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Ventriculoperitoneal Shunt-Associated Neuropsychiatric Symptoms: A Literature Review and Two Case Reports

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Abstract

Ventriculoperitoneal (VP) shunts are a common use for the treatment of hydrocephalus, which is a condition causing excessive cerebrospinal fluid (CSF) buildup in the brain. The shunt drains the CSF from the brain and into the abdomen, where it can be absorbed by the body. Although shunt placement is a widely used and successful procedure, it is unclear whether neurosurgical placement may directly cause psychiatric abnormalities when considering the dopaminergic system.
In this paper, we first review relevant literature and discuss the dopamine circuitry in the brain. The literature suggests a shunt-associated clinical syndrome, which may include a reduction in facial and/or verbal expressivity, reduced willful movement, rigidity, and vertical gaze palsy. Typical presentation occurs within three months of a shunt revision. In patients with a VP shunt, these signs may be a strong indicator of mechanical or functional shunt malfunction.

We then present two cases of hydrocephalus managed with a VP shunt, where both patients developed symptoms suggestive of psychosis and/or dopamine pathway pathology after shunt placement. We conclude that clinicians should have a suspicion for shunt-associated symptomology in patients with a history of recently treated hydrocephalus who present with psychomotor, personality, or cognitive changes.

Keywords: Ventriculoperitoneal-Shunt, Hydrocephalus, Parkinsonism, Catatonia, Psychosis

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1. Introduction

Cerebrospinal fluid (CSF) volume and pressure in the ventricular system operate in a homeostatic balance between secretion, flow, and absorption. CSF produced in the lateral ventricles travels through the interventricular foramina of Monro, into the third ventricle, through the cerebral aqueduct of Sylvius, and into the fourth ventricle. It then flows through the median and lateral apertures of Magendie and Luschka into the subarachnoid space, which leads to its absorption by the arachnoid granulations in the dural venous sinuses (1). Hydrocephalus can have many causes, including overproduction, congenital malformation, intraventricular hemorrhage, cystic lesions or tumors leading to obstruction, aqueductal stenosis, subarachnoid hemorrhage, or trauma (2). Clinical symptoms in adults can include nausea/vomiting, headache, or lethargy, and a commonly seen physical sign is papilledema (2). Surgical treatment occurs through three major procedures. First, the choroid plexus may be removed. While a choroid plexectomy is less common than other procedures, interest in this technique has increased in select patients with CSF overproduction (3, 4). Second, endoscopic third ventriculostomy can provide an outflow route from the third ventricle to the basal (interpeduncular) cistern (2). Third, and most common, extracranial shunting places a catheter between the ventricular system and another body cavity, commonly the peritoneum (2). Both patients described in this paper were treated surgically via ventriculoperitoneal (VP) shunting. Shunt malfunction can occur by a variety of mechanisms, including infection and blockage (2). More rare causes have been reported, including neurosarcoid infiltrate, knot formation, and functional etiologies involving transient increases in intra-abdominal pressure during severe constipation (5-8).

Hydrocephalus, particularly when managed with cerebrospinal fluid diversion, can affect neural systems beyond ventricular morphology. The ascending reticular activating system (ARAS), a core structural network for arousal, comprises monoaminergic, cholinergic, and glutamatergic nuclei within the brainstem and midbrain that project to the thalamus, hypothalamus, and basal forebrain, all situated near the third and fourth ventricles (9). Because of this proximity, alterations in cerebrospinal fluid pressure may transiently influence these circuits, contributing to the motivational and arousal disturbances observed in shunt-treated patients. Diffusion tensor tractography (DTT) demonstrates that periventricular arousal pathways can reorganize after shunting. In one case, a patient with traumatic brain injury and secondary hydrocephalus recovered from a vegetative to a minimally conscious state following shunt placement, with increased thalamic connectivity to the hypothalamus, basal forebrain, and frontal cortices (10). Dopaminergic involvement is also implicated in hydrocephalus-related behavioral syndromes. An 11-year-old with obstructive hydrocephalus developed akinetic mutism after subdural hematoma evacuation. Despite normal presynaptic dopamine imaging and cerebrospinal fluid homovanillic acid levels, symptoms improved with bromocriptine, suggesting pressure-related dysfunction of periventricular monoaminergic projections in the thalamus and hypothalamus (11).

Overlaying this, the mesolimbic, mesocortical, and nigrostriatal dopaminergic circuits reside in tight proximity to ventricular structures. The nigrostriatal dopaminergic system, which extends from the substantia nigra pars compacta to the dorsal striatum, can also be perturbed by ventricular pressure changes. Parkinsonism after ventriculoperitoneal shunting has been linked to reversible presynaptic dopaminergic dysfunction secondary to ventricular fluctuations or distortion, with levodopa responsiveness supporting a transient rather than degenerative mechanism (12). Disruption of dopaminergically modulated networks that connect the ventral tegmental area, anterior cingulate cortex, basal ganglia, and medial thalamus can impair the integration of emotional drive and goal-directed behavior. These circuits rely on dopaminergic input as an “energizing factor” for initiating speech and movement. Loss of this input can produce syndromes such as akinetic mutism, catatonia, or parkinsonism, reflecting a collapse of frontal–subcortical balance (13).

