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Medication-induced psychosis: A review of current evidence and future directions

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Abstract
Medication-induced psychosis is a clinically significant condition resulting from the adverse effects of various pharmacological agents on brain function. It often mimics primary psychotic disorders, complicating diagnosis and management across multiple medical disciplines. This review examines the pathophysiology, clinical presentations, diagnostic challenges, and management strategies associated with medication-induced psychosis. Neurotransmitter imbalances, medication dosages, and individual susceptibility are identified as key contributors to its development. The diagnostic process requires distinguishing medication-induced psychosis from primary psychiatric disorders through detailed patient histories and a high index of clinical suspicion. Effective management strategies focus on discontinuing the causative agent, administering antipsychotics when necessary, and implementing preventive measures, including patient education and risk mitigation. Future developments in pharmacogenomics, targeted drug design, and early detection techniques may revolutionize the prevention and treatment of medication-induced psychosis. By integrating interdisciplinary research and clinical insights, healthcare providers can improve therapeutic safety and efficacy, enhancing patient outcomes and minimizing the risks associated with psychoactive medications.

Keywords: Medication-induced psychosis**,** drug-induced psychosis**,** neurotransmitter imbalance**,** psychotropic side effects

Introduction
Medication-induced psychosis refers to a distinct subset of psychotic disorders that occur as a direct consequence of certain medications or substances. Psychosis itself is a severe mental health condition characterized by a loss of contact with reality, manifesting as hallucinations, delusions, disorganized thinking, and behavioral disturbances. Unlike primary psychotic disorders, such as schizophrenia, medication-induced psychosis arises from pharmacological effects rather than an underlying psychiatric condition. This phenomenon presents a significant challenge in clinical practice, requiring a nuanced understanding of drug actions, patient-specific factors, and differential diagnosis. Addressing this issue is crucial for ensuring patient safety and optimizing therapeutic outcomes.

Psychosis encompasses a range of clinical features, including perceptual disturbances like hallucinations, typically auditory or visual; delusions, which are false and fixed beliefs resistant to reason or evidence; disorganized thinking, marked by incoherent or illogical speech patterns; and behavioral dysregulation, which can include erratic or impulsive actions. These features are central to identifying psychosis but can also overlap with medication-induced presentations. Disentangling psychosis caused by medications from primary psychotic disorders involves assessing the temporal relationship between symptom onset and drug exposure, as well as the resolution of symptoms following discontinuation of the offending agent.

Medication-induced psychosis arises through various mechanisms, including direct pharmacological effects on the central nervous system, drug-drug interactions, and patient-specific vulnerabilities such as genetic predisposition or pre-existing psychiatric conditions. Commonly implicated drug classes include psychoactive substances like amphetamines, corticosteroids, anticholinergics, and even some antibiotics. While many cases are dose-dependent and reversible, prolonged exposure or underlying vulnerabilities can lead to more

persistent symptoms, complicating diagnosis and treatment. This condition underscores the importance of pharmacovigilance and individualized patient care in medical practice.

Historically, the recognition of medication-induced psychosis began with observations of psychotic symptoms following substance use. Amphetamine-induced psychosis, described in the mid-20th century, provided early insights into how drug effects on the dopaminergic system could mimic schizophrenia. Similarly, the psychiatric effects of medications like corticosteroids became apparent as their use expanded. Over time, diagnostic criteria for medication-induced psychosis evolved, particularly with the publication of standardized frameworks such as the Diagnostic and Statistical Manual of Mental Disorders (DSM). Earlier editions grouped these conditions under broader categories of "organic psychoses," but later versions introduced more precise criteria, emphasizing the temporal link between medication use and psychotic symptoms, alongside the reversibility of symptoms upon drug discontinuation.

The significance of medication-induced psychosis extends across multiple disciplines. In psychiatry, it challenges diagnostic processes by introducing iatrogenic factors that must be distinguished from primary mental health conditions. For pharmacology, understanding the mechanisms by which drugs can induce psychosis is critical for predicting and mitigating these effects. Additionally, medication-induced psychosis has vital implications for patient safety, emphasizing the need for vigilance in prescribing practices and monitoring for adverse effects. This topic exemplifies the intricate relationship between pharmacological treatment and mental health, underscoring the importance of interdisciplinary collaboration to safeguard patients and improve outcomes. Through a better understanding of this condition, clinicians can reduce its prevalence, enhance treatment precision, and maintain trust in medical interventions.

Methods

This review article was conducted using a systematic approach to identify and synthesize current evidence on medication-induced psychosis. Literature searches were performed in major academic databases, including PubMed, Scopus, Web of Science, and PsycINFO, using keywords such as "medication-induced psychosis," "drug-induced psychosis," "stimulant psychosis," and "anticholinergic psychosis." Articles published between January 2010 and December 2024 were included if they focused on the mechanisms, clinical manifestations, or management of psychosis linked to medications. Peer-reviewed studies, case reports, clinical trials, and reviews were eligible, while non-English and non-peer-reviewed sources were excluded. After duplicate removal, titles and abstracts were screened by two independent reviewers, followed by full-text review for relevance. Key data, including implicated medications, mechanisms, clinical findings, and management strategies, were extracted using a standardized form.

A narrative synthesis was used to organize findings into thematic categories, such as classes of medications, diagnostic challenges, and prevention strategies. Quantitative data, where available, were tabulated to highlight trends. Study quality was assessed using tools like the Cochrane Risk of Bias tool for clinical trials to ensure reliable conclusions. The scope of this review is limited by potential publication bias, reliance on English-language articles, and a lack of long-term studies on medication-induced psychosis.

Pathophysiology and Mechanisms of Medication-Induced Psychosis

Neurobiological Basis

The development of medication-induced psychosis is deeply rooted in disruptions of neurobiological systems, particularly those involving neurotransmitters and specific brain regions critical for cognition, perception, and emotional regulation. This interplay often leads to an altered state of brain function, mimicking or exacerbating psychotic disorders such as schizophrenia.

Dysregulation of the dopaminergic system is a key factor in many psychotic disorders, including those induced by medications. Hyperactivity of dopamine in the mesolimbic pathway has been strongly associated with psychosis, as it can lead to hallucinations, delusions, and other symptoms. Medications like amphetamines, methylphenidate, or levodopa increase dopamine availability by stimulating its release or inhibiting its reuptake, overwhelming the normal neural signaling mechanisms. This dopaminergic surge particularly impacts the nucleus accumbens and ventral tegmental areas, intensifying reward and salience processing to an abnormal degree. Interestingly, dopamine antagonists such as antipsychotics, which initially mitigate psychotic symptoms, may trigger rebound psychosis when discontinued, due to receptor hypersensitivity.

