ABSTRACT
The role of cerebellar pathology in psychiatric symptoms has long been recognized. Cerebellar pathology has been associated with obsessive-compulsive disorder pathophysiology, particularly with compulsive hoarding. Likewise, some cerebellum abnormalities have been described in schizophrenia, as well as in comorbidity between obsessive-compulsive disorder and schizophrenia. The authors report the case of a 32-year-old woman with obsessive-compulsive disorder and a cerebellum development variant in a family with a strong schizophrenia loading. This case emphasizes the probable role of the cerebellum in the pathophysiology of both obsessive-compulsive disorder and schizophrenia, and reconsiders the existence of a so called schizo-obsessive subtype of schizophrenia.

INTRODUCTION
The role of cerebellar pathology in psychiatric symptoms has long been recognized. In addition to its involvement in functions related to movement coordination, motor learning, and balance, cerebellum is likely implicated in higher-order mental functions, such as cognition, emotional regulation, and behavior. Traditional neuronal models of obsessive-compulsive disorder (OCD), involving dysfunction of frontal-subcortical circuits, have been supplemented by an increasing role of the cerebellum in its pathophysiology, and exploratory functional magnetic resonance imaging (fMRI) studies have unveiled its role in the neural mechanisms of decision making in compulsive hoarding.

Recent structural and functional brain imaging studies revealed that some symptoms in schizophrenia could be associated with different cerebellar abnormalities. Moreover, there is also one reported case of comorbidity between OCD and schizophrenia in a young male patient with a developmental cerebellar malformation (Dandy-Walker variant). Several authors have noted the overlap between the two disorders in the past 100 years. Nevertheless, the co-occurrence of obsessive-compulsive symptoms in psychotic illnesses has been shown to be more prevalent than was previously recognized, and the
neurobiologic dysfunction in both Cerebellum may be a common area of neurotransmitter systems and neuroanatomic systems.

**CASE REPORT**

V. F. was a 32-year-old caucasian female patient. By the age of 16, she began worrying about any object she or any family member might throw into the garbage basket, fearing that anything valuable could go along with it (apparently this fear was initially caused by the fact that her sister threw away objects). During the first couple of years after development of these thoughts, they were not very disturbing, and the patient managed to successfully complete high school and start working on her college degrees in Portuguese and English Literature. However, by little by little, she began verifying everything she or anyone in the family threw into the garbage basket and, ultimately, forbade everybody of doing so. The garbage, particularly empty containers, was meticulously inspected for anything valuable and then items were washed, folded, collected in plastic bags, and kept in her room, prompting her to sleep in the living room. The patient reported that it was the only way to get relief from the obsessive preoccupation of mistakenly throwing away valuable objects. The process of washing, according to family reports, involved strong preoccupations with symmetry. Moreover, other obsessive and compulsive symptoms were elicited. The patient repeatedly washed her hands and kitchen utensils, which she related to the poor hygienic habits of her father, diagnosed with alcohol abuse and dependence, who might have contaminated them. She also meticulously ordered decorative objects in the house, aligning them in a precise fashion. The patient became progressively secluded in her own house: the danger of any family member throwing away precious objects was too great to allow her to leave the house if anyone was left inside.

Even though the ruminations were ego-dystonic, there was only slight resistant effort concerning compulsions. Increasing symptom severity promoted intense social disability. She eventually quit college and became more and more isolated at home.

Diagnosis was reached only at the age of 23, when family members brought the patient to our outpatient clinic and she was observed for the first time by a medical team. On examination, the patient’s appearance was somewhat dishevelled, although she was able to maintain eye contact. Facial expression and mimic were normal. She had a general tone of uncooperativeness, which was sometimes punctuated with cynical remarks. Diverse obsessive thoughts and motor compulsions were evoked, though it seemed difficult for her to spontaneously verbalize them, since these symptoms remained a matter of discomfort and anxiety. No delusional thinking or errors of perception were detected. Mood was euthymic and the affective expression, although quite constricted, was not blunted. Insight to the disease was limited to recognition of undesired thoughts; judgement regarding the morbid nature of her condition and the need for treatment was partially impaired. Neurological examination was unremarkable, and we did not note any motor or posture signs suggestive of cerebellar dysfunction.

