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Meningioma and late-onset mania: about a **clinical case**

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Meningioma and mania of start late: to purpose of a case clinical

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Meningioma and late-onset mania: report of to clinical case

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Summary:

Disorder bipolar one of the conditions psychiatric higher impact may have secondary causes in its etiopathogenesis, which must be suspected and addressed, one of these are the medical causes among which are primary brain tumors, The most common of these is meningioma, having very varied clinical presentations depending on its anatomical location, these being neurological, cognitive, behavioral or behavioral alterations. neuropsychiatric symptoms. We present the case of a 69-year-old man with a history of depressive symptoms, substance abuse, as well as such as a meningioma and resection year prior to developing a picture of late-onset mania in which the approach outlined in the scientific literature is followed and an attempt is made to find the etiological relationship. of said painting for the first time at this age and given the background patient's doctors.

Keywords: meningioma, bipolar disorder, late mania, organicity, neuropsychiatric symptoms.

Abstract:

Bipolar disorder, one of the psychiatric conditions with the greatest impact, may have secondary causes in its etiopathogenesis, which must be suspected and addressed, one of these are the medical causes, among which are primary brain tumors, the most common being These meningioma have very varied clinical presentations depending on their anatomical location, these being neurological, cognitive, behavioral or neuropsychiatric symptoms. We present the case of a 69-year-old male with a history of depressive symptoms, substance use, as well as a meningioma and resection one year prior to developing late mania, in which the approach established in the scientific literature is carried out and an attempt is made to find the etiological relationship of this condition for the first time at this age and given the patient's medical history.

Keywords: meningioma, bipolar disorder, late mania, organicity, neuropsychiatric symptoms.

The authors of this manuscript declare that:

All they have participated in his elaboration and not they have conflicts of interest
The investigation HE ha done Following the Guidelines ethics international for the investigation health related with human beings made by he Council of Organizations International of the Medical Sciences (CIOMS) in collaboration with the Organization World Health Organization (WHO) https://cioms.ch/publications/product/guidelines international-ethics-for-research-related-to-health-with-beings humans/
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They have obtained the necessary permits for images and graphics used. They have preserved the identities of the patients.

INTRODUCTION

A brain tumor is a mass of abnormal cells that can present with a wide range of symptoms. They are classified according to their histopathological characteristics or their anatomical location. Brain tumors can be asymptomatic or present with neurological, cognitive, behavioral, and psychiatric symptoms. The evidence suggests that there is an increase in the prevalence of brain tumors in patients with psychiatric symptoms. In one study, the rate of primary brain tumors in hospitalized psychiatric patients was one in 1,000, which was 20 times more common than in the normal population. [1]

Meningioma is the most common type of primary tumor of the central nervous system, its location can predict symptomatology, which could cause significant morbidity and mortality. Without embargo, due to the nature of growth slow, the meningiomas are usually asymptomatic, and he Diagnosis is often made incidentally on neuroimaging or in autopsies. [1] [2]

Presenting signs and symptoms depend on the location of the tumor, ranging from generalized and focal neurological symptoms to psychiatric symptoms. [1]

A tumor in the region prefrontal dorsolateral generally she drives to executive deficits and a tumor in the region orbitofrontal she drives to the disinhibition, a tumor in the medial frontal region can cause apathy or avolition and temporal limbic tumors can present with psychosis, This shows that exists a association of psychiatric symptoms with brain tumors. [1]

More than 50% of people with brain tumors experience psychiatric symptoms, but the tumors insidious with symptoms psychiatric as the complaint of

presentation are rare. Satzer and Bond in 2016 found a low rate of brain tumors in psychiatric patients (1/10,000 adults) which meant that this still represented a very small number of psychiatric patients, brain tumors presenting with mania are even rarer; In this same study it was found that only 15% of brain tumor patients with psychiatric presentations exhibited mania. [3]

