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When the Brain Speaks through the Mind: An Intraventricular Neurocytoma Revealed by Psychiatric Symptoms - A Rare Case Report

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Abstract **Case Report**

Central neurocytoma is a rare neuroepithelial tumor, accounting for less than 1% of all intracranial neoplasms. It is most frequently located in the lateral ventricles, near the foramen of Monro, and usually manifests with signs of increased intracranial pressure or visual disturbances. Psychiatric onset is exceptional and can lead to diagnostic delay. We report the case of a 34-year-old man with no previous psychiatric history who presented with recent behavioral and mood disturbances suggestive of a manic episode. Despite appropriate pharmacological treatment, symptoms persisted, prompting brain MRI, which revealed a left intraventricular mass measuring 3.5 cm. Surgical resection achieved complete removal, and histopathological examination confirmed a central neurocytoma (WHO grade II). The postoperative course was favorable, with full remission of psychiatric symptoms. This case illustrates how a benign brain lesion can mimic a primary psychiatric disorder, emphasizing the need for neuroimaging in atypical or treatment-resistant psychiatric presentations. A multidisciplinary approach between psychiatry, neurology, and neurosurgery is essential for early diagnosis and optimal outcomes [1–4].

Keywords: Central neurocytoma; Intraventricular tumor; Psychiatric symptoms; Mania; Brain MRI; Neuropsychiatry; Case report.

INTRODUCTION

Central neurocytoma (CN) is a rare neuronal tumor first described by Hassoun *et al.* in 1982 [1]. It represents less than 1% of all intracranial neoplasms and typically arises within the lateral ventricles near the foramen of Monro [2, 3]. CN primarily affects young adults in the second to fourth decades of life, with no gender predominance [4, 5]. Histologically, it shows neuronal differentiation and is classified as a WHO grade II tumor, generally benign and slow-growing [6, 7], though recurrence may occur after incomplete resection [8].

Clinically, CN often manifests through intracranial hypertension headache, nausea, vomiting, or papilledema resulting from obstructive hydrocephalus [9]. Other symptoms may include visual or memory disturbances due to involvement of nearby structures [10]. Psychiatric onset, however, is extremely rare and may involve mood disorders, psychosis, or behavioral disinhibition related to fronto-subcortical dysfunction [11,12]. Such atypical presentations can mislead clinicians and delay neuroimaging [13]. Recognizing this possibility underscores the need for collaboration between psychiatry, neurology, and neurosurgery.

CASE PRESENTATION

A 34-year-old right-handed male, with no prior medical, neurological, or psychiatric history, was referred to the psychiatric department for recent-onset behavioral and mood disturbances. Over the course of three weeks, his family reported marked psychomotor agitation, excessive talkativeness (logorrhea), irritability, decreased need for sleep, and disinhibited behavior. The patient exhibited inflated self-esteem, impulsive spending, and grandiose delusional ideas consistent with a manic syndrome according to DSM-5 criteria. There was no history of substance use, cranial trauma, or medication intake that could explain the clinical presentation.

The initial mental status examination revealed disorganized speech, euphoric affect, pressured speech, and distractibility, without psychotic features or

cognitive impairment at that stage. Vital signs and neurological examination were unremarkable. Laboratory tests—including complete blood count, metabolic profile, thyroid function, and toxicology screening—were within normal limits.

