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EDITORIAL

Neuro-ophthalmological findings in patients with acquired prosopagnosia

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Prosopagnosia (PA) is a Greek compound word — from *prosopon* (face) and *agnosia* (non-recognition) — and signifies face agnosia [1]. In contrast to the rare acquired form, congenital PA is quite common, with a prevalence of up to 2.5 % [2], and is almost likely hereditary.

Acquired prosopagnosia (PA) is caused by different cerebral diseases. Autopsy findings of subjects with acquired PA showed extensive cerebral damage to the occipitotemporal lobes.

Causes of acquired PA and neuro-ophthalmological signs

Frequency of pathologic findings of patients with PA

I compiled the data of 147 patients with PA from the international literature to identify the frequency of this lesion's etiology, of one-sidedness, of the visual field defects, of the color vision disturbances, of additional topographagnosia, of object agnosia, and of alexia (Table 1).

Discussion

I have highlighted the essential clinical and diagnostic features revealed via modern imaging methods in patients with acquired prosopagnosia. Of interest to ophthalmologists are visual acuity and visual field defects in patients with PA. Left-sided homonymous visual field defects were detected in a high percentage of PA patients. PA was found predominantly in conjunction with strokes, and less frequently in patients with head trauma, encephalitis, cerebral tumors, or other brain pathologies.

CT or MRI examinations revealed lesions causing PA overlap with lesions causing achromatopsia as a common region in the occipitotemporal cortex. The localized brain damage also extended into the visual pathway such as the optic radiation and the lateral geniculate nucleus, explaining the homonymous visual field defects in patients [3].

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Table 1 Our examinations (data compiled from the international literature of 147 patients with acquired PA*)

	Total no. of patients	Frequency of pathological findings: *n* (%)
Etiologies	147 patients from 103 publications Mean age: 49.6 years, calculated from 143 patients (range:14-month-old child to 89 years)	Stroke: 73 (49.65 %), head trauma: 26 (17.7 %), encephalitis and meningitis: 14 (9.5 %), cerebral tumors (glioblastoma or oligodendroglioma): 6 (4.1 %), focal chronic progressive cerebral atrophy: 5 (3.4 %), brain resection in epilepsy (lobectomy): 4 (2.7 %), arterio-venous malformation: 3 (2 %), carbon monoxide intoxication: 3 (2 %), cardiopulmonary arrest: 2 (1.4 %), occipito-temporal hematoma: 2 (1.4 %), right temporal lobe abscess: 2 (1.4 %), neurosurgery of colloid cyst: 1 (0.7 %), ruptured cerebral aneurysm: 1 (0.7 %), aneurysm of right posterior cerebral artery: 1 (0.7 %), ligation of pseudoaneurysm, of left vertebral artery: 1 (0.7 %), occipital gun shot: 1 (0.7 %), birth anoxia: 1 (0.7 %), acute arterial hypotensive episode: 1 (0.7 %).
Bilateral cerebral lesions	133	74 patients (55.64 %)
Unilateral cerebral lesions	133	Total 55 patients (41.35 %); right-sided lesions: 50 (90.9 %), left-sided lesions: 5 (9.1 %) Lesions not mentioned: 4 patients
Visual field defects	147	Patients with visual field defects: 108 (73.5 %) No field defect: 23 (15.6 %) Visual fields not mentioned: 16 (10.9 %)
Unilateral field defects	108	Right visual field defect: 20 (18.5 %) Left visual field defect: 72 (66.7 %) Additional defects (scotomas, tunnel vision, double hemianopia): 16 (14.8 %)
Color vision defects (achromatopsia or dyschromatopsia)	147	44 (29.9 %)
Topographagnosia	147	43 (29.25 %)
Objectagnosia	147	17 (11.6 %)
Alexia	147	Total 11 (7.5 %); alexia: 7, alexia without agraphia: 4

*In this compilation, I used the data from case reports (publications dealing with the disease of usually one patient, rarely with two or three patients) from the international literature

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