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Review article

PSYCHOTIC MANIFESTATIONS IN PRIMARY SJÖGREN'S SYNDROME: CLINICAL CHARACTERISTICS AND PATHOPHYSIOLOGICAL INSIGHTS

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Abstract: Psychotic manifestations in primary Sjögren's syndrome (SS) represent a rare but clinically meaningful complication of the disease. The aim of this paper was to analyze the clinical characteristics, potential pathophysiological mechanisms, therapeutic approaches, and outcomes of psychotic symptoms in patients with SS.

A narrative review of the available literature was conducted, with particular focus on case reports and small case series describing psychosis in the context of SS.

Psychotic manifestations in SS may arise from central nervous system involvement, immune-inflammatory mechanisms, cytokine dysregulation, and neurotransmitter alterations, as well as from corticosteroid therapy. Pro-inflammatory cytokines, particularly interleukin-6, have been linked to dopaminergic dysfunction and the emergence of positive psychotic symptoms. Corticosteroids may induce psychotic episodes depending on dose and duration of treatment. Chronic somatic symptoms, including ocular and oral dryness, pain, and fatigue, may be vulnerable to delusional misinterpretation. In several reported cases, psychotic symptoms improved following immunosuppressive therapy, supporting a potential immune-mediated subtype of psychosis in this population.

Psychosis in primary Sjögren's syndrome likely has a multifactorial etiology. Early recognition of a possible autoimmune basis is essential, as treatment strategies may require immunomodulatory therapy in addition to standard psychiatric management.

Keywords: Sjögren's syndrome, psychosis, neuroinflammation, cytokines, corticosteroids, autoimmune diseases.

INTRODUCTION

Primary Sjögren's syndrome (SS) is a chronic systemic autoimmune disease primarily characterized by sicca symptoms, including xerostomia and xerophthalmia. In addition to glandular involvement, up to half of patients develop extraglandular manifestations, with neuropsychiatric symptoms being relatively common. While mood disorders and cognitive impairment predominate, psychotic symptoms—such as hallucinations, delusions, and disorganized thinking—are rare but clinically significant. These manifestations may result from central nervous system involvement, immune-mediated mechanisms, chronic pain, or corticosteroid therapy. Despite their clinical relevance, psychotic symptoms in SS remain poorly investigated, with existing evidence largely limited to case reports and small case series.

The aim of this narrative review is to analyze psychotic symptoms in patients with Sjögren's syndrome, with a focus on clinical characteristics, potential triggers, therapeutic approaches, and outcomes, while proposing a conceptual framework linking the pathophysiology of SS to psychotic manifestations.

PSYCHOTIC SYMPTOMS IN SJÖGREN'S SYNDROME

Sjögren's syndrome, also referred to as "autoimmune epithelitis", is a chronic, slowly progressive autoimmune disease characterized by immune-mediated dysfunction and destruction of the salivary and lacrimal glands, accompanied by a broad range of systemic manifestations (1, 2). It represents the second most common autoimmune rheumatic disease after rheumatoid arthritis and predominantly affects women (1, 3). Owing to its potential involvement of the peripheral, central, and autonomic nervous systems, Sjögren's syndrome may be associated with various neuropsychiatric manifestations (1, 4). Among these, depressive and anxiety disorders are most frequently reported, whereas psychotic disorders are less common but

remain clinically relevant (4). Although considered rare, psychotic symptoms have been consistently documented in the available literature. According to the literature, the prevalence can range from 2.5 to 60% due to the non-specific presentation of symptoms and different diagnostic criteria (5, 6). On the other hand psychosis is a clinical syndrome characterized by impaired perception of reality, leading to disturbances in cognition, behavior, and daily functioning. Core manifestations include delusions and hallucinations (7, 8).