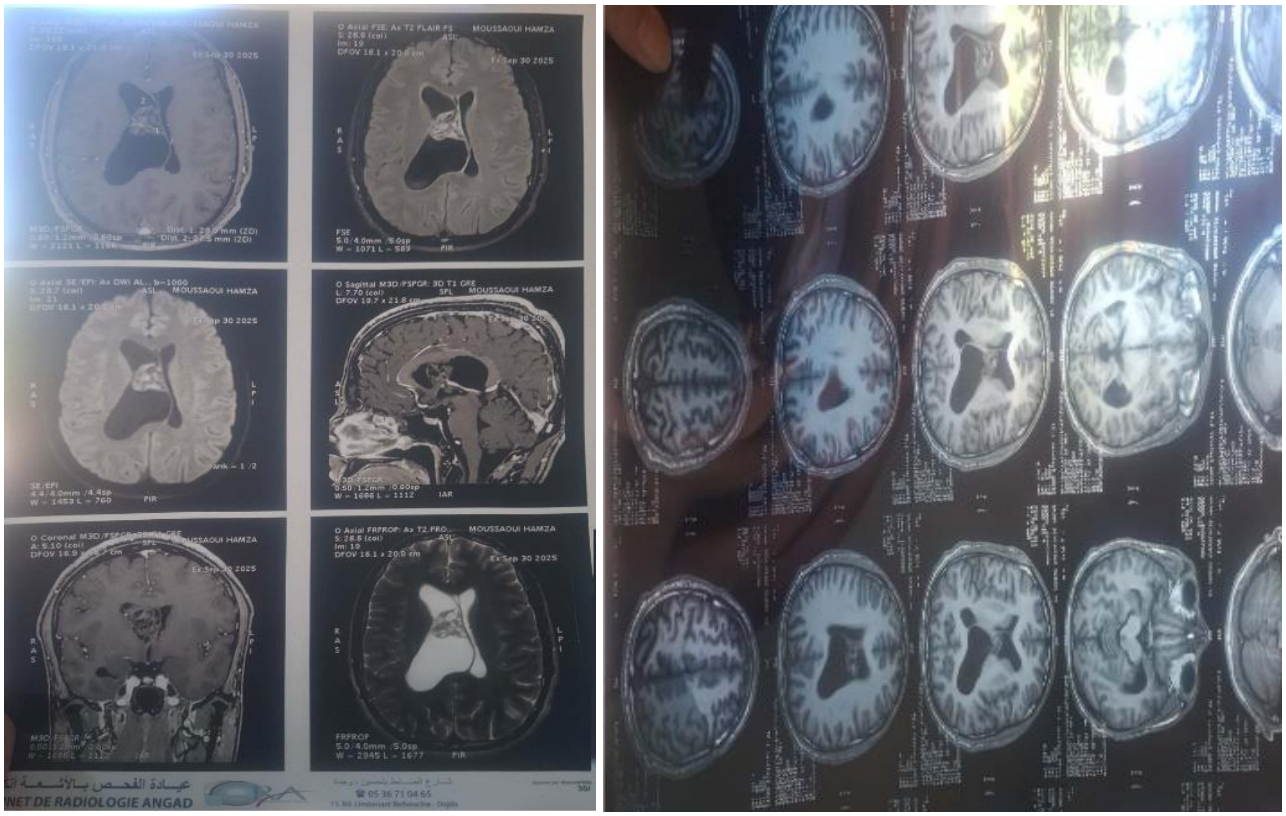
The patient was started on sodium valproate (1000 mg/day) and olanzapine (10 mg/day) under the diagnosis of a manic episode. However, after two weeks of treatment, no clinical improvement was observed, and new symptoms emerged, including memory lapses, decreased attention, and intermittent headaches. This lack of therapeutic response raised suspicion of an underlying organic cause, prompting further neuroimaging evaluation.

Magnetic resonance imaging (MRI) of the brain demonstrated a well-defined intraventricular lesion located in the left lateral ventricle, adjacent to the foramen of Monro. The mass measured approximately 3.5 × 3 cm, appeared isointense on T1-weighted sequences, hyperintense on T2-weighted and FLAIR images, and showed heterogeneous enhancement after gadolinium injection. There was a mild mass effect on the surrounding structures with partial obstruction of cerebrospinal fluid flow, but no peritumoral edema or hemorrhage.

The patient underwent left transcallosal microsurgical resection of the tumor. Intraoperative findings revealed a grayish, moderately vascularized, soft tumor, well-demarcated from the adjacent ventricular walls. Gross total resection was achieved without complications.

Histopathological examination of the specimen showed uniform round cells with small round nuclei, finely granular chromatin, and perinuclear halos arranged in sheets and clusters. Immunohistochemistry was positive for synaptophysin and neuron-specific enolase (NSE), while negative for glial fibrillary acidic protein (GFAP), confirming the diagnosis of a central neurocytoma (WHO Grade II).

