Late onset obsessive-compulsive disorder (OCD): A case report

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Dear Editor,

Obsessive-compulsive disorder (OCD) is a disease that falls within the spectrum of anxiety disorders. It is defined according to DSM-5 criteria as the presence of obsessions (recurrent and persistent thoughts, urges, or images) experienced by an individual as intrusive and unwanted. The individual tries to ignore or suppress such thoughts, urges, or images with compulsions (repetitive behaviors) that they feel driven to perform in response to the obsession, a situation which impacts significantly on their daily routine.[1]

Prevalence ranges between 0.3% and 3.1% of the population, and the greatest incidence is observed among individuals younger than 25 years of age, whereas onset in older adulthood is exceptional.[2]

Late-onset OCD, unlike OCD in young patients, affects more females (72% versus 48% in early ages) and, like other anxiety disorders, tends to be underdiagnosed, probably because it is less debilitating than other cognitive disorders, such as dementia or depression.

When late-onset OCD is encountered, an organic etiology must be ruled out. There are numerous causes, as listed in Table 1.[3,4]

The symptoms presented by patients with OCD starting in adulthood also differ from those which begin in adolescence (Table 2).[5]

With respect to treatment, clomipramine (a tricyclic antidepressant) has been the drug of choice for many years, but it has now been relegated to second line because of its side effects, especially anticholinergic effects (mouth dryness, arterial hypotension, acute urinary retention, constipation, excessive sedation, and falls) that are potentially harmful in the older population. The use of a selective serotonin reuptake inhibitor (SSRI) combined with cognitive behavioral therapy is recommended in first line: sertraline (100-250 mg/day), fluvoxamine (100-250 mg/day), citalopram (10-30 mg/day). The response rate with pharmacological treatment alone is 30%-60%, a rate that increases when combined with psychotherapy. If response is partial or non-existent, the effect of these drugs can be boosted with atypical antipsychotics, at low doses (aripiprazole 5-15 mg/day). Other alternatives, in resistant cases, are electroconvulsive therapy (ECT), stereotactic psychosurgery, and, more recently, transcranial magnetic or electric stimulation.[6-9]

Clinical Case

A 68-year-oldman was brought to the emergency room due to a self-harming incident, consisting of tracheal incision and pneumothorax after stabbing himself with a knife.

His personal history included hypertension, dyslipidemia, colorectal adenocarcinoma, and prostate cancer. Both tumors were resected, and the patient remains disease-free.

Table 1 Late-onset OCT causes

Stroke	Parkinson Disease (PD)
Traumatic brain injury	Progresive supranuclear palsy (PSP)
Infections of the central nervous system	Huntington disease
Brain tumor	Syndenham Corea
Gilles de la Tourette Syndrome	Others neurodegenerative disease: dementia

Table 2 Differences between adulthood and adolescence symptoms

SYMPTOM	OCT starting in adolescence (<25 years old)	OCT starting in adulthood (>60 years old)
Cleaning rituals	46%	70%
Dirt concert	48%	60%
Use of methods to avoid contamination	42.6%	56.7%
House cleaning	35%	45%
Need to confess	40.2%	44%
Fear of sin	19.9%	42.9%
Obsessive ideas of hurt people	36.2%	40%
Rituals of doing and undoing	30.5%	36.7%
Fear of something bad happening	48.3%	32.1%

He was diagnosed with OCD 3 years previously in another hospital, with no history of mental illness nor psychiatric admissions before that. He was receiving regular treatment with sertraline 200 mg/day, olanzapine 2.5 mg/day, diazepam 5 mg/day if needed for anxiety, pravastatin 40 mg/day, enalapril 10 mg/day, and metamizole 575 mg if needed for pain. Baseline functional status was good, with a Barthel index of 100/100, and he was retired from work.

After initial admission to the intensive care unit, where he was hemodynamically stabilized, the patient was transferred the psychiatry ward. In the first interview, the patient reported obsessive hypochondriac ideation ("I thought I had something bad in in my belly, then I thought I was going to die, and lately I thought I was going to go crazy or I was going to commit suicide") and checking rituals ("I don't know if I parked the car correctly and I go out several times a day to check that the handbrake is on"), which had led to a restriction in activities (he rarely left the house). He denied any mood disturbance. Despite receiving treatment with various psychotropic drugs in recent years, the patient's disease course had worsened progressively. At the time of the interview, he had a score of 39 on the Yale-Brown Obsessive-Compulsive Scale (YBOCS), corresponding to extreme OCD.