The serotonergic system, primarily mediated by 5-HT receptors, plays a complementary role in psychosis, particularly hallucinations and affective dysregulation. Agonism at 5-HT2A receptors, induced by drugs such as LSD or psilocybin, is strongly linked to visual and auditory hallucinations. Additionally, certain serotonin-enhancing medications, such as selective serotonin reuptake inhibitors (SSRIs) or serotonin-norepinephrine reuptake inhibitors (SNRIs), can indirectly influence dopamine systems, contributing to psychotic symptoms in susceptible individuals. This interaction demonstrates how serotonergic and dopaminergic systems interconnect to modulate cognition and perception.

Recent evidence highlights the role of glutamate, the brain’s primary excitatory neurotransmitter, in psychosis. NMDA receptor antagonists such as ketamine and phencyclidine (PCP) can induce psychotic symptoms by disrupting excitatory-inhibitory signaling in the prefrontal cortex and limbic regions. NMDA receptor dysfunction has also been implicated in schizophrenia, suggesting a shared pathway for primary and secondary psychoses. Glutamatergic dysregulation is particularly significant in medication-induced psychosis because it can amplify pre-existing vulnerabilities in neural circuits, even in individuals without prior psychiatric diagnoses.

The GABAergic system, which provides inhibitory regulation of neural activity, is another critical player. Medications that disrupt GABA signaling, such as benzodiazepines during withdrawal, can lead to heightened excitatory states, causing psychotic symptoms like paranoia and agitation. This mechanism underscores the importance of maintaining excitatory-inhibitory balance in preventing psychosis.

Several brain regions play a central role in the pathophysiology of medication-induced psychosis. The

prefrontal cortex, responsible for executive functions and reality testing, often shows hypoactivity during psychotic episodes, contributing to disorganized thinking and impaired judgment. The limbic system, particularly the amygdala and hippocampus, exhibits hyperactivity, driving emotional dysregulation and the heightened perception of threats. Disruptions in connectivity between the prefrontal cortex and limbic structures exacerbate symptoms, creating a feedback loop that sustains the psychotic state. Additionally, alterations in the thalamus and striatum, which are crucial for sensory integration and motor control, may underlie the sensory distortions and restlessness commonly observed in psychotic presentations.

Mechanisms of Induction

Medication-induced psychosis can result from diverse mechanisms, including toxicity, hypersensitivity reactions, withdrawal effects, and individual susceptibilities. Each pathway reflects the complexity of how medications interact with the brain and body.

Certain medications can directly induce psychosis by creating a toxic environment in the brain. High doses of stimulants, such as amphetamines or cocaine, lead to excessive dopamine release, which overwhelms normal neural circuits and induces psychosis-like symptoms, including paranoia, hypervigilance, and grandiosity. Similarly, corticosteroids at high doses are known to disrupt hippocampal function, contributing to excitotoxicity and the onset of psychosis. The mechanism often involves oxidative stress and mitochondrial dysfunction, which compromise neural integrity.

Idiosyncratic reactions to medications can also precipitate psychosis. Anticholinergic drugs, often used in the treatment of allergies or gastrointestinal disorders, block acetylcholine signaling, resulting in delirium and psychotic symptoms, particularly in older adults or those with compromised blood-brain barriers. Other examples include antibiotics like ciprofloxacin or isoniazid, which, though rare, can induce psychosis through poorly understood hypersensitivity pathways involving inflammation or altered neurotransmitter metabolism.

Abrupt cessation of medications, especially those affecting neurotransmitter systems, can lead to withdrawal-induced psychosis. For example, discontinuing antipsychotics may cause a rebound effect, where dopamine receptor upregulation during treatment leads to a surge of dopaminergic activity upon withdrawal. Similarly, benzodiazepine withdrawal is associated with GABAergic dysfunction, resulting in hyperexcitability and psychosis. Even substances like alcohol or opioids can induce psychotic symptoms during withdrawal, further complicating clinical presentations.

Polypharmacy, or the use of multiple medications, is an increasingly common factor in modern healthcare and can significantly contribute to the risk of psychosis. Drug-drug interactions that amplify neurotransmitter effects or disrupt metabolic pathways are frequent culprits. For instance, combining serotonergic antidepressants with other serotonergic agents may precipitate serotonin syndrome, which can include confusion, hallucinations, and agitation as part of its clinical presentation.

Role of Individual Susceptibility

Not all individuals exposed to medications that can induce psychosis will develop symptoms. The presence of underlying vulnerabilities, including genetic predispositions, pre-existing conditions, and environmental factors, plays a crucial role in determining susceptibility.

Genetic factors significantly influence an individual's response to medications. Polymorphisms in genes related to dopamine (e.g., DRD2), serotonin (e.g., 5HT2A), or glutamate receptors (e.g., GRIN2A) can increase susceptibility to psychosis. Variants in genes encoding enzymes responsible for drug metabolism, such as cytochrome P450 enzymes, can also lead to altered drug levels in the body, heightening the risk of adverse effects.

Patients with underlying psychiatric disorders, such as schizophrenia, bipolar disorder, or major depression, are at increased risk of medication-induced psychosis. These conditions are often characterized by baseline dysregulation in neurotransmitter systems, which can be exacerbated by medication effects. Additionally, the presence of a history of substance use disorder may predispose individuals to heightened sensitivity to psychoactive medications.

Physical health conditions, such as liver or kidney dysfunction, can impair drug metabolism and elimination, increasing systemic drug levels and the risk of psychosis. Older adults are particularly vulnerable due to age-related changes in pharmacokinetics and pharmacodynamics, as well as a higher likelihood of polypharmacy.

Environmental stressors, such as trauma, chronic stress, or social isolation, can act as triggers for psychosis when combined with medication effects. For instance, individuals exposed to high levels of life stress may be more likely to experience psychosis when treated with corticosteroids or psychostimulants.

