Journal of Clinical and Nursing Research, 2026, Volume 10, Issue 4
http://ojs.bbwpublisher.com/index.php/JCNR
Online ISSN: 2208-3693
Print ISSN: 2208-3685

# Research Progress on the Mechanisms and Management Strategies of Pneumonia in Patients with Schizophrenia

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**Abstract:** Patients with schizophrenia are at high risk of developing pneumonia, with significantly higher incidence and mortality rates than the general population, a public health issue that warrants urgent attention. This article systematically reviews the epidemiological characteristics of pneumonia susceptibility in schizophrenia patients, deeply explores the multifaceted risk factors, including disease-specific features, antipsychotic medication effects, behavioral and lifestyle factors, and immune dysfunction. Based on this, the article further analyzes the core pathophysiological mechanisms underlying this susceptibility and comprehensively summarizes strategies for pneumonia prevention, early identification, and treatment. By integrating the latest research evidence, this review aims to provide a theoretical basis for developing targeted comprehensive management strategies in clinical practice, thereby improving the physical health status and long-term prognosis of patients with schizophrenia.

**Keywords:** Schizophrenia; Pneumonia; Risk factors; Antipsychotics; Comprehensive management

**Online publication:** Apr 30, 2026

## 1. Introduction

The risk of pneumonia in patients with schizophrenia is significantly higher than in the general population, and this susceptibility is collectively driven by disease-specific factors, medications, behaviors, and biological factors [1]. Monotherapy is associated with a dose-dependent increase in pneumonia risk; high-dose quetiapine (≥ 440 mg/d), medium-to-high-dose clozapine (≥ 180 mg/d), high-dose olanzapine (≥ 11 mg/d), and medications with high anticholinergic burden pose particularly prominent risks; clozapine, quetiapine, and olanzapine are associated with increased rates of hospital-acquired pneumonia infection [2,3]. Concomitant use of benzodiazepines, mood stabilizers (e.g., sodium valproate), or modified electroconvulsive therapy (MECT) further increases the risk of hospital-acquired pneumonia [4,5]. Advanced age, male sex, prolonged

hospitalization, malnutrition (low serum albumin), sarcopenia (assessed by Ishii test, calf circumference, or SARC-CalF), and smoking and alcohol consumption are all risk factors [6–9]; the severity of sarcopenia is positively correlated with pneumonia risk [10]. The anticholinergic effects of clozapine lead to excessive salivation, dysphagia, and increased aspiration risk; excessive sedation suppresses cough reflex and airway clearance function; it may cause neutropenia, weakening host defenses [6,11]. Schizophrenia (especially first episode) is associated with intestinal barrier disruption, bacterial translocation (e.g., *Klebsiella pneumoniae*), and immune complex formation, linked to systemic inflammation and increased susceptibility to infection [12]. Patients with schizophrenia spectrum disorders have a higher risk of severe illness and death after SARS-CoV-2 infection [13,14], and long-term antipsychotic treatment may exert certain effects on inflammatory responses [15]. Leptin receptor (*LEPR*) gene polymorphisms are associated with pneumonia risk in clozapine-treated patients; the role of host microbiota in clozapine-induced ileus and pneumonia is being explored [16,17].