In the review of Gyawali et al. of 2019 of 52 patients with meningioma in different regions, very varied symptoms were reported depending on the affected areas, from atypical depression, anxiety, psychosis, Capgras syndrome, Anton syndrome, Godot syndrome, schizophrenia, obsessive symptoms, bipolar disorder, alcohol abuse, musical hallucinations, depersonalization, personality changes, generalized anxiety disorder, cognitive impairment, vertigo, disorientation, anorexia, agitation, restlessness and dementia-like symptoms, the diagnosis of the tumor was after an imaging study generally motivated by atypical presentations of psychiatric symptoms, poor response to psychiatric treatment or focal neurological symptoms. [1]

Treatment in these cases involves removal of the tumor and concomitant treatment of psychiatric symptoms with pharmacotherapy or electroconvulsive therapy. [1]

Bommakanti et al reported that of 20 patients with psychiatric symptoms, about 15% did not improve, 40% improved partially, and 45% improved completely after resection. In patients with partial response to psychiatric symptoms, the approach regarding the continuation of psychiatric pharmacological treatment after resection is not clear, which motivates research. of further cases individual this entity. These studies could serve to strengthen the causal association between brain tumors and the appearance of psychiatric symptoms. [4, 6, 11]

The presentation psychiatric very heterogeneous associated with the presence of meningiomas and the lack of conclusive evidence to determine the causal or secondary relationship of psychiatric symptoms with them, will open the door to continue investigating this association and the reporting of more related cases. [1]

CASE CLINICAL:

The case of a 69-year-old male, separated marital status, is presented. secondary schooling, retired, previously a carpenter, orthodox Catholic, living in a nursing home; background heredityfamily denied, background pathological personalities:

of hypertension arterial of a year of evolution treaty with losartan fifty mg, peripheral vascular insufficiency , gonarthrosis of both knees, resection of meningioma in area left frontal-temporal, presenting symptoms neurological problems that began in April 2018 with falls from their own height, adding right hemiparesis to beginning of May of the 2019, So as headache Halo cranial, dysarthria, difficulty for feeding , going with neurologist who requests NMR of simple brain showing extra-axial mass of 5.8cm x 5.2cm x 7.5 cm at front left level compatible with meningioma, like this as cortico atrophy subcortical and chronic vasculopathy Fazekas I in supratentorial white matter.

After hospitalization, surgical resection of tumor, as well as histoimmunopathological study evidencing meningioma degree Yo of agreement to the Clasification of The OMS, with receivers progesterone negative; during hospital stay develops chart acute confusional mixed handling with haloperidol and lorazepam presenting remission saying chart.

After discharge medical for improvement clinic HE gives follow-up by consultation external neurosurgery graduating alert, stable so much clinic as hemodynamically, afebrile, tolerating diet normal and via oral diuresis and evacuations present, wandering assisted, HE valued in 14 points of the scale of eat of Glasgow, pupils of 3 millimeters isochoric, normoreactive, peers cranial apparently intact, force muscular 5 of 5 in the scale of Lovett in the four extremities, normal deep tendon reflexes, Hoffman, left Trommer and Babinski bilateral, sensory and vestibulocerebellar system without alterations.

He was admitted in June 2019 for surgical wound infection, where wound reopening, drainage and antibiotic therapy were performed, and he was discharged with MSD monoparesis. 4/5, the rest of exam normal neurological.

Psychiatric history: 2 depressive episodes in adolescence and adulthood without specifying evolution and severity of the latter treated for 10 years with clonazepam unspecified dosage, non-pathological personal history of alcoholism of 4 L daily from 14 years old until 2018, positive smoking since 14 years consumption of three cigarettes a day, idle consumption from 3 months prior to hospitalization; consumption of marijuana from start to 49 years, quantity and frequency not specified, inactive consumption for 10 years, consumption of cocaine, with Irncl0 1O years ago with a frequency of every 15 days in quantity of two lines, suspending 5 years ago, methamphetamine that starts ago 6 years 3 times per week, inactive since 3 years, solvents that started to 23 years inactive for 8 years.

Denies previous episodes of hypomania or mania, suicidal attempts or psychotic periods. Tattoos denied, allergies denied.