On examination, the patient was conscious, disoriented in time and space, and oriented in person, with a marked attention disorder, and a fluctuating level of consciousness throughout the day, but with no delusions or sensorial perception changes. He presented an acute confusional state (that improved gradually until fully resolved within a few days).

Additional tests were requested to identify any underlying organic cause for the clinical picture: a complete blood count with thyroid hormones showed no findings of interest and autoimmune tests were normal. Infectious pathology was also ruled out with serologies for HIV, syphilis, hepatitis and *Borrelia burgdorferi*, and cerebrospinal fluid was normal.

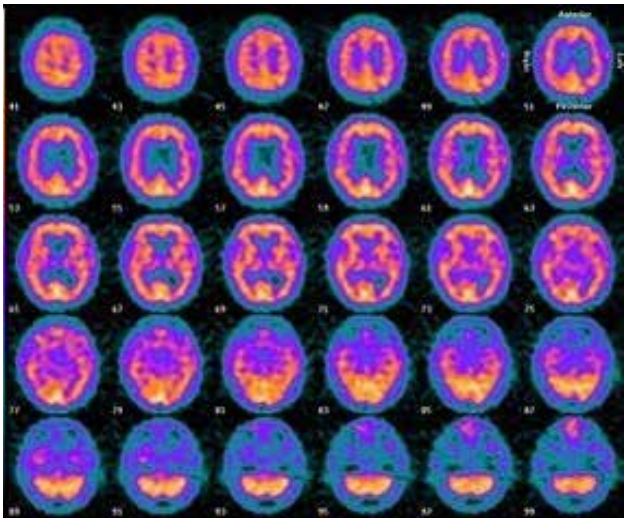

Figure 1 *Brain SPECT*

Single photon emission computed tomography (SPECT) brain scan revealed generalized changes in global cortical perfusion of ischemic etiology, significant for the age of the patient, and no perfusion pattern characteristic of primary organic dementia was observed (Figure 1).

The patient was diagnosed with a self-harm attempt in the context of obsessive thoughts of self-harm, due to OCD.

After starting treatment with fluvoxamine 200 mg/day, aripiprazole 7.5 mg/day, and quetiapine 25 mg/day, the patient's psychobehavioral symptoms improved progressively, and he was discharged home after 35 days of hospitalization, with a YBOCS score of 14 (mild disorder).

Three months after discharge, the patient attended check-ups in the psychiatry outpatient clinic, where he showed appropriate control of obsessive thoughts (Y-BOCS score of 7), and the neurology outpatient clinic, where a neuropsychological evaluation was conducted, significant for cognitive dysfunction with attentional, executive, and memory involvement. Memory was assessed using the Mini Mental State Examination (MMSE), the Hopkins verbal learning test (HVLT), and the revised Barcelona test (BCN-R), all of which showed pathological scores: MMSE 26/30; HVLT with total recall 11/36 and delayed recall 2/12, BCN-R with immediate verbal recall 7/23 and delayed verbal recall 8/23. Executive function and attention were tested using the Trail Making Test (TMT) and the Frontal Assessment Battery (FAB), and results below normal were obtained in both (TMT part A: 66 seconds, TMT part B: 144 seconds. FAB 11/18). The patient was given a diagnosis of cognitive impairment with frontotemporal involvement of probable vascular origin.

Discussion

In the diagnosis of late-onset OCD, in contrast to OCD in young patients (the etiology of which is predominantly psychiatric), organic etiology must be ruled out, because in most cases the syndrome can be explained by a cerebral factor. It is therefore essential to carry out a comprehensive study that includes blood tests with serologies and autoimmune testing, neuroimaging tests, lumbar puncture, and a cognitive assessment. With regard to cognitive assessment, it is essential to explore the executive function, attention and memory since all of these areas are involved in OCD in the presence of frontal lobe dysfunction.[10,11]

As reported in the case series published by Weiss et al.[12] and by Chacko et al.[13], traumatic brain injuries (TBI), cerebrovascular accidents, and neurodegenerative diseases are often the main factors in the development of OCD in adulthood[14,15].