## 2. Analysis of multiple risk factors for pneumonia susceptibility in patients with schizophrenia

### 2.1. Intrinsic disease factors and behavioral lifestyle risks

Patients with schizophrenia have a significantly increased risk of developing pneumonia, with intrinsic disease factors and behavioral lifestyle factors forming a complex and intertwined risk network. First, the psychiatric symptoms themselves pose a direct threat to the patient's physiological defense mechanisms. Negative symptoms, such as avolition and social withdrawal, severely impair patients' self-care abilities, leading to poor oral hygiene and increased colonization and proliferation of oral pathogens [6]. Concurrently, cognitive impairments affect swallowing coordination and the cough reflex, significantly raising the risk of silent aspiration, a key trigger for aspiration pneumonia [6]. Positive symptoms, such as delusions, may lead patients to refuse medical care or non-compliance with preventive treatments, delaying early intervention. Second, patients with schizophrenia have a very high smoking rate, reaching 50–80% [6]. Tobacco smoke directly damages the mucociliary clearance function of the respiratory tract and inhibits alveolar macrophage activity, disrupting the first line of defense against pathogens in the lungs. Additionally, substance abuse, such as alcohol and drugs, further depresses the central nervous system, weakens protective reflexes, and causes immune system dysfunction, making patients more susceptible to severe infections [6]. Malnutrition and obesity are other significant risk factors. Due to irregular eating habits, metabolic disorders induced by antipsychotics, and other reasons, patients often suffer from malnutrition or obesity. Studies have shown that decreased serum albumin levels are significantly associated with an increased risk of pneumonia in female patients with stable schizophrenia [8]. Whether it is insufficient immune protein synthesis due to malnutrition or the chronic low-grade inflammatory state caused by obesity, immune function is compromised. Finally, socioeconomic factors, such as low income, poor living conditions, and weak social support systems, limit patients' access to timely, high-quality medical care and the ability to maintain healthy living conditions, indirectly increasing infection risk [6].

### 2.2. Direct effects of antipsychotic medications

While controlling psychiatric symptoms, antipsychotic medications also directly increase the risk of pneumonia in patients through their pharmacological effects, with this association being dose-dependent and drug-specific. The primary risk stems from the sedative effects of these drugs. Especially first-generation and

certain second-generation antipsychotics with strong sedative properties can cause excessive sedation and reduced level of consciousness, significantly increasing the risk of aspiration, a key precipitant of aspiration pneumonia [6]. Second, anticholinergic side effects represent one of the core mechanisms increasing pneumonia risk. Many antipsychotics possess anticholinergic properties, leading to reduced salivation (dry mouth) and directly affecting the swallowing center, causing swallowing dysfunction [2]. A large cohort study clearly indicated that only the use of antipsychotics with a high anticholinergic burden was associated with an increased risk of pneumonia [2]. This "silent aspiration" allows oral secretions and food debris to more easily enter the lower respiratory tract. Regarding specific drug risks, clozapine stands out as the most prominent. Research confirms that clozapine use is significantly associated with an increased risk of upper respiratory infections progressing to pneumonia [18]. This risk exhibits a dose-dependent pattern, with both medium and high doses of clozapine associated with an increased risk of pneumonia (adjusted hazard ratios of 1.43 and 1.44, respectively) [2]. Furthermore, although rare, clozapine can cause severe agranulocytosis, leading to the collapse of the body's first line of defense against bacterial infections, making severe pneumonia highly likely [11]. Other drugs, such as high-dose quetiapine (≥ 440 mg/d) and high-dose olanzapine (≥ 11 mg/d), have also been confirmed to be associated with an increased risk of pneumonia[2]. Finally, the metabolic side effects of antipsychotics, such as weight gain, elevated blood glucose, and dyslipidemia, create a chronic low-grade inflammatory state, indirectly weakening the body's immune response to acute infections [6]. Notably, the combined use of antipsychotics, especially with benzodiazepines and mood stabilizers, further increases the risk of hospital-acquired pneumonia [4].