2. Literature Review

A growing body of case reports has reported abnormal psychomotor signs and symptoms associated with extracranial shunts. Considering how extracranial shunting is a popular treatment plan for patients affected by hydrocephalus, the literature theorizes that the affiliated neurological and psychiatric symptoms may be related to

hydrocephalus, the shunt itself, or a combination thereof (14).

Table 1 shows 39 reported cases in English language journals that describe the following clinical syndromes in patients with shunt-treated hydrocephalus: akinetic mutism, Parkinsonism, catatonia, blepharoclonus, frozen movement, psychosis, major neurocognitive disorder, and global rostral midbrain syndrome (7-50). Case reports describing normal pressure hydrocephalus, a different clinical syndrome, were excluded. The table represents a specific point in time at which these clinical syndromes were present and does not represent the entire clinical picture of each patient.
Thirty-five cases displayed psychomotor symptomology within three months of shunt placement or revision. Twenty-one cases use the term Parkinsonism, seven use the term akinetic mutism, five describe catatonia, and two describe "frozen movement." Twenty-one prior cases describe rigidity. Sixteen describe mutism, and another four describe hypophonia. Two describe symptomology consistent with Global Rostral Midbrain Syndrome (GRMS). Nineteen describes Parinaud's syndrome or vertical gaze palsy. Seventeen describe bradykinesia, hypokinesia, or akinesia. Nine describe expressionless facies or hypomimia. Upper motor neuron signs are seen in several cases, with nine reports of positive Babinski sign and six reports of hyperreflexia. Additional specific symptoms specific to each case report are listed where available. Twelve were responsive to further surgical intervention, including shunt revision or endoscopic third ventriculostomy. Twenty-four cases reported at least a partial response to pro-dopaminergic therapy. The clinical symptoms displayed in Table 1 lend support to the idea that neuropsychiatric symptoms may be related to the placement of the VP shunt.

Interestingly, freezing of gait is not traditionally seen in obstructive or congenital hydrocephalus, but is relatively common in idiopathic normal pressure hydrocephalus (iNPH). A 2024 meta-analysis tested this hypothesis by examining 230 pre- and postoperative video recordings of 139 patients with iNPH who underwent VP shunt surgery. Clinical observers and physiotherapists measured freezing of gait (FoG), a fundamental factor of parkinsonism and found that FoG occurred less frequently post-surgery compared to pre-surgery. However, 8% of patients still displayed FoG after VP shunt surgery (42).

However, there are a few cases in which FoG was seen in obstructive hydrocephalus. A 28-year-old male with a history of non-tumoral aqueductal stenosis presented with profound hypokinesis, best described as freezing of movement, after a third ventriculostomy following failed VP shunt. The patient was administered levodopa/carbidopa therapy, and symptoms improved to baseline wihin eight weeks (27). This report illustrated how dopaminergic agents may be useful in FoG following corrective surgery for aqueductal stenosis causing obstructive hydrocephalus.
Additionally, extrapyramidal symptoms like FoG are generally more common in patients with Parkinson’s disease than in those with hydrocephalus (43). Thus, variation in psychomotor symptoms may be attributed to the presence of the VP shunt, warranting further investigation.

One hypothesis suggests that VP shunts disrupt the internal CSF pressure gradient, increasing local pressure to the ventral midbrain and inducing global rostral midbrain dysfunction. A 19-year-old male presented with chronic headache and depression before undergoing VP shunt surgery. Three months after insertion, he was hospitalized with complaints of neurological dysfunction and drowsiness, which rapidly alleviated after recalibrating the shunt setting and reducing CSF pressure. Six months after insertion, he returned to the hospital with severe parkinsonism and akinetic mutism. After treatment with dopaminergic medications, levodopa and pramipexole, he underwent endoscopic third ventriculostomy to correct the abnormal pressure gradient and resolve psychomotor symptoms (44).

Further evidence supporting this theory is from a 2024 case study. A 52-year-old man presented with “severe consciousness disturbance” after repeated episodes of VP shunt malfunction and levodopa-resistant parkinsonism. Magnetic resonance imaging (MRI) indicated an elevated pontomesencephalic angle (PMA), which the authors attributed to a shunt-induced disruption of the CSF pressure gradient. Performance of endoscopic third ventriculostomy returned CSF flow to normal and corrected the elevated PMA, suggesting that the neuropsychiatric symptoms may have been related to shunt function (45).

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On one occasion, a 76-year-old female patient with significant akinetic mutism received VP shunt surgery for hydrocephalus following a subarachnoid hemorrhage. Her neurosurgeon noted significant bilateral injury to the pre-fronto-caudate tract (PCT), which is commonly considered a mechanism of akinetic mutism. The care team did not observe significant alterations in the patient's akinetic mutism before or after the shunt operation; however, her PCT injuries began to undergo spontaneous resolution. Shortly after PCT recovery, the patient's akinetic mutism diminished, and her normal speech capabilities returned (46).