Classes of Medications Linked to Psychosis

Medication-induced psychosis refers to the onset of psychotic symptoms, such as hallucinations, delusions, and paranoia, as a side effect of various drugs. These symptoms often arise from disruptions in neurotransmitter systems, receptor dynamics, or systemic factors caused by medications. Understanding the mechanisms, clinical features, and risk factors of psychosis-inducing drug classes is essential for timely diagnosis, management, and prevention.

Stimulants

Stimulants, such as amphetamines (e.g., Adderall, Dexedrine) and methylphenidate (e.g., Ritalin, Concerta), are well-known for their potential to induce psychosis, particularly at high doses or during misuse. These medications work by increasing dopamine and norepinephrine levels, which enhance focus, energy, and attention. However, excessive dopaminergic activity, particularly in the mesolimbic pathway, can lead to symptoms of psychosis, including paranoia, hallucinations, and delusions. Chronic misuse of stimulants, especially methamphetamine, can cause severe psychosis that mimics schizophrenia. This condition, known as stimulant psychosis, can persist even after discontinuation, suggesting long-term alterations in the dopaminergic system. Studies estimate that up to 50% of chronic stimulant users experience psychosis, while approximately 5-10% of individuals on therapeutic doses report transient psychotic symptoms. The risk is heightened in individuals with a history of mental illness, substance use disorders, or genetic susceptibility.

Anticholinergics

Anticholinergic medications, such as diphenhydramine, atropine, and benztropine, block muscarinic acetylcholine receptors in the brain, which are essential for cognitive function and sensory processing. By impairing cholinergic transmission, these drugs can cause central anticholinergic syndrome, a condition characterized by confusion, disorientation, paranoia, and hallucinations. Visual hallucinations are particularly common, often featuring vivid and bizarre images. Severe anticholinergic toxicity, typically resulting from high doses or polypharmacy, can lead to florid psychosis accompanied by tachycardia, dry mouth, and urinary retention. Older adults are especially vulnerable due to reduced acetylcholine levels and increased permeability of the blood-brain barrier. Misuse of over-the-counter medications like diphenhydramine for their sedative properties has been linked to psychotic episodes. Recognizing anticholinergic-induced psychosis is critical, as it is often mistaken for primary psychiatric disorders and requires prompt intervention to prevent life-threatening complications.

Corticosteroids

Corticosteroids, such as prednisone and dexamethasone, are widely used to treat inflammatory and autoimmune conditions. However, their potential to induce psychosis, often referred to as "steroid psychosis," is well-documented. Corticosteroids affect the brain by disrupting neurotransmitter systems, increasing glutamate activity, and reducing GABA-mediated inhibition, which are crucial for maintaining neural stability. Additionally, they dysregulate the hypothalamic-pituitary-adrenal (HPA) axis, contributing to mood and perceptual disturbances. Psychosis induced by corticosteroids typically includes symptoms such as mania, paranoia, hallucinations, and disorganized thinking. High doses, particularly above 40 mg/day of prednisone, significantly increase the risk. Symptoms usually appear within the first two weeks of treatment and may resolve upon dose reduction or discontinuation. Case studies report that up to 6% of patients on high-dose corticosteroids develop psychotic symptoms. Patients with a history of mood disorders, previous corticosteroid-induced psychosis, or concurrent use of other psychosis-inducing medications are at higher risk.

Dopaminergic Agents

Dopaminergic agents, including levodopa and dopamine agonists like pramipexole and ropinirole, are cornerstone treatments for Parkinson's disease but are frequently associated with psychotic symptoms. These medications work by enhancing dopamine activity in the brain, which improves motor symptoms in Parkinson's but may overstimulate the mesolimbic pathway, leading to psychosis. Early symptoms often include vivid dreams or minor hallucinations, which can progress to more severe manifestations such as paranoia and delusions. Advanced stages of Parkinson's disease heighten the risk due to existing dopaminergic imbalances and receptor hypersensitivity. Psychotic symptoms are dose-dependent, and management typically involves reducing the medication dose or switching to a less potent dopamine agonist. In severe cases, antipsychotics like quetiapine or clozapine, which minimally interfere with dopamine signaling, are used. Epidemiological studies indicate that 20-50% of Parkinson's patients treated with dopaminergic agents experience psychotic symptoms, underscoring the importance of careful monitoring.

Antidepressants

Both tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs) have been implicated in medication-induced psychosis. TCAs, such as amitriptyline, possess strong anticholinergic properties that can lead to confusion, hallucinations, and delirium, especially at high doses or in overdose. SSRIs, on the other hand, increase serotonin levels and may cause serotonin syndrome in severe cases. Serotonin syndrome is a potentially life-threatening condition characterized by agitation, hyperreflexia, altered mental status, and hallucinations. In individuals with undiagnosed bipolar disorder, antidepressants can precipitate manic episodes with psychotic features, such as grandiosity and paranoia. Although antidepressant-induced psychosis is relatively rare, it highlights the need for cautious prescribing, particularly in patients with mood disorders or co-administered serotonergic medications.

Anticonvulsants

Anticonvulsants like topiramate, levetiracetam, and vigabatrin are effective in managing seizures but are associated with psychotic side effects. These drugs modulate GABAergic and glutamatergic neurotransmission, and disruptions in these pathways can result in paranoia, hallucinations, and mood disturbances. Topiramate is particularly notorious for its cognitive side effects, including confusion and psychosis, which has earned it the nickname "Dopamax." Levetiracetam, while effective for epilepsy, has been linked to neuropsychiatric symptoms such as irritability, aggression, and psychosis, often requiring dose adjustments or discontinuation. Psychosis induced by anticonvulsants is typically reversible but necessitates close monitoring, especially in patients with a history of psychiatric disorders.

Immune Modulators and Biologics

Immune modulators, such as interferon-alpha and cyclosporine, are associated with significant neuropsychiatric side effects, including psychosis. Interferon-alpha, used in the treatment of hepatitis and certain cancers, can induce neuroinflammation and alter serotonin metabolism, leading to depressive and psychotic symptoms. Cyclosporine and tacrolimus, immunosuppressants used in organ transplantation, can cause neurotoxicity, resulting in hallucinations, paranoia, and confusion, particularly at high doses or in renal impairment. These medications pose a significant challenge in critically ill patients, where psychosis can complicate the course of treatment and recovery.