## 2.3. Underlying state of immune system dysfunction

Patients with schizophrenia have an inherent state of immune system dysfunction, which constitutes the biological basis for their susceptibility to pneumonia. This dysfunction exists even in medication-naïve patients. First, innate immune abnormalities are a core feature. Studies show alterations in immune markers in patients with schizophrenia, such as elevated levels of pro-inflammatory cytokines (e.g., IL-6, TNF-α), suggesting underlying immune activation or dysregulation [12]. This chronic, non-specific inflammatory background may impair effective immune responses against specific pathogens, preventing the body from mounting a precise and robust defense when facing bacterial or viral infections. Second, adaptive immune responses may also be impaired. Some studies have found that patients with schizophrenia exhibit weaker antibody responses to vaccines (e.g., influenza vaccine, pneumococcal vaccine) compared to healthy controls, suggesting potential defects in B cell function or helper T cell function [19]. A preclinical study also found that the antipsychotic risperidone disrupts antibody responses to pneumococcal vaccine in mice, which may have similar implications in clinical patients. This weakening of adaptive immune responses makes it difficult for patients to gain adequate protection through vaccination and also affects the production of specific antibodies post-infection. Furthermore, alterations in immune cell function have been widely reported. For instance, functional abnormalities in macrophages and natural killer (NK) cells suggest inherent deficiencies in immune surveillance and pathogen clearance [19]. Such functional defects in immune cells prevent the effective recognition and elimination of pathogens in the early stages of invasion, creating conditions conducive to the establishment and spread of infection. Additionally, genetic factors play a role. Research indicates that specific polymorphisms in the leptin receptor (*LEPR*) gene are associated with a significantly increased risk of pneumonia in Taiwanese schizophrenia patients treated with clozapine, with

individuals carrying certain genotypes facing a nearly 15-fold increased risk [16]. This reveals how individual genetic differences interact with the underlying immune state under drug exposure to collectively determine susceptibility to pneumonia.

## 3. Exploration of core pathophysiological mechanisms of pneumonia in patients with schizophrenia

### 3.1. Cascade mechanism from aspiration to infection

In the pathophysiology of pneumonia in patients with schizophrenia, the cascade from aspiration to infection is a central component. First, patients' defense reflex systems are often compromised due to the disease itself and pharmacological treatments. Multiple studies confirm that antipsychotics, particularly those with a high anticholinergic burden, are significantly associated with an increased risk of pneumonia [2]. Anticholinergic effects inhibit the cough reflex and swallowing reflex, allowing oropharyngeal secretions or gastric contents to easily enter the lower respiratory tract without being effectively cleared, which is a direct cause of aspiration pneumonia [6]. Second, aspirated material contains oral flora. After these bacteria colonize the lungs, the patients' impaired local immune defenses cannot effectively clear the pathogens. Studies show that patients with schizophrenia often suffer from malnutrition and sarcopenia, which can lead to decreased systemic and local immune function [7,8]. For example, decreased serum albumin level is an independent factor associated with pneumonia risk in female patients with stable schizophrenia [8]. Finally, the underlying chronic low-grade inflammatory state present in patients with schizophrenia may lead to an imbalance in the inflammatory response in the lungs upon infection. This imbalance may manifest as insufficient response, leading to pathogen dissemination, or excessive response, causing severe tissue damage and even acute respiratory distress syndrome (ARDS). Some research suggests that in patients with schizophrenia, cytokines released during infection may affect the central nervous system, exacerbate psychiatric symptoms and forming a vicious cycle [6].

### 3.2. Dysregulation of the neuro-immune-endocrine network

The susceptibility of patients with schizophrenia to pneumonia is closely related to the dysregulation of their intrinsic neuro-immune-endocrine network. Hypothalamic-pituitary-adrenal (HPA) axis dysfunction is a key component. Patients with schizophrenia often exhibit HPA axis hyperactivity, leading to chronically elevated cortisol levels. This long-term high-cortisol environment exerts widespread immunosuppressive effects, particularly inhibiting cellular immunity and appropriate inflammatory responses, thereby reducing resistance to intracellular pathogens (e.g., viruses, *Mycobacterium tuberculosis*) [6]. Autonomic nervous system dysfunction, particularly altered vagal tone, is another important factor. Vagal dysfunction may affect the function of immune cells like macrophages and interfere with the fine-tuning of systemic inflammatory responses via the cholinergic anti-inflammatory pathway. This dysregulation of neuroimmune interaction may partially explain why patients on antipsychotics with high anticholinergic burden have a higher risk of pneumonia [2]. Furthermore, there is a bidirectional relationship between infection and psychiatric symptoms. Pro-inflammatory cytokines (e.g., IL-1β, IL-6) released during infection can cross the blood-brain barrier or affect the brain via vagal afferents, potentially exacerbating existing psychiatric symptoms [6]. The worsening of psychiatric symptoms further impairs patients' self-care abilities, such as neglecting personal hygiene or inability to effectively expectorate sputum, thus increasing the risk of pneumonia occurrence or

