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- Provide complete assessment of patients with psychosis to better distinguish between primary schizophrenia and other diagnoses

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Organic Psychosis Causing Secondary Schizophrenia in One-Fourth of a Cohort of 200 Patients Previously Diagnosed With Primary Schizophrenia

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ABSTRACT

Objective: To identify the prevalence of secondary schizophrenia (organic psychosis causing a schizophrenia-like syndrome) in patients with a prior diagnosis of schizophrenia presenting to Centro Hospitalar Psiquiátrico de Lisboa, Lisbon, Portugal.

Methods: Two hundred files were retrospectively assessed through paper and electronic records of patients admitted to the hospital with an *International Classification of Diseases, Ninth Edition* diagnosis of schizophrenia (eg, code 295.x) in a 1-year time span (September 1, 2015–September 1, 2016).

Results: One-fourth of patients (n = 50, 25%) received a new organic psychosis (secondary schizophrenia) diagnosis: epilepsy-related schizophrenia-like psychosis (9.5%), dementia-related schizophrenia-like psychosis (9.5%), brain mass (3.5%), stroke-related schizophrenia-like psychosis (2.0%), and encephalitis-related schizophrenia-like psychosis (0.5%). Among patients with organic psychosis (secondary schizophrenia), the mean delay until correct diagnosis was 12 years.

Conclusions: The most striking feature of this study was the high prevalence of incorrect diagnosis of schizophrenia, with patients not receiving the minimum correct assessment before that diagnosis, resulting in negative consequences. Caution is recommended when diagnosing severely psychotic patients independent of their acute or chronic condition.

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Since 1942, thousands of psychotic patients from southern Portugal have presented to Lisbon's Hospital Júlio de Matos, even decades before the psychiatry wards opened in the general hospitals. Although functioning as an asylum, the hospital's vibrant clinical and scientific activities garnered some international attention, especially in its early years, with interesting articles on frontal leucotomy[1] and holodysphrenia.[2]

In 2013, there was a massive reorganization within the hospital, renamed Centro Hospitalar Psiquiátrico de Lisboa, and patients were now admitted to different pavilions according to their nosologic diagnosis. At that time, only inpatients with an

Clinical Points

- Many patients are incorrectly diagnosed with primary schizophrenia.
- Secondary schizophrenia is highly underestimated in clinical practice.
- Clinicians should be aware that organic psychosis may cause secondary schizophrenia.

International Classification of Diseases, Ninth Edition (*ICD-9*) schizophrenia diagnosis (eg, 295.x) were admitted to the psychiatry ward at pavilion no. 29. Naturally, my colleagues and I started focusing on this population in our outpatient clinic, both at the hospital and in the community setting, while continuing research on schizophrenia-related issues.[3–5] After review of the literature, the most recent meta-guidelines[6] were adapted to standardize an approach to our patients with schizophrenia, along with the official national Norms for Clinical Orientation.[7,8] Some of my team members were involved in the European Portuguese translation of the schizophrenia chapter of the *DSM-5*. Additionally, my colleagues and I have conducted research in the area of schizophrenia biomarker candidates.[9–12]

After 3 years of practice under the new model of care, my team members and I were interested in the characteristics of the schizophrenic population treated at Centro Hospitalar Psiquiátrico de Lisboa, specifically regarding some of the issues brought to light by the aforementioned meta-guidelines.[6] Of particular interest was the rate of secondary schizophrenia among these patients.

Secondary schizophrenia is an underestimated concept rarely used in the international scientific literature since 1968,[13] with no more than 10 results found in PubMed. However, it is a useful concept that could be applied to every condition that mimics schizophrenia or a so-called organic psychosis as codified in the *ICD* (Table 1).