In the differential diagnosis of OCD, the effects of drugs, pharmaceuticals, and other medical or psychiatric diseases must be ruled out. With regard to psychiatric diseases, it should be borne in mind that depression is up to 5 times more common in individuals with OCD and individuals with obsessive-compulsive symptoms who not meet the criteria for a diagnosis of OCD.[16]

In the clinical case we report, the patient had late-onset OCD (beginning at the age of 64 years) with no previous evidence of premorbid personality. Although the most prevalent symptoms in OCD with onset among the elderly are cleaning rituals and religious concerns (fear of sin), our patient presented primarily ritualistic behaviors (which also are slightly more prevalent in older individuals than in the young) and obsessive ideas of self-harm (fear of illness and fear of harming), leading to an incident of self-harm.

After extensive tests, TBI, CNS infection, and other focal brain lesions were ruled out, but very significant cerebral cortical perfusion changes of ischemic origin were observed. The clinical implications of this finding were determined 3 months after discharge, when the patient was stable, and a neuropsychological evaluation determined cognitive dysfunction with frontal lobe involvement.

The etiology of OCD in our patient, then, appears to be cognitive impairment onset of vascular origin. This diagnosis is supported by the fact that the patient presented an acute confusional state during hospitalization, demonstrating his limited cognitive reserve.

After intensive pharmacological intervention during admission in the psychiatry ward, with a combination of SSRIs and 2 low-dose antipsychotics, the episode could be controlled after 1 month of hospitalization; the patient made a complete psychopathological recovery and could progressively resume his activities of daily living in the months following discharge. In this respect, it should be noted that while SSRIs are the treatment of choice, they must be combined with other types of psychotropic drug (primarily atypical neuroleptics) if progress is poor or refractory.

Conclusion

Late-onset OCD is a rare entity. Organic disease, primarily neurodegenerative diseases and cerebrovascular injury, must be ruled out as the principal etiology. Therapeutic management should be individualized for each case, with the use of drugs with low side effect profiles. Psychotherapy should be prescribed (if required), and ageism must not constitute a decisive factor in the therapeutic approach.

ACKNOWLEDGEMENTS

The authors thank the Hospital Universitario October 12 for providing information on this clinical case.

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interests related with the contents of this manuscript.

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Cycloid psychosis and its longitudinal diagnosis: case report

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Dear Editor,

Psychosis constitutes a disruptive and disabling syndrome composed of alterations in thought, sensoperception, affection and personality, which subsequently lead the individual to lose contact with reality. Viewed from a phenomenological perspective, psychosis encompasses a number of characteristic symptomatic manifestations: 1) there is a serious alteration of the personality that ends in a loss of psychosocial function due to erratic behavior, 2) loss of reality's judgement and alteration of logical thinking, 3) evidenceable disorders in the intellectual, affective and volitional spheres, 4) there are often disorders in the content and course of thought, often accompanied by hallucinatory experiences or pathological illusions, 5) and finally, consciousness is clear but there is poor introspection of the disease[1,2].

Historically consolidated since the mid-19th century, schizophrenia has guided prolific lines of research, becoming the pathological archetype of psychosis. Both in the International Classification of Diseases in its tenth edition (ICD-10) and the Statistical manual and Diagnosis of Mental Diseases in its most recent version (DSM-5), psychosis is rep-

resented in strictly categorical terms: psychosis due to another medical condition, substance/medication-induced psychosis and those found in the spectrum of schizophrenia, such as schizotypal disorder and delusive idea disorder. We also can find in the affective disorder's section the specifier "with psychotic symptoms", presented in the most serious episodes (mania or depression)[3,4].

Unlike the observable in schizophrenic spectrum psychosis, cycloid psychosis is characterized by its phasic course, its tendency to repeat itself with certain periodicity and without the presence of residual symptoms. The historical development of this term begins with Kleist's marginal psychosis (1928), from other disorders described by different authors such as Wernicke's motility psychosis (1899), Westphal's primary paranoia (1876) or hallucinatory confusion of Kraff-Ebing (1879), as well as the concepts of Magnan and Legrain's *bouffée délirante* (1880). Karl Leonhard (1961) proposes a categorical grouping of the psychosis described by Kleist, defining them as an autonomous group with three subtypes: the psychosis of anguish-happiness, the confusional psychosis (incoherent-stuporous) and the psychosis of motility (akinetic-hyperkinetic). For his part, Breuning proposes six ideal types versus Leonhard's nosology, where the disorder of thought about alterations of affection stands out. The symdromatic classification of Brauning is composed of the following entities: paranoid-anxious syndrome, exalted paranoid, excitation-confusion, delayed-confusing, and hyperkinetic. On the other hand, Bräuning proposes six ideal types instead of Leonhard's nosology, which highlights the disorder of thought instead affective alterations. The Bräuning syndromatic classification is composed by the following entities: paranoid-anxiety syndrome, euphoric paranoid, confusional-excitation, delayed-perplexity and hyperkinetic[3].

