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Perspectives in Psychiatry: A Learner's Viewpoint

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Translation Perspectives in Psychiatry: A Learner's Viewpoint Editorial

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1. Introduction

This special issue of *Translation*, titled Perspectives in Psychiatry: A Learner's Viewpoint brings together a diverse collection of mini-reviews and case-studies to reflect translational medical research. These contributions highlight how psychiatry is evolving beyond scientific models of treatment and toward integration of knowledge that bridges brain, behavior, and patient care. Psychiatry sits at a unique junction between brain biology and human experience which requires clinicians and researchers to engage with neural mechanisms, behavior, and social context to understand mental illness. As neuroscience advances, the challenge is to translate novel findings into patient care that is effective and equitable, to meet real-world complexity.

2. Bridging Brain and Behavior: Updates in Neuroscience

Neurosciences are at the core of understanding clinical psychiatry. Therapeutic modalities are now being developed that attempt to directly address neural circuitry associated with mental illness. For instance, in this issue, research on MDMA-assisted psychotherapy and transcranial magnetic stimulation is reflective of the increased focus on developing novel interventions that influence an individual's neural pathways, learning, processing, and emotions. The mechanistic and clinical approach of these contributions marks a paradigm shift in applications of neuroscience; we now focus on targeting specific neurobiological and neural dynamics rather than addressing symptoms alone. This shift also highlights the exciting work in personalized medicine, where the heterogeneity of psychiatric illnesses is now being recognized and neural pathways targeted for treatments.

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3. Barriers and Opportunities: Accessing Mental Health Care

This section highlights key barriers to psychiatric treatment, with emphasis on prolonged wait times and unhoused populations. Delays in medical care can compromise effective treatment, leading to missed appointments, postponed assessments and treatment initiation. There is a disproportionate association between unhoused individuals and psychiatric disorders, with higher patient readmission rates and lower medication access and adherence, ensure psychiatric care, and reduce wait times through reforms.

4. Psychotropics and Psychotherapy: Interdisciplinary Care for Patients

This set of reviews addresses the use of psychotherapy and psychotropic medications in patient care. While psychotherapy was traditionally utilized as part of in person therapy consults, it has expanded to online modalities as well. Another less explored approach is introducing psychotherapy to medically admitted patients. Through a model-based approach, benefits and potential improvements in introducing this version of care to medically admitted patients were identified.
Additionally, antipsychotics were associated with increased risk of developing metabolic syndrome in several studies. Based on meta-analyses, metformin is a possible intervention for addressing antipsychotic-based weight gain.
Finally, interventions for misophonia may be handled in a tiered method, using approaches such as cognitive behavioral therapy, pharmacotherapy, and combined treatment models as a guide for clinical decision making. Collectively, these articles demonstrate the evolving role of psychotropic medications and psychotherapy in modern psychiatric practice.

5. The Ps of Women's Mental Health: Premenstrual, Pregnancy, and Postpartum Care

This section examines women's mental health across key reproductive stages, highlighting periods of psychiatric vulnerability, and the mechanisms, behavior, and social context to understand mental illness. As neuroscience advances, the challenge is to translate novel findings into patient care that is effective and equitable, to meet real-world complexity. importance of early recognition and care. The articles review the role of stress and inflammation in premenstrual dysphoric disorder, the association between pre-eclampsia and subsequent psychiatric risk, and new-onset psychosis during pregnancy and the postpartum period. These reviews highlight risk factors and treatment approaches. Together, these contributions emphasize the need for integrated, stage-specific mental health screening and intervention throughout reproductive care.

6. Early Intervention: Pediatric, Adolescent, and Student Mental Health

This section focuses on early identification and intervention in mental health conditions affecting children, adolescents, and students. The mini-reviews address the pediatric mental health crisis and evolving clinical guidelines, emerging concerns such as Al-related existential distress, rare psychiatric presentations such as cannibalism in youth, and evidence supporting family-centered and peer-based interventions for suicidal youth. The section also examines mental health trends among medical trainees, highlighting the impact of academic and training environments on psychological wellbeing. Collectively, these papers emphasize the value of early intervention in improving psychiatric outcomes.

7. Conclusion

As you embark on your reading journey through the space between brain biology and human behavior, we implore you to be open-minded towards the nuances that exist within the human mind and experience. This special issue displays how mental health providers and student researchers are working to provide patient-centered, optimized care for all. Through reading this issue, we invite you to take away how the complexities of the lived experience and psyche manifest themselves in the clinical setting. The art throughout the issue illustrates the key findings within each section. We hope that you enjoy reading and find this experience educational and transformative!

Bridging Brain and Behavior:

Updates in Neuroscience

Trans lation.

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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.

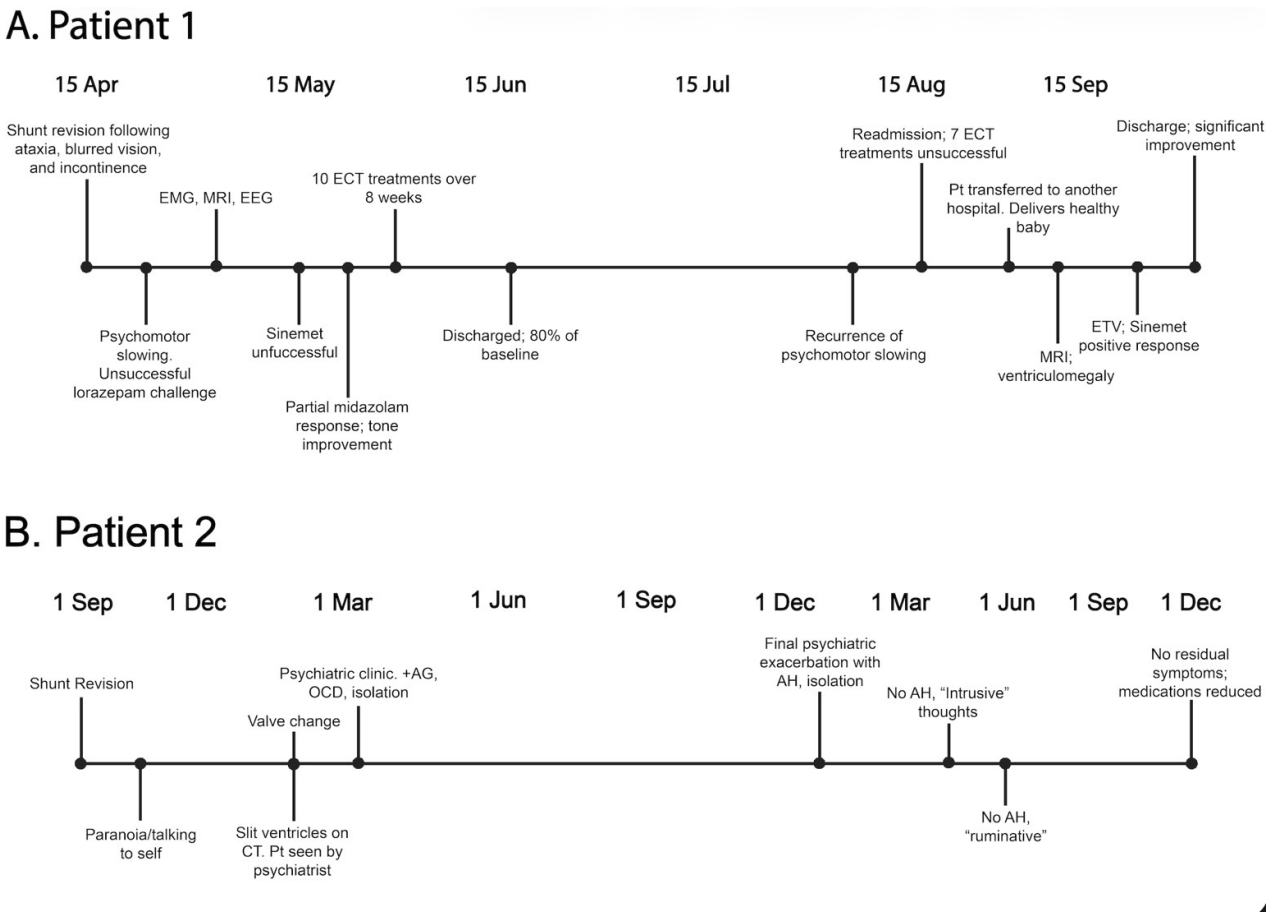

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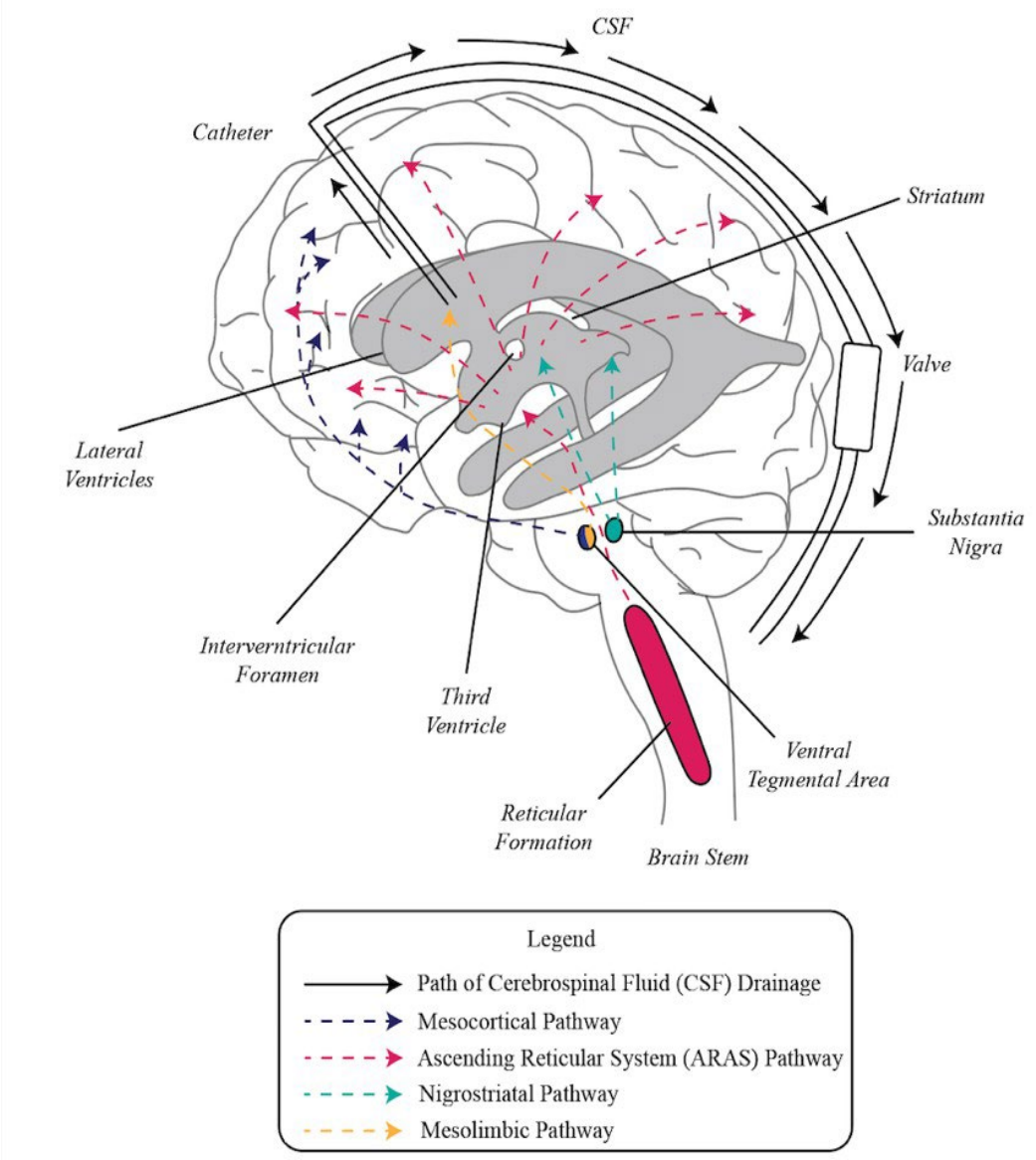
Figure 2. Anatomical relationship between a ventriculoperitoneal (VP) shunt and major dopaminergic pathways in the brain. Illustration demonstrating the trajectory of a VP shunt catheter in relation to key dopaminergic tracts, including the mesocortical, mesolimbic, nigrostriatal, and tuberoinfundibular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



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MDMA-Assisted Therapy for PTSD: Neuroplastic Change or Psychotherapeutic Catalyst?

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Abstract

MDMA-assisted psychotherapy has emerged as a promising treatment for post-traumatic stress disorder (PTSD), particularly in individuals unresponsive to conventional interventions such as SSRIs and trauma-focused cognitive behavioral therapy. As recent clinical trials report durable symptom remission, ongoing debate surrounds the mechanisms underlying its therapeutic effects. This review explores two major frameworks: a neurobiological model proposing that MDMA supports trauma reprocessing through fear extinction, memory reconsolidation, and neuroplasticity, and a relational model highlighting MDMA's prosocial and empathogenic properties that enhance therapeutic alliance and suggestibility. Evidence from both human and rodent studies indicates that MDMA reduces amygdala reactivity, increases hippocampal connectivity, and modulates serotonergic and oxytocinergic signaling. Concurrently, clinical findings suggest that the quality of the therapeutic relationship plays a critical role in treatment outcomes. Rather than viewing these processes as mutually exclusive, we propose a synergistic model in which MDMA creates a "window of emotional safety" that allows for both neurobiological and interpersonal mechanisms to support healing. Understanding this dual-action model is essential for refining treatment protocols, improving therapist training, and guiding future research in psychedelic-assisted psychotherapy.

Keywords: MDMA-assisted psychotherapy, Post-Traumatic Stress Disorder, Therapeutic Mechanisms

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1. Introduction

MDMA-assisted psychotherapy has gained traction as a novel treatment for post-traumatic stress disorder (PTSD). For patients unresponsive to conventional approaches such as selective serotonin reuptake inhibitors (SSRIs) and trauma-focused cognitive behavioral therapy (CBT). Clinical trials, studying MDMA-assisted psychotherapy, report robust and sustained symptom relief, prompting interest in its broader integration into psychiatric care (1). Yet, as implementation approaches, a central question remains: how exactly does MDMA work as a therapeutic agent? Two primary mechanisms have been proposed. The first emphasizes trauma reprocessing, suggesting that MDMA facilitates fear extinction and disrupts the reconsolidation of traumatic memories. Studies using both human and rodent models show that MDMA reduces amygdala reactivity, enhances emotional memory network connectivity, and supports neuroplasticity through serotonin, oxytocin, and brain-derived neurotrophic factor (BDNF) signaling (2-7). The second theory focuses on enhanced therapeutic alliance, arguing that MDMA's prosocial effects may amplify important psychotherapy factors such as trust, openness, and the therapist-client relationship (1, 8-10). This review aims to examine the evidence for each mechanism and reflect on how these models might shape future therapeutic interventions and psychiatric education.

2. Discussion

The growing success of MDMA-assisted therapy in clinical trials has increased interest in understanding how this treatment works, a question with direct implications for clinical practice, training, and patient selection. Two frameworks dominate the current discourse: one neurobiological and memory-centered, the other relational and context-driven.

Support for the trauma reprocessing model comes from animal studies and human neuroimaging. MDMA dampens amygdala activity, increases hippocampal engagement, and facilitates synaptic plasticity (2). These conditions may promote fear extinction and allow for reprocessing of traumatic memories in a safer state (2-7). This view aligns with traditional exposure-based therapies and suggests MDMA augments existing paradigms by making recall of the trauma more tolerable and flexible for the patient.

In contrast, growing attention is being paid to common factors in psychotherapy, especially the role of the therapeutic alliance. One study found that strength of the therapeutic alliance predicted PTSD symptom improvement, regardless of trauma-specific content (8). MDMA's effects on oxytocin and serotonin systems may heighten openness and suggestibility, increasing responsiveness to therapist input (1, 10). This model does not deny the importance of memory but instead frames MDMA as a tool that amplifies the relational environment in which the therapy occurs.

Rather than seeing these mechanisms as mutually exclusive, a more integrative view may be warranted. An interesting hypothesis suggests that MDMA appears to open a "window of emotional safety" that supports both trauma processing and therapeutic connection (4). This dual-action hypothesis suggests that effectiveness may depend on how the therapy is delivered: a strong therapeutic relationship may be necessary to guide trauma work, while the pharmacologic effects lower barriers to engagement.

Understanding these pathways matters for academic clarity, as well as for practical implementation. If trauma reprocessing is central, protocols for treatment may prioritize structured memory exposure. If alliance and context dominate, therapist training, setting, and integration practices become more critical. Future research should aim to disentangle these mechanisms in diverse populations and settings, using both qualitative and biological measures.

3. Conclusion

To guide clinical practice and training, understanding the mechanisms of MDMA-assisted psychotherapy is key. The existing evidence points to dual action: promoting neurobiological conditions for trauma reprocessing and enhancing the therapeutic relationship. In doing so, MDMA challenges the traditional boundaries between split pharmacology and psychotherapy treatments. Effective use of MDMA therapy requires attention to both its neural effects and the quality of the therapist–patient relationship.

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Transcranial Magnetic Stimulation for Nicotine Dependence: A Brief Review with Focus on Emerging Non-Tobacco Products

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Abstract

Transcranial Magnetic Stimulation (TMS) is a noninvasive treatment that has shown promise in reducing nicotine cravings and the risk of relapse. While TMS has been effective for treating tobacco use disorder, its application to newer modes of nicotine delivery, such as vaping and nicotine pouches, is understudied. This article highlights the need for updated TMS protocols that are responsive to current trends in nicotine use, especially in younger populations. Expanded research including a wide range of nicotine products will increase the generalizability and clinical relevance of TMS for the treatment of nicotine dependence**.**

Keywords: Transcranial Magnetic Stimulation, Nicotine Dependence, Non-Tobacco Nicotine Products, Immunomodulation

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1. Introduction

Transcranial Magnetic Stimulation (TMS) is a promising non-invasive neuromodulation technique for treating substance use disorders (SUDs) (1). Recent studies and clinical trials have demonstrated TMS as a method to reduce cravings in individuals with SUDs. Through regulating cortical and dopaminergic activity, TMS may assist in lowering the risk of relapse while breaking the learned behavioral patterns that lead to compulsions (2). TMS is thought to exert its effects by modulating activity in the mesocorticolimbic system, which serves a crucial role in the maintenance of addictive behaviors (3). Among the various substance disorders studied through TMS protocols, nicotine dependence, specifically tobacco use disorder, has emerged as one of the most extensively investigated.