A 2021 report details three cases of Global Rostral Midbrain Syndrome (GRMS), which contain significant abnormal neuropsychiatric symptoms. Each patient presented with GRMS following a successful VP shunt installation; spontaneous corpus callosum infarction began soon after. Over the months prior to their hospitalizations, each patient developed repeated shunt dysfunction that ultimately evolved into parkinsonism, akinetic mutism, and other signs of cognitive impairment. While parkinsonism-based symptoms responded to levodopa, other symptoms persisted. The authors noted that these cases are likely unrelated to observations of transtentorial pressure gradient behavior after shunt blockage. Rather, they suspected that the reverse transtentorial pressure gradient due to shunt overdrainage was the primary cause (47).

This literature suggests a shunt-associated clinical syndrome, which may include a reduction in facial and/or verbal expressivity, reduced willful movement, rigidity, and vertical gaze palsy. Common upper motor neuron findings, including positive Babinski sign or hyperreflexia, may be present. Typical presentation occurs within three months of a shunt revision. In patients with a VP shunt, these signs may reflect, but do not necessarily indicate, mechanical or functional shunt complications. Notably, the specific manifestation of each sign may indicate precisely how the shunt is functioning, wherein overdrainage reduces the CSF pressure gradient while tightening raises internal pressure.

3. Case Reports

The temporal relationships to shunt revision, as well as the relationship of these signs and symptoms to a current understanding of functional neuroanatomy, suggest a shunt-associated neuropsychiatric syndrome. Below, we report two cases of shunt-treated hydrocephalus presenting similar but unique neuropsychiatric symptomology. While the etiologies of hydrocephalus differed, both patients exhibited neuropsychiatric symptomology following shunt revisions, and both received psychiatric consultation as part of their clinical course.

Case 1

Patient 1 was a 28-year-old woman diagnosed with non-communicating hydrocephalus secondary to aqueductal stenosis, following headaches and lightheadedness, which did not respond to over-the-counter medication. A ventriculoperitoneal (VP) shunt was placed, and the patient's headaches resolved. Four months later, now aged 29, the patient became pregnant with her second child. Her pregnancy was unremarkable for 16 weeks, until she developed ataxia, blurred vision, and urinary incontinence. Imaging showed enlarged ventricles, and the patient's VP shunt was revised. Figure 1A shows a timeline of her clinical course of this event. MRI confirmed resolution of shunt failure and suggested a return to normal intracranial pressure (ICP). Over the next week, the patient developed dysphagia, psychomotor retardation, increased tone, and decreased consciousness. The patient was given a 2mg lorazepam challenge without effect.

Interviews with historians in the family did not reveal any history of symptoms of psychiatric illness, emotional difficulty, developmental problems, physical or sexual abuse. There was no known family history of psychotic or affective illness. No history of violence or substance abuse was described. The patient's social history included a high school education, successful employment, and marriage. The patient had no significant marital difficulties. She had significant sources of social support, including her mother and mother-in-law, who lived nearby. The patient had no history of substance use or exposure to chemicals or environmental toxins.

Evaluation of the patient revealed that she was awake and alert, but mute. She unreliably followed simple commands. She was a well-nourished, well-developed female in no acute distress. Her physical exam was unremarkable; pupils were equal, round, and reactive to light, and there was no evidence of papilledema. Her facial expression was symmetric and activated synchronously. Her

palate elevated with gag testing, but she had difficulty swallowing secondary to bradykinesia. Strength in her sternocleidomastoid and trapezius muscles was 5/5. Her spontaneous movement was minimal and bradykinetic. The patient had increased muscle tone throughout. There was a non-sustained clonus in the right ankle. There were no pathological reflexes, including grasp and palmomental. Her reflexes were 3/4 throughout and symmetrical. Plantar responses were equivocal. With assistance only, the patient took small, hesitant steps with a narrow base. She had a profound sway in Romberg testing but recovered without falling.

TSH, electrolytes, CBC, ICP, CSF chemistries, and electrolytes were normal. Anaerobic, aerobic, and fungal cultures/stains of blood and CSF were negative. Herpes simplex titers were negative. Later in treatment, the patient tested negative for Whipple's disease. Plain film shunt series was performed with steps taken to protect the fetus from radiation exposure. It revealed that the shunt was in continuity. An initial MRI of the head did not show returning ventriculomegaly. The patient had a normal swallowing study, with no evidence of aspiration. Abdominal ultrasound revealed a normal intrauterine pregnancy.
Obstetric, neurosurgical, and psychiatric services were involved in all subsequent clinical decisions. The patient was placed on levodopa-carbidopa for a presumed diagnosis of secondary parkinsonism, but her condition and symptoms worsened. The patient was given a one-time challenge with midazolam with OB/GYN and anesthesia services present. A transient improvement in tone was noted and interpreted as a partial positive response. The working diagnosis of the patient was in the catatonic spectrum, with consideration for a conversion disorder. The patient started on IV lorazepam up to 6 mg/day, and the patient maintained a moderate improvement in tone before progressing to akinesis over the next 8 days.

