguistic examination can be useful for interpreting neuropsychological performances in order not to misdiagnose PCA. It also helps us provide specific logopedic rehabilitation for patients [26].

Table 4 Neuroimaging findings in posterior cortical atrophy (PCA) and logopenic variant of primary progressive aphasia (LPPA)

		PCA (%)			LPPA (%)		
		Bilateral	Left	Right	Bilateral	Left	Right
MRI	Parietal atrophy	**14/16 (87 %)** Left > right: 3 Right > left: 3	**1/16 (6 %)**	1/16 (6 %)	**5/16 (31 %)** Left > right: 3 Right > left: 1[a]	0/16	0/16
	Temporo-parietal atrophy	**5/16 (31 %)**	**1/16 (6 %)**	2/16 (12 %)	0/16	11/16 (68 %)	1/16[a] (6 %)
	Hippocampal atrophy	5/16 (31 %)	3/16 (18 %)	3/16 (18 %)	0/16	2/16 (12 %)	0/16
SPECT	Parietal hypoperfusion	**14/16 (87 %)** Left > right: 4 Right > left: 6	**1/16 (6 %)**	1/16 (6 %)	5/16 (31 %) Left > right: 3 Right > left: 1 (left-handed)	0/16	0/16
	Hypoperfusion of the parieto-temporal junction	**12/16 (75 %)** Left > right: 2 Right > left: 4	0/16	0/16	0/16	15/16 (93 %)	1/16[a] (6 %)
	Hippocampal hypoperfusion	2/16 (12 %)	1/16 (6 %)	2/16 (12 %)	0/16	13/16 (81 %)	0/16
DAT-SPECT	Dopaminergic depletion	1/6 (16 %)	2/6 (33 %)	0/6	0/5	2/5 (40 %)	1/5 (20 %)

Abnormality in structures involved in logopenic syndrome of *PCA* patient are given in bold

[a] A left-handed *LPPA* patient with right abnormalities

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Conflicts of interest None of the authors has any conflict of interest to disclose.

Ethical standard We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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