In SUDs, the dorsolateral prefrontal cortex (DLPFC) is widely implicated in generating craving-related responses and exerting inhibitory control over them (4). The DLPFC manages stress responses by regulating the production and release of dopamine (4) and dysfunction in this pathway may reduce stress resilience and increase susceptibility to relapse. The left DLPFC is a frequent target in TMS protocols because its stimulation elicits dopamine release in the striatum. This response is believed to lead to the reductions in cue-induced craving (4).

2. Discussion

Between 2003 and 2018, at least nine randomized controlled trials (RCTs) reported significant reductions in tobacco cravings and consumption following active repetitive transcranial magnetic stimulation (rTMS) compared with sham high frequency rTMS stimulation (5). The outcomes of these nine randomized controlled trials are summarized in Table 1.

However, these studies had relatively small sample sizes, ranging from 14 to 77 participants. A more robust investigation by Zangen and colleagues, published in 2021, demonstrated a significantly higher four-week continuous quit rate (CQR) in individuals receiving active rTMS compared to those in the sham group ($p = 0.006$) (6). This study enrolled 262 participants and employed a treatment protocol involving daily rTMS to the lateral prefrontal and insular cortices for three weeks, followed by once-weekly sessions for an additional three weeks. Notably, the positive outcomes from this trial contributed to the U.S. Food and Drug Administration's approval of rTMS for smoking cessation. Despite these encouraging results, the authors emphasized that comparable large-scale studies are still lacking. It should be noted that the results from current studies can be difficult to compare due to methodological differences as well as differences in the physiology of each participant (6).

One variable that remains largely unexplored is the application of TMS for non-tobacco forms of nicotine, such as vaping and nicotine pouches. A 2019 systematic review of TMS studies for smoking revealed that each of the studies analyzed specifically dealt with smoking (7). This gap is notable given the rapid shift in nicotine use patterns among younger populations, for whom tobacco smoking is no longer the predominant mode of nicotine consumption. Preclinical data suggests that adolescence is a critical period of vulnerability to nicotine exposure; the adolescent brain exhibits increased neuroplasticity, and nicotine's rewarding effects may strengthen dependence risk (8).

The heightened vulnerability in adolescent populations underscores the need to monitor the diverse nicotine consumption methods used by young adults. Cigarette smoking among 18 to 24-year-olds dropped from 29.1% to 5.4% from 1997 to 2020. In addition, it was found that thirty-day nicotine vaping rates increased to 25.5% in adolescents by 2019 (9). Previous attempts to quantify the nicotine content in electronic cigarettes have been challenging since nicotine levels vary between brands. Despite the increasing prevalence of electronic cigarettes, no current TMS trials include participants who use electronic cigarettes as their primary nicotine source.

Another emerging trend is the increasing use of nicotine pouches, which presents new opportunities and considerations for TMS research. Recent data suggest that nicotine pouches can produce nicotine plasma levels comparable to cigarettes (10). Like electronic cigarettes, there are currently no neuromodulation studies involving nicotine pouch users, highlighting the need to incorporate these product categories into future research.

3. Conclusion

To assess possible TMS efficacy in nicotine dependence in all age groups, it is necessary to draft protocols for studies with all types of nicotine

consumption included. The rise of non-tobacco nicotine products, especially in youth populations, presents the opportunity and challenge in aiming for future TMS protocol development. Future

research should prioritize larger and more diverse sample sizes, broader age ranges, and inclusion of various nicotine delivery methods. Future studies should also adopt cue reactivity tasks to better reflect non-tobacco nicotine products, which lack the consistent sensory cues of traditional smoking, such as lighting or smoke. Because most TMS protocols rely on cue-based craving assessments, identifying cues for vaping and pouch use is essential (6, 7). The variety of these newer products makes cue-related responses more variable and harder to measure. Thus, incorporating these variables will enhance the generalizability and potential efficacy of rTMS protocols across the spectrum of nicotine dependence.

Conflict of Interest: The author declares no conflict of interest.

Table 1. Randomized Controlled Trials of TMS for nicotine dependence

Study (Author and Year)	Sample Size and Population	TMS Targetand Protocol	Duration /Sessions	Primary Outcomes	Key Findings
Eichham mer et al.,2003	n = 14 smokers	Left DLPFC; 10 Hz, high frequency rTMS	5 days	Cigarette consumpti on	Active rTMS significantly reduced daily cigaretteuse vs sham.
al., 2009	smokers	DLPFC; 10Hz, 20 min/sessio n	sessions (2 weeks)	craving & consumpti on	craving and cigarette use compared with sham.
Wing et al., 2012	n = 15 patients with schizophre nia	Left DLPFC; 20Hz rTMS	3 days	Tobacco craving	Significantly decreased craving in activegroupvs sham.
Dieler et al., 2014	n = 20 nicotine-dependent adults	Left DLPFC; intermitten t theta-burst stimulation (iTBS) adjunct to psychother apy	10 sessions	Nicotine abstinence	Higher abstinence maintenancein iTBS + psychotherapy group; pilot feasibility.
Pripfl et al., 2014	n = 14 smokers	Left DLPFC; single-session 10 Hz rTMS	1 session	Cue-induced craving& EEG delta power	Reduced craving and decreasedEEG deltapowervs sham.
Kleinet al., 2014	smokers	prefrontal +insular cortices; deeprTMS	sessions (over 3 weeks)	cessation rate	quit rate significantly higher in active group (44%)vs sham (28%).
al., 2015	smokers receiving NRT	DLPFC; 1 Hz rTMS + nicotine replaceme nt	sessions eachday for two weeks	& craving	rTMS + NRT improved abstinenceand craving reduction vs NRTalone.
etal., 2018	smokers abstinent for 24 hours	DLPFC; 20 Hz rTMS	sessions	prevention	preliminary evidence of lower relapse riskvssham.
al., 2018	smokers with schizophre nia, 14 controls)	DLPFC; 20 Hz rTMS	sessions per treatmen t week	behavior& cognition	smoking behavior; cognitive effects varied bygroup,acute administration insufficient for treatment

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Engaging Minds: A Brain Cutting Experience for Undergraduate and Pre-Medical Students

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Abstract

Brain anatomy is notoriously difficult to teach and visualize to first-time learners. To make neuroanatomy more digestible, our group designed an educational brain-cutting experience that used gross dissection of a cadaver brain alongside digital dissection through the Anatomage Table technology to elucidate more difficult structures in three dimensions. A slide presentation was also prepared to help guide the learners through the dissection and to teach structure, function, and pathology. Two sessions with undergraduate learners were performed, with the first having four learners and the second having eight. Pacing and improper cutting technique were the main obstacles encountered in the first session; however, this was remedied by proper communication, teaching, and changing the format of the visual presentation. We feel that Anatomage helped the learners better visualize each structure in three dimensions, which aided in understanding the content. Early exposure to anatomy and pathology can inspire students to pursue careers in medicine, so we hope that this experience inspires others to develop their own neuroanatomy program.

Keywords: Neuroanatomy,Education,Anatomage

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1. Introduction

Anatomy is considered the building block of medical education (1). Anatomical knowledge is essential for patient examination, correct diagnosis, and all medical specialties (2). Hands-on experience and, more recently, digital models of anatomy are crucial for medical education. Given this importance in medical education, beginning anatomical study as an undergraduate may have positive consequences. As one example, an observational study examined the impact of hands-on workshops on future medical students' motivation, confidence, and career aspirations (3). They reported an increase in median scores for motivation to pursue a medical career, theoretical, and practical knowledge. In addition, motivation to pursue a career in medicine increased after a workshop on any medical subject, regardless of the information presented (3). Reasons for learning anatomy can also extend beyond just passing a test. One study of undergraduate science students found that intrinsic interest in anatomy was a reported motivation among most of the students surveyed (4). These studies highlight the importance of incorporating hands-on experiences early in pre-medical education and the objective of this paper.

Neuroanatomy poses a unique challenge in medical education. Obtaining donated human specimens/cadavers for dissection is difficult, and it is often less expensive to use digital models of anatomical figures (5). As a result, 3D tools like Anatomage (a virtual dissection table), as well as Primal VR (a virtual reality environment for teaching and learning human anatomy), have been developed and used to fill this gap (6-7). Various studies have been conducted on the use of these virtual resources in neuroanatomy education (5-8). For example, one study assessed the effectiveness of Anatomage as a teaching tool for neuroanatomy for third-year physiotherapy students (8). The study found that those assigned to using only Anatomage for dissection had increased post-test results compared to those assigned to using only the cadaver (8). Nonetheless, there has not been a study that used a brain dissection and Anatomage simultaneously. We aimed to bridge the gap in the literature with this experience.
We designed a hands-on didactic experience to help teach neuroanatomy using an interactive, mixed-media approach that integrated cadaver and traditional presentation slides. This experience was designed to introduce brain physiology and highlight the anatomical structures that result in some vital disease pathologies. In addition, this experience was meant to inspire and motivate pre-medical students to pursue a career in medicine. There was a broad distribution of academic majors among the learners, ranging from neuroscience to electrical engineering, and only a small percentage of learners had prior neuroanatomy education.

2. Methods

Overview and objectives. Our main objective was to provide learners with a detailed understanding of neuroanatomy in three dimensions. To achieve this objective, we incorporated the Anatomage Table virtual dissection technology into a clinical dissection. Anatomical structures were first found via gross dissection; then visualized in three dimensions using the Anatomage table; then discussed in relation to function and pathology. We aimed to cultivate a positive and inviting environment for learners to ask questions and engage at their own pace. In addition, we aimed to provide a hands-on experience for pre-medical/undergraduate students.

Learning outcomes. We established three major learning outcomes for this didactic experience. At the end of this experience, learners should be able to (1) understand the three-dimensional structure and organization of the brain; (2) relate neuroanatomy to regional brain function and common medical disorders of the brain; and (3) perform a dissection of a cadaveric human brain and use digital dissection technology to identify the structures.

Equipment and supplies. This educational experience used a total of three cadaveric human brains, one per four learners, which were provided by the University of Toledo College of Medicine's Anatomy Laboratory, as originally sourced through the Anatomical Donation Program. Prior to use, each cadaveric brain was soaked in 1% saline for at least 24 hours, then transferred to fresh 1% saline for at least another 24 hours, to minimize the scent of preservative. The cadaveric brain dissection was performed on a clean, empty cadaver table with a plastic dissection platter. The tools used included a large cutting knife, a metal probe, and a scalpel. The digital dissection was performed using an Anatomage Table Clinical model (Anatomage, Santa Clara, CA) running Anatomage Table EDU version 11.0.1. The slide presentation was displayed

on multiple large external monitors throughout the lab that were connected to a standard desktop computer. Appropriate disposable personal protective equipment was provided to all learners and educators.

Educational content. We designed the experience to be as interactive as possible. The sessions were held in an anatomical laboratory within the medical school (Figure 1). We allocated one brain per four students, with one medical student guiding the cutting as a proctor. The Anatomage Table was positioned in front of the cadaver tables and was set to display the brain (see Anatomage Guide). We projected a slide presentation for the session using a central monitor, which connected to multiple TV screens in the lab that the learners could view as the session proceeded. The slide presentation included relevant anatomy, pathology, and pictures of previously cut brain slices with the anatomy labeled.

The sessions began with a slide presentation of the external brain, throughout which an external examination of the brain was performed, highlighting sulci, gyri, and meninges. We decided that making coronal slices of the brain, anterior to posterior, was the best method to view the relevant anatomical structures. Each learner took turns cutting the brain by placing the brain on its side (lying on either temporal lobe) and slicing downward in the coronal plane, and each slice was examined afterwards. The slide presentation was set up in such a way that structures would be seen and discussed in the same order as they appeared in the dissection. On the Anatomage Table, the Clipping Planes tool selected, and the Clipping Plane Slider Bar was toggled to coronally "shave" the brain to mimic the cuts being made by the learners. After we reached a structure of interest, we highlighted it on Anatomage, discussed its functions, and presented an associated pathology. The dissection proceeded from anterior to posterior, stopping in the forebrain at the level of the atrium of the lateral ventricles, and in the hindbrain at the cerebellum just behind the fourth ventricle. After the whole brain was sectioned, the tissue was placed back in its container.

Sessions. Two sessions were held. The first session was performed with four learners and one brain. The second session included eight learners, with two groups of four cutting one brain each. Figures 2-10 depict the flow of sectioning performed during the sessions, alongside anatomical structures and pathologies discussed.

3. Results

Session 1

During our first session, we found that taking a slow approach, allowing learners to ask questions and look at the brain slices, worked best. By following the pace of the learners, a better learning environment was attained as the learners had time to ask questions. Using the Anatomage Table also helped to visualize the internal structures of the brain (basal ganglia, ventricles, etc.) in three dimensions more easily than pictures or dissection alone. The Anatomage Table also has the added benefit of being able to color-code different structures and/or eliminate outer structures from the view, which helped to differentiate them for the learners. Using the Anatomage Table to remove the outer cortex from one side of the brain (leaving it intact on the other side), and simultaneously to color-code the structures of the basal ganglia, greatly enhanced the visualization of their three-dimensional structure. We found that concurrently seeing the structures in an actual brain, visualizing it on the Anatomage Table, and discussing function/pathology via a presentation made for a memorable and enlightening experience that integrated anatomy and pathology.

One limitation we noted was the consistency with cutting the brain. Since it was the learners' first time cutting a brain, it was difficult to get consistently small, even slices that mimicked the slices in the presentation. Sometimes slices were too big, and a structure was missed (i.e., amygdala) or too small, in which the slices were uneven and made future slices more difficult.

Although we dedicated a slide in the presentation to instructions on how to cut, we decided that a more involved teaching process needed to take place during the next session to ensure quality cuts. The inconsistency of cuts also led to pacing issues with the presentation. Sometimes, the presentation discussed structures we had not gotten to on the actual brain, which interrupted the workflow.

Session 2

After addressing the issues in Session 1, we doubled the class size to eight people. Figure 1 depicts the setup of Session 2 to account for more learners. This led to new challenges. For Session 2, we retained the group size of four learners per brain/table. This group size allowed the proctors to have a more personalized approach to teaching, and it encouraged an environment in which the learners felt comfortable asking questions. Having two brains also allowed room for error in case one group missed a structure. For example, one group

accidentally missed the amygdala in their cut, but the other group didn't; therefore, everyone still had an opportunity to see it. We also devoted more time in Session 2 to teaching the learners how to cut correctly. In addition, each proctor made the first cut to demonstrate proper technique. This ultimately allowed for better cuts to be made by the learners compared to Session 1. Furthermore, we addressed the pacing issue from Session 1 by adding a master structure list slide in the presentation deck. Once we reached a structure of interest, we would click on its name on the master slide, which would take us to the slides associated with that structure. This ensured that we could focus more on the brain itself than attempting to keep the same pace as the slides during the dissection.

We did encounter some limitations with the larger class size. While the pacing of the presentation was fixed, it was difficult to maintain a similar pace between proctors. There were times when one proctor would be on a future structure that the other had not reached yet. This occasionally led to confusion between each proctor and the person running the Anatomage Table. In future sessions, proper communication should be emphasized so that this does not happen. Another drawback to using more than one brain is inconsistent anatomy. One group's brain was much larger than the others, which made cutting more difficult and varied pacing to a minor degree, as they saw structures before the other group did. However, this can be remedied by attempting to select brains that are similar in size.

For both sessions, one drawback was the lack of depiction of brain pathologies on the Anatomage Table. Being able to show a normal vs. an abnormal brain on the Anatomage Table would help the learners in visualizing and understanding brain pathologies. Unfortunately, this is not a current feature of the Anatomage Table.

Hopefully, this feature will be added in a future update.

4. Discussion

Incorporating the study of anatomy into undergraduate education can help motivate students to pursue careers in medicine; this was the primary goal of the brain cutting experience. Both sessions used cadaveric human brains and Anatomage Tables. Two medical students presented specific neuroanatomical structures of the brain to undergraduate learners as they were revealed via coronal sectioning. In addition, explanations of pathologies that may arise when those structures are damaged were also presented. The Anatomage was used to showcase brain structures in three dimensions, which aided in understanding the anatomy and pathology in a more meaningful way compared to gross dissection alone. While difficulties did arise in terms of presentation pacing and cutting technique, these were addressed by the second session. In future sessions, we will address the issue of differing anatomy when using more than one brain.

5. Future Endeavors

Our future endeavors are to continue to teach medicine-inspired students about basic brain regions and functions through our guided brain cutting experience. We hope to continually improve our experience in hopes of perfecting the process. Additionally, we hope that the Anatomage program gets updated with increased detail in the brain. Currently, Anatomage seems to be more suited for learning about the whole body rather than solely the brain. An updated Anatomage would allow us to show every region of the brain that we find in our dissection. This would not only help learners identify brain regions but would also help us demonstrate certain brain regions that are difficult to see on gross dissection. Finally, an update allowing Anatomage to replicate disease states in the simulated brain would be extremely useful. This would allow learners to get a better understanding of what brain regions are affected by each disease, as well as the extent of the damage/distortion. Ultimately, our final goal is to pass our polished program onto future medical students, preserving its impact and inspiring future students to lead and expand this interactive teaching opportunity.