Shunt revision was considered unnecessary by the neurosurgical service; the patient's clinical presentation was different from her initial hydrocephalic condition and initial shunt failure, no papilledema was present, and imaging did not reveal ventriculomegaly. Based on partial response to benzodiazepine treatment and lack of response to dopaminergic therapy, the decision was made to perform electroconvulsive therapy (ECT). The patient exhibited improvement in mobility and speech after the second ECT treatment. Lorazepam was discontinued before the fourth treatment. Following the 8th treatment, continuous and significant improvements in speech, spontaneous movement, muscle tone, and oral intake were noted. After the 10th treatment, ECT was discontinued, and the patient was discharged home. Two weeks after her last ECT treatment, the patient was estimated to be at 80% of her baseline, with remaining deficits including mild bradykinesia and moderately slurred or absent speech.

The patient's condition continuously declined following the cessation of ECT treatments. The patient re-presented with somnolence, rigidity, and bradykinesia. Seven additional ECT treatments were attempted with minimal clinical response. The patient communicated her need for toileting by squeezing her husband's hand or slowly moving her legs to the side of the bed. Upon the neurologic exam, the patient had +1 reflexes in the upper extremities and +2 reflexes in the lower extremities. Rigidity was present in the upper and lower extremities but was more severe in the upper extremities. The patient had irregularity in finger movement with activation. The patient could walk but with small steps and two-person assistance. The patient had little response to a visual threat. There was no ptosis or nystagmus, but the patient did have upward gaze paralysis. Pupils were equal and reactive to light. Motor exams were not performed.
The patient underwent an additional MRI, which showed a moderate increase in the size of the lateral and third ventricles. Neurosurgical consultants suggested the patient's VP shunt was functional and did not need immediate revision. The patient was worked up for several additional conditions during this rehospitalization. Chest x-ray showed no evidence of sarcoidosis. Urine toxicology screen and heavy metal levels were normal. Liver function tests were normal. Angiotensin-converting enzyme (ACE) was measured slightly lower than normal. Lipid profile revealed elevated cholesterol and triglycerides. Rheumatoid factor, ANA, and extractable nuclear antigen testing were negative. RPR and Rubella IgM were negative. Testing was negative for Whipple’s disease, but the patient was placed on sulfamethoxazole and trimethoprim prophylactically.

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The patient delivered a healthy full-term baby girl and was transferred back to the neurology service. CT scan revealed increasing ventriculomegaly. The patient underwent an endoscopic third ventriculostomy, and her VP shunt was removed. The patient was placed on levodopa/carbidopa with a positive clinical response. The patient was discharged ambulatory without assistance, and with no remaining deficits of volitional movement or bowel or bladder control. The patient was still mildly bradykinetic at discharge. Cognitive and motor impairment improved to premorbid levels based on a follow-up interview 1 year later. The patient had no recollection of her ordeal.

Case 2

Patient 2 was a male diagnosed as an infant with congenital hydrocephalus treated by VP shunt. The patient also had a history of a right frontal stroke early in life, which left residual left-sided weakness, and a seizure disorder treated with antiepileptics. The patient developed a stutter at the age of 5. The patient had no complications or shunt failures until age 15, which was precipitated by headaches. The patient had one shunt revision at age 19, two at age 20, and three at age 21. Additionally, the patient experienced an episode of status epilepticus at age 21. The patient had moderate obsessive-compulsive symptoms as a child, but no diagnosed primary psychiatric disorder as an adult. Figure 1B shows a timeline of his clinical course of these events.

At age 22, following headache, blurred vision, and nausea, the patient's shunt was investigated and found to be nonfunctional proximally and distally, and was replaced with a Medtronic programmable shunt. His shunt was adjusted two weeks later, and then four to five times over the four months following his surgery. The patient continued to have "on and off" headaches during this time and began to have alarming psychiatric symptoms described in neurosurgical notes regarding shunt adjustments as "talking to self" and "paranoia" two months following shunt placement. The patient had several CT scans, which showed interval increases and decreases in ventricular size associated with shunt adjustments. Five months after his surgery, CT scan showed "slit ventricles." The same week, the patient was evaluated by a psychiatrist for the first time and placed on risperidone. The patient was admitted to the hospital, and his shunt valve was changed. Laboratory testing showed negative CSF culture, normal electrolytes, CBC, and renal function. The patient was discharged.