This paper addresses the question of where psychotic symptoms in Sjögren's syndrome originate. The discussion focuses on the possible mechanisms involved, including central nervous system involvement, immune processes, and treatment-related factors, particularly corticosteroid therapy. We also examine how Sjögren's syndrome may affect patients with pre-existing psychotic disorders by triggering symptom relapse or reducing treatment response. In addition, the paper considers the role of genetic and environmental vulnerability, as well as the occurrence of psychotic symptoms in patients without an identifiable predisposition. The overall aim of this work is to improve understanding of the relationship between Sjögren's syndrome and psychotic manifestations and to highlight their clinical relevance.

CNS MECHANISMS

Sjögren's syndrome can affect many parts of the central nervous system, including the brain, brainstem, spinal cord, optic nerves, and cerebellum (8).Neuropsychiatric manifestations are increasingly recognized as part of systemic autoimmune diseases, reflecting the complex interaction between immune dysregulation and central nervous system function (9). Patients may experience a range of neurological problems such as headaches, seizures, cognitive difficulties, movement disorders, and sensory deficits. Cognitive impairments often involve attention, memory, and executive functions, while psychiatric symptoms can include depression, psychosis, and catatonia. These manifestations are thought to result from immune-related dysfunction in the brain, which may disrupt normal thinking, perception, and emotional regulation. CNS involvement can therefore contribute directly to the emergence of psychotic symptoms, highlighting the need for early recognition and management (10, 11).

One potential pathway through which Sjögren's syndrome may influence psychotic symptoms is impairment in emotional processing and regulation. Several studies have investigated emotional interpretation and expression in patients with Sjögren's syndrome, a domain that is also critically impaired in psychotic disorders, often in the context of alexithymia (12, 13). It is important to remember that this chronic disease is frequently accompanied by reactive emotional symptoms, such as emotional lability, masked depression, and withdrawal, which can obscure the presence of true alexithymia. Patients often appear detached, sometimes anhedonic, and emotionally blunted. Beyond these general reactions, there is evidence of a specific difficulty in understanding and decoding both one's own and others' emotions. This impairment may reflect an organic substrate and can interact with cognitive dysfunction (6). All of this creates conditions that resemble those seen in psychosis, where patients similarly struggle with emotion recognition and regulation. Such deficits may therefore contribute to the development or worsening of psychotic symptoms in susceptible individuals (14, 15).

IMMUNE-INFLAMMATORY PATHWAYS

Sjögren's syndrome is increasingly recognized as a condition capable of inducing immune-mediated neuroinflammation that extends beyond the classic sicca manifestations and involves the central nervous system. Pro-inflammatory cytokines, including interferon-γ (IFN-γ), interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α), have been implicated in Sjögren-related CNS involvement. These mediators can activate metabolic pathways such as indoleamine-2,3-dioxygenase, leading to alterations in kynurenine metabolism and subsequent disruption of serotonergic and glutamatergic neurotransmission in the hippocampus and other brain regions (16, 17).

Importantly, similar inflammatory mechanisms have been described in psychotic disorders. Elevated levels of pro-inflammatory cytokines have been reported in patients with first-episode psychosis and schizophrenia and have been associated with cognitive deficits and impaired social functioning (18). In particular, IL-6 has beenshown to increase dopaminergic activity in striatal regions, which is closely associated with positive psychotic symptoms. Conversely, dopamine signaling can influence inflammatory pathways, indicating a reciprocal relationship between immune activation and dopaminergic neurotransmission (19).

STEROID-INDUCED PSYCHOSIS

Corticosteroids are widely prescribed due to their potent anti-inflammatory and immunosuppressive effects and are commonly used in the management of various conditions, including Sjögren's syndrome. However, their use is associated with a range of psychiatric adverse effects collectively known as corticos-

teroid-induced psychiatric disorders (CIPDs) (20, 21). These manifestations may include mood disturbances, sleep disorders, irritability, agitation, manic symptoms, and psychosis. Psychotic features can present as hallucinations, delusional thinking, disorganized thought processes, and affective instability (22, 23). Such symptoms may emerge soon after the initiation of therapy, at any stage during treatment, or even following the discontinuation of corticosteroids (24). Evidence suggests a clear dose-response relationship, with higher doses conferring greater risk. Data from the Boston Collaborative Drug Surveillance Program demonstrated psychiatric adverse effects in 18.6% of patients receiving more than 80 mg/day of prednisone or methylprednisolone, compared with 4.6% in those receiving 41–80 mg/day and only 1.3% in patients treated with doses below 40 mg/day (25). In the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), these conditions are categorized under substance- or medication-induced psychotic disorders, occurring in approximately 5–18% of patients receiving corticosteroid therapy (26, 27).