worsening, forming a vicious cycle of "infection – worsening psychiatric symptoms – further decline in self-care ability". This complex network dysregulation highlights the particular challenges and complexities of managing pneumonia in patients with schizophrenia.

### 3.3. Disruption of respiratory barrier structure and function

Disruption of the structure and function of the respiratory barrier is an important pathological basis for pulmonary susceptibility in patients with schizophrenia. First, dysfunction of the mucociliary clearance system is a common issue. The prevalent smoking habit among patients, along with the potential effects of certain antipsychotics, may lead to decreased mucociliary motility in the respiratory tract, preventing inhaled pathogens from being promptly cleared by the mucus [6]. Second, the integrity of the airway epithelial barrier may be compromised. The chronic inflammation and oxidative stress present in patients with schizophrenia may disrupt tight junctions between airway epithelial cells, increasing the risk of pathogen invasion into submucosal tissues. Studies suggest that different immune and oxidative stress pathway abnormalities exist in first-episode and multi-episode schizophrenia patients, which may extend systemically, including the respiratory barrier [12]. Finally, the alveolar surfactant system may be abnormal. Surfactant proteins play a critical role not only in reducing alveolar surface tension but also possess essential immune defense functions. It is hypothesized that certain genes associated with schizophrenia might affect the expression or function of pulmonary surfactant proteins, and their abnormalities could increase susceptibility to pneumonia [20]. Additionally, sarcopenia, a common comorbidity in patients with schizophrenia, is directly related to pneumonia risk, with its severity correlating with increased risk [10]. Muscle function decline, particularly respiratory muscle weakness, directly affects cough efficiency and airway clearance capability, further compromising the physical barrier function. These disruptions in barrier function collectively constitute structural vulnerabilities for pathogen colonization and infection development.

## 4. Comprehensive management strategies for pneumonia in patients with schizophrenia

### 4.1. Prevention strategies: Tiered intervention and active surveillance

Prevention of pneumonia in patients with schizophrenia requires a tiered intervention strategy. The core of primary prevention lies in universal measures. Given that patients with schizophrenia are at high risk for pneumonia, with a significantly higher prevalence than the general population, routine vaccination is crucial [1]. Evidence suggests that pneumococcal vaccination coverage is inadequate, and unvaccinated individuals may face severe consequences of invasive pneumococcal disease (e.g., bacteremia) [21]. Therefore, vigorously promoting and routinely administering influenza and pneumococcal vaccines to all patients with schizophrenia, and monitoring antibody responses, constitutes a fundamental public health measure to reduce infection risk. Concurrently, given the prevalent risks of malnutrition and sarcopenia in patients, nutritional support is indispensable. Studies indicate that decreased serum albumin levels and reduced calf circumference are associated with increased pneumonia risk, highlighting the importance of early nutritional assessment and intervention [8,22]. Furthermore, conducting health education for patients and caregivers to enhance awareness of early pneumonia symptoms (e.g., fever, cough, dyspnea) facilitates early identification and medical consultation.

