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Panic Disorder Revealing Congenital Hydrocephalus: A Propos D'un Cas Clinique

Tbatou Laila[1*], Stati Soukaina[1], Chaara aya[1], el Moussaoui Naouar[1], Nafiaa Hind[1], Ouanass Abderrazzak[1]

[1]Research Team of Psychiatry, Medical Psychology and History of Medicine Faculty of Medicine and Pharmacy, Mohammed V University, Rabat

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***Corresponding author:** Tbatou Laila

Abstract **Case Report**

We report the case of a 23-year-old patient with congenital tri-ventricular hydrocephalus of incidental finding during a pretherapeutic workup requested by a comorbid social anxiety disorder with chronic headache and extremity tremor. ***Discussion:*** Although the clinical picture is suggestive of a panic disorder, some elements of the observation suggest that the psychiatric symptoms are secondary to the chronic hydrocephalus. These elements are discussed in the light of the existing literature on the links between psychiatric disorders and hydrocephalus and of neuroimaging studies finding ventricular enlargement in patients suffering from anxiety disorders. ***Conclusion:*** There are links between hydrocephalus and psychiatric manifestations. A better understanding of these links could shed light on the etiopathogeny of anxiety disorders.

Keywords: Panic Disorder Congenital Hydrocephalus.

1. INTRODUCTION

Panic disorder is a common anxiety disorder, characterized by the presence of recurrent and unexpected panic attacks generating persistent anxiety, its prevalence is high in the general population estimated at 1.3%. Its onset is usually early in young adults. This disorder has a chronic evolution with an index of chronicity ranging from 71 to 80%.

The presence of psychiatric symptoms in a patient suffering from neurological pathologies is frequent. In clinical psychiatric practice, it is necessary to know how to look for and identify neurological pathologies that may express themselves, during their evolution, by psychiatric symptoms by means of a standardized neurological examination to be completed by imaging examinations, in order to help the specific diagnostic and therapeutic approach, which is often complex for the psychiatrist.

Hydrocephalus is a neurological condition characterized by pathological enlargement of the cerebrospinal fluid (CSF)-containing ventricles of the brain, usually due to obstructed flow or reduced CSF absorption. Chronic hydrocephalus can lead to motor problems, including gait instability and abnormal movements of the arms and hands [1]. Memory and more subtle cognitive deficits are also observed.

In the clinical setting, patients with hydrocephalus frequently present with affective and emotional disorders, the most frequently observed neuropsychiatric symptoms were apathy, followed by anxiety [2], the course of which is positively correlated with the anxiety response, including panic disorder and social anxiety.

2. METHODS

Through a clinical case and a brief review of the literature based on current research, we will review the anxious manifestations accompanying chronic hydrocephalus and the causal links between hydrocephalus and anxiety disorders as well as the pharmacological treatment in these manifestations.

To extend this questioning, it is interesting to note that some authors have put forward the hypothesis that hydrocephalus would be linked to an alteration of the mechanics and dynamics of the CSF [3], which is responsible for the psychiatric symptomatology.

3. PRESENTATION OF A CASE

Mr. A. Y is 23 years old, single, native and resident of Salé, without children and living with his mother in an apartment. He works as a cab driver. According to his mother, he has always been isolated, shy and introverted since his childhood.

Mr A.Y presented himself in psychiatric consultation of the Ar-Razi hospital, 7 months ago for the management of a rapidly progressive installation of a strong feeling of anxiety associating spontaneous panic attacks or triggered by certain situations such as his presence in front of an agent of authority or in front of police barricades, as well as an exaggerated fear of the crowd, an intense and durable fear of the contact with others and trembling while speaking in front of an audience with a tendency to avoid the social situations.

The symptomatology was complicated by neurological signs of chronic headache, photophobia and tremor of the extremities. The diagnosis of a panic disorder comorbid with social anxiety was retained according to the DSM 5 criteria [5]. An evaluation using the HAMILTON anxiety scale was performed, showing an anxiety with a score of 31, indicating severe to profound anxiety. A pre-therapeutic workup was requested, including a CBC, complete ionogram, lipid and thyroid workup, liver, syphilis and HIV serology, and an electrocardiogram.