Prolonged use of corticosteroids affects the hypothalamic–pituitary–adrenal (HPA) axis, leading to changes in the body's response to stress and in normal feedback regulation. These disturbances, together with imbalances in neurotransmitter systems, can result in various psychiatric symptoms. Corticosteroids influence dopamine pathways, which may increase the risk of developing psychotic symptoms. In addition, long-term corticosteroid exposure has been linked to structural changes in the brain, such as hippocampal atrophy associated with memory problems and depression, as well as increased activity of the amygdala, which contributes to anxiety and emotional instability. These mechanisms help explain the wide range of psychiatric effects seen with corticosteroid use (28).

SOMATIC SYMPTOM MISINTERPRETATION

The next important question we wanted to address is the relationship between Sjögren's syndrome and psychosis. More specifically, we aimed to explore how the key symptoms of Sjögren's syndrome may affect patients with psychotic disorders, and in what ways these symptoms can worsen existing psychosis or lead to the development of new psychotic symptoms. We were particularly interested in identifying which symptoms are more likely to exacerbate psychosis and how they might be experienced or interpreted by patients who are already psychotic.

Primary Sjögren's syndrome is defined by chronic ocular and oral dryness resulting from autoimmune damage to lacrimal and salivary glands, with sicca symptoms present in the majority of patients. These persistent somatic symptoms have been associated with a range of psychiatric manifestations, including psychosis, and case reports describe patients with psychotic features occurring alongside or following prominent dryness symptoms (29). These symptoms provide persistent, ambiguous bodily sensations that are vulnerable to delusional misinterpretation in patients with impaired reality testing. Severe dry eye disease is associated with burning, foreign-body sensation, blurred or fluctuating vision, and visual discomfort (30, 31). In psychotic individuals, this may be experienced as externally caused or threatening, thereby becoming incorporated into persecutory or somatic delusions. Similarly, xerostomia and the oral discomfort seen in Sjögren's syndrome often lead to a range of oral symptoms, including speech difficulties, trouble swallowing (dysphagia), changes in taste perception, and a burning sensation in the mouth (32). Psychotic patients often report disturbances in the way they perceive and interpret bodily sensations, including unusual bodily experiences and alterations in body representation (33). Case reports and clinical observations indicate that these individuals may misinterpret physical discomfort or other bodily sensations, and these misinterpretations can sometimes intensify psychotic symptoms when patients are unable to accurately contextualize or articulate their experiences (34). These sensations may be distorted and incorporated into somatic delusions, depersonalization, and cenesthetic or ksenopathic phenomena, where patients experience abnormal bodily perceptions or feel as if external forces are acting upon their bodies. Several case reports of primary Sjögren's syndrome have documented severe psychiatric features, including persecutory and referential delusions, paranoia, anxiety, auditory hallucinations, and even suicidal ideation (35, 36, 37). Catatonic phenomena such as mutism, negativism, immobility, and Gegenhalten have also been reported in association with neuropsychiatric involvement in Sjögren's syndrome (36, 37). In some of these cases, the psychotic symptoms appeared to be closely intertwined with the underlying autoimmune process and bodily distress, and improved with immunosuppressive therapy rather than with antipsychotics alone. Besides the typical dry eyes and mouth, many patients with Sjögren's syndrome experience persistent fatigue and widespread pain. These ongoing physical symptoms can make individuals more sensitive to perceived threats and may shape the way they interpret their bodily discomfort, which can in turn affect their psychological well-being (37).

THERAPEUTIC IMPLICATIONS

Antipsychotics remain the cornerstone of treatment for psychotic symptoms in primary psychiatric disorders (38, 39). Despite their well-recognized adverse effect profile, they occupy a central position in the therapeutic hierarchy for psychosis and are among the most frequently prescribed classes of psychotropic medications overall (40, 41).

