INTRODUCTION

This paper presents a transient ischemic attack (TIA) in the vertebral-basilar sector, manifested by three categories of signs: visual, writing–reading language, and color perception.

The particularity of the case lies in the order of the normalization of the impaired functions. Usually, the first to normalize are the visual elementary functions (the visual field), followed by the ability to write and read and finally by the color perception (6).

The patient D.S., aged 58, was brought urgently at 7:40 in the morning with a central anamnesis, being sent to the Neurology Clinic with this diagnosis by the emergency service of the Ophthalmology Clinic.

On admission, the patient was restless and anxious, suggesting a psychogenic blindness rather than a cortical blindness. The assumption for a cortical blindness was very plausible, suggested by the absence of the fright blink reflexes and by the pathological antecedents of the patient.

The patient has a long history of hypertension, showing at the admittance AT = 190/115 mmHg, being recently diagnosed with ischemic heart disease.

About 10 minutes after his arrival in the hospital emergency room, the patient declared textually: “I begin to see in front of me but I can’t see anything neither to the right nor to the left, neither down nor up”. The new examination of the fright blink reflex showed its presence, increasing the probability of a psychogenic blindness.

In the neurology department, the patient declared that he began seeing well to the left but that he saw nothing to the right. About 30 minutes after his admittance, a brief neurological examination was carried out, stating the presence of a right homonymous hemianopsia, aspect confirming the onset by cortical blindness. (20)

Three hours from the admittance, a detailed neurological examination was carried out; the patient was in a very good overall condition, he was calm and cooperative. The patient repeated that he sees nothing to
the right and when he looks to the left, although the objects appear clearly outlined, the colors are whitish, sometimes dirty-whitish. The examination confirmed a right homonymous hemianopsia.

With the patient having a right hand preference, a Pötzl syndrome was suspected, objectively existing hemianopsia and subjective disorders of color perception. (10)

The reading and writing ability examination was performed. The reading was impossible. The writing after dictation resulted in illegible literal forms. The normality of the oral language imposed the diagnosis of alexia with agraphia. (8) The examination of colors emphasized the confusion to name some of the colors he had seen. Thus, the naming of the colors red, yellow and green was correct, while blue was mistaken with violet and vice versa, and orange was randomly named red, respectively yellow. When the colors violet, blue, green, yellow and orange were presented to him, the patient showed correctly the color requested by the examiner. He separated correctly the pure blue samples from the blue-green ones, as well as the straw-yellow from the khaki yellow samples. (3)

The patient became restless and when he was asked about the reason of such behavior, he declared that he did not understand why he cannot read or write. After 5 hours from his admittance, he asked for paper and pencil, trying to write. He succeeded to write deformed with small letters: „I am an engineer, my wife is laboratory-assistant, I have a son, student at the faculty of constructions.” After writing this down, he said: „Again one cannot understood what I wrote.” He was surprised when the examiner red the text he spontaneously wrote, without concomitant reading, (after the examiner’s suggestion). The patient was given the book „Physiopathology of the nervous system having written at the bottom of the cover only „the nervous system” with capital letters of about 3 cm. He was unable to read this segment of the thesis.

At the examiner’s request, he followed with his finger the outline of the letters, naming them successively. After repeating the mioarthrokinetic literal lexia, he managed to pronounce „the nervous system”: the word blindness was differentiated from the central blindness even before the differentiation of the agnosia from aphasia, which is still difficult in the practice nowadays. (7)

Seven hours from the admittance, his writing became fluent, confirming the Pötzl syndrome diagnosis, represented by the triad: right homonymous hemianopsia, writing and reading agnosia, color agnosia. The patient said that at 4 p.m. the word comprehension became possible and his right eyesight improved, aspects confirmed at the evening visit at 6:30 p.m. He also declared to sees the colors, which lost their whitish shade. The color agnosia evaluation was repeated and the color perception was found normal. (2)

The MRI examination carried out two days later, showed a normal aspect, except a mild diffuse cortical atrophy.