The term *secondary schizophrenia* has rarely been used in the past few decades[14]; however, some authors have provided insights that psychiatrists should be aware of with regard to the concept:

- It may have an environmental etiology such as occurs in brain injury.[15]
- It arises in the presence of systemic physical illness, and psychopathology is not helpful in distinguishing secondary from primary schizophrenia.[16]
- It is a disparate range of brain disorders that can, uncommonly, give rise to schizophrenia-like symptomatology.[17]
- It is often not considered in the diagnostic workup of patients with schizophrenia-like illnesss.[18]
- It includes many different clinical conditions that may cause schizophrenia-like psychosis such as drug intoxication, temporal lobe epilepsy, traumatic brain injury, cerebrovascular disease, storage disorders, mitochondrial disorders, leukodystrophies, normal pressure hydrocephalus, brain tumors, demyelinating disease, infections, velocardiofacial syndrome, Alzheimer's disease, Prader-Willi syndrome, Friedreich ataxia, Wilson disease, Huntington's disease, and Fahr syndrome.[19]

Table 1. Organic Psychosis Codes of Conditions Causing Secondary Schizophrenia

Classification	Code	Description
ICD-9, 1975	294.8	Other persistent mental disorders due to conditions classified elsewhere
ICD-10, 1992	F06.2	Organic delusional (schizophrenia-like) disorder
ICD-11, 2019	6E61	Secondary psychotic syndrome

Abbreviations: *ICD* = *International Classification of Diseases*.

Table 2. Patients With Organic Psychosis Causing Secondary Schizophrenia

Diagnosis	n	%	Delay Until Diagnosis (y)
Epilepsy	19	9.5	14
Dementia	19	9.5	12
Brain mass	7	3.5	9
Stroke sequelae	4	2.0	7
Encephalitis	1	0.5	6

The main objective of this study was to identify the prevalence of secondary schizophrenia (organic psychosis causing a schizophrenia-like syndrome) in patients with a diagnosis of schizophrenia presenting to Centro Hospitalar Psiquiátrico de Lisboa.

METHODS

Clinical records, both paper and electronic, of all patients with an *ICD-9* diagnosis of schizophrenia (inpatients and outpatients) treated by my team in a 1-year time span (September 1, 2015–September 1, 2016) were examined. All of these patients had been, at some point (before or after admission), assessed by the following diagnostic protocol: computed tomography (CT) brain scan, electroencephalography (EEG), routine blood work, urine drug tests, and personality and intelligence evaluation plus neuropsychological assessment.

Data were analyzed with SPSS version 25 (IBM Corp, Armonk, New York) for possible correlations between different variables to answer the following research question: What is the prevalence of organic psychosis (secondary schizophrenia) among our patients with a previous diagnosis of schizophrenia?

RESULTS

In 1 year (during 2016), 200 patients with a presumed diagnosis of *ICD-9* schizophrenia (eg, 295.x) were admitted to the hospital. Most of the patients were male (59.5%). The mean age of the patients was 47 years; the youngest was 25 years and the oldest was 93 years.

After finalizing the diagnostic protocol, 50 patients (25%) received a new *ICD-9* organic psychosis (secondary schizophrenia) diagnosis (293.x), mostly due to findings on CT brain scan (40 patients, 20%) and EEG (25 patients, 12.5%). This subsample (Table 2) included patients with a classic differential diagnosis such as brain mass or stroke sequelae but also some other neurologic pathologies of interest (some already published as case reports), such as frontal meningioma,[20] epileptic psychosis,[21] Huntington's disease chorea,[22] Dandy-Walker variant syndrome,[23] autism spectrum disorder with splinter skills,[24] and anti–*N*-methyl-D-aspartate (NMDA) receptor encephalitis.[25] Careful review of clinical records revealed a mean delay until correct diagnosis of 12 years.

DISCUSSION

Fifty patients presented to the hospital with an incorrect diagnosis of schizophrenia, which highlights the importance of following recommended protocols during initial medical workup of first-episode psychosis,[26] especially regarding the exclusion of organic brain dysfunction that might be causing or contributing to the physiopathology of psychosis.

Unfortunately, most of these patients were already psychotic for a considerable amount of time before the first CT brain scans and EEGs were performed. This delay in diagnostic examination occurred mostly because they were seen by other clinicians who assumed the diagnosis of schizophrenia, sometimes disregarding clinical history (eg, seizures or memory impairment) and bypassing proper exclusion of neurologic lesion or dysfunction. Although the temporal sequence criteria of causality is not completely certain, these patients did indeed have organic psychosis, as their neurologic lesions or dysfunction were in areas easily recognizable as having psychotogenic potential. The following sections discuss the subgroups of the 50 patients to explain the probable criteria of causality.

