457 - VERY LATE-ONSET SCHIZOPHRENIA-LIKE PSYCHOSIS… A DIAGNOSTIC DILEMMA…

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Background: The nosology and etiological underpinnings of very late-onset schizophrenia-like psychosis (VLOSLP) have remained controversial. This case report highlights its diagnostic complexity.

Case report: A 64-year-old woman, with a previous history of hypertension, diabetes, mild cognitive decline, right grade-4 hemiparesis as sequelae of an ischemic-stroke (three years before), started persecutory and partition delusions. After six months, the delusions were accompanied by complex visual hallucinations (scenic, lilliputian and holocampine), elementary auditory, tactile, olfactory, and gustatory hallucinations, causing a profound daily life impact, consequently she was hospitalized. Neither negative symptoms nor formal thought disorders were present. Electroencephalography and laboratorial evaluations were unremarkable (including thyroid function, folic acid, cyanocobalamin, infectious serologies and anti-gliadin/transglutaminase antibodies). Neuroimaging displayed subcortical microvascular lesions in the left centrum semiovale, bilateral thalamic and basal ganglia lacunes. Neuropsychological examination revealed mild/moderate impairment in working-memory, sustained-attention, executive functions, abstract thinking, and visuospatial abilities. Mini-mental state examination (MMSE) scored 20/30. Clozapine was started. As psychotic symptoms ameliorated cognitive deficits also improved (MMSE score: 25/30). She was discharged with residual symptoms.

Discussion: Late-life psychosis implies a thorough investigation, bringing about challenges in diagnosis. Several medical causes, including neuroinflammatory/immunologic, were ruled out. This two-stage progression, with partition delusions and multimodal hallucinations, in the absence of formal thought disorder and negative symptoms is typical of VLOSP. It is arguable to ascribe our patient's psychosis to a previous vascular dementia or to VLOSLP. Almost half of VLOSLP patients may develop dementia. It is still debatable whether this propensity is a true characteristic of VLOPSL or reflects an initial misdiagnosis. Some neuropathological studies suggest a restricted limbic tauopathy underlying VLOSP. Notwithstanding, cognitive impairment is common in VLOSLP, including in those patients who do not develop dementia. Neuroimaging studies evidence that lacunar infarction in the basal ganglia alongside chronic white matter small vessels ischemic disease, may underlie the pathophysiology of psychosis via a disruption in the frontal-subcortical pathways. Nevertheless, cases of post-stroke psychosis usually resolve in few months. In conclusion, the neurobiological underpinnings of VLOSLP are complex and multifaceted. More systematized studies using biomarkers and neuroimaging are needed so clinicians can perform a more accurate diagnosis of VLOSLP.

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