Perris and Brockington (1981) in their definition of cycloid psychosis mention a number of essential diagnostic criteria for identifying these disorders. A psychotic condition unrelated to drug use and without any organic basis is needed. The debut usually occurs between the ages of 15 and 50, appearing suddenly and with a rapid progression into a psychotic state. At least four of the following criteria are needed: confusion, incongruous humor, delusions, hallucinations, intense anxiety, feelings of happiness or ecstasy, alterations of akinetic or hyperkinetic motility, death ideas and discreet mood swings. Symptomatology usually varies greatly[4,5].

The epidemiological knowledge of cycloid psychosis is insufficient since there is a lack of statistical support with representative samples. In the literature it is possible to find observational studies of limited magnitude, where we can find series of sociodemographic and etiopathogenic characteristics that seem to meet in these patients. Apparently, they are more frequent in the female sex, the age of onset is, on average, at 30 years, however, as mentioned in the Perris and Brockington Criteria, the first outbreak can occur between 15 and 50 years old. The triggering factor is usually related to the puerperium, personal history of perinatal insults and alterations in glycine metabolism. Family studies in these patients suggest certain genetic conditioning, finding up to 40% cases of cycloid psychosis in first-degree relatives, and some cases where Prader-Willi Syndrome coexists with the appearance of episodic psychosis with cycloid characteristics. Fukuda (1990) points out that these atypical psychoses could be related to the pathophysiological mechanisms of epilepsy, since some convergent characteristics meet, such as the compromise of consciousness, the abrupt onset of episodes and electroencephalographic alterations (paroxysmal dysrhythmias); this way, the appearance of psychotic symptoms could be explain as a product of Landolt's phenomenon or forced normalization[3,4,6-8].

Although the categorical classification of mental illnesses makes it possible to create a standardized language for diagnosis in any part of the world, it is undeniable that this same homogenization rules out entities of a nature as particular as cycloid psychosis. ICD-10 includes cycloid psychosis within the spectrum of schizophrenia (acute polymorphic psychotic disorder with or without symptoms of schizophrenia), although as mentioned above, the morbid presentation and evolution are of a different nature. The diagnostic complexity in this type of psychosis is represented by the usual overlaps with the criteria of four large psychiatric entities: brief psychotic disorder, bipolar disorder type I, schizophrenia and schizoaffective disorder. The difference usually lies in the evolution of the condition (acute, episodic and with complete remission) and syndromic presentation, since it has been seen that sudden changes in mood, intense anxiety, perplexity, delusional ideas not congruent with the state of mood and hallucinations, both visual and auditory, are more prevalent in cycloid psychosis[3,9-11].

Recommendations regarding the pharmacological management of cycloid psychosis come from clinical experience, some uncontrolled studies and anecdotal case reports. As it has been pointed out, to date there are no controlled studies, mainly due to this categorical absence in the nosological systems CIE-10 and DSM-5. Some studies suggest the application of electroconvulsive therapy, with greater benefit in those clinical presentations with predominance of motor symptoms. Atypical neuroleptics seem to be effective as abortive acute episodes, with weak evidence in reducing relapses, unlike lithium. The advocacy of maintaining conservative management (only treating acute episodes and not installing prolonged pharmacological schemes), has an apparent logical support if we consider that cycloid psychosis is characterized by its phasic presentation and surprising benignity[12,13].

The following case exposes the typical and episodic syndromatic evolution of cycloid psychosis throughout the life

of a patient. As will be seen, the clinical characteristics do not depart completely from the wide nosological field delimited by classical psychoses. It is crucial to make a timely and accurate diagnosis in this type of psychosis since treatment and prognosis completely diverge from psychotic disorders of the schizophrenic spectrum.