Acknowledgements

Thank you to the generous body donors, their families, and the Anatomical Donation Program of the University of Toledo College of Medicine, who granted us the privilege to conduct the brain cutting experience. We are honoring the donors' final wishes by using their bodies for medical education. We also acknowledge the University of Toledo Interprofessional Immersive Simulation Center for providing the Anatomage Tables used in this experience, as well as providing training and technical support.

Supplementary Information

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Figure 1: Session Setup. Representation of the brain cutting experience setup. For session 2, we had 2 groups of 4, each with a dissection table and a brain. We had multiple screens for the presentation, as well as the Anatomage and the Anatomage Navigator.

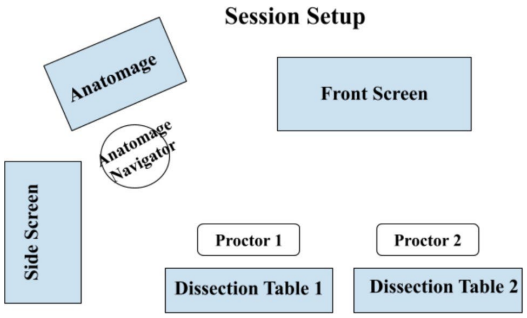


Figure 2: Whole Brain. Shows a whole brain with the arachnoid mater still attached to half of the brain. Discussed the normal structure and function of the various brain lobes, the pre- and post-central gyri, visible cranial nerves, visible arteries within the Circle of Willis, brainstem structures (midbrain, pons, medulla, and cerebellum), and meninges. Also discussed Gerstmann Syndrome and hemispatial neglect.

Figure 3: First coronal cut. Shows white and grey matter. Discussed how the structure of neurons is correlated with white/grey matter.

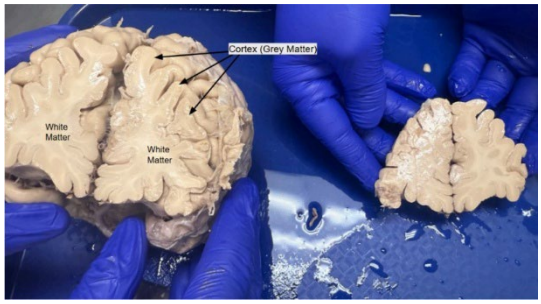


Figure 4: Second coronal cut. Showed/discussed the normal structure and function of the head of the caudate and lateral ventricles. Also explained the function of cerebrospinal fluid and the pathophysiology of hydrocephalus, specifically normal pressure hydrocephalus.

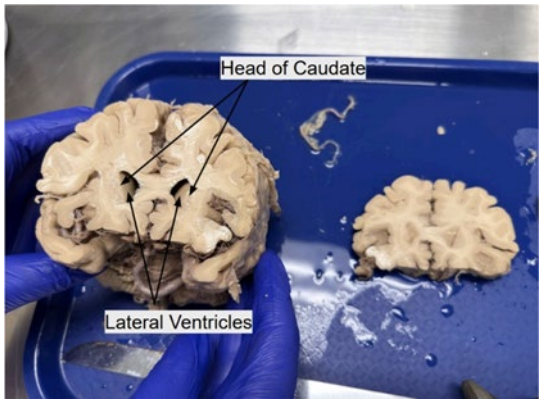


Figure 5: Third coronal cut. Showed/discussed the normal structure and function of the caudate, putamen, internal capsule, corpus callosum, and the basal ganglia. Discussed Huntington's Disease.

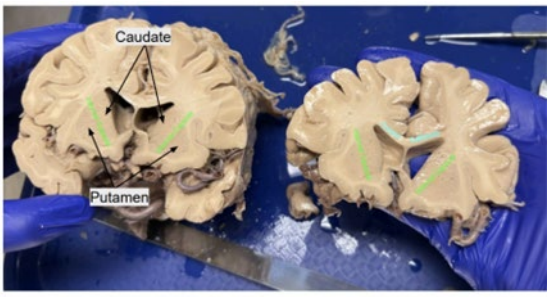


Figure 6: Fourth coronal cut. Showed/discussed normal structure and function of the 3rd ventricle, pituitary gland, anterior commissure, septum pellucidum, and amygdala. Discussed Kluver-Bucy Syndrome.

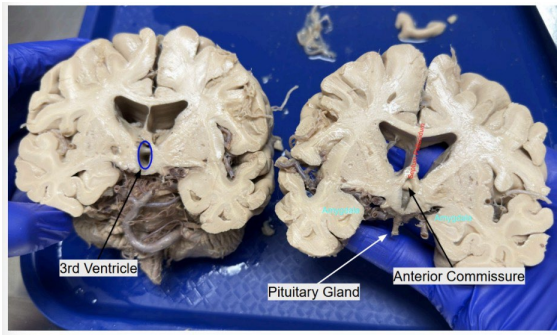


Figure 7: Fifth coronal cut. Showed/discussed normal structure and function of choroid plexus, thalamus, substantia nigra, midbrain, pons, medulla, putamen, globus pallidus, and mammillary bodies. Discussed Parkinson's Disease and Wernicke-Korsakoff Syndrome.'

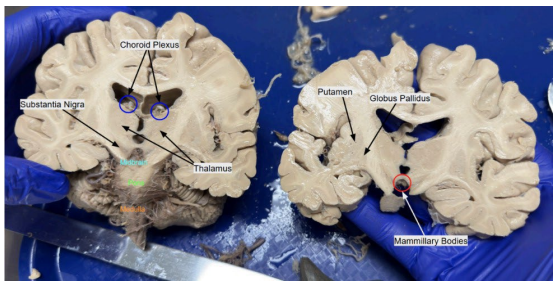


Figure 8: Sixth coronal cut. Showed/discussed the normal structure and function of the cerebral aqueduct and hippocampus. Discussed Alzheimer's Disease.

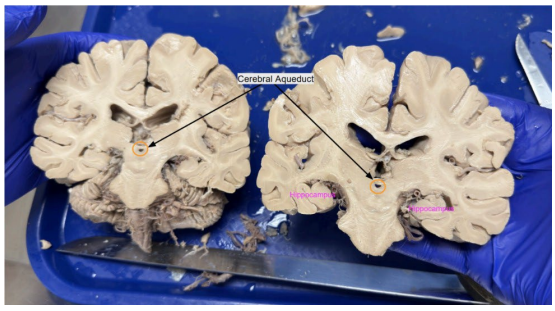
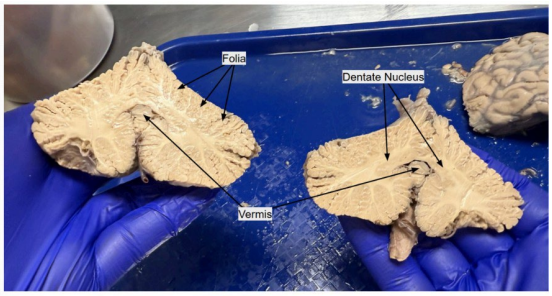


Figure 9: Seventh coronal cut. Showed/discussed the normal structure and function of the 4th ventricle.

a,

Figure 10: Coronal cut through cerebellum. Showed/discussed the normal structure and function of the cerebellum and its structures (folia, dentate nucleus, and vermis). Discussed Chiari II malformation.



Additional Files

Anatomage Guide

Author Disclosure:

The authors declare no conflicts of interest.

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Mind Over Microbes: The Role of the Microbiota-Gut-Brain Axis in Depression and Anxiety

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1. Introduction

Depression and anxiety affect approximately 10% of the global population and significantly impact patient functioning (1). Due to their debilitating nature, it is essential to better understand the underlying pathophysiology of these disorders to develop more effective treatments.
Growing research on the microbiota-gut-brain axis (MGBA) has proven promising, implicating this system in the pathogenesis of depression and anxiety. The MGBA is a bidirectional communication network between the central nervous system (CNS) and the gastrointestinal (GI) tract. The gut microbiota consists of diverse microorganisms essential to this system. Evidence suggests that gut dysbiosis, a disruption in gut microbiota composition, causes dysregulation in immune activation and neurotransmitter production, leading to neuroinflammation and onset or worsening of mental health disorders (2). This review explores MGBA communication routes, microbiota alterations in depression and anxiety, and the therapeutic potential of probiotics, dietary modulation, and fecal microbiota transplantation (FMT).

2. Discussion

I. *MGBA Communication Routes*
The gut microbiota is a dynamic ecosystem of over a trillion microorganisms including bacteria, viruses, fungi, and archaea involved in digestion, neurotransmitter release, and immune function (1,3). Conversely, the brain influences gut microbial composition and function. The MGBA involves bidirectional communication through neuronal, endocrine, and immune pathways (4). Disruptions in this system are linked to the pathogenesis and severity of depression and anxiety (3).

Ia*. Neuronal Pathways: ENS, Vagus Nerve, and Neurotransmitters*
The enteric nervous system (ENS) innervates the gut and regulates motility and responses to microbial metabolites. ENS development is influenced by early-life bacterial exposure. Studies with germ-free (GF) mice exhibited underdeveloped ENS that improved with microbiota restoration (4).

The vagus nerve mediates gut-brain communication by transmitting signals from the GI tract to the brain either directly or through enteroendocrine cells and hormones. One study found that lactic acid bacteria reduced anxiety-and depressive-like behavior in mice via GABA receptor modulation (4). Certain gut bacteria also affect brain neurotransmitters: *Lactobacillus* and *Bifidobacterium* produce GABA and histamine, while *Escherichia Coli* synthesizes serotonin, dopamine, and noradrenaline, metabolites that influence the CNS and mental health (4).

Ib*. Endocrine Pathways: HPA-axis and Stress*

The hypothalamic-pituitary-adrenal (HPA) axis regulates stress and the MGBA through endocrine signaling. HPA axis-produced stress hormones increase gut permeability, causing bacterial translocation that further activates the HPA axis and immune system (4). Dysregulation of this axis is a reliable biological marker of stress-related disorders. In one study, rats with activated stress circuits displayed anxious and depressive behaviors, which normalized after stress removal (5). GF mice also exhibit HPA axis dysfunction, increasing vulnerability to stress and cognitive disorders, further implicating MGBA involvement (4).

Ic*. Immune Pathways: Inflammation and Neuroimmune Signaling*
Gut-brain communication also occurs through immune mechanisms. Gut microbes regulate immune activity through dietary metabolites such as short-chain fatty acids (SCFAs) and bacterial fermentation products that are transported to the brain and modify immune responses (4). Dysbiosis can compromise intestinal barrier integrity, triggering immune activation and production of pro-inflammatory cytokines (i.e. IL-6, IL-1β, and TNF-α). These cytokines can cross the blood-brain barrier and disrupt neurotransmitter balance, contributing to depression and anxiety (6, 7). In one, *Bifidobacteria* supplementation reduced levels of IL-6, INF-!, and TNF-" in rats compared to controls (4).

II. *Dysbiosis in Depression and Anxiety*
Research describes characteristic changes in gut microbial composition in depression and anxiety. Depression is associated with reduced microbial diversity, elevated *Firmicutes* levels, and decreased *Bacteroides* and *Lactobacillus* levels. Anxiety correlates with decreased SCFA-producing bacteria and increased *Proteobacteria* levels (2). Dysbiosis also alters gut bacteria stress reactivity,

resulting in further anxiety. Microbial shifts, such as increased *Eggerthella* bacteria and decreased *Coprococcus* and *Subdoligranulum* are observed in both disorders (8). Overall, research strongly supports a correlation between depression, anxiety, and microbial dysbiosis.

I. *Probiotics, Diet, and FMT*

Probiotics show promise in reducing anxiety and depression, although results vary across strains and durations. One eight-week multi-strain probiotic trial showed significant depressive symptom reduction, while another study found reduced cortisol responses and somatic symptoms under stress with probiotic use (9).

Dietary modifications can also be beneficial by enhancing microbial diversity, decreasing inflammation, and producing favorable metabolites. Diets rich in omega-3 fatty acids, whole foods, and fiber are linked to decreased risk and severity of mental health disorders. FMT, transplantation of healthy microflora, is another promising treatment that restores healthy gut microbial composition and function (8).

3. Conclusion

The MGBA represents a compelling paradigm shift in the understanding and treatment of depression and anxiety. Gut microbiota modulates neuroinflammation, stress responses, and neurotransmitter production, making them a potential therapeutic target for psychiatric disorders. Probiotics, dietary modulation, and FMT have all been suggested as promising therapeutic interventions, although clinical efficacy varies among studies and further research is necessary. Integrating microbiome science with psychiatric care has potential to create an effective alternative to traditional pharmacological agents and enhance treatment outcomes in affected patients.

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An Essay on Parkinson's Disease and the Gut-Brain Connection

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1. Introduction

Parkinson's Disease (PD) is a neurodegenerative condition characterized by a wide range of motor and non-motor symptoms. According to Parkinson's Foundation, it is estimated that more than 10 million people are suffering from PD worldwide, and 90,000 individuals are diagnosed with PD in the U.S. per year, making it the second most common neurodegenerative disease following Alzheimer's disease (1). PD has also affected several prominent public figures, including Muhammad Ali, Neil Diamond, and Michael J. Fox, highlighting its broad clinical and societal impact. Although PD was depicted early on in ancient Chinese and Indian texts, it was only named in 1817 after James Parkinson, an English physician, who described it as "shaking palsy" and "paralysis agitans" (2). Parkinson later described the disease in his book, *An Essay on the Shaking Palsy,* as "Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported" (3). After Parkinson's findings, French Neurologist Jean-Martin Charcot proceeded to characterize the clinical spectrum of the disease, noting the bradykinesia, which hinders the daily lives of those affected (3). In addition, he highlighted there are two models of the disease: the rigid and the tremorous form (3). This discovery led to the rejection of the portrayal of the disease as "paralysis agitans" or "shaking palsy," since those affected were not debilitated and did not always experience tremors. While it was suggested in the late 19th century that motor symptoms were due to the selective loss of dopamine neurons in the substantia nigra pars compacta, it was not until the 1960s that levodopa (dopamine replacement therapy) was developed as the mainstay clinical treatment for PD (4). This marks the "dopamine era," in which most efforts at treatment development were aimed at targeting the loss of dopamine neurons. This was later replaced by the "alpha synuclein era," at which efforts were aimed at targeting the other hallmark pathological finding of PD patients: Lewy bodies (2, 4). Lewy bodies are misfolded aggregates of the presynaptic neuronal protein alpha-synuclein (α-synuclein) found throughout the brains of PD patients (5). Despite these ongoing efforts to develop alternative disease-modifying treatments for PD, the lack of success over the years may be attributed to the fundamental question about PD that remains unsolved: What exactly causes the loss of these

dopamine neurons? There are numerous hypotheses to the etiology of PD, some of which include genetic predisposition, environmental toxicity, inflammation, and alterations in the gut-brain connection (6-9). In recent years, the involvement of the gut-brain connection in the pathogenesis of PD has gained considerable attention.

Recent work has found a link between the gut microbiome and the development of PD. Furthermore, emerging studies focusing on the gut-brain axis found that α-synuclein aggregates

(which form Lewy bodies) may stem from the gut and move to the brain via the vagus nerve (10). Understanding the mechanisms by which α-synuclein travels to the brain is significant to our understanding of how the gut may be involved in the pathogenesis of PD. The aim of this essay is to examine the role the gut-brain connection has in the pathogenesis of PD, apply this to the clinical presentation of a PD patient, and elucidate implications the gut-brain connection may have on the development of future PD treatments.

2. Background

Symptoms and Diagnosis

To contextualize the potential role of the gut in PD, it is necessary to first consider how the disease manifests clinically. PD is an age-related disease with the risk of the disease increasing with age. Only 25% of those afflicted are below the age of 65, and 5-10% of those afflicted are below the age of 50 years old (11). The incidence of PD is higher in men than women, however, women tend to experience greater "motor and non-motor response functions," which is said to be attributed to their lower body weight (11). The disease is characterized by two distinct features: motor and nonmotor symptoms. The cardinal motor symptoms include bradykinesia (slowness of movement), rigidity, resting tremor, and postural instability (12). PD symptoms, like many other diseases, vary in progression and severity for each patient. Motor symptoms typically begin as unilateral and progress to bilateral, followed by bulbar dysfunction in mature cases, (characterized by dysphagia (trouble swallowing) and dysarthria (trouble with speech)) (11, 12). Non-motor symptoms include cognitive deficits, sleep abnormalities, autonomic dysfunction, psychiatric disturbances, and sensory symptoms (12). Sleep abnormalities, specifically REM sleep behavioral disorder, typically occur early in the disease course, prior to motor deficits (13, 14). Psychiatric disturbances may include anxiety, depression, and hallucinations (14). Autonomic symptoms include drenching sweats, dyspnea, seborrhea, and gastrointestinal dysfunction (14). Sensory signs consist of tingling, akathisia, in addition to olfactory deficit (15). Cognitive deficits appear late in the disease course and consist of visuospatial or executive function deficits (14-16).

There are two theories as to how the gut is involved in the pathogenesis of PD, the brain-first and the gut-first hypotheses (17). The brain-first hypothesis states that the disease starts in the brain and spreads to the gut. Patients whose disease course is consistent with this hypothesis may have motor symptoms without early prodromal nonmotor symptoms such as REM Sleep Behavior Disorder or gastrointestinal dysfunction (17). On the other hand, the gut-first hypothesis states that the disease begins in the peripheral nervous system where misfolded α-synuclein proteins travel to the brain. Patients whose disease course is consistent with this hypothesis may have early autonomic dysfunction, gastrointestinal dysfunction, and REM Sleep Behavior Disorder prior to the onset of motor symptoms (17).

As a result of the clinical heterogeneity of PD, a wide range of diagnostic approaches is employed. In addition to a thorough physical exam and patient history, a variety of diagnostic tests may be run. Magnetic resonance imaging, dopaminergic imaging, transcranial ultrasound of the substantia nigra, genetic testing, autonomic function tests, olfactory tests, as well as tremor analysis, are examples of diagnostic methods (11).