The patient continued to have psychiatric symptoms and was seen longitudinally at an outpatient psychiatric clinic for 9 months. He presented with flat affect, isolation, fear of leaving home, poor concentration, fatigue, feelings of guilt, anxiety, dependence on parents, and auditory hallucinations of a male voice. The patient had motor abnormalities associated with hemiparesis and stuttering, but no other neurological abnormalities. The patient was given risperidone 3mg daily and citalopram 20mg daily based on a diagnosis of cognitive disorder NOS, Psychosis NOS, and depression NOS. Medications were increased over the following four months as the patient continued to have daily auditory hallucinations. The patient had a significant exacerbation of symptoms, stating "I can't take it anymore," in respect to "the voices" and "impulses." His medications were adjusted to their highest level, at risperidone 2mg BID and citalopram 60mg daily.

Over the next 4 months, the patient's symptoms improved, with auditory hallucinations giving way to "intrusive thoughts" and subsequently "ruminative thoughts" and mild auditory hallucinations when stressed. The patient experienced hyperprolactinemia, a well-documented side effect of high-potency antipsychotics. Six months later, the patient had no residual psychosis and denied depressive symptoms. Four months later, he was assessed to have no residual psychiatric symptoms and was tapered from those medications without recurrence of symptoms. Following a lack of seizure activity, antiepileptic therapy was also discontinued by the patient's neurology team.

4. Discussion

Both patients contribute meaningfully to the existing literature on this topic. Patient 1, consistent with many prior cases, presented shortly after a shunt revision with diminished volitional movement, facial expression, and vocalization. Her neurological signs are closely aligned with those previously reported. Notably, the use of ECT during her treatment, her unique diagnostic considerations, and the presence of a viable intrauterine pregnancy make her case a novel addition to the literature. Patient 2, whose presentation diverged from previously described cases, offers important insight into the

complex neurobiological underpinnings of psychosis.

The diagnostic landscape for both patients was intricate. For Patient 1, key considerations included secondary parkinsonism, akinetic mutism, and catatonia, which are all commonly reported in the literature. Additional possibilities, such as conversion disorder, infectious etiologies, and autoimmune processes, were also considered, but the patient tested negative for common tests for these disorders. Following a positive response to a benzodiazepine challenge, a working diagnosis of catatonia was pursued, and ECT administered with initial success, though symptoms later recurred. It is important to note that the response to ECT does not exclude secondary parkinsonism or akinetic mutism, as ECT may enhance dopaminergic transmission in neural circuits involved in all three conditions (51). The conclusion of symptoms after VP shunt revision and third ventriculostomy following full-term labor supports the conclusion that the patient's symptoms were not due to the initiation of a primary psychotic disorder.

The assessment of Patient 2 was similarly complex. Given his age, primary psychiatric disorders were initially considered, particularly considering his auditory hallucinations and delusions. However, the duration of his symptoms exceeded the criteria for brief psychotic disorder and schizophreniform disorder. A diagnosis of schizophrenia was ultimately deemed inappropriate due to the significant influence of an underlying medical condition on his neurological function. His subsequent recovery without further psychotic episodes supports the conclusion that his symptoms were not due to a primary psychiatric disorder.

5. Shunt Association

The symptomology of both cases represents a significant departure from each patient's baseline. Neither patient had a prior psychiatric history to explain such profound changes, and the close temporal relationship between shunt manipulation and symptom onset strengthens the argument for a shunt-associated mechanism.

In Patient 1, symptoms emerged within one week of VP shunt revision and ultimately resolved within a year following an endoscopic third ventriculostomy, supporting the idea that shunt-related factors contributed to her condition. Similarly, Patient 2 developed psychiatric symptoms approximately two months after programmable shunt placement and in the context of multiple shunt adjustments. Following an episode of slit ventricles, the valve of his VP shunt was replaced. His psychiatric symptoms persisted in a crescendo-decrescendo fashion for roughly 9 months and then did not recur. Both patients returned to their baseline functioning within a year, further supporting the notion that their symptoms were reversible and likely secondary to the shunt-related changes.

In the case of Patient 1, the response to ECT suggests a neurotransmitter hypofunction that was amenable to stimulation. Furthermore, her eventual response to pro-dopaminergic therapy suggests that, as in many reported cases, her ascending dopaminergic tract may have been hypofunctional. The psychosis of Patient 2 differs from the neurologic deficits seen in Patient 1 or the reported cases above. Furthermore, Patient 2 was treated by dopaminergic antagonism. This may suggest that the crescendo of Patient 2's symptomology was not slowed, but rather exacerbated, by antipsychotics.

Based upon the presented case reports, it is suggested that mechanical or functional disruptions related to shunt placement and adjustment may have transiently altered neural circuits involved in motor control and cognition. Cycles of shunt malfunction or fluctuating intracranial dynamics could have subtly impacted the spatial configuration of arousal and limbic structures, including regions of the basal ganglia and thalamus implicated in the regulation of psychosis and voluntary movement. Figure 2 provides an illustration of the anatomical course of a VP shunt in relation to the brain's major dopaminergic pathways. This overlay emphasizes the potential proximity of the shunt catheter to these tracts, which may help explain the dopaminergic involvement underlying the neuropsychiatric symptoms observed in Patients 1 and 2.