Clinical Case

R.M.S, a 27-year-old female, mestiza, currently in free union, a mother of two sons, a housewife, highest education: high school. She has a history of psychotropic substance use during adolescence, which she permanently suspended at age 21. Without other important medical records.

At 16 years old, she manifests a behavioral pattern of floating anxiety, excessive worry, social withdrawal and decreased attention. It is until the age of 18 when she presents her first psychotic episode, acutely and concomitantly to cannabis use. The clinical picture consisted of self-absorption, delusional paranoid ideation, simple auditory and visual hallucinations, global insomnia and symptoms of adrenergic hyperactivity. She was admitted to a Juvenile Integration Center for two weeks and discharged with restitutio ad integrum. Antipsychotic and mood modulator treatment was prescribed, which she suspended six months after her discharge.

At 21 years of age, during the first trimester of pregnancy, she presented a new psychotic episode with similar characteristics to the previous ones, being admitted to our unit for the first time (prior evaluation of the binomial in gynecology service) and subsequently discharged by her rapid clinical improvement. The response to the neuroleptic and benzodiazepine scheme occurred within the first five days of hospitalization. During the second week of her discharge she suspended the pharmacological treatment on his own will. During the puerperium she remained asymptomatic, with a satisfactory performance in maternal care. At her third episode, at age 25, after a stressful psyco-stressor, she presents global insomnia, disabling anxiety and delusions of persecution and harm. She received homeopathic treatment, with a favorable response achieving complete remission within seven days.

She is admitted to our unit for preseting her fourth psychotic episode. The clinical presentation already manifested in the preceding episodes begins to settle gradually after the birth of her second child, hatching during the late puerperium. First-order symptoms, blocking and interception of thought are added. Anxiety is intense, accompanied by adrenergic discharge, without conditioning external stimuli and mixed insomnia. In the continuous admission service, the patient is found with disorganization of thought and negativism attitude to the interrogatory. When admitted, and during the first hours of hospital stay, she exhibits an aspect of perplexity that alternates with moments of emotional lability. During the first three days of hospitalization, management with haloperidol is administered intramuscularly, however, it is suspended due to stiffness in thoracic and pelvic limbs and slight tongue fasciculations. It was necessary to indicate an anticholinergic corrector. When the neuroleptic side effects disappeared, she presented catatonic signs, such as ambitency, mutism and maintenance of induced postures. Therapeutic trial was performed with lorazepam orally, with complete remission of psychomotor alterations, so it was decided to add risperidone and sertraline to the scheme. During the second week there is a clear improvement, with remission of psychotic symptoms. She is discharged without residual data of the episode and with improvement of the sleep pattern.

Discussion

Since they were first described, cycloid psychoses have inevitably led to a gray area of diagnostic misunderstandings. It is necessary to emphasize its particular characteristics: form of abrupt onset, short duration, complete recovery, tendency to repeat and clinical that includes psychotic, anxious and affective symptoms.

From the case described, it is possible to objectify the essential features of cycloid psychosis, highlighting the integral interepisodic remission and without data suggesting a state of chronic deterioration. The rapid response to antipsychotic drug schemes, usually between one to two weeks and the contribution of the puerperal state in her last episode, is highlighted. During his monthly follow-up she has been in total remission of both emotional and psychotic symptoms.

Performing a psychopathological analysis it is possible to detect in our patient, throughout her condition (Table 1), symptomatic elements of the three areas of marginal psychoses described by Karl Leonhard: confusional (confusing-dreamlike states, perplexed attitude), anxious (apprehensive anticipation, adrenergic symptoms) and psychomotor (incomplete catatonic syndrome) (Figure 1).

Adjusting to the essential criteria of Perris and Brockington, the patient has the debut of a psychotic condition at age 18. The beginning of each episode occurs between a few hours to days, that is, the transition to a psychotic state is of abrupt and overwhelming presentation. Of the five additional criteria it meets four: state of confusion or perplexity, delusions with incongruous humor, an overwhelming sense of anxiety of remarkable vegetative courtship and hallucinatory experiences. It is curious that in some cases of cycloid psychosis, motor skills are affected more frequently than schizophrenic psychoses, predominantly stiffness and hypokinesia states, even with minimal doses of atypical antipsy-