Pathophysiology

Beyond its clinical presentation, PD reflects progressive dysfunction of the dopaminergic system and related neural circuits. Neuronal loss within the substantia nigra pars compacta disrupts basal ganglia signaling required for smooth, coordinated movement (18). In parallel, α-synuclein adopts misfolded conformations that aggregate into Lewy bodies across the central and enteric nervous systems, including the gastrointestinal (GI) tract (18-20). As a result of the diverse genetic, inflammatory, and environmental stressors that can damage dopaminergic neurons, multiple interacting risk factors have been implicated in the development of PD. Environmental risk factors including toxins, methanol exposure, aging, carbon monoxide poisoning, as well as trauma to the head, may result in neurodegeneration (7, 18). Genetic lesions, including mutations in synuclein alpha (SNCA), leucine-rich repeat kinase 2 (LRRK2), and glucosylceramidase beta (GBA), may predispose individuals to developing PD (15). Similarly, mitochondrial dysfunction and increased oxidative stress, which can be provoked by the environmental or genetic factors listed above, can lead to neuronal energy depletion in addition to loss of function (15).

3. The Gut-Brain Function

Overview

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The enteric nervous system (ENS) is a "nerve network composed of neurons and glial cells that regulates the motor and secretory functions of the GI tract" (19). The GI tract is a long structure between the mouth and the anus that houses all the organs essential for the breakdown and absorption of nutrients (22). In addition to these digestive functions, the GI tract contains a diverse community of microorganisms, collectively termed the gut microbiome. Deterioration of the gut microbiome can lead to harmful effects on the blood-brain barrier (19). The ENS and the nervous system are connected to the brain through the gut-brain axis (GBA), allowing the nervous system to send signals to the brain and vice versa. The vagus nerve plays an important role in the gut-brain axis, transmitting signals related to digestion and mood reciprocally between the central nervous system and the gut (20).

Recent Studies

Early researchers of PD found that GI symptoms manifested much earlier than other symptoms, leading to investigation of the gut in PD patients (13). Braak, a German anatomist and pathologist, conducted an autopsy-study in which tissue from the brain, distal esophagus, and the gastric wall of PD patients were investigated for Lewy body pathology (21). Braak found a buildup of Lewy body pathology in gastrointestinal structures as well as in the dorsal motor nucleus of the vagus nerve (DMV) in the brainstem, which connects the gut to the brain (21, 22). This work led to what is now referred to as the "Braak hypothesis" (21, 23), which states that PD pathology may originate in the GI tract, where an external agent triggers the misfolding and accumulation of α-synuclein aggregates (22, 24). In turn, the aggregates of α-synuclein travel retrogradely to the brain via the vagus nerve (22). This formation of Lewy bodies then leads to neurodegeneration of the vital dopaminergic structures in the basal ganglia.

On the contrary, not all neuropathological findings are consistent with Braak hypothesis. Several other autopsy studies conducted of patients with PD did not indicate any irregular buildup of Lewy bodies in relevant structures (25). This discrepancy raises an important question: Is vagotomy a viable treatment method for PD? A vagotomy is a surgical procedure involving the transection of the vagus nerve. There are two types of vagotomies, total and selective. A total vagotomy involves cutting the nerve before it branches, while selective vagotomy preserves some vagus nerve function in the intestines (26). A study conducted using nationwide Swedish registers of 9,430 vagotomized patients (3,455 truncal and 5,978 selective) showed that overall, vagotomy was not related to the risk of PD (27). Nevertheless, the evidence remains controversial as to how exactly the vagus nerve may be involved in the pathogenesis of PD.

An alternative method for targeting the gut in PD involves improving the gut microbiota as a method of reducing the risk of the disease as well associated symptoms. One study aimed at improving the imbalance of microorganisms in the gut (often referred to as gut dysbiosis) prescribed PD patients probiotics (living organisms that aid in our digestion) (21). The results indicated a reduction of non-motor symptoms associated with the disease compared to those given a placebo (21). Fecal microbiota transplantation (FMT) is another approach aimed at reducing symptoms in PD patients. FMT involves taking fecal matter from healthy donors and injecting it into PD patients to diversify the gut microbiota. One study found that after FMT, quality of sleep and life, anxiety and depression were improved (28). The study used PSQI (Pittsburgh Sleep Quality Index), HAMD (Hamilton Depression Rating Scale), HAMA (Hamilton Anxiety Rating Scale), PDQ-39 (Parkinson's Disease Questionnaire-39), NMSQ (Non-Motor Symptoms Questionnaire), and UPDRS-III (Unified Parkinson's Disease Rating Scale - Part III) in n=12 PD subjects (31). A statistically (p-value = 0.001 or < 0.001) significant improvement was reported compared to initial test scores (28). Specifically, changes in symptoms from baseline were observed as follows: PSQI decreased by 34%, HAMD and HAMA decreased by 55%, PDQ-39 decreased by 50%, NMSQ decreased by 35%, and UPDRS III decreased by 43% (28).

4. Patient Interview

In addition to reviewing experimental and clinical studies on PD, a real-world clinical case was examined through a live patient interview conducted as a part of Neuroscience 1000, a one-credit hour course focused on diagnosis, disease, and the pathophysiology of the nervous system. The course emphasizes live patient interviews, didactic question and answer sessions with patients, and the integration of disease pathophysiology with neuroanatomy. A summary of the patient's experience with PD is presented below.

The patient examined in class was a sixty-year-old Caucasian male diagnosed with PD in 2016. The patient experienced a prolonged diagnostic

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process. Since the patient was not exhibiting signs of tremor at the time of testing, establishing a diagnosis of PD was challenging. As a result, tests were done to rule out other diseases, and the diagnosis was ultimately determined to be PD. The patient displayed symptoms of rigidity in his left arm, difficulty moving, limping, as well as loss of mobility. He reports that his walking difficulties started in 2020, which were characterized by a shuffling gait. In addition, the patient experienced unsteadiness, lack of coordination, as well as difficulty with day-to-day tasks that required fine motor skills. The patient's wife also noted reduced olfactory function. Bradykinesia, a primary clinical feature of PD, was observed multiple times in this patient. Specifically, the patient had reduced facial expression, reduced blink rate, and bradykinesia in the jaw (slightly opened jaw). Two tests were conducted in class to highlight the patient's symptoms of bradykinesia. The first test performed was the finger tapping test, in which the patient tapped his index finger and thumb together repeatedly and as quickly as possible. Bradykinesia was noted as a reduced amplitude over time with finger tapping. The other test involved setting a 60-second timer to see how many times the patient blinks in that time. The patient blinked only once in those 60 seconds, compared to the average blink rate 15-20 times per minute, which is a drastic difference.

The patient was prescribed carbidopa-levodopa to treat his PD symptoms. He reported mobility improvement in his left arm weeks after starting levodopa treatment. Levodopa is the precursor to dopamine, which is converted to dopamine in the brain, allowing the body to replace the lost dopamine. Levodopa is administered in combination with carbidopa, which inhibits its peripheral conversion to dopamine, thereby increasing the amount of levodopa that reaches the brain. Given levodopa's relatively short half-life of about four hours, the patient's physicians shortened the dosing interval to every three hours to improve symptom control. Taking the drug consistently minimizes the fluctuations in dopamine levels and allows better control of PD symptoms. The patient reported being diligent with his medication, daily routine, and exercise, which has contributed to an improvement in his PD symptoms. Despite the adversity the patient has experienced, he sustained a highly positive mindset, which he linked to his strong support system.

5. Conclusion

PD may be difficult to diagnose due to the heterogeneous manifestation of symptoms amongst patients. PD may result from a genetic predisposition, exposure to environmental toxins, or a combination of both, a process known as the "double-strike theory." Considering recent work on the relationship between the gut-brain connection and PD pathophysiology, methods such as fecal microbiota transplant or probiotic prescription aimed at diversifying the gut microbiome may be useful treatments in the future. However, current evidence for these gut-targeted interventions remains preliminary.

More research regarding the use of vagotomy in treating PD patients should be performed to better understand the potential of this intervention for PD patients, as current evidence is not conclusive. Vagotomy is not currently an accepted treatment for PD due to the preliminary nature of current research regarding this as a potential treatment. In the future, several factors may be considered when evaluating the potential use of vagotomy for treatment in PD patients, including disease progression, severity of symptoms, prior treatments, and whether the patient's symptoms are consistent with the gut-first hypothesis.

Patients with symptoms that are consistent with the gut-first hypothesis may be more likely to benefit from vagotomy than patients with symptoms that are consistent with the brain-first hypothesis, but this possibility requires further investigation.

In summary, while dopaminergic replacement therapy, such as levodopa, is effective in alleviating and minimizing PD symptoms, the development of alternative treatments is needed. Increasing evidence in recent years has shed light on the connection between neurodegenerative diseases like PD and the gut. Nevertheless, more research is required to develop and validate gut-targeted therapeutic strategies as viable options for PD management.

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Translation.

BARRIERS AND OPPORTUNITIES:

ACCESSING MENTAL HEALTH CARE

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Impact of Wait Times on Psychiatric Outcomes: A Mini-Review

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Abstract

Prolonged wait times in psychiatric care (in the emergency department, outpatient setting, or between diagnosis and treatment initiation) have been associated with worsening clinical outcomes. This mini-review explores how wait times in psychiatric care contribute to worsening symptoms and emphasizes the need for earlier intervention. Psychiatric patients admitted through the emergency department stayed in the emergency department 3.2 times longer compared to nonpsychiatric admission. Following in-patient psychiatric care, patients are typically referred to outpatient visits. From 2011 to 2022 an increase in the median waiting time for outpatient psychiatic visits from 15 to 50 days was reported. After outpatient visits, patients still experience delays in their treatment initiation. Longer intervals between diagnosis and treatment initiation have been associated with worsening clinical outcomes. All these delays in psychiatric care highlight critical periods where early intervention can improve symptom progression and clinical outcomes.

Keywords: Psychiatric Wait Times, Symptom Progression, Mental Health Care Delays

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1. Introduction

Wait time in medicine is a common source of frustration for patients. Wait times delay medical care and serve as a barrier to adequate behavioral health care. Longer wait times were linked to a decreased likelihood of appointment attendance, with a 5% reduction in attendance per additional week of waiting (1). Wait times for psychiatric treatment can be categorized into three phases: in the emergency department, awaiting outpatient clinic visits, and the interval between clinic assessment and treatment initiation. A retrospective cohort study from 2007–2008 found that psychiatric patients admitted through the emergency department (ED) remained in the ED 3.2 times longer than nonpsychiatric admissions, with average stays of 1,089 minutes vs 340 minutes (2). Similarly, an Australian observational study comparing data from 2011 and 2022 reported a rise in median wait times for in-person outpatient clinical psychiatric care, from 15 to 50 days, highlighting a growing delay in outpatient access (3). Additionally, longer intervals between diagnostic assessment and treatment initiation in patients with major depressive disorder (MDD) were associated with worse treatment outcomes, underscoring the clinical implications of delayed care (4).

2. Discussion

Prolonged wait times in psychiatric care occur across various settings, including emergency departments, outpatient clinics, and the interval between diagnosis and treatment initiation. These delays are consistently associated with worsening clinical outcomes, regardless of where they occur. The ED is often the frontline resource for patients in psychiatric crisis. However, the ED is a suboptimal setting for individuals seeking psychiatric care due to limited psychiatric resources and a busy atmosphere. Geriatric and pediatric patients, two vulnerable populations, have experienced longer ED wait times for psychiatric consultation, alongside a rise in mental health–related visits, respectively (5, 6). These increased wait times for psychiatric consultation increase their risk of experiencing worsening symptoms and prolonged stay in the hospital (5, 6).

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After hospital discharge, patients are typically referred for follow-up with a psychiatrist. Many report the time between discharge and the first outpatient appointment feels “too long,” highlighting a critical window for targeted intervention (7). These prolonged wait times were significantly associated with increased psychological distress, worsening symptoms, and engagement in maladaptive coping behaviors (7). A cohort study of 139,694 children and adolescents found that patients who received mental health follow-up within seven days showed a significant reduced risk of suicide during the initial 6-month discharge period (8). Being able to efficiently help patients during these critical windows could improve psychiatric outcomes. Additionally, the long wait times for outpatient psychiatric appointments does not only affect patients, but also adds strain on providers, limiting their ability to provide adequate care (3).
Delays between appointments and treatment initiation are another form of “wait time” that patients experience. Studies exploring both MDD and psychosis show that delayed intervention results in worse long-term outcomes (4, 9). The delay in treatment initiation represents a critical window during which symptoms could be addressed, yet many patients are left waiting. A 2024 systematic review found that brief, low-intensity interventions (e.g., psychoeducation, parental coaching, remote support, etc.) can mitigate symptom deterioration during high-risk delays in patient care (10). Minimizing the delay in treatment initiation can allow for improved treatment outcomes.

3. Conclusion

Increased wait times for patients with psychiatric symptoms result in worse clinical outcomes. Delays in patient care represent critical periods during which early interventions could improve symptom progression and overall prognosis. Despite studies showing that simple, brief interventions can help improve psychiatric symptom progression, systemic reform is needed to help reduce wait times across all aspects of psychiatric care. Increased support for telepsychiatry, rapid-start treatment pathways, and access to urgent care clinics where psychiatric needs can be met should be prioritized. Addressing these wait times through meaningful systemic reform and targeted interim interventions will help alleviate the growing burden of psychiatric illness.

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Access to Psychiatric Medications in Unhoused Populations: Implications for Hospitalization and Readmission Rates

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Abstract

Persons experiencing homelessness (PEH) face disproportionately high rates of psychiatric illness and hospitalization, with mental illness prevalence reported as high as 76.2%—significantly higher than in the general population. Psychiatric treatment for PEH is often hindered by systemic barriers to care, including medication nonadherence stemming from fragmented healthcare delivery, stigma, transportation challenges, and unstable housing. This discontinuity contributes to elevated readmission rates, with studies showing 30-day psychiatric readmission rates up to 2.04 times higher for unhoused individuals. Medication access is further complicated by inappropriate prescribing patterns and difficulty in maintaining treatment regimens for chronic mental illnesses such as schizophrenia and bipolar disorder. Multifactorial barriers—spanning patient-level, treatment-related, and structural factors—worsen adherence. Evidence-based interventions such as Assertive Community Treatment, Customized Adherence Enhancement with long-acting injectables, and Housing First models have shown success in improving treatment continuity and reducing psychiatric symptomatology. Emerging solutions like street medicine and mobile outreach deliver low-barrier care directly to shelters and encampments. Addressing psychiatric medication adherence among PEH is not only a clinical priority but a public health imperative, requiring integrated, compassionate, and housing-informed care models.

Keywords: Psychiatry, Unhoused, Medication Adherence

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1. Introduction

Access to psychiatric care is an important factor for mental health outcomes, yet there are significant disparities among unhoused populations. Persons experiencing homelessness (PEH) are disproportionately impacted by psychiatric disorders with studies finding a reported prevalence as high as 76.2% for any mental illness, much higher than within the general population (1). Effective treatment for psychiatric disorders often relies on sustained access to medications which may be frequently disrupted for PEH. There are several barriers to access including disconnected healthcare delivery, limitations in transportation, stigma, and unstable housing which all compound to exacerbate mediation nonadherence. These issues are exacerbated by discontinuity in care leading to worsening psychiatric symptoms, increased reliance on emergency services, and higher hospitalization and readmission rates (2-4).

2. Discussion

Psychiatric Readmission for PEH

PEH with psychiatric disorders face disproportionately higher rates of hospitalization and readmission, potentially driven and worsened by unmet treatment needs and limited outpatient follow-up. A retroactive study found that 30-day psychiatric readmission rates occurred within 42.8% of unhoused patients compared to only 19.9% for matched patients with secured housing (4). Other similar studies have found readmission rate discrepancies for unhoused patients were 22.2% compared to only 7.0% for matched and securely housed controls (3). A cohort analysis of 3907 PEH discharged from psychiatric hospitals revealed that 70% had severe mental illness. Adjustments made for covariates displayed that individuals had a 2.04-fold increased risk of 30-day readmission and a 1.65-fold increase at 90 days (2).

Medication nonadherence and limitationstherein may play a central role in these outcomes. Psychiatric medications such as antipsychotics, mood stabilizers, and antidepressants effectively reduce symptom relapse and hospitalizations, though they often require consistent use and close monitoring. Unhoused patients with schizophrenia or bipolar disorder experience high rates of potentially inappropriate prescription – up to 88.4% in one study – or no appropriate prescription at all (5). Failures in providing effective treatment may exacerbate the psychiatric symptoms and reinforce the detrimental cycle of hospitalization and institutional care for patients.

Barriers to Medication Adherence

There are several interrelated factors that contribute to the increased rate of behavioral health medication nonadherence in PEH. Factors include patient-related challenges (cognitive impairment, substance use, mistrust in healthcare), pharmacotherapy-related issues (detrimental side effects, lack of long-acting medications) and social determinants (income insecurity, lack of medication storage) (6). Patients with a post-traumatic stress disorder (PTSD) diagnosis were less likely to have nonadherence identified – potentially due to underreporting and reluctance from individuals to self-report their nonadherence or diagnosis (6). Mental health services are critical for the success of psychiatric treatment. Factors leading to improved care, medication adherence, and length of homelessness include receipt of primary care, care from a mental health specialist, and receipt of long-acting injectable antipsychotics (7, 8).

Interventions and Evidence-Based Solutions

Intervention strategies aimed at improving mediation access and adherence among PEH must be multifactorial. Models such as Assertive Community Treatment, Customized Adherence Enhancement combined with Long-Acting Injectable medications, and Housing First have shown strong evidence for improved psychiatric medication adherence (9). Common themes within these interventions rely on the combination of follow-up services and improved healthcare delivery, which allow them to be well-suited for individuals facing housing instability.

Notably, the Housing First programs are particularly of interest in benefiting these patients as they provide immediate access to stable housing without typical preconditions such as sobriety or compliance with treatment. Introduction and maintenance of stable housing environments has been associated with both better medication adherence as well as a reduction in psychiatric symptomatology. Stabilization of psychiatric symptoms can facilitate improved engagement with other social services and the health care systems, paving a path for long-term recovery.