The postoperative course was uneventful. The patient experienced complete remission of psychiatric symptoms within two weeks after surgery, along with gradual cognitive recovery confirmed by neuropsychological assessment. Follow-up MRI at six months showed no residual or recurrent tumor. The patient remained asymptomatic and psychiatrically stable during one year of follow-up under multidisciplinary care involving psychiatry and neurosurgery.



IRM Cérébral

DISCUSSION

Central neurocytoma (CN) is a rare benign neuronal tumor that usually presents with neurological symptoms such as headache, signs of intracranial hypertension, or visual disturbances caused by obstructive hydrocephalus [1,2]. However, psychiatric manifestations as the first clinical sign remain extremely

uncommon, with only a few cases reported in the literature [3–6]. These atypical presentations may result from the tumor's anatomical location and interference with the fronto-subcortical circuits, which are critically involved in the regulation of behavior, affect, and executive functions [7,8].

Lesions of the frontal lobes, caudate nucleus, and anterior limbic system can manifest through behavioral disinhibition, mood elevation, or psychotic symptoms, mimicking primary psychiatric disorders such as mania or schizophrenia [9,10]. In our case, the intraventricular location of the tumor, close to the left frontal horn and caudate nucleus, likely explains the manic-like presentation, as described in other reports of frontal or subcortical involvement [11,12].

The delay in diagnosis is often due to the absence of neurological signs and the predominance of psychiatric symptoms, leading to initial psychiatric management without neuroimaging [13]. Several authors emphasize the need for systematic brain imaging, particularly MRI, in any case of atypical, resistant, or late-onset psychiatric syndrome [14,15]. In the present case, the absence of improvement after adequate mood-stabilizing therapy and the appearance of cognitive symptoms were key elements that prompted further investigation.

Radiologically, CN typically appears as a well-circumscribed, intraventricular mass, often attached to the septum pellucidum or the ventricular wall, isointense on T1-weighted and hyperintense on T2-weighted MRI, with heterogeneous enhancement after gadolinium injection [16,17]. These features may resemble other intraventricular tumors such as oligodendroglioma, ependymoma, or subependymoma, making histopathological confirmation essential [18].

Histologically, CN exhibits small, uniform, round cells with neuronal differentiation, and immunohistochemical staining is positive for synaptophysin and neuron-specific enolase (NSE), but negative for glial fibrillary acidic protein (GFAP), distinguishing it from glial tumors [19,20]. The WHO classifies CN as a Grade II neuronal tumor, with generally favorable prognosis when gross total resection is achieved [21].

Surgical resection remains the treatment of choice and is usually curative. Several studies have shown excellent long-term survival rates exceeding 90 % after total removal [22]. In cases of incomplete excision, adjuvant radiotherapy may reduce recurrence risk [23]. Postoperative follow-up with periodic MRI every 6 to 12 months is recommended, as recurrence may occur years after surgery [24].

From a neuropsychiatric standpoint, this case underscores the tight interplay between brain structure and mental function, illustrating how an organic lesion can mimic a primary psychiatric disorder. Early recognition of such atypical cases is crucial for timely neurosurgical intervention and prevention of prolonged psychiatric morbidity.

In summary, this observation emphasizes the importance of multidisciplinary collaboration between psychiatry, neurology, and neurosurgery in the assessment of new-onset, treatment-resistant, or atypical psychiatric presentations. The overall prognosis remains favorable, particularly when total surgical resection is achieved.

CONCLUSION

This observation highlights the close interplay between the brain and mind. Although rare, central neurocytoma can manifest solely through psychiatric symptoms such as mood or behavioral changes [3–6]. In the absence of neurological signs, clinicians must maintain a high index of suspicion and perform neuroimaging in atypical, resistant, or late-onset psychiatric cases [14–16]. Collaboration among psychiatrists, neurologists, radiologists, and neurosurgeons enables early detection, proper surgical management, and favorable outcomes [11, 17, 21].

Recognizing such atypical presentations reinforces the importance of a neuropsychiatric approach that bridges psychiatry and neurology, ensuring timely diagnosis and improved quality of life [7, 8, 20].

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