The results of the biological check-up and the ECG came back without any particularity and the patient was put on paroxetine 20 mg /d and alprazolam in progressive degression with referral to a psychologist for cognitive-behavioral therapy sessions.The evolution was marked by a slight improvement of the psychiatric symptomatology. The HAMILTON scale of evaluation of control anxiety was done, the score went from 31 to 25, for a duration of 45 days, but without improvement of headaches or tremors of the extremities. Thus a brain scan was requested showing tri-ventricular hydrocephalus without signs of transependymal resorption. The patient was referred to a neurologist for evaluation and management.

During a neurological consultation, a brain MRI, a fundus and a visual field evaluation were requested by the treating neurologist.The results of the workup confirmed tri-ventricular hydrocephalus and showed a stenosis of the aqueduct of Sylvius. The evaluation of the bi-ocular visual field showed a relative diffuse deficit with peripheral scotomas in the right eye and a borderline normal visual field in the left eye.

Thus the patient was put on acetazolamide (diamox) 250mg daily, with regular monitoring. Later, the patient was seen in consultation, he was put on venlafaxine 75mg per day due to the slight clinical improvement and the side effects of paroxetine (erectile dysfunction).

The evolution was marked by a clinical improvement with a change in the Beck score from 16 to 8 and the HAMILTON anxiety assessment scale was done showing an anxiety with a score of 20. Since he was put on acetazolamide (diamox), his clinical condition has stabilized, with no recent changes, and there were no additional neurological clinical signs that would have led to fears of worsening hydrocephalus and/or intracranial hypertension. Puncture or placement of an internal ventricular shunt to evacuate excess fluid was suggested to our patient in case of headache exacerbation.

4. SUMMARY

Mr A Y presented a tri-ventricular hydrocephalus without signs of transependymal resorption, due to a congenital stenosis of the aqueduct of Sylvius, asymptomatic and untreated by shunt until the age of 23 years, the inaugural clinical picture is purely psychiatric. The hydrocephalus was associated with intracranial hypertension.

In the majority of cases, intracranial hypertension is a neurological emergency [4]. However, in Mr AY, the intracranial hypertension was progressive, with minimal CSF resorption and was not well tolerated clinically, producing chronic headaches and photophobia, but stabilized to date with medical treatment (diamox) and Venlafaxine. It is therefore possible that the anxiety symptomatology was secondary to the resulting structural change in intracranial hypertension.

5. DISCUSSION

The pathophysiological mechanisms of hydrocephalus-induced brain injury result in significant changes in the brain, not only in its morphological characteristics, but also in its circulation, biochemistry, metabolism, and maturation [5]. The ventricles compress the surrounding parenchyma, cause changes in cerebral blood flow and neurochemistry in various neurotransmitters and metabolites [6].

Adjacent to the ventricular system are the mesolimbic and mesocortical dopaminergic system, which innervates the nucleus accumbens, and the prefrontal and anterior cingulate cortex system [7]. These areas are involved in fear and anxiety and are innervated by dopaminergic fibers from the Ventral Tegmental Area (VTA) and the medial part of the compact gray matter [8], which are sensitive to ventricular dilation in chronic hydrocephalus. In this study, we noted a significant increase in the anxiety response in hydrocephalic rats, suggesting that a deficit in the dopaminergic systems of the VTA is related to anxiety.

Several reports have documented functional damage to dopaminergic cells and decreased dopamine in experimental models of hydrocephalus [9].

In a 2001 study by Webster RA on Neurotransmitters, Drugs and Brain Function found decreased tyrosine hydroxylase (TH) immunoreactivity in VTA in the hydrocephalus, thereby limiting the rate of catecholamine synthesis, including dopamine [7]. TH is a rate-limiting enzyme in the process of dopamine and norepinephrine production. As hydrocephalus developed, the number of tyrosine hydroxylase neurons decreased significantly as well as the intensity of axonal fibers.

