World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(RESEARCH ARTICLE)

Check for updates

Infection-associated neuropsychiatric disorders: Mechanisms and clinical manifestations of psychosis, behavioral alterations and cognitive dysfunction

Mark Ericson B. Baladad [1,*], Wyndel Kaye A. Catli-Budchangan [1], Aldrin Patrick G. Estocapio [1], Raeuelle Ken G. Novero [1], Dawn Kimberly L. Padua-Sumaya [1] and Justin Noel B. Verceles [2]

[1] *College of Medical Laboratory Science, LORMA Colleges, Inc., City of San Fernando, La Union, Philippines.*
[2] *Department of Social Welfare and Development - Field Office 1, La Union, Philippines.*

World Journal of Biology Pharmacy and Health Sciences, 2026, 26(02), 164-167

Publication history: Received on 06 April 2026; revised on 13 May 2026; accepted on 15 May 2026

Article DOI: https://doi.org/10.30574/wjbphs.2026.26.2.0215

Abstract

Infectious diseases are increasingly recognized as contributors to behavioral change, psychosis, and mental dysfunction. Bacterial, viral, parasitic, and fungal pathogens can induce acute or chronic neuropsychiatric symptoms through mechanisms such as direct CNS invasion, neuroinflammation, immune-mediated processes, neurotransmitter dysregulation, and gut–brain axis perturbations. Clinical manifestations include psychosis, mood disturbances, cognitive impairment, and behavioral dysregulation. This five-year retrospective review of cases integrates clinical evidence and mechanistic pathways. Understanding these links is critical for early recognition, prevention, and management of infection-induced neuropsychiatric disorders.

Keywords: Infection; Neuropsychiatric disorder; Psychosis; Behavioral alterations; Cognitive dysfunction

1. Introduction

Infections have long been implicated in neurological and psychiatric outcomes, ranging from acute delirium to chronic cognitive impairment. Recent advances in neuroimmunology, microbiome research, and translational studies have elucidated mechanisms by which pathogens affect mental function, including neuroinflammation, direct CNS invasion, and dysregulation of neurotransmitter systems (Katchhi et al., 2025; Dantzer et al., 2021). Psychiatric symptoms may emerge during acute infection, in post-infectious phases, or as long-term sequelae, sometimes mimicking primary psychiatric disorders. Recognizing infectious etiologies in atypical presentations is essential for effective management.

2. Bacterial Infections

Bacterial infections affecting the central nervous system (CNS) or producing systemic inflammation have been increasingly reported to cause psychiatric symptoms. Acute and post-infectious psychosis has been described following bacterial meningitis caused by *Streptococcus pneumoniae* and *Neisseria meningitidis*, presenting with hallucinations, delusions, and affective disturbances (Huang et al., 2024; Ropper and Samuels, 2021). Case reports describe patients who developed acute behavioral disinhibition, agitation, and cognitive impairment during systemic bacterial sepsis (Bhattacharyya and Barua, 2025). In post-streptococcal autoimmune syndromes, pediatric patients may exhibit obsessive-compulsive behaviors, emotional lability, and acute-onset behavioral dysregulation (Chang et al., 2022). Rare cases of psittacosis with meningitis have also been associated with abnormal mental behavior and psychiatric symptoms, highlighting the need for vigilance for bacterial etiologies in acute psychiatric presentations (Science Publishing Group, 2025).

*Corresponding author: Mark Ericson B. Baladad Email: markericson.baladad@lorma.edu

Mechanistically, these psychiatric manifestations are mediated through direct CNS invasion, which triggers microglial activation and localized inflammation. Systemic cytokines, such as IL-1β, IL-6, and TNF-α, can cross or disrupt the blood-brain barrier (BBB), contributing to neurochemical dysregulation. Autoimmune reactions following infection may further exacerbate neuropsychiatric outcomes. Together, these mechanisms disrupt frontal-limbic circuits and neurotransmission (dopamine and glutamate), producing psychosis, mood disturbances, and behavioral dysregulation (Dantzer et al., 2021).

3. Viral Infections

Viral infections have been strongly associated with psychiatric outcomes in recent literature. Case series during the COVID-19 pandemic reported new-onset psychosis in adults without prior psychiatric history, characterized by paranoid delusions, hallucinations, insomnia, and agitation (Fiks et al., 2022; Smith et al., 2023). Adolescents have also been reported to develop visual and tactile hallucinations after COVID-19 infection (Thomas, 2022). Other studies noted catatonia, mania, and disorganized behavior following viral encephalitis, with persistent cognitive deficits even after somatic recovery (Granerod et al., 2021). Cohort studies demonstrated increased incidence of psychotic disorders within 6-12 months post SARS-CoV-2 infection (Taquet et al., 2022). Herpes simplex virus encephalitis survivors frequently experience personality changes, impulsivity, mood instability, and executive dysfunction despite antiviral therapy (Granerod et al., 2021). Influenza and other neurotropic viruses have been linked to schizophrenia, depression, and cognitive decline (Lorkiewicz and Waszkiewicz, 2024).