RCAT showed a high left side retinal hypotension (a difference of minus 25 mmHg at the right side) suggesting most probably a carotidian stenosis upstream the eye artery emergence. (18)

Fig.1 Doppler ultrasonography of the carotidian artery
The Doppler ultrasonography examination confirmed the diagnosis of left carotid artery stenosis in the pre-retinal segment. (fig. 1)

For the moment, the patient refuses the angiographic investigation aimed to state the necessity of a surgical intervention.

DISCUSSIONS

This case is a typical Pötzl syndrome, the expression of a vertebral-carotidian hemodynamic embolism caused by a carotidian stenosis. This was described by Schott in 1961, (22) being the second syndrome of this type after the description of the embolism („steal”) subclavian syndrome. The acquired embolism syndrome was described initially, and only later the syndrome due to a congenital anomaly. (1) Later, (1965) it was described the steal syndrome between the anterior cerebral arteries through the anterior communicating artery. Usually, the vertebro-carotidian embolism manifests clinically by vertebro-cerebral reactions, by the ischemia of the cerebral trunk and of the cerebellum. (22)

The cortical manifestation of the syndrome might suggest, despite the carotidian stenosis confirmed both by the Doppler and ophthalmic pressure (RCAT) examinations, a vertebral embolism from a atheromatous plaque at point where the vertebral artery emerges from the subclavian artery (4) or even by an embolism from an upper segment of the vertebral artery or from the basilar artery (17). The onset by cortical blindness refutes such supposition, confirming that ischemia in this situation was initially bilateral. A thrombotic TIA can be determined only by a small-sized white embolus, and a junction embolus of the basilar artery is by definition a large red embolus, resulting in a cerebral attack with persistent cortical blindness. (13)

The existence of agraphia might suggest a double embolization of cortical and vertebro-basilar origin, with affection of the angular gyrus and the existence of alexia with agraphia. (5) The explanation called for in this case is nevertheless of neuropsychologic type. Agraphia was in this case less manifest than alexia and diminished within 5 hours from the onset, while the other two symptoms lasted about 9 hours. In the case of an angular gyrus syndrome, the agraphia syndrome is dominating, being persistent within the Gerstmann syndrome, and alexia – if present - is of subangular nature (Greenblatt central pure alexia), with transitory character. (6) Here agraphia occurs as a consequence of the abolition of the visual control upon the writing and of the delay in the replacement of this control by the kinestezic one.

The occurrence of the Schott embolism syndrome (21) at the left side might be explained by the asymmetry of the communicating arteries (9), (13), the right side communicating artery being hypoplastic, or by the calibre similarity of the left communicating artery with the first segment of the left posterior cerebral artery (Zolog, 1994)

In 1993, Ross et al. (12) by MR-angiography of the two actually existing variants, of the direction of the flow through the posterior communicating arteries, ascertained that in 92% of the healthy subjects, the flow is directed anterior-posterior from the internal carotid artery towards the posterior cerebral artery, and the internal carotid artery contributes effectively to the flow of the posterior posterior cerebral artery. In 8% of the cases, the flow is directed posterior-anterior, the vertebro-basilar system contributing to the establishment of the sylvian flow. It seems that this cerebral-anatomical localization is the only one where the flow may show individual variations in the flow direction. These are ultimately dictated by the size ratio between the carotid arteries and the basilar artery. Previously, the reversal of the flow direction in the ophthalmic artery had been ascertained, but this is a physiopathological phenomenon (without visual functional consequences), consecutively to the preophthalmic carotidian stenosis. (15)

CONCLUSIONS

The fast symptomatological changes, within minutes or tenths of minutes from the clinical onset of a cerebral ischemia (TIA or IA in progression) are suggestive for a determination syndrome. (19)

The embolism syndromes assume anatomical-functional specific features which fall among congenital anomalies (and variants). (20)
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