It starts at the end of May 2020 with sadness and irritability, increased energy, verbosity, disinhibited and disorganized behavior, paranoid and grandiose delusions, decreased need of sleep, increased non-directed activity, as well as heteroaggression, going to a psychiatrist who prescribes olanzapine 10 mg every 12 hr, valproic acid 500 mg every 24 hr, Pregabalin 150 mg every 24 hr and Lorazepam 2 mg every 24 hr, with poor response to treatment.

to the exam mentally alert, conscious, cooperator, oriented in 3 spheres, memory without alterations, clothing according to the context and gender, good hygiene and seasoning, march with a broad base of support and with help cane, contact adequate visual, hyperthymic affect with humor I agreed, language verbose with increase of the volume, thought illogical with drain of ideas and ideas delirious, denying hallucinations and illusions, with risk of self-harm and heteroaggression, altered judgment, no insight.

Psychiatric hospitalization is carried out for diagnostic, therapeutic and risk containment adaptation, an approach is carried out for a patient with a psychotic episode. of first time having a diagnosis of manic episode with mixed characteristics, according to the criteria of the Manual diagnosis and statistical of the disorders mental in its Sta. Edition (DSM-5) (APA, 2015) with laboratory studies consisting of blood biometry, blood chemistry, blood function tests, general urine examination, thyroid profile, 5-element urine screening (THC, opioids, benzodiazepines, methamphetamines and amphetamines), viral panel for HBV, HCV and HIV1NIH2, as well as computed axial tomography skull, electroencephalogram, as well as vitamin B12 levels and SARS-coV-2 PCR as a general protocol for patients admitted to our hospital due to the current pandemic, presenting results without alterations, in terms of CT without tumor or evidence of hemorrhage. [eleven]

During internment of fifteen days sample effects adverse to neuroleptics such as slight stiffness in extremities and fine tremor as well as progressive clinical improvement with the already mentioned scheme, leaving conscious, alert, oriented in 3 spheres, immediate memory without alterations, recent and remote alterations, language without alterations, in euthymia with corresponding mood, thinking logical experience content!, normal clinical intelligence, normal general information background, denying hallucinations, delusions and illusions, without alterations in instinctive life, denies ideas of self-aggression and heteroaggression.

Montreal Cognitive Assessment (MOCA) test is applied with a result of 24 points.

Mania of start late: a diagnosis of exclusion.

The rate of prevalence of the bipolar I disorder in the general population decreases 1.4% in people aged 18 to 44 years to 0.1-0.4% in the group aged 65 years or older, early diagnosis and accurate is difficult in the practice clinic, already that the appearance of the disorder
bipolar HE characterizes Commonly by symptoms nonspecific. [7 - 9]

Mania is an emergency psychiatric which is most frequently observed in BD-I and schizoaffective disorder, but brain conditions or neurosurgical procedures can also produce secondary mania. [3]

A manic episode can occur at any age, and late-onset mania is defined as the first mania in life in patients over 50 years of age; The prevalence of late-onset mania in hospitalized patients is approximately 6.0%; approximately one-third of elderly patients with mania are experiencing their first manic episode, as is the case with our patient. The possible differences between the disorder bipolar early start and late They have been the subject of many studies. Much less researched is late-onset mania, which has a heterogeneous origin, such as late-onset bipolar disorder, pre-existing depressive disorder that develops into bipolar disorder, or secondary mania caused by illness or medication. [9]

No there is studies that describe systematically the etiopathogenesis and the course of mania in old age which makes diagnosis difficult. In these patients, neurological abnormalities have been reported in 17-36%. With increasing age, manic symptoms increasingly show underlying organic factors. For these cases, the concept of “secondary mania,” meaning: a mania caused by a somatic condition or the use of medications. [9]

The factors somatic they can be a true cause of mania (mania secondary), which should be considered especially in patients with a first manic episode after of the 40 years, mania with symptoms atypical (p. e.g. hallucinations visuals or
olfactory) or atypical course (e.g. e.g. single manic episode or refractory mania), or neurological symptoms. [3 . 1st]

So same these factors somatic they can trigger mania as first demonstration of disorder bipolar in a person with vulnerability latent and with either

no history of depressive episodes. Somatic comorbidity can also be a chance finding without any causal relationship with mania. [10]