In contrast, the management of psychotic symptoms occurring secondary to Sjögren's syndrome appears to differ substantially. Based on case reports published over the past decade, treatment strategies in this context often extend beyond conventional antipsychotic therapy (35, 36, 37). Given that Sjögren's syndrome is primarily an immune-mediated systemic disease, immunosuppressive and immunomodulatory treatments are frequently employed and, in many cases, lead to significant improvement of psychiatric symptoms. This therapeutic response mirrors observations in other immune-mediated neurological conditions, where similar treatment strategies have proven effective (42). The aforementioned supports the hypothesis that psychosis associated with Sjögren's syndrome may be driven by underlying immune and inflammatory mechanisms rather than by primary psychiatric pathology.

CONCLUSION

Psychotic symptoms in primary Sjögren's syndrome constitute a rare but clinically meaningful manifestation of the disease. Evidence from published case reports and small case series suggests that psychosis in this context may arise from multiple, potentially interacting mechanisms, including immune-mediated neuroinflammation, cytokine dysregulation, central nervous system involvement, corticosteroid exposure, and the psychological and perceptual impact of chronic somatic symptoms.

The observation that psychotic symptoms in several reported cases improved with immunosuppressive or immunomodulatory therapy—sometimes more robustly than with antipsychotic treatment alone—strengthens the hypothesis of an immune-driven subtype of psychosis in this population.

Clinicians should maintain a high index of suspicion for autoimmune etiologies when encountering atypical, treatment-resistant, or late-onset psychosis, particularly in patients presenting with sicca symptoms, systemic autoimmune features, or neurological signs. Early identification is critical, as treatment strategies may differ substantially from those used in primary psychiatric disorders and may require targeted immunotherapy.

Future prospective studies are needed to determine the true prevalence of psychotic manifestations in Sjögren's syndrome, to better characterize their pathophysiology, and to establish evidence-based treatment algorithms. Improved recognition of this association may enhance both psychiatric and rheumatologic outcomes and contribute to a more integrated understanding of immune–brain interactions in psychosis.

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Note: Artificial intelligence was not utilized as a tool in this study.

Sažetak

PSIHOTIČNE MANIFESTACIJE U PRIMARNOM SJOGRENOVOM SINDROMU: KLINIČKE KARAKTERISTIKE I PATOFIZIOLOŠKI MEHANIZMI

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Psihotične manifestacije u primarnom Sjögrenovom sindromu (SS) predstavljaju retku, ali klinički značajnu komplikaciju bolesti. Cilj rada je analiza kliničkih karakteristika, mogućih patofizioloških mehanizama, terapijskih pristupa i ishoda psihotičnih simptoma kod bolesnika sa SS.

Sproveden je narativni pregled dostupne literature sa posebnim osvrtom na prikaze slučajeva i manje serije bolesnika koje opisuju pojavu psihoze u kontekstu SS.

Psihotične manifestacije u SS mogu se javiti kao posledica zahvatanja centralnog nervnog sistema, imuno-inflamatornih mehanizama, disfunkcije citokina i poremećaja neurotransmisije, kao i usled terapije kortikosteroidima. Proinflamatorni citokini, naročito interleukin-6, povezani su sa dopaminergičkom disfunkcijom i pojavom pozitivnih psihotičnih simptoma. Kortikosteroidi mogu indukovati psihotične epizode u zavisnosti od doze i trajanja primene. Hronični somatski simptomi, poput suvoće očiju i usta, bola i umora, mogu biti podložni pogrešnoj interpretaciji i inkorporaciji u sumanute ideje. U pojedinim slučajevima zabeleženo je poboljšanje psihotičnih simptoma nakon imunosupresivne terapije, što ukazuje na moguću imunološku osnovu poremećaja.