Furthermore, the contrasting treatment responses highlight the complexity of the underlying pathophysiology. The pro-dopaminergic therapy in Patient 1 and dopaminergic antagonism in Patient 2 suggest that VP shunt-related deviations may manifest along a spectrum of neuropsychiatric symptoms. Further research is needed to clarify these mechanisms and guide tailored therapeutic strategies.

The management of Patient 1 was further complicated by a viable intrauterine pregnancy, which may have contributed to her shunt

dysfunction. Although ECT was employed in her treatment, the patient ultimately delivered a healthy, full-term infant without complication. Her case highlights the importance of multidisciplinary care and vigilance in managing pregnant patients with VP shunts, while also emphasizing that pregnancy-related factors may compound shunt-associated risks.

Although these findings contribute to the literature surrounding hydrocephalus, several considerations arise when examining the potential associations of neuropsychiatric symptoms and VP shunt placement. There is existing evidence suggesting that dopamine-related neuropsychological signs (apathy, executive dysfunction, gait slowing/bradykinesia and parkinsonism) are a common component of communicating hydrocephalus, most commonly idiopathic normal pressure hydrocephalus (iNPH) and as a chronic syndrome (52). In this literature review, normal pressure hydrocephalus was excluded, which therefore excluded a large subset of reports on communicating hydrocephalus. On the other hand, obstructive hydrocephalus is less likely to cause neuropsychiatric syndromes, but when they do occur, they tend to present as clinically striking parkinsonism (24). Although there are numerous case reports and literature reviews discussing neuropsychological syndromes across both obstructive and communicating hydrocephalus, there is no epidemiological study that directly compares the incidence of neuropsychological symptoms related to dopamine pathway dysregulation. Considering both patients in this case series presented with an obstructive hydrocephalus, it is plausible that some unknown functional change in the VP shunt may have led to the observed symptoms. However, we are unable to make any definitive conclusions due to our finite understanding of how shunt pressure dynamics, neurological, and dopaminergic responses interact in shunt-treated hydrocephalus.

It also remains unclear whether these neuropsychiatric symptoms were caused by the shunt revision procedure or the function of the shunt itself. Without imaging of ventricular size or volume studies before and after intervention, we cannot determine whether mass effect or mechanical changes in CSF flow may have disrupted the dopaminergic pathways. Regardless, the immediate onset of neuropsychiatric symptoms following shunt placement suggests a physiologic link rather than a coincidental progression of disease. This becomes particularly interesting regarding Patient 1, who was diagnosed with hydrocephalus later in life, while Patient 2's condition was congenital. Both had differing brain maturity and age, making it difficult to infer whether there is a temporal pattern to the onset of the observed symptoms.

These cases also raise a broader question posed in prior reports: Are the unexpected neuropsychiatric symptoms a manifestation of restoration of normal shunt function, the underlying disease, or related to surgical manipulation itself? This distinction is particularly relevant as some case reports, such as our Patient 1, result in an endoscopic third ventriculostomy as an alternative treatment. This procedure is thought to alter CSF dynamics differently than VP shunting, which could lead to distinct neuropsychiatric changes. Further research incorporating imaging and neurologic analyses is needed to better clarify how VP shunt changes relate to dopaminergic neuropsychiatric outcomes. Ultimately, while many clinical factors can inform our understanding of VP shunt placement and function, the most meaningful measure of treatment success is the patient's functional outcome.

Taken together, these cases highlight the need for heightened clinical awareness of neuropsychiatric symptoms that may emerge in the context of VP shunt interventions, particularly when presentations deviate from primary psychiatric disorders. They emphasize the importance of considering shunt-related pathophysiology in the differential diagnosis and call for further investigation into the nuanced ways in which shunt function, neuroanatomical disruption, and individual patient factors interact to produce these complex clinical pictures.

6. Conclusion

This literature review and two case reports highlight a clinical syndrome characterized by psychiatric and movement abnormalities associated with shunt-treated hydrocephalus. A growing body of literature suggests that the onset of bradykinesia, personality changes, and cognitive dysfunction may be associated with VP shunt-treated hydrocephalus, independent of initial symptoms. The cases described here display interesting symptomology and clinical courses of neuropsychiatric complications that can occur in shunt-treated hydrocephalus, particularly associated with shunt revisions. Clinicians should have a high degree of suspicion for shunt-

associated symptoms when evaluating motor or psychiatric abnormalities seen in a patient with shunt-treated hydrocephalus.

7. Supplemental Information

Table 1. Previous Shunt-Associated Studies. A: Akinesia, B: Bradykinesia, M: Mutism, PS: Parinauds, EF: Expressionless facies, BS: Babinski sign, HR: Hyperreflexia, G: Gait disturbance and posturing, I: Incontinence, T: Tremor, R: Rigidity, LC: Levadopa/Carbidopa, Br: bromocriptine, E: Ephedrine, S: Surgery, Mo: Months, VP: Ventriculoperitoneal.