Table 1 Characteristics of the episodes

Age	Triggers	Clinical	Pharmacological treatment	Resolution time
18 years old	Cannabis use	Social isolation Delusional ideation of harm and reference, auditory hallucinations, visual hallucinations, global insomnia, anxiety with adrenergic hyperactivity	Antipsychotic and mood modulator (Risperidone and Lamotrigine, doses not specified by the patient)	14 days
21 years old	Pregnacy	Same symptoms as in the first episode	Antipsychotic and benzodiazepine (Risperidone and clonazepam, doses not specified by the patient)	5 days
25 years old	Relationship problem	Delusional ideation of harm and reference anxiety with episodic adrenergic hyperactivity, global insomnia	Homeopathic treatment	7 days
26 years old	Late puerperium	First-order symptoms, perplexity, emotional lability, anxiety with adrenergic discharge, global insomnia, extrapyramidal syndrome, catatonic signs	Haloperidol intramuscularly (10 mg / day, suspended on the 3rd day) Lorazepam orally (1 mg / day, suspended when remitting catatonic signs) Risperidone and Sertraline (3 mg and 100 mg / day, respectively; installed during the last hospital week and at hospital discharge)	14 days

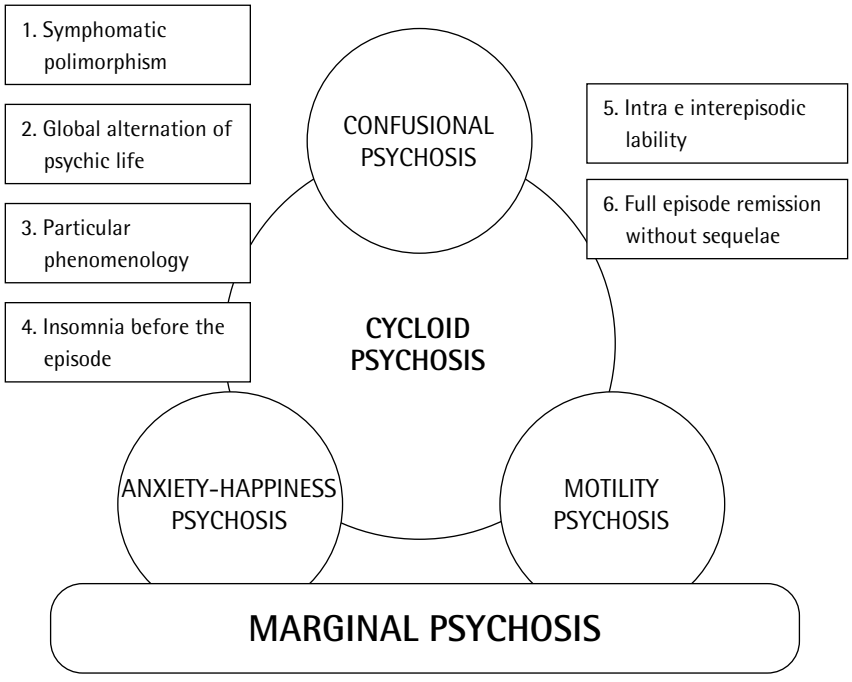

Figure 1 *The typical classification of cycloid psychosis is carried out in three areas: anxiety-happiness, motility and confusion. The symptomatic domains are usually varied and present symptoms of anxiety and affective lability. The complete remission of the psychotic clinic is the essential criterion in this type of psychosis*