Secondary prevention focuses on targeted interventions for high-risk patients. Identifying high-risk

factors is key, including advanced age, use of specific antipsychotics (especially clozapine, olanzapine, quetiapine, particularly at high doses), concomitant use of benzodiazepines or mood stabilizers, receiving modified electroconvulsive therapy (MECT), and presence of dysphagia or sialorrhea symptoms [2,5,6]. For these patients, routine swallowing function assessment should be conducted. If abnormalities are found, consider adjusting antipsychotic medication (e.g., switching to drugs with fewer anticholinergic side effects) and implementing physical interventions such as postural adjustments during eating and modification of food consistency. Enhancing oral care interventions to reduce oropharyngeal pathogen colonization is also an important aspect of preventing aspiration pneumonia [2].

Tertiary prevention aims to prevent complications, primarily targeting patients with schizophrenia who already have chronic respiratory diseases (e.g., chronic obstructive pulmonary disease). This patient group inherently has a high prevalence of respiratory diseases[1] and requires enhanced chronic disease management to ensure regular medication and reduce acute exacerbations. In psychiatric inpatient settings, strict implementation of infection control protocols is particularly important. Hospitalization duration is an independent risk factor for hospital-acquired pneumonia, and polypharmacy during hospitalization (including antipsychotics combined with MECT, benzodiazepines, mood stabilizers) significantly increases pneumonia risk [3,4]. Therefore, optimizing the inpatient environment, shortening unnecessary hospital stays, carefully evaluating the necessity of combination therapies, and strictly enforcing infection control measures such as hand hygiene and environmental disinfection are crucial for reducing nosocomial infection risk.

## 4.2. Treatment strategies: Multidisciplinary collaboration and individualized medication

The treatment of pneumonia in patients with schizophrenia presents unique challenges and requires multidisciplinary collaboration and individualized medication. Early identification and diagnosis are primary challenges. Due to potential communication difficulties or atypical symptom presentation, healthcare professionals must maintain a high index of suspicion for non-typical manifestations. For instance, sudden worsening of psychiatric symptoms, lethargy, or refusal to eat may be the initial or sole symptoms of pneumonia [6]. Therefore, for any patient with unexplained deterioration in health status, timely detailed physical examination and chest imaging should be performed to avoid delayed diagnosis.

The choice of anti-infective therapy requires special consideration. Given the high aspiration risk in patients with schizophrenia, empirical antibiotic selection should cover aspiration-related anaerobes and Gram-negative bacteria. Close monitoring for drug interactions is particularly important. Certain antibiotics (e.g., fluoroquinolones, macrolides) can prolong the QTc interval, and co-administration with many antipsychotics may synergistically increase the risk of arrhythmias [2]. Additionally, the inhibition of antipsychotic-metabolizing enzymes (e.g., CYP1A2) can lead to drug toxicity and increase pneumonia risk, so the impact on liver enzymes should be considered when selecting antibiotics [16].

Adjustment of psychiatric medications is a core component of treatment. During severe pneumonia, it is necessary to assess whether to temporarily reduce the dose or switch to antipsychotics with weaker sedative effects and fewer anticholinergic side effects to reduce aspiration risk due to excessive sedation and improve ventilatory function[2]. However, this decision requires careful balancing against the risk of psychiatric symptom relapse. Studies show that antipsychotic monotherapy, especially with high anticholinergic burden drugs, is associated with increased pneumonia risk [2]. For patients on clozapine, considering dose reduction during upper respiratory tract infections may help prevent progression to pneumonia [18]. However, any

adjustments should be made under the guidance of a psychiatrist with close monitoring of psychiatric status.

Supportive care and rehabilitation are crucial for prognosis. Ensuring adequate oxygenation, meticulous fluid management, and nutritional support is fundamental. Given the strong association of malnutrition and sarcopenia with pneumonia occurrence and poor outcomes [8–10], active nutritional intervention is indispensable. Early implementation of respiratory rehabilitation training, such as deep breathing exercises, effective coughing, and postural drainage, can help improve lung function and promote sputum clearance. Concurrently, active management of comorbidities, such as glycemic control and management of chronic cardiopulmonary diseases, is essential. For elderly or critically ill patients, multidisciplinary team collaboration can be considered, and the potential of combining antipsychotics with traditional Chinese medicine may be explored, as the latter may help shorten hospital stays when used in combination [23].