Reported Diagnosis	Clinical Description
Akinetic Mutism (7, 19, 28-30, 44-46, 48)	A, B, BS, EF, G, HR, I, M, PS, R, Spasticity, Apathy, Rare horizontal movement, T
Parkinsonism (3-6, 8, 15, 17, 18, 20, 25, 26, 32-34, 36, 37, 39-41, 44, 48)	A, B, BS, EF, G, HR, I, M, PS, R, T, Hypophonia, Drooling, Dysphagia, Hypomimia, Somnolence, Emotional Lability, Cognitive deficits, Dysarthria
Catatonia (21-24, 38)	A, B, M, R, Abulia, Negativism, Agitation, Incoherence, Waxy flexibility, Hypertonia, Apragmatism, Anorexia, Sleep disturbance, Preservation, Hyperventilation
Blepharoclonus (15)	B, EF, G, R, T, Blepharoclonus
Frozen Movement (27)	B, EF, R, Soft Voice
Psychosis (49, 50)	Depersonalization, Derealization, Erratic behavior, Depression, Paranoia, Guilt, Hallucinations, Fatigue, Flat affect, Poor concentration
Major Neurocognitive Disorder (49)	Episodes of depersonalization and derealization, erratic behavior
Global Rostral Midbrain Syndrome (44, 47)	Vertical gaze palsy, light-near dissociation, convergence nystagmus, ptosis, oculomotor palsy, contralateral weakness

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Figure 1. (A) Clinical disease progression timeline for Patient 1. (B) Clinical disease progression timeline for Patient 2. Each timeline illustrates the chronological sequence of major clinical events, treatments, and disease state transitions for each patient. Key milestones include symptom onset, therapeutic interventions (e.g., medications, surgeries), and outcomes such as recovery, relapse, or complications. Time is represented in months and covers major clinical events.