Antibiotics

Certain antibiotics, such as ciprofloxacin, isoniazid, and metronidazole, have been reported to cause psychosis. The mechanisms may involve neurotoxic effects, disruption of GABAergic signaling, or alterations in neurotransmitter synthesis. Psychotic symptoms, including auditory and visual hallucinations, agitation, and paranoia, are rare but more likely to occur in elderly patients or those with pre-existing neurological conditions. Severe cases, particularly

with isoniazid, may involve seizures or delirium. Recognizing antibiotic-induced psychosis is critical to prevent misdiagnosis and unnecessary psychiatric treatment.

Opioids and Opioid Antagonists

Opioids, such as morphine and fentanyl, can induce psychotic symptoms during intoxication or withdrawal. These symptoms are thought to arise from dysregulation of dopamine signaling in the mesolimbic pathway. Withdrawal psychosis is characterized by agitation, paranoia, and hallucinations, often requiring medical intervention. Opioid antagonists like naloxone, used to reverse opioid overdoses, can also precipitate transient psychosis as part of an acute withdrawal reaction. While opioid-induced psychosis is less common than other drug classes, its occurrence highlights the importance of monitoring during opioid detoxification and overdose reversal.

Diagnostic Challenges in Medication-Induced Psychosis

Diagnosing medication-induced psychosis poses significant challenges due to the overlap of its clinical presentation with primary psychotic disorders and other medical or psychiatric conditions. Identifying the condition requires careful attention to the relationship between medication use and symptom onset, along with a comprehensive evaluation of the patient's medical and psychiatric history. Clinicians must use diagnostic tools judiciously and consider potential confounding factors to arrive at an accurate diagnosis. Failure to correctly identify medication-induced psychosis can lead to unnecessary or inappropriate treatments and delay resolution of symptoms.

Differentiating Medication-Induced Psychosis from Primary Psychotic Disorders

The primary challenge in diagnosing medication-induced psychosis is distinguishing it from primary psychotic disorders, such as schizophrenia or bipolar disorder with psychotic features. One key differentiator is the temporal relationship between the onset of symptoms and medication use. In medication-induced psychosis, symptoms often appear shortly after starting a new drug, increasing the dose, or abruptly discontinuing a medication. For example, psychosis caused by corticosteroids frequently arises within the first two weeks of initiating high-dose treatment, while anticholinergic-induced psychosis can emerge rapidly following acute toxicity. Symptoms tend to improve or resolve entirely once the offending drug is discontinued or its dose is reduced, which is less likely in primary psychotic disorders.

Another distinguishing factor is the absence of a prior psychotic history. Patients with medication-induced psychosis often lack a history of psychotic episodes or a family history of psychotic disorders. In contrast, individuals with schizophrenia or bipolar disorder typically have a history of gradual symptom progression or episodic psychosis. However, pre-existing psychiatric conditions, such as major depressive disorder or anxiety, may complicate the diagnosis, as these conditions can predispose patients to drug-induced psychosis or exacerbate its presentation.

The nature of symptoms can also provide diagnostic clues. While hallucinations and delusions are common to both conditions, certain features may hint at a drug-induced origin. For instance, stimulant-induced psychosis often involves persecutory delusions and auditory hallucinations with heightened agitation, whereas anticholinergic toxicity frequently presents with vivid visual hallucinations and paranoia, often accompanied by systemic effects like dry mouth or tachycardia. Dopaminergic psychosis in Parkinson's patients may initially manifest as benign hallucinations, such as seeing animals or deceased relatives, before progressing to more complex psychotic features.

Diagnostic Tools

Although the diagnosis of medication-induced psychosis is largely clinical, diagnostic tools can support the evaluation and help exclude alternative causes.

A detailed clinical history is essential, focusing on the timing of symptom onset, any recent changes in medication or dosages, and the patient's medical and psychiatric history. Tools such as the Positive and Negative Syndrome Scale (PANSS) can quantify the severity of psychotic symptoms, allowing clinicians to monitor changes over time and assess the response to treatment adjustments. The Clinical Global Impression (CGI) scale is also valuable for evaluating overall psychiatric status and gauging the impact of medication changes on symptom severity.

Laboratory tests can provide additional insights, particularly in cases where medication toxicity or drug interactions are suspected. For example, toxicology screens can identify the presence of stimulants, opioids, or other psychoactive substances that may be contributing to the symptoms. Measuring serum drug levels is especially useful for medications like lithium or anticonvulsants, where toxic levels are strongly associated with psychosis. In cases of anticholinergic toxicity, tests for serum anticholinergic activity can confirm the diagnosis. Similarly, cortisol levels may support the diagnosis of corticosteroid-induced psychosis.

Neuroimaging may be employed to exclude structural brain abnormalities or other organic causes of psychosis. While structural imaging techniques such as MRI or CT scans are often sufficient, functional imaging modalities like PET or SPECT scans may reveal alterations in dopaminergic activity in cases of stimulant- or dopaminergic-induced psychosis. Advanced imaging may also detect neuroinflammation in cases of immune-modulator-induced psychosis, although its routine use is not yet established.

Common Misdiagnoses

Medication-induced psychosis is frequently misdiagnosed due to its similarity to other psychiatric and medical conditions. Among the most common misdiagnoses are schizophrenia, bipolar disorder, and delirium, each of which requires distinct management approaches.

Schizophrenia is often misdiagnosed in cases of medication-induced psychosis because both conditions share core symptoms, such as hallucinations, delusions, and disorganized thinking. However, the absence of a prior psychotic history, along with the resolution of symptoms after discontinuing the offending medication, strongly favors a drug-induced diagnosis. Mislabeling medication-induced psychosis as schizophrenia can lead to unnecessary long-term antipsychotic treatment, stigmatization, and failure to address the underlying cause.

Bipolar disorder with psychotic features may also be mistaken for medication-induced psychosis, particularly in cases where drugs like corticosteroids or antidepressants

trigger manic-like symptoms. Differentiating between the two requires a careful evaluation of the patient's mood state, longitudinal psychiatric history, and the temporal relationship between symptom onset and medication use. Bipolar disorder typically involves recurring manic or depressive episodes over time, whereas medication-induced psychosis resolves with drug cessation.

Delirium is another condition that can be confused with medication-induced psychosis, especially when the causative drug affects cognition or causes systemic toxicity. This is particularly true for anticholinergic drugs or sedative-hypnotics. Delirium is characterized by fluctuating levels of consciousness, global cognitive impairment, and an acute onset, which distinguishes it from isolated psychosis. Recognizing delirium is critical because it often signals a serious underlying medical condition requiring immediate intervention.