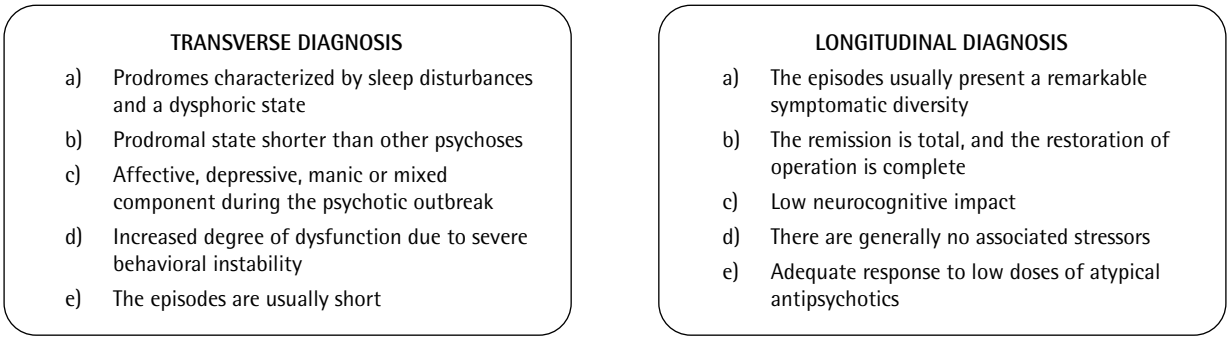

Figure 2 — *The characteristics of the psychotic episodes of cycloid presentation have typical elements, but not pathognomonic. These can be classified from a transversal and longitudinal perspective. Very useful for the diagnosis of certainty*

chotics, a phenomenon observed in the last hospitalization of our patient[11].

It is of special interest to proceed to a differential diagnostic exercise. Schizophrenia does not appear to be compatible with interepisodic complete recoveries (despite showing catatonic signs); bipolar or schizoaffective spectrum disorder cannot be diagnosed because an important affective condition that accompanies psychotic symptomatology cannot be seen; and a reactive psychosis seems inappropriate because of the frequency with which these conditions are repeated in the patient (without identifiable triggers in some of them). For all the above, we are inclined to diagnose cycloid psychosis, which is more in line with the nosological conception and the specific criteria in the whole condition. It should be noted that the patient's diagnostic stability is one decade, his clinical condition has required relatively low doses of atypical antipsychotics maintained for very short periods (this is partly due to irregular therapeutic compliance) (Figure 2).

The foregoing is consistent with what has been stated in the literature regarding the notable involvement of atypical antipsychotics during acute periods, but not for the prevention of future recurrences, even if you have made the distinction of discontinuation of any antipsychotic drug may lead to increased relapses Only lithium or carmabazepine appear to have a prophylactic effect[10-12,14,15].

Currently the patient has the same pharmacological treatment of progress in reduction doses. During the subsequent interviews, he has only reported persistence of certain mild intensity anxiety symptoms.

Conclusions

The significance of the diagnosis of cycloid psychosis goes beyond what concerns *nosotaxia*; The clinical and heuristic value of its concept seems evident. Within the ICD-10 nosological system, cycloid psychosis is in the category of acute and transient psychotic disorders, however, the classic diagnostic criteria are not mentioned. Within the DSM-5 there is no mention of that entity, but a spectrum of conditions related to schizophrenia are proposed.

The lack of interest in the cycloid psychosis construct may be secondary to the intrinsic characteristics of the Westphal-Kleist-Leonhard nosological spectrum: 1) the atipicity in the evolution of the disease, usually showing mildness and rapid remission of symptoms, 2) a varied interepisodic and interepisodic symptomatic constellation; 3) Therefore, it is totally contrary to the phenomenological and categorical conception of schizophrenic psychosis.

Throughout the article a longitudinal approach has been described in the diagnosis of cycloid psychosis, however, there are some characteristics in the episode, either debut or recurrence, which suggest us to face this particular entity: 1) prodromal symptoms characteristics, such as dysphoria or sleep disorders (the stage that precedes the psychotic outbreak is shorter than that of other psychoses), 2) predominance of an affective component (manic or depressive), 3) greater behavioral instability during the episode (notable impairment in function, unlike other non-cycloid psychotic conditions), 4) faster recovery of the psychotic condition (the difference in the response to drug treatment is usually not significant)[16].

Some studies indicate a possible phenomenon of underdiagnosis and overlap of these psychotic entities with the criteria proposed in the most popular diagnostic categories and wide clinical limits, we speak of acute polymorphic psychotic disorder and brief psychotic disorder. Given this risk, we must not forget the value of longitudinal analysis in all psychiatric conditions, and the evaluation of

the patient's overall state through a rigorous psychopathological semiology.

So far it is unknown if it is necessary to maintain a pharmacological therapeutic line, an issue that faces different authors, since one of the most discussed aspects is its close relationship with physiopathogenesis of epilepsy, so if we start from this premise it would be necessary to establish a long-term antiepileptic treatment. We are still far from having therapeutic guidelines based on clinical evidence for cycloid psychosis, and it will not be possible until it is contemplated in the large nosological systems.

ACKNOWLEDGMENTS

To the executive staff of the Héctor H. Tovar Acosta Psychiatric Hospital of the Mexican Social Security Institute for their support in the academic, research activities of their resident and attached physicians.