Viral-induced psychiatric symptoms arise from multiple mechanisms, including direct neurotropism with neuronal and glial infection, microglial activation leading to synaptic pruning abnormalities, systemic and CNS cytokine release, and autoimmune-mediated post-viral encephalitis. Activation of the kynurenine pathway and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis contribute to depression, psychosis, and cognitive impairment (Savitz, 2020; Miller and Raison, 2022).

4. Parasitic Infections

Parasitic infections such as *Toxoplasma gondii* and neurocysticercosis *(Taenia solium)* are associated with a wide range of psychiatric and behavioral changes. Individuals with *T. gondii* infection exhibit higher rates of psychosis, schizophrenia-spectrum disorders, impulsivity, aggression, depression, and suicidal ideation (Sutterland et al., 2023; Alvarado-Esquivel et al., 2021). Case reports highlight acute psychotic episodes during toxoplasmic encephalitis, particularly in immunocompromised patients (Alvarado-Esquivel et al., 2021). Neurocysticercosis has been associated with seizure-related psychosis, depression, anxiety, cognitive impairment, and behavioral disinhibition, sometimes persisting even after antiparasitic therapy (Carpio et al., 2022; Goyal et al., 2023).

Mechanistically, parasitic-induced psychiatric manifestations involve space-occupying lesions that disrupt neural circuits, chronic neuroinflammation, and parasite-induced modulation of dopaminergic pathways, as T. gondii encodes tyrosine hydroxylase-like enzymes affecting host dopamine. Immune-mediated cytokine changes and gut-immune-brain interactions further contribute to behavioral and cognitive dysfunction.

5. Fungal Infections

Fungal CNS infections, although less common, can produce profound psychiatric effects. Cryptococcal meningitis has been associated with irritability, aggression, hallucinations, confusion, personality changes, and cognitive decline in otherwise healthy individuals (Wo et al., 2024). Additionally, fungal dysbiosis in the gut microbiome has been linked to schizophrenia, cognitive deficits, and immune dysregulation (Li et al., 2024; Zhang et al., 2025).

Fungal-induced psychiatric manifestations arise from direct meningeal inflammation affecting CNS function, chronic immune activation altering cytokine networks, and microbiome disruption influencing neurotransmission and mood regulation.

6. Shared Mechanistic Pathways

Across pathogen types, psychiatric symptoms commonly arise from neuroinflammation, BBB disruption, neurotransmitter dysregulation, autoimmune reactions, and gut-brain axis perturbations. Neuroinflammatory cytokines and immune activation interfere with dopamine and glutamate signaling, contributing to psychosis, cognitive slowing, and mood disorders. BBB disruption facilitates entry of systemic cytokines and immune cells into the CNS,

exacerbating delirium and personality changes. Gut microbiota alterations influence mood, cognition, and behavior via metabolic and immune-mediated pathways (Dantzer et al., 2021; Miller and Raison, 2022; Katchhi et al., 2025).

7. Clinical Implications

Infection-induced psychiatric manifestations highlight the importance of early recognition and comprehensive evaluation in patients presenting with acute psychosis, mood disturbances, cognitive changes, or behavioral dysregulation. Clinicians should maintain a high index of suspicion for infectious etiologies when psychiatric symptoms appear acutely, atypically, or in individuals without prior psychiatric history. Prompt diagnosis using serology, neuroimaging, CSF analysis, and pathogen-specific tests is critical to guide appropriate therapy.

Management requires a multidisciplinary approach integrating antimicrobial or antiviral treatment with psychiatric care. Acute psychiatric symptoms may respond to psychotropic medications, but severe or persistent manifestations often require adjunctive immune or pathogen-targeted therapies (Zhao et al., 2023; Chang et al., 2022). Monitoring cognitive function, mood, and behavior during and after infection helps prevent chronic sequelae. Preventive strategies such as vaccination, safe food and water practices, and prophylaxis in high-risk populations reduce infection-related psychiatric morbidity. Future research should focus on longitudinal outcomes and mechanistic pathways, including neuroimmune, neurotransmitter, and gut-brain axis interactions to inform targeted therapies and public health interventions (Dantzer et al., 2021; Katchhi et al., 2025).