Other conditions, such as substance-induced psychosis, dementia, or organic brain syndromes, may also mimic medication-induced psychosis. For instance, stimulant-induced psychosis can resemble the psychosis seen in drug withdrawal, while anticholinergic-induced psychosis may be mistaken for early Alzheimer's disease. A thorough medication history, combined with an understanding of the patient's age, comorbidities, and clinical course, is essential to avoid diagnostic errors.

Clinical Manifestations of Medication-Induced Psychosis

Medication-induced psychosis encompasses a spectrum of symptoms that often resemble primary psychotic disorders, such as schizophrenia or bipolar disorder with psychotic features. However, its clinical presentation varies widely depending on the type of medication, dosage, and individual factors like pre-existing health conditions and genetic predisposition. A thorough understanding of the onset, progression, and specific symptomatology is essential for identifying and managing this complex condition.

Onset and Progression

The onset of medication-induced psychosis can be either acute or subacute, depending on the offending drug and the patient's response to it. Acute presentations often occur within hours to days of initiating a new medication, increasing its dose, or reaching toxic levels. For example, stimulants such as amphetamines or cocaine can induce psychosis within hours, characterized by heightened paranoia, agitation, and hallucinations. Similarly, anticholinergic toxicity, as seen with drugs like diphenhydramine or atropine, may cause sudden onset psychosis accompanied by confusion and physical symptoms such as tachycardia and dry mouth.

In contrast, subacute presentations emerge more gradually, over several days to weeks, often due to cumulative drug effects or prolonged use. Dopaminergic medications for Parkinson's disease, such as levodopa or dopamine agonists, often cause subtle initial symptoms like vivid dreams or mild illusions before progressing to more pronounced psychosis, including hallucinations and paranoia, over weeks or months. Corticosteroids, especially at high doses, may also induce subacute psychosis with manic or grandiose features, typically within the first two weeks of treatment. The progression of symptoms often depends on the duration of exposure to the medication. Acute symptoms may resolve promptly after discontinuing the offending drug, while subacute or chronic cases may persist and require additional pharmacological or therapeutic interventions.

Symptoms

The symptom profile of medication-induced psychosis includes hallucinations, delusions, agitation, and disorganized thinking or behavior. While these symptoms mirror those of primary psychotic disorders, their nature and accompanying features often provide diagnostic clues to the drug-induced etiology.

Hallucinations are a hallmark feature and may involve various sensory modalities.

- Auditory hallucinations, such as hearing accusatory or commanding voices, are common with stimulants like methamphetamine and serotonergic agents. These closely resemble the hallucinations seen in schizophrenia.
- Visual hallucinations are frequently reported with anticholinergic drugs and dopaminergic agents. Patients may describe seeing small animals, distorted faces, or even deceased loved ones. While these hallucinations may initially be benign, they can become distressing as the condition progresses.
- Tactile hallucinations, such as the sensation of bugs crawling on the skin (formication), are strongly associated with chronic stimulant use, particularly methamphetamine.

Delusions are another common symptom, with their content often reflecting the neurochemical effects of the drug

- Persecutory delusions (e.g., beliefs of being watched or followed) are prevalent in stimulant-induced psychosis and are frequently accompanied by heightened anxiety or hypervigilance.
- Grandiose delusions, such as exaggerated self-importance or extraordinary abilities, are characteristic of corticosteroid-induced psychosis and may coexist with manic-like behavior.
- Paranoid delusions, including irrational fears of being poisoned or harmed, are commonly seen with dopaminergic agents and anticholinergic drugs.

Agitation and Behavioral Changes are particularly pronounced in psychoses caused by stimulants, corticosteroids, or withdrawal from sedative-hypnotics. Patients may display impulsive or aggressive behavior, restlessness, and a decreased need for sleep. These symptoms can escalate in overstimulating environments, increasing the risk of self-harm or harm to others.

Disorganized Thinking and Behavior is frequently observed and may manifest as incoherent speech, tangential or circumstantial reasoning, or bizarre and unpredictable actions. For example, anticholinergic-induced psychosis often involves profound confusion and wandering behaviors, reflecting significant cognitive disruption. In severe cases, patients may engage in dangerous activities due to impaired judgment and loss of reality testing.

Case Studies of Medication-Induced Psychosis

Medication-induced psychosis is a multifaceted condition that varies significantly in presentation based on the type of medication, dosage, and patient-specific factors. The following case studies illustrate the variability of symptoms, onset, and outcomes associated with different drug classes.

Each case highlights the importance of accurate diagnosis and appropriate management.

Neuropsychiatric Effects of Trimethoprim-Sulfamethoxazole

A rare case study by Omri *et al.* (2024) [1] demonstrated the potential for trimethoprim-sulfamethoxazole, a commonly prescribed antibiotic, to induce acute psychosis. In this case, an immunocompetent patient developed vivid hallucinations and paranoid delusions shortly after starting the medication. The symptoms resolved entirely upon drug discontinuation. This study suggested two potential mechanisms: the drug's interference with folate metabolism in the central nervous system, which disrupts neurotransmitter synthesis, and neuroinflammation triggered by the medication.

The findings emphasize the importance of considering medication as a possible cause in new-onset psychosis, particularly when the patient lacks a psychiatric history. Misdiagnosis could result in unnecessary psychiatric treatment, delaying appropriate care. Future research should focus on the prevalence of these adverse effects and identify risk factors such as genetic predispositions or underlying metabolic vulnerabilities.

Steroid-Induced Psychosis in Oncology Patients

Álvarez *et al.* (2024) [2] detailed a case of steroid-induced psychosis in a patient undergoing high-dose corticosteroid therapy for mediastinal lymphoma. The patient exhibited paranoid delusions and hallucinations, which resolved following a gradual reduction in the steroid dose. This study linked the psychosis to glucocorticoid receptor overactivation, which dysregulates dopamine and serotonin signaling in the brain. High doses of corticosteroids (>40 mg prednisone equivalents daily) were particularly implicated in triggering these symptoms. This research reinforces the dose-dependent nature of steroid-induced psychosis and highlights the importance of monitoring patients closely, especially during high-dose therapy. It also underscores the need for patient and caregiver education to identify early neuropsychiatric symptoms. Exploring safer anti-inflammatory options, such as biologics or JAK inhibitors, could reduce reliance on high-dose corticosteroids and mitigate these risks.