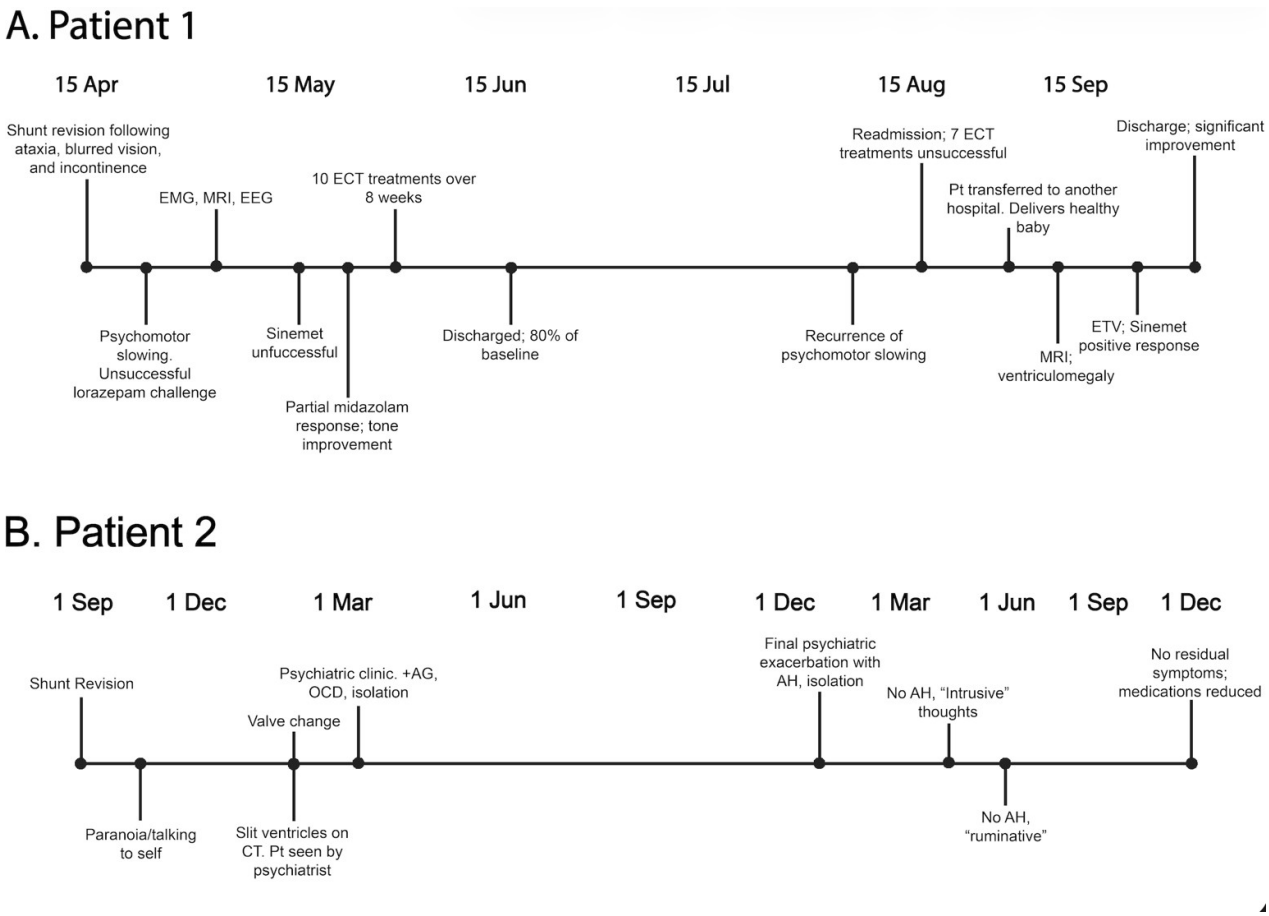

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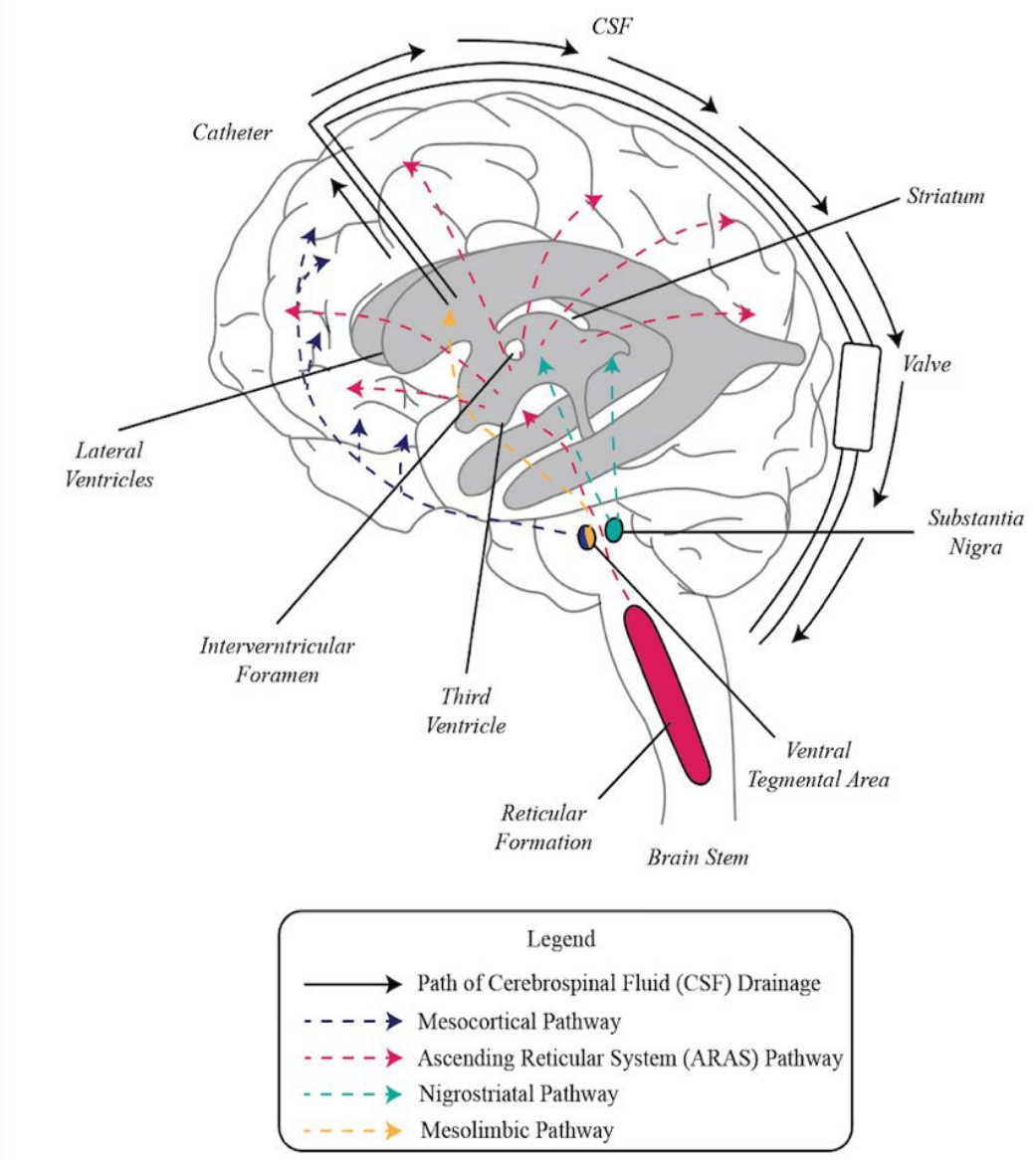
Figure 2. Anatomical relationship between a ventriculoperitoneal (VP) shunt and major dopaminergic pathways in the brain. Illustration demonstrating the trajectory of a VP shunt catheter in relation to key dopaminergic tracts, including the mesocortical, mesolimbic, nigrostriatal, and tuberoinfundibular pathways. This overlay highlights potential anatomical proximity that may contribute to neuropsychiatric symptoms through disruption of dopaminergic signaling.



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