The reality of the daily experiences that PEH faces both require and deserve specialized approaches

to healthcare delivery which bridge treatment and care for mental, behavior, and substance use disorders. Street medicine and mobile psychiatric outreach are examples of emerging modalities which effectively bring this care to PEH in low-barrier environments such as shelters and encampments. By meeting people where they are, these programs can help overcome barriers to care (10).

3. Conclusion

Unhoused persons living with psychiatric illness represent a population at a crossroads between healthcare, housing, and social service failures. Elevated prevalence within psychiatric conditions among this population in conjunction with heightened hospitalization and readmission rates are driven in part by poor access and adherence to psychiatric medication and fragmented continuity of care. Interventions show promising success in improving access to psychiatric treatment, including long-acting formulations of medications, low-barrier clinics, and integrated care models. These interventions reduce hospitalizations and promote greater housing stability. Bringing to light psychiatric medication adherence among PEH must be viewed as an important clinical concern as well as a broader public health issue.

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TRANSLATION.

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Psychotropics and Psychotherapy:

Interdisciplinary Care for Patients

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The Potential Utility of Supportive Psychotherapy for Medically Admitted Patients

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Abstract

The use of psychotherapy in inpatient settings is underexplored but shows promising benefits. This review summarizes the literature on its application in both inpatient psychiatric and medical units, with a focus on supportive psychotherapy as a potential modality for providing medically hospitalized patients with psychotherapeutic care. To further explore this, we piloted an acting internship in which fourth-year medical students provided supportive psychotherapy to medically admitted patients of the psychiatry consult-liaison service. The model proved feasible and anecdotally beneficial for patients. It underscores the need for further investigation into the role of psychotherapy, and particularly supportive psychotherapy, in the care of medically admitted patients.

Keywords: Supportive Psychotherapy, Inpatient, Hospitalized

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1. Introduction

Many envision talk therapy occurring within the comfort of a therapist's office, or more recently, on a screen from one's couch. The current literature and practice reflect this sentiment. However, one might question whether untapped benefits exist for admitted patients. Recently, we explored this idea through the implementation of an acting internship in which fourth-year medical students provide supportive psychotherapy to medically hospitalized patients.

Psychotherapy as adjunct treatment within psychiatric units, though not ubiquitous, is increasingly integrated. While the literature is not abundant, the practice is validated by several meta-analyses on the subject. Depressive disorders are the most thoroughly investigated with a recent meta-analysis illustrating small to moderate, but sustained benefit in depressive symptoms for those who receive combination pharmacologic and psychologic therapies while admitted (1). A broader meta-analysis found moderate improvement in symptomatic and functional measures of psychiatric symptoms for those who received psychotherapy in addition to control treatments while admitted (2). Individual studies have investigated more specific clinical scenarios. Notably, a randomized control trial found that in patients hospitalized for a suicide attempt, the addition of brief CBT reduced readmission rates and subsequent suicide attempts at 6-month-follow-up (3).

However, within medical inpatient settings the application of psychotherapy is nearly unexplored. There is related meta-analytical evidence which suggests survival benefits provided by psychosocial support interventions in medical settings (4). Notably, this data did not focus on quality-of-life benefits and included data from outpatient settings. The lack of investigation into psychotherapy specifically is likely due, in part, to systemic barriers to its application. These barriers are frequently acknowledged in publications about their use within psychiatric inpatient settings. They are also likely exaggerated within medical units less accustomed to the provision of psychotherapy.

In my experience, these included frequent interruptions leading to less efficient or less productive sessions, pain or confusion making sessions more difficult to navigate, and unpredictable discharges resulting in abrupt endings to therapeutic relationships. However, the hospital itself is a significant stressor, and patients requiring care from a psychiatry consultation
service often come with psychiatric comorbidities. Many of these diagnoses are well established to benefit from outpatient psychotherapy, yet access to such therapy while in the hospital remains scarce. The lack of literature or practice on the subject, and question of potential benefit for both patient and learner encouraged us to confront these barriers.

2. Discussion

The model centered around identifying patients of the psychiatry consult-liaison service who could benefit from supportive psychotherapy. Patient selection was led by an attending physician, and selected patients were seen for ~50-minute sessions for the duration of their hospital stay as available. This included ~20 sessions encompassing 8 different patients with one patient receiving 6 sessions between two admissions. The challenges with such a model were, as discussed, largely predictable. Nurse interruptions for medication administration, other service interruptions, sessions being cut short for diagnostic studies, and waxing and waning patient mental status. While these factors necessitated simplification of therapeutic goals, they are also what made sessions subjectively valuable to patients. The sessions focused on simple goals such as building rapport, sharing of ego strength, encouraging outpatient follow up when relevant, and addressing areas of anxiety. Though each patient had unique narratives, diagnoses, and internal conflicts, there were some commonalities. Several patients experienced significant anxiety relating to physical or occupational therapy or declining health status. Validation of these fears was a centerpiece of care for several patients. Two patients found that sessions helped them cope with emotions surrounding their physical therapy. There was an importance placed on forming a strong therapeutic alliance with patients within a session or two. This proved successful with most patients, strengthening the argument that it is possible to do so quickly in the hospital setting. Most patients were interested in speaking for the duration of their hospital stay, and several reported sessions to be cathartic. One patient with undiagnosed hoarding disorder left the hospital having taken her first major steps to decluttering

her home. Patients reported a variety of meaningful benefits, and the model proved to be implementable over a month with plans to continue. Anecdotally the experience indicated that there may be value in further exploring the utility of inpatient supportive or behavioral therapies, particularly, for improving PT and OT participation rates and outcomes.

3. Conclusion

The hospital is anything but solitary — yet many of these patients lacked an empathetic listener. It was clear that 50 minutes as a listener with distinct, but simple, therapeutic goals, provided some with significant catharsis. While implementation was feasible in the context discussed, and patients reported meaningful benefits, further investigation is necessary to more formally support the efficacy and use of supportive psychotherapy in medically admitted patients.Looking forward, studies that explore whether inpatient psychotherapy affects objective clinical outcome measures in addition to subjective patient experiences may be of particular benefit.

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The Utility of Metformin in the Management of Metabolic Syndrome Induced by Atypical Antipsychotics

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Abstract

Keywords: Antipsychotics,Metabolic Syndrome,Metformin

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1. Introduction

Metabolic syndrome is characterized by dyslipidemia, hypertension, central obesity, hyperglycemia, and insulin resistance. Together, these conditions lead to a significantly elevated risk of cardiovascular disease, type 2 diabetes, and cerebrovascular disease (1). All antipsychotic medications, particularly atypical or second-generation antipsychotics (SGAs), increase the risk for metabolic syndrome. The prevalence of metabolic syndrome in individuals taking antipsychotics ranges from 37% to 63% (2). The most significant metabolic side effects are associated with clozapine and olanzapine (3). In addition, diabetes and morbid obesity associated with SGA use may develop within as little as 6-12 weeks of treatment, whereas in the general population, these conditions typically take up to 5-10 years to manifest (3). Recent studies suggest that antipsychotics dysregulate glucose metabolism through CNS pathways, specifically through changes in the autonomic nervous system, neurotransmitter receptors, neuropeptide expression, AMP-activated protein kinase, and central insulin action (4). Moreover, antipsychotics block central glucose sensing, which impairs peripheral glucose metabolism (4).
Metformin is consistently utilized as a first-line medication in the treatment of type 2 diabetes. Its primary functions involve increasing insulin sensitivity, reducing gluconeogenesis, and increasing the peripheral uptake of glucose (5). Metformin is also being investigated as an adjunctive medication in patients using SGAs to attenuate insulin resistance and metabolic syndrome. Notably, metformin may lead to weight loss, reduced cardiovascular risk, and improved total cholesterol, LDL, and triglyceride levels. This review examines the efficacy of metformin in treating atypical antipsychotic-induced metabolic abnormalities, including insulin resistance and weight gain.

2. Discussion

Metformin's glucose-lowering properties, coupled with its ability to improve insulin sensitivity, may counteract the associated weight gain and glucose dysregulation that results from antipsychotic therapy. A recent study found that 78.8% of patients using first- and second-generation antipsychotics have an elevation in their baseline

doi: 10.46570/utjms-2025-1676 weight by > 7% (6). This raises a major concern regarding the metabolic changes associated with antipsychotic use. However, metformin may be a solution to this challenge. A meta-analysis was performed using global data from 14 studies that investigated the concomitant initiation of metformin with an antipsychotic, which found a statistically significant reduction in the weight gain with metformin (−3.12 kg) when compared with the placebo group, resulting in a 5.1% reduction in body weight change (7). A second meta-analysis of 12 studies found a mean difference in weight between metformin-treated and placebo groups of -3.27 kg (8). In addition, metformin was more efficacious in stopping weight gain when initiated prior to the development of insulin resistance compared to patients with chronic antipsychotic use who have already undergone weight gain (8). Taken together, these studies suggest that metformin may have greater efficacy in antipsychotic-naive patients to proactively stop weight gain. A different double-blind study was performed to evaluate the administration of metformin to attenuate olanzapine induced weight gain in drug-naive patients with schizophrenia. This study demonstrated that only 16.7% of subjects in the metformin group gained >7% of their initial body weight, compared with 63.16% in the control group, highlighting a substantial difference in weight gain between the two groups (6). According to current guidelines, weight loss of > 7% is the cutoff point for clinically significant weight gain (6). These findings suggest that metformin led to a statistically significant as well as clinically significant reduction in weight gain, which may attenuate the associated morbidity and mortality.

The current guidelines recommend obtaining baseline screening before or shortly after the initiation of any antipsychotic medication, including BMI, waist circumference, blood pressure, fasting glucose, and lipid profile. BMI should be reassessed at 4, 8, and 12 weeks after SGA initiation, and quarterly thereafter. Fasting glucose, lipid profile, and blood pressure should be reevaluated at 3 months, followed by annual assessments of blood pressure and plasma glucose, and lipid testing at 5-year intervals (9). Despite these guidelines, however, monitoring practices have been reported to be suboptimal (9).

The first evidence-based guidelines
were recently published recommending metformin

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for preventing antipsychotic-induced weight gain (AIWG). They recommend co-initiation with high-risk antipsychotics (olanzapine, clozapine), co-initiation with medium risk antipsychotics (quetiapine, paliperidone/risperidone) in patients aged 10-25 years or with cardiometabolic risk factors, and commencement with any antipsychotic if baseline weight increases >3% within the first year (10). Despite these novel guidelines, shared decision-making, a collaborative process between healthcare professionals and patients to make decisions about their care, will be necessary for implementation (10).

3. Conclusion

This review highlights metformin use as an advantageous adjunctive therapy in mitigating the metabolic dysfunction associated with second-generation antipsychotics, particularly weight gain and insulin resistance. Meta-analyses and clinical trials consistently demonstrate that metformin leads to statistically and clinically significant reductions in weight gain, notably when initiated early in treatment. Newly established guidelines for concomitant use of metformin in managing antipsychotic-induced metabolic effects represents an important step toward standardizing care, though successful implementation will rely on shared decision-making and continued evaluation.

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Toward a Treatment Algorithm for Misophonia: Integrating CBT and Pharmacotherapy

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1. Introduction

Misophonia is a condition marked by extreme sensitivity and diminished ability to tolerate specific auditory stimuli and related sensory cues such as breathing and chewing (1). These triggers cause a range of unpleasant responses, including anger, irritation, disgust, and anxiety. In addition to these emotional responses, there is also associated autonomic arousal such as increased muscle tension, heart rate, and sweating (2). These symptoms can lead to significant mental distress and impairment in daily functioning, with severity varying between individuals, contexts, and settings.

The reported incidence rate of misophonia varies due to limited research and the absence of standardized diagnostic tools (3). The symptoms of misophonia are often first observed during childhood or adolescence, a period critical for social and emotional development, and as such this condition can have profound consequences on relationships, academics, and overall quality of life (4). Thus, there is a clear need for the development of a systematic approach to treating individuals with misophonia. Although several studies have explored various treatment modalities, there is no standardized regimen (5). This review therefore proposes a structured, tiered treatment algorithm to act as a conceptual framework and inspire future research on this topic.

2. Discussion

Clinical Overview

While the pathophysiology of misophonia remains unclear, recent studies support that the etiology may be due to aberrant neural connections between auditory and limbic regions of the brain. These connections may cause heightened emotional reactivity to specific sounds and triggers (6). Furthermore, functional imaging studies show hyperactivation of the anterior insular cortex and abnormal salience network processing, suggesting that auditory triggers in individuals with misophonia may be assigned inappropriately elevated emotional significance (6). These findings align with the clinical observation, that individuals with misophonia experience increased emotional and autonomic arousal in response to trigger stimuli.

Individuals with more severe misophonia symptoms reported higher levels of anxiety and depressive symptoms as well as personality disorder traits including borderline and avoidant traits (7). These observations suggest that misophonia may exist within a broader psychological framework of emotional dysregulation and avoidance.

Given these findings and the large range of severity of symptoms for those with misophonia, a multi-tiered treatment algorithm should provide a flexible and pragmatic approach to address the heterogeneity of symptoms while also managing potential treatment side effects.

Proposed Treatment Algorithm

Based on a recent systematic review summarizing the existing interventions for misophonia including interventions such as Cognitive Behavioral Therapy (CBT), pharmacotherapy, and combined treatment approaches (5), this tiered algorithm integrates evidence-based modalities to support clinical decision making:

Tier 1: CBT Monotherapy

The first-line treatment is CBT as it targets maladaptive emotional responses and encourages exposure-based desensitization consistent with the strongest evidence level (Randomized Controlled Trial (RCT)) present in this review. In addition, CBT has the lowest side effect profile, making it an ideal treatment (5).

Tier 2: Selective Serotonin Reuptake Inhibitors (SSRIs) and CBT Combination Therapy:

For individuals with moderate or severe misophonia that do not respond to CBT alone, or that have significant comorbid anxiety and depressive symptoms, SSRIs combined with CBT could be added, to address both misophonia symptoms and comorbid disorders. This approach was effective in individuals that also experienced anxiety, depression, avoidance symptoms, and obsessive-compulsive disorder (OCD), with evidence limited to a handful of case studies. SSRIs likely would not be the ideal first line treatment as there is the potential for side effects such as nausea, appetite changes, decreased libido, QT prolongation, and serotonin syndrome (8).

Tier 3: Adjunctive Pharmacotherapy for Refractory Cases

In refractory cases where misophonia persists despite combined psychotherapy and SSRI

intervention, adjunctive use of benzodiazepines or atypical antipsychotics could potentially be considered, based on limited evidence showing efficacy. This treatment tier is the last line due to the significant lack of RCTs, with the potential for an even greater side effect burden.
Benzodiazepines carry a risk of cognitive, sensory, and motor impairment, as well as a high potential for dependence (9). While atypical antipsychotics are associated with weight gain, QT prolongation, metabolic syndrome, and in some cases extrapyramidal symptoms (10).

3. Conclusion

This proposed three-tiered treatment algorithm offers a structured yet flexible framework for managing misophonia, a condition with significant heterogeneity in symptom presentation and severity. By prioritizing CBT as a first line intervention and escalating to pharmacological treatments, this approach seeks to balance positive clinical outcomes, symptom severity, and minimize the potential side effect burden.
This proposed approach is not without drawbacks. The current evidence base for misophonia interventions remains limited, consisting largely of case studies and small trials. As such, this algorithm should be viewed as a conceptual framework rather than a definitive treatment guideline. Future research, including randomized controlled trials and larger cohort studies are essential to validate these strategies and to refine treatment plans for this understudied condition.

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TRANSLATION

Special Issue 2026

THE P'S OF WOMEN'S MENTAL HEALTH

Premenstrual, Pregnancy and Postpartum Care

- Role of Stress and Inflammation in the Etiology, Treatment, and Management of Premenstrual Dysphoric Disorder
- Pre-eclampsia and the Risk of Development of Psychiatric Disorders
- New Onset Psychosis throughout Pregnancy and Postpartum: Risk Factors and Maintenance Therapy

COLLEGE OF MEDICINE AND LIFE SCIENCES
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Role of Stress and Inflammation in the Etiology, Treatment, and Management of Premenstrual Dysphoric Disorder (PMDD)

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Abstract

According to the DSM-5, Premenstrual Dysphoric Disorder (PMDD) is defined as a mood disorder characterized by physical, emotional, and behavioral symptoms that occur during the luteal phase and cause significant distress or impairment. While epidemiological studies highlight that PMDD occurs in 1.8-5.8% of menstruating individuals, this is a vast underestimation prevalence due in part to cultural and systematic factors that impact diagnosis. That said, it is imperative to conduct research on this debilitating condition and its impact on overall wellbeing. The objective of this review is to explore the role stress and inflammation plays on etiology, treatment, and management of PMDD. To ensure a thorough review, the inclusion criteria focused on studies published in peer reviewed journals on etiology (stress and inflammation in relation to PMDD) and the treatment and management of PMDD. Studies conducted on topics pertaining to genetics and serotonin dysregulation were not included. Additionally, research conducted prior to 2005 were also excluded from the review process. Overall, existing literature demonstrates that stress and inflammation has been found in individuals with PMDD. However, more research must be done on the potential therapeutic value of Yaz, other COCs, and anti-inflammatory agents in treatment of PMDD.

Keywords: Inflammation, Women's Health, Depression, Stress, Suicide

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1. Introduction

Premenstrual Dysphoric Disorder (PMDD), as defined in DSM-5, is a mood disorder marked by physical, emotional, and behavioral symptoms during the week before menses, the luteal phase. Diagnosis requires at least five symptoms related to mood, and additional physical and behavioral symptoms, which must cause significant distress or impairment limited to the luteal phase. For a full diagnosis, symptoms must be documented prospectively over two menstrual cycles, as retrospective reports only warrant a provisional diagnosis. PMDD affects an estimated 1.8–5.8% of menstruating individuals, although the actual prevalence may be higher due to patient difficulties with documentation (1). Considering PMDD's strong association with suicidality and significant impact on quality of life, further investigation is needed (2). This manuscript will review the role of stress and inflammation in the etiology, treatment, and management of PMDD. By viewing PMDD as a maladaptive stress response to luteal cycle hormonal fluctuations, it offers a framework for treatments targeting both the hormonal fluctuations and the physiological response.