A 45-year-old woman with systemic lupus erythematosus was prescribed high-dose prednisone (60 mg/day) for a lupus flare. Within one week, she developed grandiose delusions, believing she had been chosen to lead a global peace initiative. Her family noted increased energy, decreased need for sleep, and reckless spending behavior. She denied any history of psychiatric illness or prior episodes of mania. The prednisone dose was tapered gradually, and the patient was closely monitored for symptom improvement. Within two weeks of dose reduction, her psychotic symptoms resolved completely, with no further episodes reported on lower maintenance doses (10 mg/day) (Dubovsky *et al.*, 2012) [10]. This case highlights the manic and psychotic effects of corticosteroids, which are dose-dependent and often emerge within the first two weeks of treatment. High doses (>40 mg/day of prednisone or equivalent) are a known risk factor for steroid-induced psychosis. Studies indicate a 1.8-6% prevalence of psychiatric symptoms, including psychosis, in patients receiving corticosteroid therapy (Dubovsky *et al.*, 2012) [10].

Elliott *et al.* (2024) [7] examined the effects of high-dose corticosteroids on pediatric oncology patients, documenting mood instability and psychosis as significant side effects. The study found that children were particularly vulnerable to these effects due to the developing central nervous system and hormonal systems. Symptoms included hallucinations and severe mood disturbances, which necessitated dose adjustments and close monitoring. The findings emphasize the need for tailored dosing protocols and vigilant monitoring in pediatric patients receiving corticosteroids. Caregivers should be educated to recognize early signs of psychosis to facilitate prompt intervention. Future research should explore age-specific risk factors and alternative anti-inflammatory agents that are safer for children.

Dopaminergic Dysregulation in Psychosis

Andreou *et al.* (2024) [3] investigated the role of dopaminergic dysregulation in medication-induced psychosis by examining structural abnormalities in the caudate nucleus of patients. They found significant overlap in the neural pathways affected in both primary psychosis and psychosis induced by medications such as dopaminergic agents. The study highlighted overactivation of the mesolimbic system as a shared mechanism underlying psychosis, with structural changes in the caudate nucleus correlating with symptom severity. These findings have critical implications for the management of psychosis in patients receiving dopaminergic medications, such as those with Parkinson's disease. They suggest that selective dopamine receptor targeting could mitigate psychosis risk while maintaining therapeutic efficacy for motor symptoms. Future research should focus on developing dopamine receptor-specific drugs and using neuroimaging biomarkers to identify early signs of dopaminergic dysregulation.

A 68-year-old man with Parkinson's disease on levodopa therapy began experiencing visual hallucinations of his deceased wife. Initially benign, these hallucinations became distressing over time, accompanied by paranoid delusions that his neighbors were attempting to poison him. The symptoms developed gradually after a dose increase to manage worsening motor symptoms. The levodopa dose was reduced, which alleviated the psychotic symptoms but resulted in mild worsening of motor function. The patient was subsequently started on quetiapine, an antipsychotic with minimal dopaminergic blockade, which provided further symptom relief without significant motor side effects (Aarsland *et al.* 2007) [11]. Psychosis in Parkinson's disease is often related to excessive dopaminergic stimulation from medications. Hallucinations, particularly visual, are common and may progress to paranoia and delusions if untreated. A study by Aarsland *et al.* (2007) [11] found that up to 50% of Parkinson's patients on dopaminergic therapy develop psychotic symptoms, underscoring the importance of balancing symptom management with medication side effects.

Tardive Dyskinesia and Psychotic Symptoms

Besag *et al.* (2024) [4] explored the connection between tardive dyskinesia (TD) and psychosis in patients with chronic mental illness treated with long-term antipsychotics. The study revealed a significant correlation between the duration of antipsychotic exposure and the risk of developing both TD and psychotic symptoms. Patients with TD were more likely to experience worsening psychosis,

suggesting a shared underlying mechanism involving dopamine receptor hypersensitivity. This study underscores the importance of regular monitoring for motor and psychotic symptoms in patients on long-term antipsychotic therapy. Early detection allows for timely adjustments to medication regimens or the introduction of adjunct treatments. Research into alternative antipsychotics with reduced dopamine receptor antagonism could help minimize these risks in long-term treatment.

Lifestyle Interventions for Medication-Induced Weight Gain: Hui *et al.* (2021) [5] developed a lifestyle intervention program to address weight gain associated with psychosis-inducing medications, such as antipsychotics and corticosteroids. The program included behavioral counseling, dietary modifications, and physical activity recommendations. Participants showed significant reductions in weight gain and improvements in metabolic profiles, which are critical given the link between metabolic syndrome and secondary psychotic symptoms. This study highlights the value of integrating non-pharmacological strategies into the management of medication-induced psychosis. Addressing metabolic side effects can improve overall health outcomes and reduce the risk of secondary psychiatric complications. Larger studies are needed to validate these findings and explore the use of digital tools, such as wearable devices, to enhance compliance and monitoring.

Hydralazine-Induced Psychosis in Transplant Patients
Dogrul *et al.* (2024) [6] reported on a kidney transplant patient who developed visual and auditory hallucinations attributed to hydralazine therapy. Symptoms resolved upon drug discontinuation, implicating hydralazine as the cause. The study suggested that polypharmacy and altered drug metabolism in transplant patients heightened their vulnerability to neuropsychiatric side effects. This case underscores the importance of vigilant monitoring for neuropsychiatric symptoms in transplant patients, particularly when using medications like hydralazine. Clinicians should consider alternative vasodilators with lower psychosis risks for this population. Future studies should investigate the prevalence of hydralazine-induced psychosis and identify safer antihypertensive options for transplant recipients.

Stimulant-Induced Psychosis
A 32-year-old man with no history of psychiatric illness presented to the emergency department with paranoia and auditory hallucinations. He reported hearing voices accusing him of being a thief and believed his neighbors were spying on him through hidden cameras. The symptoms began after three days of continuous methamphetamine use, during which he consumed progressively higher doses to maintain euphoria. On examination, the patient was hypervigilant, agitated, and unable to sit still due to his fear of surveillance. Physical findings included dilated pupils, tachycardia, and sweating. The patient was treated with supportive care, including hydration, a quiet environment, and reassurance. Benzodiazepines were administered to manage agitation. The symptoms resolved within 48 hours of abstinence, and the patient was referred to a substance use counseling program for long-term management (Glasner-Edwards & Mooney, 2014) [8]. This case demonstrates the acute onset of psychosis due to stimulant use, characterized by persecutory delusions and hallucinations. The dopaminergic surge caused by methamphetamine is the primary mechanism underlying the psychosis. Studies suggest that up to 40-50% of chronic methamphetamine users experience psychosis, with higher doses and prolonged use increasing the risk.

