Noah Syndrome (animal hoarding) in a Pseudo-Ornithologist With Secondary Schizophrenia

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Noah syndrome is a variant of Diogenes syndrome that presents as animal hoarding in squalid conditions, with well-known predisposing factors such as loneliness, psychosocial stress, and diverse psychopathology, due to neurologic and/or psychiatric conditions.[1] When treating patients with Noah syndrome, it seems relevant to prepare community interventions based on collaborative and solution-focused approaches; however, there is a need for more research on further successful diagnosis, intervention, and follow-up strategies.[2] Here, a case is presented of a patient with Noah syndrome, secondary to mental retardation complicated by organic psychosis, misdiagnosed as schizophrenia for 25 years.

Case Report

A 56-year-old Portuguese man, living alone in a poor rural area, was admitted for aggressive behavior, resisting any help provided by his newly assigned primary caretaker. He had been found in his house, which was in squalid condition, with very poor hygiene and in extreme self-neglect surrounded by plastic bottles full of urine and a collection of moribund birds in soiled birdcages. He had auditory hallucinations that he attributed to distant neighbors, interpreted with persecutory delusions, usually insulting him because of his obviously unkept home. He proudly assumed to be a bird collector (*passarinheiro*). But although he spent all his income buying new animals every month, he could not, paradoxically, take them to bird contests or even care for them as a true birdman or ornithologist would do. The situation had worsened in the last 3 years after the death of his mother, an overwhelmed caregiver who had helped him deal with his animal hoarding issue.

According to his clinical records, he had 9 psychiatric admissions over the past 25 years, with the official diagnosis of schizophrenia. However, information from the records was more compatible with mental retardation complicated by psychosis not otherwise specified.

The patient had previously been on various typical and atypical antipsychotics, anticonvulsants, and benzodiazepines. Full recovery was never achieved because of an insufficient gain in insight and poor adherence to clinical treatments and social support. During infancy and adolescence, he failed to complete primary school due to undisclosed serious learning disabilities. He also had untreated obesity, dyslipidemia, arterial hypertension, benign prostatic hyperplasia, and seborrheic dermatitis. There were no known psychiatric illnesses in his family. Results of blood and urine tests, electrocardiography, chest radiograph, and electroencephalography were found in his old and recent clinical records, all with no important findings. However, for the first time in his life, he underwent a brain computed tomography scan, which revealed asymmetric frontal and temporal cortical atrophy, more evident on the left side, with unspecific calcification of the anterior third of falx cerebri. His first-ever neuropsychological assessment documented cognitive deficit, with a maximum value of 22/30 on the Mini-Mental State Examination,[3] and an important and predominant impairment of the prefrontal cortex functions. The patient received new diagnoses of disorder of intellectual development and secondary psychotic syndrome (*ICD-11* codes: 6A00 and 6E61, respectively). He was started on olanzapine 20 mg every night. After almost 6 months of admission on the acute psychiatric ward, he achieved a full remission of his psychotic symptoms. But, due to little improvement in activities of daily living, the patient was discharged to a nursing home to guarantee a reduction in animal hoarding numbers, ongoing monitoring, education, and continuous psychosocial support.

Discussion

When treating a patient with schizophrenia-like psychosis, clinicians should take extra care when making the differential diagnosis, as organic psychosis is many times overlooked or underestimated. In my experience, organic psychosis can represent up to one-fourth of what once seemed to be true schizophrenia. Caution is always recommended when diagnosing schizophrenia in psychotic patients, independent of their acute or chronic condition.[4]

Many imitators affecting the central nervous system can mimic schizophrenia, thus diagnoses of secondary schizophrenia or pseudoschizophrenia[5] should always be discarded before assuming primary schizophrenia. Clinicians should be aware of schizophrenia as one of the greatest imitated syndromes of medicine.[6]

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