Stress, Inflammation, and PMDD

Proposed etiologies for PMDD include genetics, serotonin dysregulation, and stress and inflammation. Studies show individuals with and without PMDD experience similar levels of peripheral ovarian hormones during the luteal phase; however, those with PMDD respond differently to luteal fluctuations, possibly due to the impact of stress and inflammation. A study of almost 4000 women found trauma and PTSD to be independently associated with PMDD. Repeated life stressors and social stressors have been shown to increase inflammatory markers such as C-reactive protein (CRP), interleukin-6, and interleukin-1β (3). Mood disorders have consistently been associated with inflammation, with increased pro-inflammatory cytokines seen in patients with mood disorders (4). In PMDD, there is a relative increase in inflammatory markers in the luteal phase. Additionally, elevated hs-CRP levels have been linked to emotional, behavioral, and physical symptoms of PMDD (5).

Treatment of PMDD

PMDD treatment targets both hormonal fluctuations and the resultant systemic response. Combination oral contraceptives (COCs) are a first-line treatment that stabilize luteal cycle fluctuations by preventing ovulation. Despite wide usage, COCs vary in composition, and research on their efficacy in treating PMDD remains limited. Drospirenone/Ethinylestradiol is the only FDA approved medication for treatment of PMDD, as it has been shown to improve emotional and physical symptoms in comparison to placebo (6). Its unique formulation and dosing schedule, 24 active pills followed by 4 placebo pills, differs from the standard 21-active, 7-placebo schedule that other COC's possess. Its longer dosing schedule for hormones is thought to contribute to its effect on mood in PMDD (7). Additionally, its progesterone derivative, drospirenone, possesses anti-androgenic and diuretic effects. Lastly, the systemic response to luteal cycle fluctuations has historically been addressed through selective serotonin reuptake inhibitors (SSRIs), luteal specific or continuous, alongside cognitive behavioral therapy (CBT), which has been effective at reducing mood symptoms of PMDD. (7).

2. Discussion

Fleshner (2017) proposed a mechanism linking stress, inflammation, and mood pathology, theorizing that exposure to stressors triggers the release of danger-associated molecular patterns (DAMPs) from injured neurons, which prompts creation of inflammatory markers increasing tryptophan breakdown, thus impairing serotonin synthesis and the body's stress response (8). In addition to existing literature, this supports the hypothesis that stress and inflammation play key roles in the pathophysiology of PMDD.
The unique formulation and dosing schedule of Drospirenone/Ethinylestradiol likely plays a key role in alleviating PMDD symptoms. Compared to levonorgestrel-based contraceptives, drospirenone has been shown to reduce water retention and weight gain (9). Prescribers should especially consider Drospirenone/Ethinylestradiol for patients experiencing these issues. For severe cases of PMDD, clinicians should explore extended dosing regimens, which can mitigate mood disturbances tied to hormone withdrawal during placebo intervals. Aside from potential breakthrough bleeding, extended dosing regimens have not been linked to significant adverse effects.

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Furthermore, the elevated inflammatory markers seen in PMDD suggest potential benefit from anti-inflammatory agents for PMDD, including dietary curcumin, omega-3 fatty acids, acetyl-salicylic acid, etc. These therapies have shown some success in mood disorders; however, they require more investigation as potential treatments for PMDD. Lastly, although CBT is established as the core psychotherapy intervention for PMDD, emerging therapies warrant exploration. Specifically, given the association between PMDD and PTSD, therapies such as eye movement desensitization and reprocessing, which reduces psychophysiological stress responses and enhances parasympathetic tone in PTSD populations, may be helpful as adjunct therapies (10).

3. Conclusion

Existing literature has shown a connection between stress, inflammation, and the pathophysiology of PMDD. However, additional research on the therapeutic value of Drospirenone/Ethinylestradiol and other COCs, as well as anti-inflammatory agents is necessary. In summary, a multidisciplinary approach addressing hormonal fluctuations, stress, and inflammation should inform clinical practice and guide development of therapies for PMDD.

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Pre-eclampsia and the Risk of Development of Psychiatric Disorders

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Abstract

Pre-eclampsia (PE) is a severe disease of pregnancy occurring most often around 20 weeks gestation and marked by symptoms of new-onset hypertension, proteinuria, and end organ damage. Given its high prevalence and serious risk in causing complications, it is important to understand the etiology and potential outcomes of PE. Emerging research suggests that PE during pregnancy may be associated with an increased risk of severe psychiatric disorders in the mother. In this study, we aim to review the current literature examining whether women with PE are more likely to develop subsequent psychiatric disorders including psychosis, mania, and PTSD.

Keywords: Psychosis,PTSD,Preclampsia

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1. Introduction

Pre-eclampsia (PE) is a severe disease occurring most often around 20 weeks' gestation and comprises 2-8% of pregnancy-related complications, contributing to more than 50,000 maternal deaths worldwide. Marked by symptoms of new-onset hypertension, proteinuria, and end organ damage, this disorder poses a significant risk to both maternal and fetal health. Given its high prevalence and serious risk of causing complications, it is important to understand the etiology and potential outcomes of PE. Emerging research suggests that PE during pregnancy may be associated with an increased risk of severe psychiatric disorders in the mother including psychosis, mania, etc. In this study, we discuss the current literature examining whether women with PE are more likely to develop subsequent psychiatric disorders.

2. Discussion

PE and Neural Impact

Recent studies have been exploring the potential link between PE and the development of psychiatric disorders in mothers. PE as a hypertensive disorder is a known systemic condition with poor understanding of its etiology. The main proposed etiology presently includes the development of uteroplacental ischemia leading to systemic inflammation, vascular dysfunction, and immune dysregulation (1). A major mechanism of neurovascular dysfunction caused by PE is thought to be through its ability to impair cerebral blood flow autoregulation. This exacerbates hyperperfusion of the brain, leading to elevated blood brain barrier permeability and causing neuroinflammation, which in turn may disrupt mood regulation capabilities (2). In patients with genetic predisposition to psychotic illness, the inflammatory or neurovascular stress associated with PE may serve as a potential trigger for onset of illness. PE is one of the most prevalent immune-mediated disorders and like disorders such as postpartum psychosis, is strongly linked to first pregnancies (3). This overlap between the two leads to the idea that women with PE may be associated with a higher risk of developing severe postpartum psychiatric illness.

PE and Mood Disorders

Many studies have shown evidence of PE being a risk factor for postpartum depression and anxiety. Only a few studies, however, have suggested a strong connection between PE and mood disorders. For example, one large Danish population study found that primiparous women are at risk of first onset psychiatric disorders including psychosis, mania, and schizophrenia spectrum disorders (4). More specifically, women with PE were associated with a four times increased risk for first onset of these disorders. Other population-based studies support these findings, with one study finding that patients with pre-eclampsia were at a higher risk for developing postpartum depression and postpartum acute stress reaction than patients without PE (5). While several studies suggest that PE may contribute to the development of psychiatric disorders in the postpartum period, there is also evidence indicating psychiatric illness may emerge antepartum in the context of PE. One documented case report described the onset of acute psychosis in a patient with PE, introducing a potential causal relationship between the two conditions (6). Although the patient had additional risk factors for psychosis, including previous psychiatric history and chronic substance use disorder, the patient's psychiatric symptoms resolved following treatment of PE and delivery. This further suggests that psychosis may have been precipitated by the hypertensive nature of PE.

PE and PTSD

A significant number of women who have experienced PE during their pregnancy report their labor as a traumatic event (7). This is consistent with the fact that PE often leads to the need for more intervention during labor and birth at an earlier gestation than expected. Therefore, there may be a relationship between PE and resultant development of PTSD following birth. There have been studies produced that show evidence of this, with one particularly noting that women who reported a history of PE were over four times as likely to screen positive for PTSD symptoms than women with normal pregnancies (8). However, a recent cohort study showed different results, finding no statistical significance difference in PTSD prevalence at six months postpartum in women with PE compared to those with normotensive pregnancies (9). There are limitations in measuring PTSD symptoms which may explain the difference

in results. Given that the perception of a traumatic birth may vary amongst patients and is a subjective measure, it can be hard to effectively define (10).

3. Conclusion

PE is a hypertensive complication of pregnancy that leads to inflammatory and neurovascular stress posing as a potential trigger for onset of psychiatric illness. Women with PE during their pregnancy may be at an increased risk to developing various psychiatric disorders including psychosis, mania, schizophrenia spectrum disorder, and PTSD. There is a need for continued research on PE, however, to clarify this and support targeted screening and early intervention strategies for pregnant women. As more focused studies examine the relationship between hypertensive disorders of pregnancy and severe psychiatric outcomes, better management, care, and support for affected patients is possible.

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New Onset Psychosis throughout Pregnancy and Postpartum: Risk Factors and Maintenance Therapy

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1. Introduction

New-onset psychosis during pregnancy and the postpartum period is a rare but severe psychiatric emergency with potentially life-threatening consequences for the mother and infant. Postpartum psychosis (PP) is most likely to present within the first two weeks after birth and is characterized by hallucinations, delusions, insomnia, mood fluctuation, irritability, and disorganized or risky behavior (1, 2). The condition is estimated to affect 1 to 2 women per 1,000 deliveries, with higher risk among those with underlying bipolar disorders or prior episodes of PP (3). Although prevalence is highest among this population, around 40% of PP cases occur in women with no prior psychiatric history, which complicates detection and timely intervention (4). Despite the fact that PP is not its own diagnosis in the DSM-5, it is classified as a specifier for mood or psychotic disorders with “peripartum onset,” defined as symptoms emerging during pregnancy or within four weeks postpartum (5). The American College of Obstetricians and Gynecologists (ACOG) recommends universal mental health screening at least once during the perinatal period, with additional screening for mood and anxiety disorders at the postpartum visit (6). Despite its severity, there are no formal guidelines for long-term maintenance treatment of PP (1). Clinical decisions are often guided by expert opinion and extrapolated from data on bipolar disorder, rather than psychosis-specific studies. This mini review aims to examine the clinical risk factors and treatment strategies for new-onset psychosis during pregnancy and the postpartum period.

2. Discussion

PP is relatively unique in that a clear etiological event can be defined, although the specific mechanism is unclear at this time. Many potential risk factors have been identified, ranging from biological to psychosocial to peripartum complications. One plausible biological trigger for psychosis includes hormonal withdrawal, as levels of estrogen and progesterone sharply decline after birth. However, immediate post-birth administration of estrogen in a small case series demonstrated no impact in women at risk for postpartum mood disorders (1). Immune system activation may also contribute to the development of acute PP, which is supported by evidence of the co-occurrence of PP with thyroiditis and autoimmune encephalitis. Sleep deprivation is the most consistently reported finding in women with psychosis. Labor can cause initial disruption of circadian rhythm, which is further compounded upon by newborn awakenings, which may contribute to emergence of psychiatric symptoms. Also, specific genetic variants of serotonin gene receptors are potentially implicated (1). Lastly, additional biological risk factors may include PP experience with previous births and family history. Furthermore, psychosocial vulnerabilities, such as lack of partner support, low socioeconomic status, exposure to adverse childhood events, and perinatal stress have also been outlined (1, 4). In addition, peripartum complications, such as preeclampsia, emergency cesarean section, postpartum hemorrhage, and ICU admission were more frequently observed in women who developed psychosis, suggesting that physical stress on the body may be involved in PP development (7). Lastly, studies are suggesting that PP episodes may indicate an underlying bipolar disorder. For instance, 14% of women with depressive episodes during the first postpartum month were later diagnosed with bipolar disorder within 15 years, compared to only 4% whose onset was unrelated to childbirth (1). This reinforces the need for more research to better characterize both the pathophysiological evolution and the treatment of PP.

Lithium remains the most evidence-based pharmacologic option for both the acute and maintenance treatment of PP. In the largest study to date (N = 64), lithium demonstrated robust efficacy in stabilizing acute symptoms and preventing relapse in the long term (1). When initiated within 24 hours postpartum in high-risk individuals, lithium maintenance therapy reduced relapse rates from 40–60% to under 20% (8). Given that women who experience PP are at a 31% risk of recurrence in subsequent pregnancies, early intervention and sustained mood stabilization are essential (1). Additionally, adjunctive antipsychotics are frequently used during the acute phase of PP, particularly in cases involving mania and agitation. Agents such as olanzapine and quetiapine are most commonly used, though clinicians must remain mindful of teratogenic risks and the potential for transmission through breast milk (8). Lastly, electroconvulsive therapy (ECT) is an especially valuable option for severe, treatment-resistant, or life-threatening presentations of PP, including those

complicated by suicidality or catatonia. Studies have reported remission rates as high as 80–90% in small case series, noting ECT's favorable safety profile in the postpartum period (8).

3. Conclusion

This review underscores the complexity of new onset peri- and post-partum psychosis and emphasizes the importance of risk factor recognition, routine screening, and long-term follow-up. Evidence consistently shows a multifactorial etiology of PP, which may include biological predisposition, psychosocial vulnerabilities, and peripartum complications. Lithium, antipsychotics and ECT have demonstrated efficacy in the treatment of PP, however, further research and standardized clinical guidelines are essential to optimize care.

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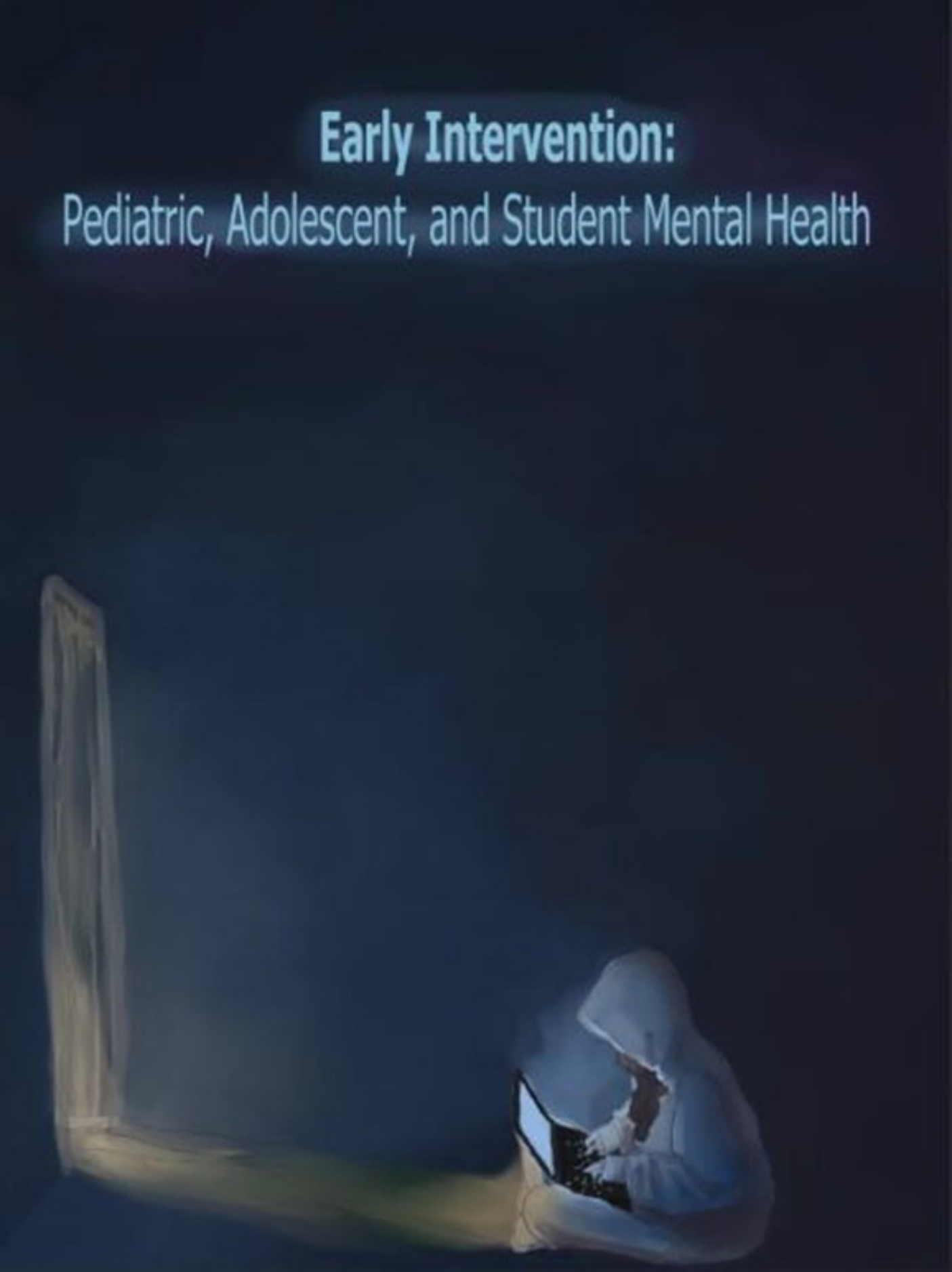
Early Intervention:

Pediatric, Adolescent, and Student Mental Health



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Pediatric Mental Health Crisis: Current Guidelines and Adjustments

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Abstract

Pediatric anxiety, depression, and suicide rates are on the rise across the U.S. and Ohio. This mental health crisis has placed significant burden on both the emergency department and primary care providers. The emergency department does not have the resources for the long-term care of pediatric patients struggling with mental health. Furthermore, primary care providers do not feel their training has equipped them to manage the psychiatric conditions plaguing today's youth. To help address this crisis and decreased the number of patients resorting to emergency care, primary care and pediatric residency programs can implement seminars, simulations, and trauma-informed care trainings. Additionally, robust screening protocols, discussions about pediatric mental health in office, and increasing connections between physicians and mental health services will ensure patients are appropriately screened, diagnosed, and treated for psychiatric conditions.