Anticholinergic-Induced Psychosis
A 70-year-old woman with a history of insomnia and anxiety presented with confusion, paranoia, and visual hallucinations of “tiny creatures” crawling on her walls. Her family reported that she had been taking increasing doses of over-the-counter diphenhydramine (up to 150 mg daily) for the past month to help with sleep. On physical examination, she was disoriented to time and place, with dilated pupils, dry mucous membranes, and tachycardia. The patient was diagnosed with anticholinergic toxicity and treated with supportive care and the administration of physostigmine, a cholinesterase inhibitor. Within hours, her confusion and hallucinations improved significantly. Diphenhydramine was discontinued, and she was advised on safer alternatives for managing insomnia (Tune, 2001) [13], this case illustrates the hallucinatory and cognitive effects of anticholinergic toxicity, a condition often seen in older adults due to age-related reductions in acetylcholine reserves. Visual hallucinations and paranoia are hallmark features, with systemic signs such as dry mouth and tachycardia providing diagnostic clues. According to Tune (2001) [13], medications with anticholinergic properties are a frequent cause of delirium and psychosis in elderly populations.

Antidepressant-Induced Psychosis
A 29-year-old woman with a history of major depressive disorder was prescribed an SSRI (fluoxetine) for worsening depressive symptoms. After three weeks, she developed agitation, confusion, and auditory hallucinations. She also exhibited hyperreflexia and clonus on physical examination. These symptoms were consistent with serotonin syndrome. Fluoxetine was discontinued immediately, and the patient was treated with cyproheptadine, a serotonin antagonist. Her symptoms resolved within 48 hours, and she was transitioned to a different class of antidepressants under close monitoring (Boyer and Shannon, 2005) [12]. This case highlights the risk of serotonin syndrome, a potentially life-threatening condition characterized by excess serotonergic activity. While rare, psychotic symptoms such as hallucinations and confusion can be prominent features. According to Boyer and Shannon (2005) [12], serotonin syndrome is most commonly triggered by high doses, polypharmacy, or interactions with other serotonergic drugs.

Management Strategies for Medication-Induced Psychosis
The management of medication-induced psychosis requires a comprehensive, multi-faceted approach that addresses acute symptoms, prevents recurrence, and promotes long-term patient well-being. Interventions must be tailored to the causative medication, the severity of the psychosis, and the individual needs of the patient. Immediate stabilization is crucial, followed by structured strategies to reduce the risk of recurrence and provide ongoing support. Collaborative care between specialists further enhances outcomes.

Immediate Interventions

The initial step in managing medication-induced psychosis is to stabilize the patient and address the underlying cause. Timely identification and intervention are critical to prevent harm to the patient or others.

Discontinuation or Tapering of the Offending Medication: Identifying and discontinuing the causative medication is the cornerstone of treatment. In many cases, symptoms resolve promptly after cessation of the drug. However, abrupt discontinuation may not always be feasible, particularly for medications like corticosteroids or dopaminergic agents, which can cause withdrawal effects or exacerbate underlying conditions. In such cases, a gradual tapering schedule is recommended to minimize risks. For example:

- **Corticosteroids**: High doses should be tapered cautiously to prevent adrenal insufficiency while reducing the psychotic symptoms.
- **Dopaminergic agents**: In Parkinson's disease, lowering doses of levodopa or dopamine agonists can help alleviate psychosis but must be balanced against the risk of worsening motor symptoms.

Substitution with a safer alternative is often an effective strategy. For instance, replacing anticholinergic drugs with non-anticholinergic alternatives can help prevent recurrence in elderly patients prone to anticholinergic toxicity.

Use of Antipsychotics or Sedatives

In cases of severe psychosis, pharmacological management may be necessary to stabilize the patient. Antipsychotic medications, particularly atypical agents like quetiapine, olanzapine, or risperidone, are preferred for their efficacy in treating hallucinations and delusions. These agents are often chosen based on the underlying condition and the need to avoid side effects. For instance:

- Quetiapine or clozapine are preferred in Parkinson's disease to minimize the impact on motor function.
- In stimulant-induced psychosis, antipsychotics can quickly alleviate paranoia and hallucinations.

Benzodiazepines, such as lorazepam or diazepam, are effective for calming severe agitation or managing anxiety, especially in stimulant- or withdrawal-induced psychosis. In anticholinergic toxicity, physostigmine (a cholinesterase inhibitor) may be used in severe cases to reverse psychotic symptoms and cognitive impairment.

Hospitalization for Severe Cases

Patients exhibiting violent behavior, suicidal ideation, or severe distress may require inpatient care for close monitoring. A calm, low-stimulation environment can aid in reducing agitation, while emergency care teams address acute symptoms. Physical restraints should be used only as a last resort in cases where there is an immediate threat to the patient or others.

Long-Term Management

Once the acute episode has resolved, the focus shifts to preventing recurrence, educating the patient and caregivers, and addressing underlying vulnerabilities that may predispose the patient to future episodes of psychosis.

Monitoring for Recurrence: Patients who have experienced medication-induced psychosis are at higher risk of recurrence if re-exposed to the same or similar drugs. Regular follow-ups are essential to monitor for early signs of psychosis, particularly during high-risk periods such as during corticosteroid or stimulant therapy. Prophylactic strategies, such as the use of mood stabilizers like lithium or valproate, may be considered for patients requiring medications with a known risk of inducing psychosis.

Clinicians should regularly review medication regimens to identify potential interactions or dose-dependent risks. Reducing polypharmacy, especially in elderly patients, can significantly lower the likelihood of psychosis. Monitoring plans should include assessments of mental health, adherence to medications, and potential side effects.

