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Letter to the Editor

Progression of oculomotor deficit in a patient with posterior cortical atrophy

Abbreviations:
AD
Topic:
Alzheimer's disease
PCA
Topic:
posterior cortical atrophy
NPT
Topic:
Neuropsychological testing
PET
Topic:
Positron Emission Tomography
FDG
Topic:
Fluorodeoxyglucose
SC
Topic:
Superior Colliculus
MRI
Topic:
Magnetic Resonance Imaging
PPC
Topic:
Posterior Parietal Cortex
SWJs
Topic:
Square Wave Jerks
CSF
Topic:
cerebrospinal fluid*Keywords:*
Alzheimer's disease
Dementia
Eye movements
Visual perception
Visual agnosia

Dear Editor,

High order visual abnormalities, including visual agnosia/inattention and visuo-spatial memory loss, are common features of Alzheimer's disease (AD), often associated with oculomotor defects [1]. Particularly, high-order visual changes are characteristic of posterior cortical atrophy (PCA), a progressive syndrome with decline in visuoperceptual, visuo-spatial, literacy, and praxic skills [2], whose neurodegeneration, affecting parietal-occipito-temporal cortices, is attributable to AD pathology in most cases, while less frequently Lewy-body disease or corticobasal syndrome are responsible [2]. While changes in saccadic performance have often been described in typical AD [3], oculomotor functioning and changes progression are less investigated in PCA, despite the prominent visual involvement. Longer saccadic latencies, reduced saccadic amplitude and frequent large intrusive saccades have been reported [3].

We describe here oculomotor characteristics at baseline and follow-up in a 78-years-old patient, presenting with 5-years history of progressive alexia and agraphia. At first visit, visual agnosia, prosopagnosia and visuo-spatial memory loss were evidenced. Neurological, neuro-ophthalmological evaluations, visual field testing and Visual Evoked Potentials were otherwise normal. Neuropsychological testing (NPT), brain Magnetic Resonance Imaging (MRI), Fluorodeoxyglucose (FDG) Positron Emission Tomography (PET) scan and cerebrospinal fluid (CSF) examination were performed (Table 1). PCA was diagnosed according to the Tang-Wai criteria [2]. Nine-month follow-up showed a worsening of visual symptoms; NPTs were globally deteriorated (Table 1).

Eye movements were recorded with an eye-tracker device (ASL504, Applied Science Laboratories, Bedford, MA, USA). Methods have been extensively described elsewhere [4]. We recorded horizontal (10°–18°) visually-guided saccades: pro-saccades with gap paradigm and antisaccades (40 trials for each amplitude), and fixation task (Fig. 1-legend) at baseline and 9-month follow-up. Saccadic parameters, rate of antisaccade errors with corrections, antisaccadic latencies and fixation abnormalities were compared with 7 healthy controls (mean 71-years-old, range 63–81 years) with Kruskal test for non-normal distributions (Fig. 1-legend). All subjects gave their informed consent; the study was approved by the Regional Ethics Committee.

During the first recording, the patient's pro-saccadic latency was significantly longer than controls; saccades were hypometric at 10° with significant reduction of peak velocity and increase of duration (Fig. 1A, Table 2). Other parameters were normal. The antisaccade error rate was higher than controls, with low corrections (Table 2). The patient's mean latencies of correctly executed antisaccades, erroneous prosaccades and corrective antisaccades were significantly higher (Fig. 1B, Table 2). SWJs were identified (frequency: 20/min) (Fig. 1C, Table 2).

At follow-up saccadic latency had not changed significantly; gain and peak velocity were reduced at 18°, with increased duration (Fig. 1D, Table 2); accuracy was significantly worsened at 18°. In the antisaccade task, the patient performed only erroneous pro-saccades (Fig. 1E), with unchanged latency. SWJs increased to 70/min, with a significant reduction in amplitude. Large intrusive saccades were identified (Fig. 1F, Table 2).

In summary, our patient showed: increase of saccade latencies, progressive inability to suppress reflexive saccades, fixation changes and reduced saccade velocity.

Progressive alexia and agraphia, associated with visual agnosia and prosopagnosia, indicate respectively an involvement of left angular gyrus, temporo-parietal junction and occipito-temporal cortex. In addition, increased latencies, reduced accuracy, of pro and anti-saccades and fixation changes may be the expression of a Posterior Parietal Cortex (PPC) contribution. In humans, PPC lesions are associated with increased latency of pro and antisaccades, greater variability of amplitude and hypometria [5]. The PPC computes target position based on stimuli localization and memory of its position, both of which were abnormal in our patient, and thus longer latency would indicate uncertainty in

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