Keywords: Pediatric Primary Care,Mental Health,Residency Training

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1. Introduction

Anxiety, depression, and suicide are increasingly prevalent in the U.S., culminating in a mental health crisis. As a result, suicide is the second leading cause of death among children 10-19 years old (1). The United Health Foundation's *America's Health Rankings 2024 Annual Report* showed that 19.9% of children ages 3-17 are diagnosed with a mental health condition in the U.S. (Table 1) (2). In Ohio, 22.1% of children 3 to 17 years old are diagnosed with a mental health condition (Table 1) (2).

In response to this crisis, pediatric primary care providers are recommended to screen for anxiety, depression, and suicide. However, many providers do not always ask patients about their mental health. About 76% of primary care physicians (PCPs) believe it is important to talk to adolescent patients about their mental health; however, only 46% ask about their patients' mental health at every visit (1). Although there have been calls to improve mental health training within pediatric residency programs and the ACGME requires a developmental and behavior pediatrics rotation, many pediatricians still do not feel fully equipped to treat patients with mental health conditions (3). In this mini review, we discuss the current guidelines for pediatric mental health management, pediatric residency training in mental health, the uptick of mental health-related emergency department (ED) visits, and methods in which PCPs can address this demand.

Current Pediatric Mental Health Guidelines

Per the current guidelines, pediatricians are recommended to screen for anxiety, depression, and suicide risk (1, 4, 5). Screening instruments for anxiety and depression are recommended for patients ages 8-18 years and 12-18 years respectively (1, 5). The most used anxiety screening tools are Screen for Child Anxiety Related Disorders (SCARED) and Social Phobia Inventory (SPIN) (5). The 9-item Patient Health Questionnaire (PHQ-9) is the most used screening tool for depression (1). Screening for suicide involves asking patients about current and past suicidal ideation, self-harm, and suicide attempts (1). In addition, pediatricians should consider factors that increase the risk of these conditions, including personal and family history of mental illness, age, sex, race, ethnicity, and adverse childhood experiences (1, 5). Pediatric clinical systems are advised to collaborate with mental health specialists to enhance their knowledgebase, consult mental health services and refer to specialists as needed to provide adequate support (1, 4, 5). This ensures patients are appropriately screened, diagnosed, and treated.

Resident Training in Pediatric Mental Health

The ACGME requirements for pediatric residency training list diagnosing, assessing, and treating behavioral and mental health (B/MH) conditions as a core competency (6, 7). Since only about half of B/MH referrals from pediatric offices reach mental health specialists, this burden falls on PCPs and pediatricians (8). Sadly, many physicians feel their residencies did not prepare them to manage B/MH conditions. While 86% of pediatric residents felt that those entering pediatric subspecialties should be trained in B/MH competencies, only around 52% feel confident in their ability to assess and treat them (9). There have been improvements to residency training, however. The Be ExPeRT (Behavioral Health Expansion in Pediatric Residency Training) program is one example. After an initial half-day seminar and four monthly case-based seminars, 23 residents reported improved confidence in assessing, diagnosing, and treating depression, anxiety, and suicidal ideation (8).

Emergency Department Trends in Mental Illness Among Youth

The rising prevalence of pediatric mental illness has been accompanied by a significant increase in mental health–related ED visits. In the U.S., pediatric mental health-related ED visits increased from 4.8 million (7.7% of all pediatric ED visits) in 2011 to 7.5 million (13.1% of all pediatric ED visits) in 2020 (10). Among the pediatric mental health-related ED visits, suicide-related visits increased the most, increasing from 0.9% in 2011 to 4.2% in 2020 (10). These trends of mental health-related ED visits demonstrate the burden the mental health crisis has placed on the ED.

2. Discussion

Pediatric mental health conditions are not being treated effectively. Patients often are unable to see mental health specialists, so the burden falls to PCPs and pediatricians. However, many physicians do not feel prepared to treat these conditions. Pediatric patients are then forced to seek treatment at the ED, which increases their burden. Seeing as one of the ACGME's core competencies

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is treating mental health conditions, pediatricians need robust training. This can include seminars, simulations, and trauma-informed care. Some programs have implemented new strategies, such as Be ExPeRT, which has increased physician competency in diagnosing and treating mental health conditions. With consistent screening and management of these conditions in primary care settings, patients will not have to resort to emergency care.

3. Conclusion

Anxiety, depression, and suicidal ideation are increasingly prevalent among pediatric patients, and rates have continued to rise. Much of the burden of management falls to PCPs, pediatricians, and EDs, since it is difficult to see mental health specialists. However, PCPs and pediatricians do not feel prepared to handle these conditions, and EDs are often overwhelmed by psychiatric visits. Pediatricians and PCPs need to be equipped to manage mental health conditions.

Supplementary Content

Table 1. Ohio and U.S. Mental Health and Suicide Statistics.

Mental health conditions include ADHD, depression, anxiety or behavior or conduct problems diagnosed by a healthcare provider. Mental health conditions, anxiety, and depression data is from 2022-23. Suicide data is from 2020-22. Mental health statistics for Ohio and the United States are from the United Health Foundation's *America's Health Rankings 2024 Annual Report* (2).

	Ohio	United States
Diagnosed Mental Health Conditions (% children ages 3-17)	22.1	19.9
Anxiety (% children ages 3-17)	12.7	10.7
Depression (% children ages 3-17)	5.7	4.4
Suicide (deaths per 100,000 adolescents ages 15-19)	9.6	10.5

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AI and Existential Dread: A Rising Concern in Pediatric Psychology

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1. Introduction

Artificial intelligence (AI) is reshaping modern life, from how we communicate and learn to how we perceive our own abilities. For adolescents, who are in the formative stage of identity development, AI introduces psychological challenges that remain poorly understood. As generative models now author essays, compose music, and solve complex problems, young people are increasingly exposed to comparisons that question the uniqueness of human thought.

Digital media has long been associated with increased anxiety and depressive symptoms in youth (1). But AI may take this further, challenging foundational beliefs about human relevance. Adolescents may begin to wonder: What is the point of cultivating skill or meaning in a world where machines are more efficient? What was once an abstract concern for adults is now a daily dilemma for teenagers. Mental health providers are starting to encounter this distress, yet few frameworks exist to guide clinical understanding. This paper explores the rise of AI-related existential anxiety in adolescents, relevant psychological theories, and implications for pediatric mental health care.

2. Discussion

AI Exposure and the Changing Mental Health Landscape

In the last decade, mood disorders and identity-related struggles have surged among adolescents (1). While social media and academic pressures have received significant attention, AI introduces a distinct psychological burden. Teens now routinely interact with generative tools like ChatGPT or image generators, which perform tasks once considered exclusive to human creativity and intellect (2,3).

Some adolescents report feelings of inadequacy and irrelevance after seeing AI produce superior creative or intellectual output (4). In clinical settings, providers are beginning to hear youth voice fears of being replaceable or unnecessary. Although large-scale studies remain limited, emerging qualitative evidence suggests these reactions are not isolated. Unlike previous technology-related anxieties tied to social comparison or attention span, AI-related distress appears more ontologically centered on meaning, identity, and purpose (4).

Psychological Frameworks Explaining the Response

To understand why AI provokes such distress, it is helpful to integrate developmental and existential theories. Erik Erikson's psychosocial model characterizes adolescence as a period of identity formation (5). During this phase, adolescents explore values, goals, and roles that contribute to a cohesive sense of self. When AI encroaches into academic or creative spaces, it can destabilize this process. If a machine can outperform a student's best effort, questions about their value or future contributions may arise.

Existential psychology further illuminates this experience. Irvin Yalom outlined core human concerns, including meaninglessness, isolation, and the threat of non-being (6). Adolescents exposed to narratives of AI dominance may experience these concerns prematurely and more intensely. Alvin Toffler's "future shock" offers another lens, describing how overwhelming technological acceleration can induce psychological disorientation (7). Today's teens are not just forming identity online; they are doing so in an era where AI is increasingly portrayed as surpassing human potential.

Clinical Implications for Pediatric Psychiatry

As AI continues to permeate learning environments, social media, and entertainment, clinicians must be prepared to recognize AI-related distress. Standard intake forms and checklists may overlook existential fears such as obsolescence or purposelessness. Instead, mental health professionals may need to ask open-ended questions about AI use and related emotions, especially when adolescents are present with identity confusion, demotivation, or a sense of futility (8).

Therapeutic approaches grounded in meaning-making and value-based living may be particularly useful. Narrative therapy can help youth reclaim authorship of their identity in a machine-dominated world (9). Acceptance and Commitment Therapy (ACT) encourages patients to accept difficult thoughts and commit to actions aligned with personal values, offering a practical way to address existential anxiety without resistance (10). These interventions shift focus

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from comparison with machines, such as emotional depth, moral reasoning, and lived experience.

Psychoeducation also plays a critical role. Helping adolescents understand AI's limitations and lack of consciousness can reframe unhelpful comparisons. Applying a biopsychosocial model, clinicians can situate AI anxiety within developmental and societal contexts, ensuring treatment reflects the complexity of adolescent experience. Families and educators can further reinforce messages of intrinsic human worth and encourage engagement in activities that foster purpose beyond productivity.

3. Conclusions

AI represents more than a technological shift; it is a cultural and psychological one. For adolescents already navigating the vulnerable process of identity formation, generative models introduce new existential stressors. Feelings of displacement, inadequacy, and disconnection are emerging in clinical settings and must be taken seriously. Although research in this area is just beginning, existing theories and early observations support the concern that AI may be reshaping adolescent mental health. Pediatric providers must guide youth through this transformation with compassion, evidence-based interventions, and a renewed focus on meaning, resilience, and what it truly means to be human.

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Feasting on Flesh: A Case Report and Literature Review of Cannibalistic Ideation in Children & Adolescents

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Abstract

Human cannibalism is an extremely rare and violent act that is poorly understood in the modern era, and pediatric cannibalism is even less understood. This is due to methodological hurdles when investigating the topic, including a lack of data from the sheer rarity of the act as well as bias in case analysis. Here, we will detail a case of an adolescent with cannibalistic ideation. This 14-year-old male presented to the ED due to ego-syntonic thoughts of cannibalism and was promptly admitted for an inpatient psychiatric stay for four days. Through this case report, we aim to improve medical providers' understanding of pediatric cannibalism and cannibalistic ideation and provide psychodynamic context.

Keywords: Cannibalism, Pediatric Psychiatry, Psychiatry, Forensic psychiatry

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1. Introduction

The word cannibalism – the eating of one's own kind – evokes a myriad of feelings, including disgust, horror, and even morbid curiosity. It does not currently exist as a diagnosis in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR), and there is limited research and professional experience on cannibalism, particularly in children. The existing literature primarily focuses on the rarity of such behaviors and the complexities of their underlying causes. S Jesus et al. (1) explores the occurrences of cannibalism among individuals, including children, faced with extreme psychiatric disorders. This study suggests that extreme cases of cannibalism often arise either from or in conjunction with severe mental illness or trauma (1). Furthermore, a connection between severe abuse or neglect can give rise to the emergence of atypical behaviors, including cannibalism, in children (2). While the acts or thoughts of cannibalism among children are extremely rare, the reported cases that do exist are often linked to psychiatric disturbances or exposure to traumatizing environments (3). As such, with the minimal research surrounding the topic of cannibalism in children, these studies emphasize the necessity for further research and investigation to help physicians and social workers understand the factors contributing to such rare and extreme behaviors in children. With more awareness and research into the topic, our goal of this paper is to encourage providers to investigate ways to mitigate the emergence of these behaviors.

We will direct our focus to a case of a 14-year-old male who presented to the emergency department with a chief complaint of the desire to eat his peers. He described these thoughts as enticing, causing his mouth to water at the sight of his peers' skin. With the potentially violent nature of his expressed thoughts, he was admitted from the emergency room to the child & adolescent inpatient psychiatric unit at The University of Toledo Medical Center. With minimal research and standardized practices involved with the emergence of cannibalism in children, limited guidance was available on best practices in clinical management of this pediatric patient. As such, the aim of this paper is to increase awareness of the presentation of cannibalism in children and to help guide care teams in effectively treating these complex but rare cases.

2. Case Report

Mr. X, a 14-year-old male, presented to the emergency department in May 2024 after telling his school psychologist that he had been having thoughts of cannibalism. He was promptly admitted to the pediatric inpatient psychiatric unit, where history was collected from the patient and his family. Prior to this admission, he had a history of suicidal ideation and no pertinent medical history. Family history was significant for bipolar disorder and a suicide attempt in his maternal grandmother and anxiety and depression in his three half siblings. *History of Present Illness* During his initial interview at admission, the patient reported that he had been having thoughts of wanting to bite, chew, and eat random people he sees, especially his peers at school. These thoughts had been occurring for a few months but were becoming more intense and more difficult to control, according to the patient. The patient reported an interest in tasting different skin textures including eczema or specific body parts including the eyes, ears, and fingers, which made his mouth water to think about. He said he never acted on these thoughts, but he did want to hurt others. He denied feeling bad about having thoughts in the moment but did come to regret them later. He denied any current suicidal or homicidal ideation. The patient denied any psychotic features including visual or auditory hallucinations but did report having two imaginary friends who helped him decide what to do in life. The patient stated that after he started having these thoughts, he watched a documentary on Jeffrey Dahmer, a cannibalistic necrophiliac serial killer who committed 17 murders between 1978 and 1991. Mr. X believed that he could relate with him and became fascinated with the idea of being able to do what he wanted without having to suppress his thoughts. This admiration of Jeffrey Dahmer alarmingly extended to other serial killers as well, including Ted Bundy. *Collateral Information* At the time of admission, the patient's mother and father were interviewed as well. According to them, this was the first time they were made aware of the thoughts their son had been having and were shocked at the revelation. Interestingly,
his father described him as a master at manipulation and questioned his honesty. Regarding the history of violence, they denied any animal cruelty but did state that the patient was physically aggressive with his younger sister, even choking her at one point and wishing her dead. The parents noted a history of the patient saying bizarre things including comments about death,

playing Russian roulette, and setting the house on fire. They also reported concern that the patient would frequently talk to himself when alone in his room and would hear him having both sides of the conversation, but they did not believe he was hallucinating. The parents stated that the patient had endorsed suicidal ideation beginning at age 7 years and previously used clonidine which was prescribed by a local behavioral health center. His mother also reported that the patient was previously sexually active with a 25-year-old man whom she did not know. There were previous allegations of sexual abuse by a family member, however no evidence was found, and the allegations were dropped.

Social History

The patient was a student in Toledo, Ohio and lived with his mother who was pregnant at the time, father, older brother, younger sister, and grandmother. His familial relationships were described as fair. He had no history of behavioral issues at school and scored highly on state testing. He had no history of substance use and a urine toxicology screen was negative at the time of admission. He was exposed to marijuana while in utero. The patient expressed a romantic interest in males and experimented with wearing dresses and purses occasionally. He denied a history of abuse, neglect, or trauma other than the aforementioned.

Hospital Stay

During his stay at the inpatient unit, the patient was formally diagnosed with Episodic Mood Disorder and started on sertraline. He was found to exhibit some traits of narcissistic and antisocial personality disorders. His stay was complicated by an episode of hoarding acetaminophen with the stated intention of collecting enough to kill himself. After a four-day hospitalization, he felt ready to connect with his support system, expressed forward thinking, tolerated medications, and was subsequently discharged home with his parents.

3. Discussion

The 14-year-old male Mr. X with a pertinent past psychiatric history of suicidal ideation since age 7, self-harm since age 9, and a history of physical violence toward a younger sibling presented to the emergency department after months of cannibalistic ideation with increasing intensity and associated symptoms of salivation and craving. He stated he felt no guilt or shame while having these cannibalistic thoughts, only later feeling a sense of guilt and regret. He also reported having a fascination with Ted Bundy and Jeffrey Dahmer as he envied their freedom from guilt and believed that he could relate to Dahmer. Importantly, he noted he experienced cannibalistic ideation prior to media exposure to Dahmer. After a 4-day inpatient psychiatric stay, the patient was discharged home with a diagnosis of Episodic Mood Disorder after developing a stronger coping mechanism for stress and a change in medication.

The question of what makes a person become a cannibal has been a topic of interest in forensic science for quite some time. While cannibalism is relatively common in the animal kingdom, anthropophagy, or human cannibalism, it is often regarded as one of the greatest taboos in our society (4). However, it has occurred in various populations throughout history including hominids, the Crusaders, soldiers in World War II (5), and the culture-bound-syndrome of Wendigo psychosis (6).

Cannibalism, from a psychoanalytic standpoint, can be seen as the incorporation, introjection, or identification with an object, as well as oral sadism. Additional themes associated with cannibalism include domination, vengeance, and punishment. Sigmund Freud postulated that cannibalism stems from an unconscious memory common to all humans, suggesting that the act of primal cannibalism could be linked to the Oedipus complex. He proposed that there were several phases of sexual development, the first of which being the “oral cannibalistic” stage. Freud explained that during this stage, expressions of love, aggression, and pleasure from both nourishment and sexuality are integral to human experience. He also emphasized the importance of identification with others and how these influence one’s sense of self. He stated that the act of consuming someone involves taking them in both physically (i.e. the act of eating) and psychologically (i.e. integration of their personality
or behaviors into one’s own identity). Melancholic identification can occur when a person becomes fixated on an object or person of their desires. In this situation, there is a drive to cannibalize the object, thereby absorbing and destroying it. It is also possible, according to Freud, for violent tendencies and identification to become directed toward the self, resulting in autocannibalism. By regressing to a cannibalistic developmental stage, the melancholic individual reenacts a primal desire to eliminate and replace the father figure.

However, due to the taboo surrounding cannibalism, the libido initially directed toward the object is distorted and redirected, causing the melancholic individual to turn it against themselves (7).