Patient Education and Counseling:

Education plays a central role in empowering patients and caregivers to recognize early warning signs of psychosis and take preventive measures. Patients should be informed about the causative role of certain medications and the importance of adhering to prescribed regimens. Key points to discuss include:

- Avoiding self-medication and over-the-counter drugs with similar properties (e.g., anticholinergics).
- Reporting any new or worsening symptoms immediately.
- Maintaining regular follow-up visits to monitor mental health.

Caregiver involvement is particularly valuable in patients with cognitive impairments or those unable to independently manage their medication regimens. Behavioral counseling or psychotherapy may also help patients cope with the psychological impact of experiencing a psychotic episode.

Addressing Underlying Risk Factors:

Patients with predisposing conditions, such as psychiatric disorders, substance use disorders, or cognitive decline, require additional interventions to minimize future risk. For example:

- Behavioral therapy, such as cognitive-behavioral therapy (CBT), can help patients with substance use disorders avoid triggers for psychosis.
- Patients with bipolar disorder may require mood stabilizers to prevent manic episodes triggered by medications like corticosteroids or antidepressants.

Lifestyle modifications, such as maintaining a regular sleep schedule, engaging in physical activity, and reducing stress, can also support long-term mental health and reduce the risk of psychosis.

Interdisciplinary Approaches

Medication-induced psychosis is a complex condition that benefits from collaboration among healthcare providers across multiple disciplines. An interdisciplinary approach ensures comprehensive care tailored to the patient's needs.

Psychiatrists are integral to diagnosing and treating psychotic symptoms. They oversee the use of antipsychotics or sedatives, provide psychotherapy when needed, and address co-occurring psychiatric conditions. Their expertise is particularly crucial in patients with recurrent psychosis or a history of psychiatric disorders.

Clinical pharmacists play a vital role in identifying drug interactions, reviewing medication regimens for psychosis-inducing agents, and recommending safer alternatives. Their input is invaluable in cases involving polypharmacy or

complex dosing schedules, such as in elderly patients or those with chronic conditions.

Primary care providers (PCPs) are often the first to recognize medication-induced psychosis and coordinate care among specialists. They are responsible for managing chronic conditions requiring psychosis-inducing medications, such as corticosteroid-treated autoimmune diseases or Parkinson's disease. PCPs also monitor long-term health and provide continuity of care, ensuring that patients adhere to treatment plans and follow-up visits.

Future Directions in Medication-Induced Psychosis

Medication-induced psychosis presents significant challenges that demand targeted research, innovative pharmacological solutions, and ethical frameworks for optimal patient care. While advancements have improved our understanding and management of this condition, significant gaps remain in identifying, predicting, and mitigating its occurrence. Future efforts should prioritize addressing these challenges to improve outcomes for vulnerable populations.

Addressing Research Gaps

Significant gaps in the research landscape hinder comprehensive understanding and prevention of medication-induced psychosis. One of the most pressing needs is for longitudinal studies to explore the long-term trajectory of psychosis following medication exposure. Such studies could clarify whether symptoms resolve completely, persist, or recur after subsequent drug exposures. They would also provide insight into the cumulative effects of prolonged or repeated exposure to psychosis-inducing medications.

Another critical area of research is the development of biomarkers to identify individuals at higher risk. Current diagnostic methods rely on clinical observation and patient history, which often fail to detect subtle predispositions. Genetic studies focusing on polymorphisms in dopamine and serotonin receptor genes, as well as neuroimaging approaches using functional MRI or PET scans, could help predict susceptibility. Similarly, the identification of inflammatory or metabolic markers might provide a systemic perspective on psychosis risk. These advancements would enable preemptive screening and more personalized treatment strategies.

Advances in Pharmacology

The development of safer medications and precise prescribing practices is key to reducing the burden of medication-induced psychosis. For many conditions, such as Parkinson's disease or autoimmune disorders, medications like dopaminergic agents and corticosteroids are indispensable. However, ongoing efforts to design drugs with reduced psychosis risk could improve patient safety without compromising efficacy.

Future drugs should prioritize targeted action to minimize effects on pathways linked to psychosis. For instance:

- Dopaminergic agents with selective action on motor pathways and extended-release formulations could reduce the likelihood of psychotic symptoms in Parkinson's patients.
- Corticosteroid alternatives, such as biologics or JAK inhibitors, could offer anti-inflammatory benefits without the neuropsychiatric side effects associated with high-dose steroids.
- Anticholinergic medications with minimal CNS penetration could address peripheral conditions without inducing cognitive or psychiatric side effects.

Advances in genetic testing and artificial intelligence (AI) could allow clinicians to tailor drug regimens to individual risk profiles. AI-driven analysis of clinical and pharmacological data may predict psychosis risk, enabling safer prescribing practices.

Prophylactic use of antipsychotics, such as low-dose quetiapine or clozapine, may help mitigate psychosis risk in high-risk populations, such as Parkinson's patients requiring high-dose dopaminergic therapy. This approach represents a cost-effective strategy to reduce psychosis incidence while leveraging well-established safety profiles.

Integration of Interdisciplinary Care

Medication-induced psychosis is best managed through a collaborative approach involving psychiatrists, pharmacologists, primary care providers, and disease-specific specialists. Psychiatrists provide expertise in managing psychotic symptoms, while pharmacologists optimize medication regimens to minimize risk. Primary care providers monitor chronic conditions and coordinate care, ensuring alignment among specialists.

Tailored care plans developed by interdisciplinary teams can address individual patient needs. For example, in Parkinson's patients experiencing dopaminergic psychosis, neurologists may adjust medication regimens while psychiatrists manage psychotic symptoms using low-impact antipsychotics. Similarly, pharmacists can review complex drug regimens in elderly patients to reduce cumulative risks associated with polypharmacy.

Conclusion

Medication-induced psychosis highlights the complex relationship between pharmacological agents and brain function, with implications that extend beyond psychiatry into broader medical fields. Timely recognition and intervention are critical to minimizing morbidity and improving outcomes. This review explored the pathophysiology, clinical presentations, and diagnostic challenges of the condition, emphasizing the roles of neurotransmitter imbalances, medication dosages, and individual susceptibility. Differentiating medication-induced psychosis from primary psychotic disorders requires clinical vigilance and comprehensive patient histories.

Management focuses on discontinuing the offending medication and using antipsychotics as needed, along with preventive strategies like patient education. However, the lack of predictive biomarkers and robust longitudinal studies underscores the need for further research. Future advancements in pharmacogenomics, drug design, and early detection may enable personalized strategies to reduce risk.

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