## 4.3. System optimization and future research directions

Optimizing the management of pneumonia in patients with schizophrenia requires systemic innovation and promotion of cutting-edge research. The primary task is to establish integrated care models. The current fragmentation between physical and mental health services must be addressed by actively promoting deep collaboration between mental health institutions and respiratory, infectious disease, and geriatric departments in general hospitals. Establishing dedicated physical health management pathways for patients with severe mental illness can ensure they receive timely, equitable, and high-quality medical care for physical illnesses. This model facilitates the systematic implementation of preventive strategies and early detection for treatment.

Drug development should focus on novel targets. Existing antipsychotics, especially clozapine, olanzapine, and quetiapine, whose sedative and anticholinergic effects, along with potential impacts on immune function, are important factors increasing pneumonia risk [2,11]. Future research and development should aim to create novel antipsychotics with fewer effects on immune function and metabolism, or that do not impair protective reflexes like cough and swallowing. Concurrently, in-depth research into the mechanisms of interaction between antipsychotics and the immune system, for example, their effects on immune responses via muscarinic receptors, may provide clues for repurposing old drugs or mitigating risks [24].

Identifying reliable biomarkers for risk prediction is key to precision prevention. Future research should focus on discovering biomarkers capable of predicting pneumonia risk in patients with schizophrenia. Potential directions include specific inflammatory cytokine profiles, such as leptin receptor gene polymorphisms associated with clozapine-induced pneumonia; salivary or gut microbiome characteristics, as research suggests host microbiota may be related to clozapine-associated ileus and pneumonia; and easily measurable physical indicators, such as sarcopenia screening tool (SARC-CalF, SARC-F+EBM) scores or calf circumference [9,16,17,22]. These markers could be used for early identification of high-risk individuals to implement intensive prevention.

Immunomodulatory interventions represent a potentially promising yet cautiously explored area. Basic research shows activation of neuroimmune and oxidative pathways in schizophrenia, and pneumonia itself is an intense inflammatory process [12]. Theoretically, using anti-inflammatory drugs or immunomodulators (e.g., inhibitors targeting cytokines like IL-6) could potentially improve immune function while addressing core psychiatric pathology. However, such interventions must be evaluated with extreme caution regarding the potential increased risk of infection. Future translational research, under rigorous monitoring, is needed

to explore the safety and efficacy of specific immunomodulatory strategies in defined patient subgroups, avoiding further increasing infection susceptibility due to inappropriate interventions.

## 5. Conclusion

The high incidence of pneumonia in patients with schizophrenia represents a complex clinical challenge stemming from a combination of intrinsic disease pathophysiology, external therapeutic interventions, and broad psychosocial factors. From an expert perspective, the core of this issue lies in understanding that schizophrenia is not an isolated brain disorder but a systemic health crisis involving multi-system dysregulation. The disease itself may impair respiratory defenses through neuroimmune and neuroendocrine pathways; antipsychotics, while managing psychiatric symptoms, may suppress cough reflexes, increase sedation and metabolic side effects, indirectly elevating aspiration and infection risks; and patients often have concomitant cognitive impairment, diminished self-care capacity, unhealthy lifestyle habits (e.g., smoking), and potential barriers to accessing medical care and social support, collectively creating conditions conducive to pneumonia.

Therefore, future clinical practice and research must resolutely move towards integration and precision. In terms of management, prevention is far superior to treatment. Establishing an integrated preventive system encompassing vaccination, oral care, smoking cessation, and nutritional support is crucial, requiring deep collaboration among psychiatry, pulmonology, general practice, and community nursing. During treatment, a high index of suspicion is needed to recognize pneumonia that may present atypically with apathy or delirium, and careful management of complex interactions between anti-infective and psychiatric medications is essential.

## Disclosure statement

The author declares no conflict of interest.

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