After some discussion of her case, the patient accepted referral to the inpatient unit, where she began treatment with fluvoxamine (100mg three times daily). Routine blood tests, a formal neuropsychological evaluation, and karyotype were normal. Brain magnetic resonance imaging (MRI) revealed a cerebellum variant caused by inferior right venous development abnormality. Because the patient’s sister was diagnosed with schizophrenia with some obsessive features, we also requested a brain MRI for the sister, which came back normal.

Our patient was discharged one month later, with partial improvement of obsessive thoughts and compulsions. Soon after discharge, however, the patient discontinued both her medication and the follow-up visits.

We admitted the patient to the inpatient unit four additional times under the Portuguese Mental Health Act and on account of the increasing severity of the hoarding behavior. Symptom clusters were almost the same as in first admission, although she progressively lost insight into her disease. In one episode, the patient presented with slight paranoid ideation. She was treated once with paroxetine (20mg twice daily), which did not worsen her paranoid symptoms, and twice with clomipramine (75mg three times daily) (once in association with flupenthixol, 20mg biweekly intramuscularly). After every hospitalization, the patient was discharged with partial improvement of obsessive thoughts and compulsions. After the first discharge, we referred her to our day hospital unit, which she refused to attend after four weeks. She was also referred for cognitive behavior therapy (CBT) but dropped out after two sessions. Once home, after each hospitalization, she would stop medication immediately and discontinue follow-up visits within the first month. The patient once explained that the reason she did not adhere to her medication was due to her fear of mismanaging it.

Interesting to note, the patient’s mother, though permanently refusing psychiatric care, had a likely diagnosis of schizophrenia without prominent negative symptoms. She exhibited continuous morbid behavior with suspicions about the quality of the food (frequently throwing it away) and of the daughter’s medications (telling
her not to go to follow-up appointments and to stop pharmacological treatment. We did not identify classical depressive symptoms in the patient throughout the longitudinal follow-up visits. However, she told us she felt profoundly unhappy about her condition.

During her last admission, we decided to initiate treatment with intravenous clomipramine, building to 75mg in association with risperidone 2mg once daily. During a 10-day period, we noticed a striking transition from generally hostile behavior to one of cooperation with the treatment and remarkable social interaction with other patients and professionals, which was at odds with previous inpatient behavior. However, when we switched her to oral clomipramine (titrated up to 225mg once daily) this outstanding effect was lost, and the patient returned to her baseline behavior.

One month after this last admission, the patient was discharged with a therapeutic plan that included CBT, in which, once again, she refused to follow up. which is related to compulsion, passive acceptance of obsessive intrusiveness, isolation, and a striking lack of adherence to treatment. These features remind us of an ancient—yet controversial—diagnosis proposed by several authors: obsessive psychosis—here understood in its original sense, which was related to a particular experience of being ill rather than to specific psychotic symptoms. Moreover, our patient did have, albeit transiently, paranoid ideation. Nevertheless, the impaired judgment this patient undoubtedly presented about her illness has been associated with cerebellar dysfunction in patients with schizophrenia. We must take in consideration the fact that schizophrenia is associated with above-average rates of midline cerebral malformations. Therefore, the cerebellar variant disclosed in this case may also be considered as an accidental finding, with no relationship to the clinical description. In addition to the aforementioned evidence supporting the role of cerebellar abnormalities in some symptoms of schizophrenia, the cerebellum itself has been recognized to be involved in the pathophysiology of OCD and, specifically, compulsive hoarding, with which this patient presented.

In this case, we could hypothesise that cerebellum dysfunction along with some genetic sharing with the schizophrenic relatives, could have contributed to this kind of evolution and account for the symptom co-expression.

**REFERENCES**