Psihoza u primarnom Sjögrenovom sindromu najverovatnije ima multifaktorsku etiologiju. Pravovremeno prepoznavanje autoimmune osnove psihotičnih simptoma od ključnog je značaja, jer terapijski pristup može zahtevati primenu imunomodulatorne terapije pored standardnog psihijatrijskog lečenja.

Ključne reči: Sjögrenov sindrom, psihoza, neuroinflamacija, citokini, kortikosteroidi, autoimmune bolesti.

REFERENCES

1. André F, Böckle BC. Sjögren›s syndrome. J Dtsch Dermatol Ges. 2022; 20(7): 980-1002. doi: 10.1111/ddg.14823.

2. Bombardieri M, Argyropoulou OD, Ferro F, Coleby R, Pontarini E, Governato G, et al. One year in review 2020: pathogenesis of primary Sjögren's syndrome. Clin Exp Rheumatol. 2020; 38Suppl 126:3-15.

3. Wong JK, Nortley R, Andrews T, D'Cruz D. Psychiatric manifestations of primary Sjögren's syndrome: a case report and literature review. BMJ Case Rep. 2014; 2014: bcr2012008038. doi: 10.1136/bcr-2012-008038.

4. El-Ouakhoumi A, Joulal H, Yousfi J, Benjilali L, Zahlane M, Essaadouni L. Refractory depressive disorder as the first manifestation of adolescent Sjögren's syndrome, successfully treated with rituximab. Eur J Case Rep Intern Med. 2024; 11(7): 004621. doi: 10.12890/2024_004621.

5. Wang N, Jin Y, Lu S, Li G, Li Y, He J. Central nervous symptoms as the prominent manifestation of Sjögren's disease: a case report. BMC Neurol. 2026; 26(1): 97. doi: 10.1186/s12883-026-04630-0.

6. Morreale M, Marchione P, Giacomini P, Pontecorvo S, Marianetti M, Vento C, et al. Neurological involvement in primary Sjögren syndrome: a focus on central nervous system. PLoS One. 2014; 9(1): e84605. doi: 10.1371/journal.pone.0084605.

7. Kilciksiz CM, Keefe R, Benoit J, Öngür D, Torous J. Verbal memory measurement towards digital perspectives in first-episode psychosis: a review. Schizophr Res Cogn. 2020; 21: 100177. doi: 10.1016/j.scog.2020.100177.

8. Perzyńska-Mazan J, Maślińska M, Gasik R. Neurological manifestations of primary Sjögren's syndrome. Reumatologia. 2018; 56(2): 99-105. doi: 10.5114/reum.2018.75521.

9. Skorić J, Pavković B. Infectious mononucleosis and autoimmune hepatitis. Sanamed. 2021; 16(1): 91-4. doi: 10.24125/sanamed.v16i1.502.

10. Barsottini OGP, Moraes MPM, Fraiman PHA, Marussi VHR, Souza AWS, Braga Neto P, et al. Sjogren's syndrome: a neurological perspective. ArqNeuropsiquiatr. 2023; 81(12): 1077-83. doi: 10.1055/s-0043-1777105.

11. Peschard-Franco M, Piña-Rosales E, García-Santos A. A 56-year-old woman with initial severe electrolyte imbalance who developed altered mental status, psychosis and catatonia. Neurohospitalist. 2025; 16(1): 19418744251364125. doi: 10.1177/19418744251364125.

12. van Leeuwen N, Bossema ER, Van Middendorp H, Kruize AA, Bootsma H, Bijlsma JW, et al. Dealing with emotions when the ability to cry is hampered: emotion processing and regulation in patients with primary Sjögren's syndrome. Clin Exp Rheumatol. 2012; 30(4): 492-8.

13. Alessitimia e pathway immunoendocrino nel lupus eritematoso sistemico e nell'artrite reumatoide [Alexithymia and immunoendocrine parameters in patients affected by systemic lupus erythematosus and rheumatoid arthritis]. Reumatismo. 2008; 60(1): 50-6. doi: 10.4081/reumatismo.2008.50. [Article in Italian].