CONFLICTS OF INTEREST

The authors state that they have no conflicts of interest in this article.

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Pica disorder as a symptom of depression in a patient with bipolar disorder and intellectual disability

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Dear Editor,

Pica is a little studied and poorly understood disorder. It is associated with certain medical pathologies and psychiatric disorders; it is observed more frequently in people with intellectual disabilities. Paradoxically, it has also been docu-

mented in some stages of life such as childhood and pregnancy.

We present the case of a 27-year-old female patient diagnosed with bipolar II disorder and a mild intellectual disability (ID) (WAIS IV: IQ56). Her biological sister was diagnosed with bipolar I disorder. At the age of 18 the patient developed a depressive episode with catatonic symptoms that responded to electroconvulsive treatment (19 sessions) associated with venlafaxine (225mg/day). At the age of 20, the patient began a Pica disorder of high severity and frequency (> 500 episodes/year), ingesting buttons, knives, scalpels, radio antennas, tree branches... that necessitated endoscopies, laparoscopies, and enterotomies. Blood and neuroimaging tests were always normal. Treatment with antiepileptics (carbamazepine 400mg/day (7.3mg/L), oxcarbazepine 900mg/day), and antipsychotics (amisulpiride 500mg/day, aripiprazole 30mg/day) was not effective. Lithium treatment (1200mg/day; Litemia: 0.87 mEq/L) was started in combination with olanzapine (25mg/day), achieving a decrease in the frequency of the Pica that allowed us to observe for the first time that some days before the Pica episodes, the patient would present depressive symptomatology in the form of hypothymia, isolation, rumination, and daytime hypersomnolence. During these phases, hypomanic episodes were observed in the form of tachypsychia, tachylalia, hyperthymia, inappropriate laughing, increased socialization, and decreased need for sleep, which occurred approximately 1-2 times a month. Concomitant treatment with topiramate (450mg/day) in combination with lithium and olanzapine was initiated with an anti-impulsive objective. After 3 months with this treatment, the severity and frequency of the pica decreased markedly, limited to the intake of soft objects (toothbrushes and plastics) with a frequency of 1 episode/3 months and no observed intake of sharp objects. At the same time, the depressive and hypomanic phases became much less frequent, reaching an euthymia which is currently maintained.

Discussion

We considered it to be of interest to carry out a review of the literature on Pica disorder, as well as its relationship with other psychiatric disorders.

Pica disorder is defined as the need to ingest non-nutritive or non-food substances for a minimum period of one month[1], with a very wide variety of substances. It is marked by the intake substances ranging from soil and other minerals (geophagy) to ice cubes (pagophagia), feces (coprophagy), and more dangerous objects such as knives, scalpels, and batteries. This behavior is frequently observed in childhood (10-15%)[2,3], in pregnant women (27%)[4], and in dialysis patients (42%)[5]. Although in the groups mentioned above Pica behavior is usually limited to geophagy or pagophagia, in people with intellectual disabilities or other psychiatric disorders intake is usually of a greater severity and frequency. From an etiological point of view theories have been proposed involving zinc and iron deficit[5], the serotorinergic[6] and dopaminergic[7] circuits, the opioid system[8], and low levels of education and culture[4].

In the population with intellectual disability, Pica disorders are reported in 4-26%, more commonly in the population with severe ID and/or associated autism spectrum disorders[9]. A higher prevalence of this disorder has been observed in Prader-Willi syndrome[10], associating pica with the hyperphagia presented by these patients.

There are few publications on the association of pica with other psychiatric disorders. Mehra (2018) describes the clinical case of a recurrent depressive disorder that was associated with pica (pagophagia) and that improved after the introduction of antidepressant treatment with venlafaxine. In people with intellectual disabilities, it is more frequent to observe the appearance or worsening of an existing pica during depressive episodes[11,12], but studies in this regard are very scarce.

The case we present is the first described in the literature involving a pica disorder associated with depressive phases in a patient with bipolar II disorder. In our case, the euthymizing treatment with antiepileptics and antipsychotics did not achieve disappearance of the disorder, but it did achieve a remarkable improvement. Based on the serious medical-surgical consequences of pica disorder, we consider it very important to develop the psychotherapeutic and pharmacological treatment of this psychiatric disorder.

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