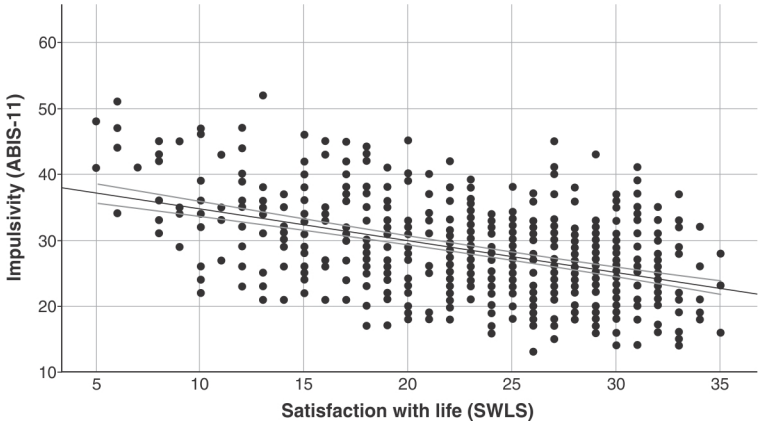

Figure 1 Pearson correlation between impulsivity and satisfaction with life (r = -0.469, p < 0.001). ABIS-11 = Abbreviated Barratt Impulsiveness Scale 11; SWLS = Satisfaction with Life Scale.

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Catatonia – not only a schizophrenia subtype: a case report of Wilson's disease

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Catatonia is a psychomotor syndrome first described by Kahlbaum, who acknowledged psychiatric, neurologic, and general medical etiologies.[1] Under the influence of Kraepelin, catatonia was considered a schizophrenia subtype for many years. The DSM-5 described catatonia across the manual, regardless of associated conditions.[2] Wilson's disease (WD) is a genetic condition that causes copper to accumulate in several organs. Approximately 20% of patients present with behavioral symptoms at disease onset. Psychotic phenomena and catatonia are rare,[3,4] but may occur due to copper accumulating in the brain.[5-7]

A 24-year-old single man was referred to the psychiatric ward of the hospital of Universidade Federal de Pernambuco due to reports of unusual and refractory depression. Two years before admission, the patient developed insomnia and refused food. During the first year, he still interacted with friends and family. After 6 months, he isolated himself, became mute, and developed self-injurious behavior. He was taken to a primary care service where escitalopram 20 mg/day, levomepromazine 25 mg/day, clonazepam 2 mg/day, and lithium carbonate 300 mg/day were prescribed. Twenty months after the initial onset of symptoms, he had significant impairment in activities of daily living, reduced mobility, repetitive masticatory movements, mutism, and posturing. The patient had no

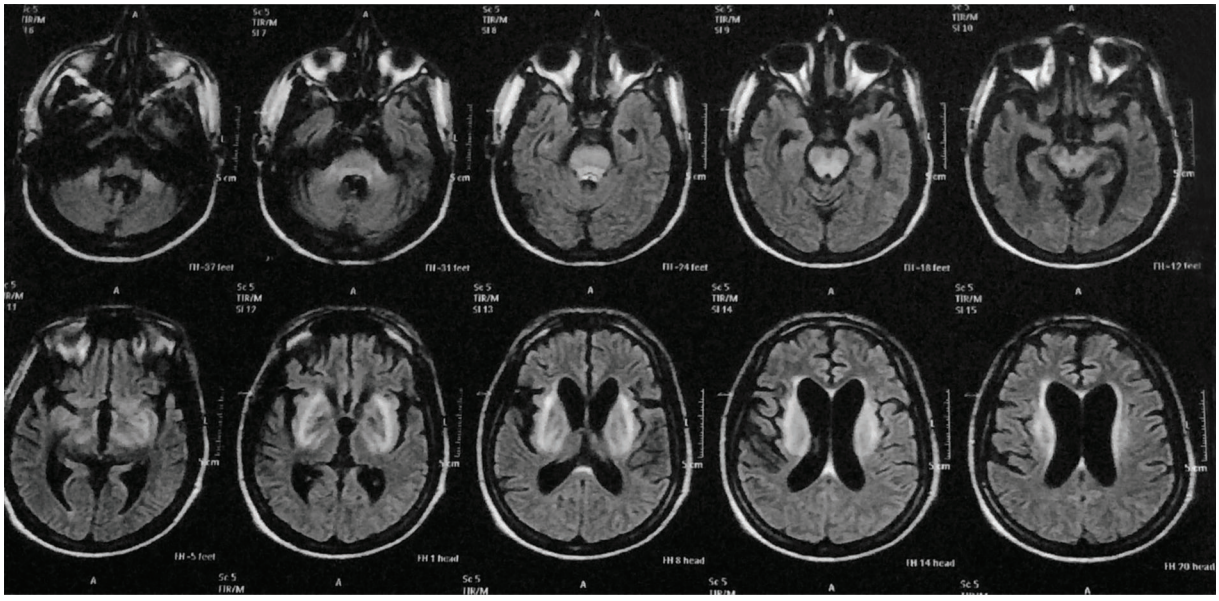

Figure 1 MRI of brain showing diffuse volumetric reduction with adaptive dilation of the ventricular system and bilateral hyperintensity in the basal ganglia on T2/FLAIR sequences, especially in the periphery of the putamen, alterations which are consistent with Wilson's disease.

previous history of psychiatric disorders or psychoactive substance use. During the admission evaluation, he remained mute, with his head down and fists clenched. When called, he glanced at the doctor. Upon request, he tried to stand up, but failed. He scored 27 on the Bush-Francis Catatonia Rating Scale (BFCRS), and 0 on the Katz scale.