14. Ozdemir E, Xiao Z, Griffiths H, MacBeth A. Alexithymia in schizophrenia and psychosis vulnerability: a systematic review and meta-analysis. J Clin Psychol. 2025; 81(6): 410-24. doi:10.1002/jclp.23788.

15. van der Velde J, Swart M, van Rijn S, van der Meer L, Wunderink L, Wiersma D, et al. Cognitive alexithymia is associated with the degree of risk for psychosis. PLoS One. 2015; 10(6): e0124803. doi: 10.1371/journal.pone.0124803.

16. de Oliveira FR, Fantucci MZ, Adriano L, Valim V, Cunha TM, Louzada-Junior P, et al. Neurological and inflammatory manifestations in Sjögren's syndrome: the role of the kynurenine metabolic pathway. Int J Mol Sci. 2018; 19(12): 3953. doi: 10.3390/ijms19123953.

17. de Oliveira FR, Appenzeller S, Pasoto SG, Fernandes MLMS, Lemos Lopes ML, de Magalhães Souza Fialho SC, et al. Recommendations on neurologic, cognitive, and psychiatric manifestations in patients with Sjögren›s disease by the Brazilian Society of Rheumatology. Adv Rheumatol. 2025; 65(1): 7. doi: 10.1186/s42358-025-00438-7.

18. Catalán A, Aymerich C, Rodríguez-Sánchez JM, Pedruzo B, Salazar de Pablo G, Gil P, et al. Peripheral inflammation and neurocognitive functioning in early psychosis: specific associations of TNF-α and IL-6 with social cognition. Eur Psychiatry. 2025; 68(1): e95. doi: 10.1192/j.eurpsy.2025.10063.

19. Speers LJ, Bilkey DK. Inflammation in schizophrenia: the role of disordered oscillatory mechanisms. Cells. 2025; 14(9): 650. doi: 10.3390/cells14090650.

20. Gostoli S, Carrozzino D, Raimondi G, Subach R, Gigante G, Rafanelli C. Corticosteroid-induced manic and/or psychotic symptoms: a systematic review. Front Pharmacol. 2025; 16: 1628765. doi: 10.3389/fphar.2025.1628765.

21. El Kodsi S, Elouadoudi J, Denani M, Alfa MA, Laffinti MA. Severe corticosteroid-induced mania with psychotic fea-

tures in an HIV-positive patient: a case report. Cureus. 2025; 17(12): e99486. doi: 10.7759/cureus.99486.

22. Sofía-Avendaño-Lopez S, Rodríguez-Marín AJ, Lara-Castillo M, Agresott-Carrillo J, Lara-Cortés LE, Sánchez-Almanzar JF, et al. Molecular, pathophysiological, and clinical aspects of corticosteroid-induced neuropsychiatric effects: from bench to bedside. Biomedicines. 2024; 12(9): 2131. doi: 10.3390/biomedicines12092131.

23. Cerullo MA. Corticosteroid-induced mania: prepare for the unpredictable. Curr Psychiatr. 2006; 5: 43-50.

24. Bachu AK, Davis V, Abdulrahim M, Harbaugh L, Prasad S, Kotapati VP, et al. Corticosteroid-induced psychosis: a report of three cases. Cureus. 2023; 15(5): e39221. doi: 10.7759/cureus.39221.

25. Nasereddin L, Alnajjar O, Bashar H, Abuarab SF, Al-Adwan R, Chellappan DK, et al. Corticosteroid-induced psychiatric disorders: mechanisms, outcomes, and clinical implications. Diseases. 2024; 12(12): 300. doi: 10.3390/diseases12120300.

26. Canessa-Muñoz S, Yelmo-Cruz S, Hamilton-Lopez A, Baez-Marrero C. Corticosteroid-induced psychosis: a report of two cases and review of the literature. Cureus. 2025; 17(3): e81515. doi: 10.7759/cureus.81515.

27. National Academies of Sciences, Engineering, and Medicine; Health and Medicine Division; Board on Health Care Services; Committee on Selected Immune Disorders and Disability. Selected immune disorders and disability. Washington (DC): National Academies Press; 2022.