Epilepsy-Related Schizophrenia-Like Psychosis

Nineteen patients had epileptic psychosis: 4 cases of frontal epilepsy with secondary generalization affecting the temporal lobes and 15 cases of temporal epilepsy with predominance of left temporal lobe dysfunction (7 in the left temporal lobe, 4 in the right temporal lobe, and 4 with bilateral temporal EEG anomalies). All of these patients had a history of at least 1 episode of epileptic seizures recorded in their clinical files. Their mean age was 49 years, with a mean delay before the correct diagnosis of 13 years.

Epileptic patients have a well-known increased risk of psychosis,[27] especially those with temporal epilepsy[28]; however, psychotic symptoms resembling schizophrenia (paranoid delusions and auditory hallucinations) may not be exclusively associated with temporal lobe epilepsy.[29] On the other hand, it is well known that epilepsy may be associated with psychotic disorders, but it is less widely recognized that relapsing psychotic phenomena may be the first and only symptom of epilepsy.[30] As the presence of many common features did not fully support the theory that psychosis after epilepsy and epilepsy after psychosis were 2 distinctly different entities, a necessary reconceptualization of psychosis in epilepsy is suggested.[31] All of our patients with epileptic psychosis were sent for neurologic evaluation, and most were started on anticonvulsants in addition to standard treatment with antipsychotics.

Dementia-Related Schizophrenia-Like Psychosis

In the 19 patients with dementia-related psychosis, 8 had frontal and temporal lobe atrophy (frontotemporal dementia resembling Pick's disease), 7 had diffuse ischemic white matter disease (vascular dementia resembling Binswanger disease), 3 had bilateral basal ganglia calcification (resembling Fahr syndrome), and 1 had diffuse brain atrophy (with genetic testing positive for Huntington's disease). Their mean age was 64 years, with a mean delay before the correct diagnosis of 12 years.

Dementia may occur with a wide variety of symptoms. It is estimated that behavioral and psychological symptoms affect up to 90% of all dementia patients over the course of their illness, irrespective of its subtype, and may even present as schizophrenia-like symptoms such as delusions or hallucinations.[32] Although dementia in schizophrenia seems to be a real entity with a neuropsychological signature similar to that of frontotemporal dementia, structural imaging abnormalities are not characteristic of that particular cognitive deterioration,[33] and clinicians should be aware of the real risk of frontotemporal dementia mimicking schizophrenia, even in young patients.[34] Vascular dementia should also be considered as an important cause of psychosis.[35] Last but not least, other rare causes of cognitive decline, such as Fahr syndrome[36,37] or Huntington's disease chorea,[38] should also be taken into account in the differential diagnosis of schizophrenia. All of the patients with dementia-related psychosis were sent for neurologic evaluation, and most were put on antidementia drugs in addition to standard treatment with antipsychotics.

Brain Mass–Related Schizophrenia-Like Psychosis

Seven patients had a brain mass with various locations: 2 cysts affecting the pineal gland, 2 meningiomas in the falx cerebri affecting the corpus callosum white matter, and 2 cysts of the sublenticular area and 1 cyst of the right temporal lobe, all 3 affecting the auditory pathways. The mean age of these patients was 38 years, with a mean delay before the correct diagnosis of 9 years.

Tumors occurring in various parts of the brain have been associated with psychotic illness, and resolution of psychotic symptoms has been documented after surgical treatment in several instances.[39] This association might be especially true for areas such as the pineal gland,[40] the corpus callosum,[41–44] the sublenticular auditory pathways,[45,46] and the temporal lobe.[47,48] Although all of the patients were referred to neurosurgical assessment, none were candidates for brain surgery, and antipsychotics were kept as the main treatment after discharge.

Stroke-Related Schizophrenia-Like Psychosis

Four patients had stroke-related psychosis, all with white matter ischemic disease in various locations of the left side of the brain: 2 in the frontal lobe, 1 in the temporal lobe, and 1 in the pons. Their mean age was 39 years, with a mean delay before the correct diagnosis of 7 years.