While the psychoanalytic perspective provides a symbolic interpretation of cannibalism rooted in unconscious desires and early developmental stages, more contemporary views approach the topic through social, cultural, and ethical lenses, focusing on its implications and meanings in modern society. As cannibalism may occur due to several distinct reasons, researchers have attempted to classify cannibalism into various subtypes. Some may differentiate by the act itself, including self-cannibalization (autosarcophagy), the act of only drinking human blood (vampirism), and cannibalistic homicide (murder for food), which may be further subdivided into kin-cannibalism and non-kin-cannibalism (5, 8, 9). Others differentiate by motive, including revenge, medicinal, lust, delusional, self-defense, humiliation, fantasy, sexual fantasy, or sadism (10, 11, 12).

One review article detailed cannibalism classification into 3 broad types. The first type they detailed as nutritional or gastronomic, which includes the subtypes of medicinal, famine-induced, and placentophagy. The second was ritual cannibalism, which is often tied to a religion or belief system. And the third type they named pathological cannibalism, which includes cannibalistic homicide as well as autosarcophagy (5). One could consider trichophagia as a form of autosarcophagy as well (13).

In yet another classification system, authors posit that cannibalism could be separated by the victim's social group in relation to the perpetrator. This results in two main types, exocannibalism and endocannibalism. In exocannibalism, the victim is from an outside social group to the perpetrator, while in endocannibalism, the victim is from the same social group as the perpetrator (14). These various classifications help to separate between socially acceptable and non-acceptable forms of cannibalism. And when it comes to pathological cannibalism, they also help to elucidate the various reasons why an individual enacted such an extreme act of violence (5).

Many are interested in the psychiatric associations with cannibalism as well. In a recent case study of 5 cannibals, cannibalism was found to be associated with schizophrenia as well as mixed personality disorder (11). These researchers found that those diagnosed with schizophrenia participated in cannibalism as an extension of self-defense, believing that their survival depended on the assimilation or the annihilation of the other. Those diagnosed with a mixed personality disorder chose to engage in the act as they had felt humiliated by their victim, which prompted the individual to overcome their humiliation through this violent act. Another study investigating cannibalistic homicide found that cannibals that victimized their kin were statistically more likely to have a higher mental health score than cannibals that did not (8). This association indicates that those with a higher severity of psychiatric illness were more likely to cross yet another social norm by cannibalizing their own family member (8).

The association between obsessive compulsive disorder (OCD) and cannibalistic ideation is another comorbidity to consider. In one report detailing a case of Mesial Temporal Sclerosis and a 6-year history of seizures, a patient presented with a recent onset of psychosis and OCD with an urge to eat his son and wife. Notably, these thoughts were so ego-dystonic for that patient as he drank his own blood and attempted suicide to avoid harming his family and quell these ideations (15). Others living with OCD may experience obsessions with cannibalistic themes, including intrusive thoughts, urges, or a fear of becoming a cannibal through the ingestion of one's own body (autosarcophagy) or another's, either accidentally or intentionally. This often includes the fear of ingesting skin cells or bodily fluids, resulting in a compulsion to use skin barriers, avoiding eating around others, or putting flesh in or near their mouths (16).

Additionally, many researchers are interested in the childhood upbringing of homicidal cannibals. In one study comparing the background of cannibalistic serial killers and non-cannibalistic serial killers, it was found that the cannibal group had a statistically higher amount of brain abnormalities, medical complications at birth, low socioeconomic status in youth, and abandonment by a parental figure (17). This provides additional evidence that cannibalistic serial killers are likely a result of childhood influences and prior adverse life events rather than environmental factors at the time of homicide.

Lastly, comorbidity of cannibalism with paraphilias must be considered. Vorarephilia, or 'vore' as it is often called, is a paraphilia in which people are sexually aroused by thoughts of being eaten, eating another, and/or watching the activity for sexual release (18). While there are numerous online communities dedicated to fantasy

vorarephilia content (e.g., Gaia Online Exeon Vore Guild, www.mindlessconsumption.com, www.vore.net), there have been a handful of case reports related to sexual cannibalism and vorarephilia documented in the literature. In one case related to vorarephilia, a man referred to as "The Turkey Man," repeatedly hired a dominatrix to "cook him" within an oven facsimile comprised of a cardboard box and have her describe in detail cooking him and eating him, resulting in him orgasming without physical stimulation from just the fantasy alone (19). Another case detailed a man with the sexual fantasy of being cooked and eaten by the witch from the fairy tale of Hansel and Gretel, and a third case detailed a middle-aged man seeking psychiatric care for paraphilias, including the fantasy of being consumed, destroyed, and defected by a very large and dominating woman (19). However, the closest case to actually enact vorarephilia would likely be the case of Armin Meiwes, a man who killed and ate his male partner with his consent in 2002. A videorecording documented the entire event, including the consent of both individuals, the restraint of the victim, the severing and attempted consumption of the victim's penis, and the killing and eating of the victim's flesh. Notably, Meiwes reportedly began to seek out partners in his adolescence willing to role-play being consumed by him (20).

In our case report, we found evidence of ego-syntonic cannibalistic ideation without evidence of compulsions with a sadistic motive, a prior history of psychiatric diagnoses (Reactive Attachment Disorder, elevated depression scores, and Episodic Mood Disorder), low socioeconomic status in his

youth, and a history of homicidal ideation and physical violence towards a kin member. These traits are consistent with some features of other cannibals detailed in this discussion and provide evidence of these traits within a pediatric patient. To our knowledge, Mr. X did not actually engage in cannibalistic behavior. However, medico-legal issues surrounding duty to protect were raised. With cannibalism that has occurred, an issue for the filing of proper legal charges may be whether it occurred after death (necrocannibalism) or whether the victim was killed before ingestion (homicidal cannibalism) (5). This duty was believed to be appropriately discharged by hospitalization and subsequent resolution of cannibalistic thoughts.

4. Conclusion

Pediatric cannibalism involves a complex interplay between psychiatric and environmental factors as delineated in the case of Mr. X. Moreover, his case represents a unique combination of notable psychiatric history, ego-syntonic cannibalistic ideation, and fascination with criminals who had known cannibalistic behavior. While there were similarities between this case and other documented cases, Mr. X's lack of compulsion and sadistic motives were unique, emphasizing the idea that pathologic cannibalism develops differently from patient to patient. The culmination of our findings contributes to the limited understanding behind cannibalism, including the role that early adverse childhood experiences and psychiatric disorders play in developing such ideation. More research is necessary to better address the interrelated factors that bring about this rare psychiatric phenomenon, especially regarding pediatric cases.

Conflicts of Interest

The authors declare no conflicts of interest.

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A Mini Review: The Impact of Family-Centered Interventions and Peer Support in Improving Mental Health Outcomes for Suicidal Youth

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1. Introduction

Effective long-term treatment following acute crisis intervention is essential to reduce suicidal ideation and prevent future suicide attempts in at-risk youth. Since 2019, research has emphasized the critical role of family involvement during both an acute crisis and recovery phases, as well as the addition of peer support workers in mental health treatment. This review explores specific family-centered interventions and peer support strategies aimed at improving mental health outcomes among suicidal youth.

2. Discussion

strategy that can be used for adolescent patients to address interpersonal problems, communication skills, and problem-solving strategies related to depression. An ultra-brief version, IPT-A SCI, was tested as an emergency outpatient intervention for suicidal adolescents in acute crisis (1). The goal was to provide immediate support to reduce suicide risk and develop a plan for ongoing care. The intervention included five weekly sessions, with parental involvement in sessions 1 and 5 (1).
Session 1 focused on suicide risk assessment, depression evaluation, and creating a safety plan with coping strategies when suicide risk is increased (2). Session 5 reviewed treatment progress and the safety plan to prevent relapse. Patients and parents also received four follow-up emails over 12 weeks to reinforce the safety plan and interpersonal skills developed, as well as additional resources and emergency contact information. IPT-A SCI demonstrated feasibility, safety, and reduced suicidal ideation compared to a waitlist control. It also resulted in lower post-treatment dropout rates compared to standard care, which included inpatient, day treatment, or outpatient treatment combined with follow up therapy (1). This intervention equips parents with tools to manage future crises and connects families with community mental health resources, which can ultimately improve outcomes for suicidal youth.

Research has demonstrated that incorporating family care into outpatient treatment plans leads to improvement in patient depression and anxiety, family conflict, suicide risk, and help-seeking behaviors (3). Intensive outpatient therapy (IOP), designed for patients requiring more support than conventional outpatient care, offers an opportunity to implement family-centered interventions (3). One case report describes a 15-year-old female patient with major depressive disorder who, after discharge from inpatient services following a suicide attempt, received family-centered care throughout her IOP treatment. This approach included medication management, individual case management, measurement-based care, dialectical behavior therapy (DBT), and the *CHATogether* intervention.

Medication management facilitated ongoing communication between the family and psychiatrist regarding diagnosis, medication options, side effects, and treatment goals. Case management assisted the family with logistical challenges, such as coordinating with the patient's school, and provided education on coping strategies specific to their daughter's needs, as well as ways to create a safer home environment during crises. The parents also participated in weekly peer support groups, where they found connection and reassurance that they were not alone in supporting their child through a mental health crisis (3). Measurement-based care involved collecting data from validated questionnaires assessing social stress, locus of control, depression, hyperactivity, and emotional sensitivity, gathered from both patient and family throughout treatment. This information guided clinicians, the patient, and family in monitoring their daughter's progress through IOP, and allowed necessary adjustments to be made to the treatment plan. DBT modules focusing on mindfulness, emotional regulation, distress tolerance, and interpersonal effectiveness led to improved family communication and emotional co-regulation. The *CHATogether* intervention provided a safe space to address family-specific conflicts and helped the parents recognize how their own childhood trauma influenced their parenting and communication styles with their daughter. By the end of IOP, the family reported developing new coping skills and a more compassionate understanding of the patient's mental health challenges. They continued with individual and family therapy even after completing the program (3). Family-centered therapy supports the well-being of the patient and empowers caregivers with the skills and resources needed to provide effective support. Overall,

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family engagement is associated with better treatment adherence and higher attendance rates in mental health services and ensuring optimal outcomes (4,5).

In addition to family education and support, peer support can improve outcomes for youth with a history of suicidal ideation or attempts. Over one year, two peer support workers were integrated into the treatment plan of a chronically suicidal 17-year-old female patient. The patient reported that this intervention led to reduced loneliness and increased hope. Through regular meetings, the patient formed “normal” connections with people who treated her with genuine interest and respect, helping her feel valued and less isolated. The peer workers provided a safe space for her to openly discuss suicidality, the loss of peers to suicide, and struggles with family relationships. These were all topics she reported she did not feel comfortable addressing with other mental health staff. Connecting with peer support workers gave her hope, as they encouraged her to look ahead to the future and her goals, such as obtaining further education. Overall, the patient experienced a decrease in suicidal tendencies and a stronger sense of connectedness and community (6).

3. Conclusion

Family-centered care and peer support interventions can reduce suicidal ideation and improve mental health outcomes in youth with a history of suicide attempts or ideation. However, it is important to recognize that not all adolescents have access to supportive family members or peer support resources. Additionally, families may face limitations in time, finances, or availability that prevent full participation in intensive, family-centered care. These disparities highlight the need to continue developing and implementing effective, accessible psychiatric treatment strategies that support at-risk youth of all different socioeconomic backgrounds.

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Mental Health in Medical Trainees: What the Data Tells Us

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1. Introduction

It was my first day of clinical rotations, and I had not felt this kind of anxiety since starting medical school. For years, I had studied this world in books and lectures, but now I was caring for real people. As I entered the inpatient psychiatry unit, my mind raced. The fear of not knowing enough, the pressure to perform, and the uncertainty of what lay ahead were impossible to ignore.

Medical school is a test of intellect and emotion, often in equal measure. Global meta-analyses show that about one in three medical students (33.8%) struggles with anxiety, one in four (27.2%) screens positive for depression, and one in ten (11.1%) has contemplated suicide (1,2). These numbers are not abstract; they sit beside us in lectures and clinical rotations, quietly signaling a system under strain.

Later that morning, the attending physician in the psychiatric unit gathered us for a lecture on antidepressants. He began writing the names of SSRIs on the whiteboard: citalopram, escitalopram, sertraline, and so on. When he reached fluoxetine, he paused. “I’ve been taking fluoxetine since residency, and it’s helped me a lot,” he shared. “I try to normalize mental struggles.”

His honesty broke from the usual script of clinical instruction. It highlighted how rarely we witness such vulnerability in medicine, despite anxiety and depression among medical students occurring at rates 2 to 3 times higher than their non-medical peers (1, 2). This pattern often intensifies over time, with depressive symptoms increasing by 14% during training (2).

2. Discussion

Despite being trained to recognize and treat anxiety and depression in others, a striking disconnect exists when addressing our own mental health. Only one in six medical students (15.7%) who screen positive for depression seeks psychiatric care (2). Reluctance to seek help is often driven by fears of peer judgment, concerns about treatment appearing in official records, and worries about the impact on residency and career prospects (2-4). Unaddressed mental health concerns have been linked to poor academic performance, academic dishonesty, and increased alcohol and substance use (5). For practicing physicians, depressive symptoms are associated with a higher incidence of medical errors (6).

Stigma remains one of the most persistent barriers to mental health care in medicine, fueled by interpersonal attitudes and what educators call the hidden curriculum (3, 4, 7-9). These unspoken lessons and cultural norms within the hidden curriculum often discourage vulnerability, making trainees feel that seeking support could threaten their professional reputation (3, 4, 7, 8). Mentors may unintentionally reinforce this by rarely discussing their own mental health experiences and by modeling stoicism (7). As a result, many students and residents learn to conceal their struggles, perpetuating a cycle of silence and avoidance of support (7-9). However, research shows that when faculty are transparent about their own experiences with mental health care, it reduces stigma, encourages trainees to reach out, and fosters a more supportive learning environment (8, 9).

Beyond the hidden curriculum and interpersonal stigma, structural barriers at the policy level have further discouraged physicians from utilizing mental health services. Historically, many state medical licensure applications required physicians to disclose any history of mental health treatment, regardless of whether it impacted their ability to practice (3, 4, 7-9). A national survey of 5,829 U.S. physicians found that nearly 40% were reluctant to seek mental health care due to concerns that disclosure could negatively affect their license (3). Recently, growing recognition of these consequences has prompted meaningful reform. As of May 2025, thirty-seven state medical boards have revised their licensing applications, removed intrusive mental health questions unless directly related to current impairment (10). This aligns with recommendations from the American Medical Association and the Federation of State Medical Boards, which urge that only current impairment, not diagnosis or treatment history, should be relevant (3, 10). While further changes are needed, this is a crucial step toward giving physicians the privacy and compassion they offer to others.

3. Conclusions

The principle of “see one, do one, teach one” has long shaped how clinical skills are passed from physician to trainee. But when it comes to mental health, this tradition breaks down. If mentors do not model openness or vulnerability, students are left without a template for these behaviors and may instead learn to remain silent. That is why

my attendees' brief comment about fluoxetine mattered so much. That authenticity created space for something deeper: the possibility of a profession where honesty about mental health is not whispered but welcomed. Perhaps meaningful change begins not just in what we are taught, but in what we are willing to share. Every small act of honesty lays the groundwork for openness – shaping what future physicians see, do, and one day, teach.

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Closing Editorial

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This issue of Translation highlights novel interventions for psychiatric conditions, structural challenges in psychiatric treatment, and evolving trends in the pediatric population affecting treatment. These pieces are relevant – the recommendations for initiation of metformin with atypical antipsychotics come at a time when more children and adolescents are being prescribed atypical antipsychotics off-label than ever before. Identifying ways to mitigate treatment side effects and prevent long-term morbidity is of utmost importance, especially with the pediatric population, given the downstream effects of the early onset of obesity and metabolic syndrome. Pediatricians and child psychiatrists alike additionally face new issues in adolescent care, from targeting different formulations of nicotine dependence, to recognizing the impact of artificial intelligence on teens' self-worth and mean-making, during the time of their identity development. Lyons and Edgington, as well as Nelson et al., place these issues in context for us. The burden of addressing these issues is magnified for pediatricians, as described by Roehrs et al., given the lack of training pediatricians receive during residency in pediatric psychiatry, and the rise of psychiatric needs in the pediatric population.

It is interesting that despite the rising psychiatric needs, the ACGME does not mandate child psychiatry rotation during pediatrics residency. Perhaps this is something that we will see in the coming years. In the meantime, supporting pediatrics colleagues through collaborative care efforts and the like may help to mitigate some of these structural challenges.

Systemic and structural challenges are present in many other aspects of providing psychiatric care. Roth et al. and Davuluri et al. showcase this in their pieces on the challenges of medication access for people experiencing homelessness and the impact of wait times on psychiatric outcomes. Among medical specialties, psychiatry continues to have some of the longest wait times for outpatient referrals, due to the shortage of psychiatrists relative to the demand.

It is now well-established that prolonged untreated bipolar disorder, schizophrenia, or repeated episodes of alcohol and/or sedative-hypnotic withdrawal are harmful to the brain and reduce the success of treatment.

Psychiatric research continues to shed light on new ways of understanding the brain. The role of the gut-brain axis in depression, anxiety, and Parkinson's, the mechanisms through which both the peripartum period and preeclampsia increase risk of psychosis– these advances continue to shape our understanding of the brain as an organ

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not only responsible for dictating the body's actions, but also sensitive to the fluctuating stressors throughout the other organ systems. Coming into the new year, may this issue be a reminder to you to be intentional in your approach to patients, deliberately seek out new literature, and consider the whole person living with the condition. From the person experiencing homelessness, to the peripartum woman with psychosis, to the teen being treated with atypical antipsychotics, to the older adult with Parkinson's, be steadfast in your commitment to providing evidence-based care.

Thank you for reading, and peace and joy to you and yours.

Sincerely,
Emily Cooper, MD