In hospital, the patient was treated with lorazepam 12 mg/day (progressing over 2 weeks and continuing for up to 4 weeks) and olanzapine 5 mg/day, considering a possible psychotic etiology. Throughout the first 2 weeks of hospitalization, we noticed an improvement in psychomotor stiffness. The relevant lab results were: C-reactive protein, 10 mg/L; gamma-glutamyl transferase, 150.4 U/L; and ferritin, 465.9 ng/mL, with normal transaminases. Electroencephalography was unremarkable. Abdominal ultrasound showed a liver with heterogeneous texture and regeneration nodules. MRI of the brain (Figure 1) showed diffuse volumetric reduction and bilateral hyperintensities in the basal ganglia on T2/FLAIR sequences, especially in the periphery of the putamen. A 24-hour urine copper test (128.8 μg) and ceruloplasmin level (6.7 mg/dL) were requested, as well as an ophthalmologic evaluation, which revealed Kayser-Fleischer rings. Olanzapine was discontinued immediately upon diagnosis of WD. Due to a limited response to benzodiazepines after 4 weeks, bitemporal electroconvulsive therapy was performed, which did not show any benefit after six sessions. Specific treatment for WD with the use of copper chelation therapy agents was initiated.[5] After 6 months, the patient was able to sit, eat, and cooperate with caregivers. Nine months after starting chelation therapy, the patient scored 10 on the BFCRS and 2 on the Katz scale. In three previously described cases, chelation had mixed.[4,8,9]

It is important to remember that catatonia is not exclusively a subtype of schizophrenia, and may be a manifestation of other clinical conditions. Clinicians should be aware of this and institute treatment for the underlying cause of catatonia whenever one is identified.

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Suicide by jumping from high places in a Brazilian city: regional peculiarities as a determining factor of variation in suicide methods

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Considering the findings reported by Borges-Santos & Wang in their letter "Suicide by hanging in Brazil: challenges to mitigating its escalation," which reported a proportional increase of 51.1% in suicide by hanging over 20 years (1997-2017),[1] we hypothesized that, in some regions of Brazil, these rates would vary as a result of local specificities. Jumping from high places is a growing method of suicide in cities where suicide "hotspots" are popular, as we believe is the case of Natal (population 1,485,505), the capital of the northeastern state of Rio Grande do Norte, where the construction of a high-level bridge may have increased the number of suicides by jumping. We conducted a search of Brazilian Ministry of Health data (DATASUS), analyzing deaths from self-inflicted causes (ICD-10 codes X60-X84) in the city of Natal over the same 20-year period (1997-2017), and compared the number of suicides before and after the new bridge was opened (on November 21, 2007).

Overall, during this 20-year period, 488 people committed suicide in the city: 47.3% by hanging (the most observed method), followed by fire (13.7%) and self-inflicted gunshot wounds (10.4%); jumping accounted for only 8.4% of suicides. The second period (2007-2017) conserved hanging as the leading cause (52,6%), but already showed suicide by jumping in second, sharing the same proportion with lesions caused by fire (both representing 11,3% of suicides). In 1997, hanging represented 41.6% of all suicides, and no suicides by jumping occurred. In 2017, 52.9% of suicides were by hanging (a 27.1% increase), followed by jumping (now representing 21% of all completed suicides). This represents an important disparity to the data reported by Borges-Santos & Wang. While hanging remained as the leading method of suicide, we observed a downward trend in its proportion, while suicide by jumping steadily rose in popularity. Our data suggest that this occurred particularly after 2007, in a clear overlap with the opening of the bridge (Figure 1).

In different regions of the world, certain structures (such as bridges) have gained notoriety as "hotspots" for suicide by jumping. Jumps from such sites may increase the risk of copycat acts, considering their fatality rate, the distress or physical harm caused to bystanders, and prominent media coverage.[2] Recent studies reported evidence for prevention after the erection of barriers, with an overall reduction in deaths of 86% and little evidence of substitution by other jumping sites.[3,4] In the particular case of this Brazilian city, we observed a diverted trend in the increase of suicide methods, when compared with national data, which could point to regional variables – such as the presence of a suicide "hotspot" – potentially leading to copycat acts and thus increasing the rate of suicide by jumping.

These observations reinforce the need for an evaluation of suicide risk and its most common methods that takes regional characteristics into account. This could lead to a better comprehension of this phenomenon and improve the odds of developing more efficacious actions to prevent suicide from a public health standpoint.