The diagnosis in a patient with mania at a late age focuses on detecting or excluding bipolar disorder, an organic condition (affective disorder due to neurological, systemic or substance causes, and delirium), and dementia. [10]

This includes a good history and interview with the family, complete physical and neurological examination, laboratory studies, brain imaging studies, electroencephalogram, application of MMSE or neuropsychological tests if necessary. [10]

Such causes to discard include: event cerebral vascular, tumor cerebral, head trauma, epilepsy, multiple sclerosis, encephalopathy, infection (including syphilis, HIV, HBV, HCV and cryptococcosis), systemic hyperthyroidism, Addison's disease, Cushing's disease, vitamin B12 deficiency, anemia, depression with agitation, use of corticosteroids, dopamine agonists, sympathomimetics, antibiotics, estrogens, thyroid hormone, antidepressants, atypical antipsychotics, benzodiazepines, drugs (especially cocaine and amphetamines), alcohol and anabolic steroids. [10]

If secondary mania is suspected, the underlying condition and symptomatic treatment should be addressed first. may also be necessary and is basically the same as the treatment of mania in the context of bipolar disorder, one must take consider the elderly susceptibility to the effects secondary, the first goal is stabilization and the reduction of symptoms. [10]

In a single manic episode in which an organic cause has been demonstrated and has occurred a recovery complete symptomatic and functional, No there is indication for maintenance treatment preventive. [10]

Regarding the neurobiological alterations associated with bipolar disorder type one, thanks to studies functional NMR has found that the typical locations of The lesions fit current models of bipolar disorder, which involve hyperactivity of left hemisphere reward-processing brain areas and hypoactivity of bilateral prefrontal emotion-modulating regions. [3]

Injury studies complement these models by suggesting that the hypoactivity of the brain limbic of the hemisphere right, either a imbalance left/right, can be relevant for the pathophysiology of mania, mainly frontal regions, regions cerebral limbic temporary and subcortical, in particular the

prefrontal cortex, the medial temporal lobe, and the basal ganglia and their connecting pathways. [3]

Causation between lesions and mania is most clearly demonstrable when the lesions are acute, discrete, and circumscribed (e.g., stroke and neoplasms). [3]

The contribution of lesion studies is to demonstrate that the structural brain lesions most reliably associated with secondary mania (namely, stroke and brain tumors) are clearly associated with hypofunction, thus providing support for this hypothesis. [3]

In both primary BD-1 and secondary mania, the balance of brain activity seems be displaced toward the hyperactivity of the side left (for example, on the left side epileptogenic foci) and/ or hypoactivity on the right side (e.g., right-sided heart attacks). In contrast, several studies show that secondary depression is associated with destructive lesions of the left hemisphere. [3]

This seems be expected, already that BD-1 is almost with certainty a disease heterogeneous, and even the most consistent imaging findings of left-sided limbic hyperactivity and right-sided structural lesions will not explain the pathophysiology of mania in all patients. [3]

Integration of the case and conclusion:

Given this information, we know that brain tumors can cause psychiatric symptoms, in this case episodes of secondary mania, in patients with or without an emotional history, and that these lesions may or may not have a possible causality in the outbreak of the disorder. chart maniac of first time, in patients with starting manias late or with atypical or neurological data, the intentional search for secondary causes is a priority, likewise in our patient with a history of left frontotemporal meningioma and subsequent surgical resection and previous depressive symptoms, subsequent manic symptoms late, no left clear the relationship between the injury and the specific causality, it can be hypothesized that his first manic episode had a secondary to his tumor or resection prior to or due to his late presentation of a bipolar disorder given his emotional history and that his previous tumor has contributed to his vulnerability to developing said condition given the advances in functional and neurobiological studies of the circuits involved in it disorder bipolar, specifically the mania in the which HE ha involved the hypoactivity of right frontotemporal areas and the hyperactivity of the left ones, given this is priority continue with the investigation of this guy of cases for

elucidate the associations between injuries cerebral and the mechanisms neurobiological involved in the mania either in he disorder bipolar, already be in paintings secondary and in bipolar disorder both early and late onset.

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111 Psychiatry

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