Stroke may result in psychotic symptoms with varied clinical characteristics depending on the location of the stroke within the brain,[49] even in patients with silent vascular brain lesions.[50] The 4 patients with psychosis secondary to stroke were sent for neurologic evaluation, and most were put on a stroke relapse prophylaxis protocol in addition to standard treatment with antipsychotics.

Anti-NMDA Receptor Encephalitis–Related Schizophrenia-Like Psychosis

One patient (aged 34 years) had anti-NMDA receptor encephalitis, a diagnosis that was officially categorized in 2007 and since then has been recognized as a neuropsychiatric entity secondary to autoimmune response primarily against NMDA receptors.[51] Although new-onset acute prominent psychotic syndromes in patients with NMDA receptor encephalitis have been well documented, there is a lack of case studies on differential diagnosis and treatment of anti-NMDA receptor encephalitis after a long-term diagnostic history of functional psychotic disorders.[52] Recent guidelines[53] may help navigate through the differential diagnosis. This unique patient with NMDA receptor encephalitis had been labeled as schizophrenic for 6 years before the correct diagnosis was made. Only after a severe catatonic episode was he finally sent to an internal medicine autoimmune outpatient consultation for immunomodulatory treatment in conjunction with standard treatment with antipsychotics. Catatonia is a very particular syndrome that clinicians should investigate thoroughly so as to exclude an organic cause.[54]

CONCLUSION

Efforts to find the correct diagnosis for patients with organic psychosis misdiagnosed as schizophrenia were successful. Nevertheless, clinicians should be cautious in the emergency department, as that is where most of the incorrect diagnoses were made.

The main limitation of this study was the small sample size, which was studied in a transversal and retrospective design. Also, it would be beneficial to look at some of the social factors such as, for example, many patients from the sample were living as homeless in Lisbon, which is the largest city in Portugal.

There are also many limitations with the *ICD-9* (eg, schizoaffective disorder coded as a schizophrenia subtype). Unfortunately, in Portugal, the Ministry of Health still uses the *ICD-9* for diagnosis codification purposes, so there is no option other than to conduct research with this classification. Adoption of the *ICD-10* (or even *ICD-11*) nosology may generate more accurate diagnoses, yielding better treatment for patients and thus better datasets for future research. It is hoped that this work will increase awareness among clinicians that organic psychosis may cause secondary schizophrenias, which unfortunately is underestimated in the international scientific community.

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POSTTEST

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1. **A 69-year-old man was assessed at a routine appointment for antipsychotic prescription renewal for treatment of chronic disorganized schizophrenia. He had been admitted to a psychiatric hospital 33 years ago, in a different country, following a probable psychotic episode with persecutory delusions and auditory hallucinations, but you have no access to clinical records. According to the article, what action should you take for the best follow-up?**
   a. Renew the antipsychotic prescription for the patient.
   b. Ask the patient to provide a copy of previous clinical records.
   c. Order brain imaging, among other routine tests.
   d. Ask the patient about the presence of first-rank symptoms.

2. **A 44-year-old man was admitted to the psychiatric ward of a general hospital for aggressive behavior and paranoid delusions of poisoning, probably secondary to cacosmia, and auditory hallucinations. His extensive clinical records revealed many admissions over the last 22 years, following various episodes of what could have been an undifferentiated schizophrenia. According to the article, what action should you take for the best follow-up?**
   a. Ask the patient if he had any previous history of seizures.
   b. Order electroencephalography, among other diagnostic tests.
   c. Consult with Neurology before initiating treatment with anticonvulsants.
   d. Consult with Neurology after initiating treatment with anticonvulsants.

3. **A 22-year-old woman has been admitted with catalepsy and waxy flexibility, highly suggestive of what was once described as catatonic schizophrenia. She was homeless, probably using various kinds of drugs for months, and largely immobile for several days before admission, neglecting hygiene and self-care. According to the article, what action should you take for the best differential diagnosis?**
   a. Consult with Neurology before sending the patient to a social worker.
   b. Consult with Neurology after initiating treatment with benzodiazepines.
   c. Order electroencephalography and urine drug screen, among other diagnostic tests.
   d. Order lumbar puncture and urine drug screen, among other routine diagnostic tests.