28. Tashbayev B, Rusthen S, Young A, Herlofson BB, Hove LH, Singh PB, et al. Interdisciplinary, comprehensive oral and ocular evaluation of patients with primary Sjögren's syndrome. Sci Rep. 2017; 7(1): 10761. doi: 10.1038/s41598-017-10809-w.

29. Ngo DYJ, Thomson WM, Nolan A, Ferguson S. The lived experience of Sjögren's syndrome. BMC Oral Health. 2016; 16: 7. doi: 10.1186/s12903-016-0165-4.

30. Pienkos E, Giersch A, Hansen M, Humpston C, McCarthy-Jones S, Mishara A, et al. Hallucinations beyond voices: a conceptual review of the phenomenology of altered perception in psychosis. Schizophr Bull. 2019; 45(Suppl 1): S67-77. doi: 10.1093/schbul/sby057.

31. Reeves RR, Torres RA. Exacerbation of psychosis by misinterpretation of physical symptoms. South Med J. 2003; 96(7): 702-4. doi: 10.1097/01.SMJ.0000078686.34109.11.

32. Lin CE. One patient with Sjogren's syndrome presenting schizophrenia-like symptoms. Neuropsychiatr Dis Treat. 2016; 12: 661-666. doi: 10.2147/NDT.S97753.

33. Hammett EK, Fernandez-Carbonell C, Crayne C, Boneparth A, Cron RQ, Radhakrishna SM. Adolescent Sjogren's syndrome presenting as psychosis: a case series. Pediatr Rheumatol Online J. 2020; 18(1): 15. doi: 10.1186/s12969-020-0412-8.

34. PT S, Jacob JA, Modi U, George D. Primary Sjogren's syndrome presenting as acute psychosis with catatonic symptoms. Indian J Psychiatry. 2022; 64(Suppl 3): S687-8. doi: 10.4103/0019-5545.342028.

35. Rosado SN, Silveira V, Reis AI, Gordinho A, Noronha C. Catatonia and psychosis as manifestations of primary Sjögren's syndrome. Eur J Case Rep Intern Med. 2018; 5(6): 000855. doi: 10.12890/2018_000855.

36. Inagaki T, Kudo K, Kurimoto N, Aoki T, Kuriyama K. A case of prolonged catatonia caused by Sjögren's syndrome. Case Rep Immunol. 2020; 2020: 8881503. doi: 10.1155/2020/8881503.

37. Mardale DA, Opriș-Belinski D, Bojincă V, Bojincă M, Mazilu D, Păsăran E, et al. The physical and psychosocial impact of fatigue among patients with Sjögren's syndrome: a systematic review. J Clin Med. 2023; 13(6): 1537. doi: 10.3390/jcm13061537.

38. Sampogna G, Di Vincenzo M, Giuliani L, Menculini G, Mancuso E, Arsenio E, et al. A systematic review on the effectiveness of antipsychotic drugs on the quality of life of patients with schizophrenia. Brain Sci. 2023; 13(11): 1577. doi: 10.3390/brainsci13111577.

39. Lazović B, Blažić I, Zlatković-Švenda M, Zugić V. Severe pneumonia caused by antipsychotic drugs – what does not suit, the patient or the drug? Sanamed. 2018; 13(3): 307-10. doi: 10.24125/sanamed.v13i3.257.

40. Mohamed AA, Almulhim AS, Alnijadi AA, Abd Aziz Fb, Alajmi KK, Al-Mudhaffar AA, et al. Evaluation of prescription patterns of antipsychotics in schizophrenia patients: a single-center prospective study. J Clin Med. 2025; 14(9): 2941. doi: 10.3390/jcm14092941.

41. Vallianatou K. Antipsychotics. Medicine (Baltimore). 2024; 52(9): 573-6. doi: 10.1016/j.mpmed.2024.06.016.

42. Inić T, Stanković Stevanović N, Čuturić B, Cvjetković Bošnjak M, MešiBosić V. Psychotic presentation as a first clinical manifestation of anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis – case report. MD-Medical Data. 2025; 17(3): 179–183.

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