Other authors [10] reported that normal rats under stress showed decreased tyrosine hydroxylase expression in the amygdala and elevated glucocorticoids. They demonstrated that anxiety induction involved modulation of the central dopaminergic system in the amygdala.

In a study involving patients with idiopathic hydrocephalus. In a study involving patients with idiopathic hydrocephalus [2], they observed that a quarter of the patients with idiopathic hydrocephalus have anxiety-like behavior. The authors mentioned that the anxious symptoms could be attributed to brain lesions in the anterior cingulate cortex and orbitofrontal area.

In addition, a previous clinical study suggested that hypoperfusion in the anterior cingulate cortex and thalamus may be involved in the anxiety response in patients with idiopathic hydrocephalus [2].

The results of a study demonstrate that hydrocephalus produces a significant increase in cholecystokinin (CCK) expression in periventricular hypothalamic areas, including the arcuate nucleus, CCK and agonists of its receptors have anxiogenic effects, play an important role in anxiety-related behavior [11].

They found that CCK in the hypothalamus inhibits the dopaminergic system, which may implicate the anxiogenic effect in a potential CCK-dopamine interaction. Concomitantly, hydrocephalic rats showed a substantial decrease in neuropeptide Y (NPY) immunoreactivity in all these areas, as NPY is well known for its stress-related anxiolytic effects [12].

Changes in CCK expression and NPY expression may reflect the anxiogenic effects of hydrocephalus progression. In a recent study [13], reported an interaction between dopamine and CCK in the hypothalamus.

Data found in animal experimental studies suggest that dilation of the ventricle in animals with induced hydrocephalus may result in increased anxiety responses and functional dopaminergic impairment in the VTA.

Hydrocephalus can lead to motor problems, including gait instability as well as arm and hand dysfunction [1].

As hydrocephalus progressed, affected rats decreased activity and locomotion, exhibited spasticity in the extremities, and showed little weight gain compared to control cases.

All abnormal symptoms except decreased activity subsequently resolved in most animals. Rats with chronic marked hydrocephalus exhibited hindlimb paraparesis with spasticity, slow movements, and finally chronic lethargy.

Structural and/or functional lesions of the neostriatum can result in motor functions, including abnormal gait and posture, as well as intellectual and emotional impairment. These changes have been well documented in Parkinson's disease and other neurodegenerative disorders [14].

The basal ganglia are located adjacent to the ventricular system and the respiratory system, play an important role in reinforcement and emotional processing, as well as in the initiation and control of voluntary movements [15].

The abnormal behavior that developed resolved over time. Similarly, in a clinical setting, residual abnormalities in patients with chronic hydrocephalus may be too subtle to lead to typical parkinsonism .Studies have indicated that parkinsonian symptoms do not appear in patients until there is an 80% or greater decline in dopamine levels in the striatum and substantia nigra [16].

Hydrocephalus is characterized by diffuse cortical and subcortical lesions, which distinguishes it from other pathologies resulting from discrete lesions [17]. Cognitive impairment occurs not only in the pediatric population, but also as a secondary symptom in patients with chronic hydrocephalus. It is well documented that children with hydrocephalus can benefit from shunt surgery and that CSF shunting has improved cognitive impairment.

Intellectual impairment has been linked to altered neuronal innervation in the septo-hippocampal cholinergic nuclei of the basal forebrain (learning and memory impairments), the dopaminergic ATV (emotional control), locus ceruleus, and noradrenergic subcerulus is involved in cognition [18].

6. CONCLUSIONS

Through this clinical case, we found that there would be a psychopathological link between hydrocephalus and anxiety disorders in which the progression of hydrocephalus was positively correlated with the anxiety response.

Further research is needed to clarify the interactions between the various components of the neurotransmitter systems.

Appropriate management must be multidisciplinary, including neurology, neurosurgery, and psychiatry, to prevent negative impact on all areas of these patients' lives.

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