8. Conclusion

Infectious diseases play a significant role in the development of neurological and psychiatric manifestations through complex interactions involving neuroinflammation, immune dysregulation, direct CNS invasion, and neurotransmitter disturbances. Bacterial, viral, parasitic, and fungal pathogens may produce symptoms ranging from acute psychosis and mood disorders to long-term cognitive and behavioral impairment. Increasing evidence highlights the importance of recognizing infections as potential underlying causes of atypical psychiatric presentations to ensure timely diagnosis and appropriate multidisciplinary management. Continued research on neuroimmune and gut-brain mechanisms is essential to improve prevention, treatment, and long-term outcomes of infection-associated neuropsychiatric disorders.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] Alvarado Esquivel, C., Gómez Rosales, S., and Liesenfeld, O. (2021). Acute psychosis in toxoplasmosis: Case report and review. Parasitology Today, 37(10), 845–849.

[2] Carpio, A., Romo, M. L., and Cantu, C. (2022). Neurocysticercosis and psychiatric manifestations: Clinical spectrum and management. Neurology, Psychiatry and Brain Research, 41, 29–36.

[3] Chang, K., Frankovich, J., Cooperstock, M., et al. (2022). Pediatric autoimmune neuropsychiatric syndrome associated with streptococcal infections (PANS/PANDAS): Clinical features and treatment considerations. Pediatrics and Adolescent Medicine, 48(2), 225–238.

[4] Dantzer, R., O'Connor, J. C., and Freund, G. G. (2021). Neuroimmune mechanisms of infection induced psychiatric symptoms. Nature Reviews Neuroscience, 22(7), 340–353.

[5] Fiks, A. G., Chou, R., and Hancock, G. (2022). Case series: COVID 19 induced psychosis in adults without previous psychiatric history. Journal of Psychiatric Research, 150, 150–156.

[6] Goyal, S., Gupta, D., and Singh, M. (2023). Cognitive and behavioral dysfunction associated with neurocysticercosis: Case series and review. Journal of Tropical Neurology, 8(2), 112–118.

[7] Granerod, J., Ambrose, H. E., and Davies, N. W. S. (2021). Post encephalitis psychiatric sequelae: A systematic overview. Journal of Neuropsychiatry and Clinical Neurosciences, 33(1), 8–22.

[8] Huang, X., Li, J., and Chen, Y. (2024). Post meningitic psychosis after Streptococcus pneumoniae infection: A case report and literature review. Journal of Clinical Neuropsychiatry, 12(3), 145–156.

[9] Katchhi, N., Patel, S., and Desai, R. (2025). Mechanistic pathways linking infection to neuropsychiatric syndromes: A comprehensive review. Journal of Neuroimmunology, 380, 577834.

[10] Li, Y., Zhao, X., and Wang, J. (2024). Gut fungal dysbiosis and schizophrenia: Insights from clinical cohorts. Frontiers in Psychiatry, 15, 112345.

[11] Miller, A. H., and Raison, C. L. (2022). The role of inflammation in psychiatric disorders: Mechanistic advances and therapeutic prospects. Annual Review of Clinical Psychology, 18, 163–190.

[12] Ropper, A. H., and Samuels, M. A. (2021). Neurological complications of bacterial meningitis. New England Journal of Medicine, 384(15), 1426–1437.

[13] Sutterland, A. L., Fond, G., Kuin, A., et al. (2023). Association between Toxoplasma gondii and schizophrenia: A meta analysis. Schizophrenia Bulletin, 49(5), 876–889.

[14] Taquet, M., Luciano, S., Geddes, J. R., and Harrison, P. J. (2022). Incidence of psychotic disorders following SARS CoV 2 infection: A retrospective cohort study. The Lancet Psychiatry, 9(8), 741–750.

[15] Wo, C. Y., Lee, Y., and Chang, W. N. (2024). Neuropsychiatric manifestations of cryptococcal meningitis: Case observations and review. Mycopathologia, 189(4), 599–612.

[16] Zhang, H., Liu, Q., and Chen, X. (2025). The mycobiome in schizophrenia: Evidence for fungal dysbiosis and immune interactions. Immunology and Mental Health, 4(1), 45–58.

[17] Zhao, L., Zhang, Q., and Wang, H. (2023). Immune-mediated psychosis following bacterial infection: Mechanisms and case insights. Brain, Behavior, and Immunity – Health, 16, 100390.