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What Should a Cardiologist Know About Psychiatric Medications? A Narrative Review of Cardiovascular Safety, Monitoring, and Clinical Management

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Abstract

Background: Psychotropic medications including antipsychotics and antidepressants, are among the most commonly prescribed medications. Despite the use of newer-generation agents, cardiovascular (CV) side effects still affect patients using these medications. They are widely used for the treatment of bipolar disorder, major depressive disorder, anxiety disorders, schizophrenia and many other psychiatric conditions. Many of these medications are associated with CV adverse effects ranging from mild orthostatic hypotension to life-threatening arrhythmias, myocarditis, and metabolic complications. Currently, There are more people and more indications for using antipsychotics, so, cardiovascular side effects should be studied properly.

Objective: The aim of this review is to summarizes the current evidence regarding the CV side effects of antipsychotic and antidepressant medications, explores the underlying pathophysiological mechanisms, compares the CV safety profiles of different drug classes, and discusses practical strategies for CV risk assessment, monitoring, and clinical management.

Methods: We conducted literature review by searching major biomedical databases, including PubMed, Scopus and Google Scholar, for relevant articles related to our topic, including recent articles. Original studies, systematic reviews, meta-analyses and clinical practice guidelines addressing CV complications associated with psychotropic drugs were reviewed.

Results: Cardiovascular adverse effects vary considerably among psychotropic medications. Antipsychotic drugs are associated with QT interval prolongation, torsades de pointes ventricular arrhythmias, metabolic syndrome, orthostatic hypotension, myocarditis and cardiomyopathy. Antidepressants have class-specific CV effects especially conduction abnormalities with tricyclic antidepressants. Selective serotonin reuptake inhibitors have a relatively favourable CV profile. The risk of adverse cardiovascular outcomes was influenced by several factors including patient-related factors, concomitant medications, electrolyte abnormalities, and presence of CV disease.

Conclusion: In order to minimize CV morbidity and mortality, psychotropic medications should be carefully selected, CV risk assessment should be individualized, appropriate electrocardiographic, metabolic monitoring, and early recognition of adverse effects are essential. Collaboration between psychiatrists, cardiologists, primary care physicians, and clinical pharmacists is essential for optimizing treatment outcomes and maintaining CV safety.

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Keywords: Antipsychotics; Antidepressants; Cardiovascular toxicity; QT prolongation; Arrhythmias; Metabolic syndrome; Electrocardiography; Cardiovascular monitoring

1. Introduction

Psychotropic medications are among the most frequently prescribed medications worldwide and constitute the cornerstone of treatment for many psychiatric disorders, including schizophrenia, bipolar disorder, major depressive disorder, anxiety disorders, obsessive-compulsive disorder, and many neuropsychiatric diseases. During the past two decades, their use has increased substantially due to expanding clinical indications, greater recognition of psychiatric diseases, and better access to mental health care. Although newer psychotropic drugs have generally demonstrated improved neurological tolerability compared with earlier medications, cardiovascular side effects remain an important cause of morbidity and, in some patients, mortality [1].

Cardiovascular (CV) complications associated with psychotropic medications include a broad spectrum of manifestations ranging from relatively benign electrocardiographic (ECG) abnormalities to life-threatening ventricular arrhythmias, myocarditis, cardiomyopathy, venous thromboembolism, orthostatic hypotension, and long-term metabolic disturbances that accelerate atherosclerotic CV disease. Drug-induced corrected QT (QTc) interval prolongation remains one of the most clinically significant adverse effects because it may predispose susceptible individuals to dangerous ventricular arrhythmias and sudden cardiac death. In addition, several psychotropic medications adversely influence body weight, glucose metabolism, lipid homeostasis, and blood pressure, increasing long-term CV risk [2,3].

Antipsychotic medications continue to represent the basis of treatment for schizophrenia and related psychotic diseases. Their indications have increased beyond schizophrenia to include bipolar disorder, treatment-resistant depression, behavioural disturbances associated with dementia, autism spectrum disorders, delirium, and several off-label conditions. Antipsychotics are traditionally classified into first-generation (typical) and second-generation (atypical) agents, each exhibiting distinct pharmacological and CV safety profiles (Table 1). While second-generation antipsychotics generally produce fewer extrapyramidal adverse effects and better treatment adherence, several members of this class are associated with significant metabolic abnormalities and CV complications [4].

Table 1: Classification of Antipsychotic Medications.

First-generation (Typical)	Second-generation (Atypical)
Chlorpromazine	Aripiprazole
Haloperidol	Clozapine
Fluphenazine	Olanzapine
Perphenazine	Quetiapine
Thioridazine	Risperidone
Pimozide	Ziprasidone
Thiothixene	Lurasidone

Similarly, antidepressants are among the most commonly prescribed medications in clinical practice. Their use include major depressive disorder, generalized anxiety disorder, panic disorder, neuropathic pain, fibromyalgia, migraine prophylaxis, and other chronic medical conditions. Although selective serotonin reuptake inhibitors (SSRIs) and other newer antidepressants show favorable CV safety compared with tricyclic antidepressants (TCAs), clinically important adverse effects—including QT prolongation, blood pressure changes, orthostatic hypotension, and clinically significant drug–drug interactions—remain relevant, especially in older adults and patients with pre-existing CV disease [5,6].

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Differences in receptor-binding affinity, ion channel effects, autonomic nervous system, inflammatory pathways, metabolic consequences, and pharmacokinetic interactions affects the cardiovascular safety profile of psychotropic medications. Other factors affecting the likelihood of adverse CV outcomes include patient's age, sex, structural heart disease, electrolyte abnormalities, renal impairment, and use of other QT-prolonging drugs. Therefore, individualized risk assessment before starting treatment and proper CV monitoring during therapy have become essential components of modern psychopharmacology [7,8].

Many clinicians remain unfamiliar with the relative CV safety of individual agents or with current recommendations for CV risk assessment and monitoring, despite increasing awareness of psychotropic-associated CV toxicity. A comprehensive understanding of psychotropic drugs adverse effects is essential for psychiatrists, cardiologists, internists, primary care physicians and clinical pharmacists involved in the care of these patients [7,9].

The aim of this review is to provide a comprehensive and evidence-based overview of the CV effects of antipsychotic and antidepressant medications, emphasizing the underlying mechanisms of CV toxicity, comparative safety profiles of individual drug classes, current recommendations for cardiovascular monitoring, and practical clinical strategies to minimize CV complications without affecting the optimal psychiatric outcomes.

2. Literature Search Methodology

A comprehensive narrative literature review was conducted to identify published evidence regarding the CV effects of antipsychotic and antidepressant medications. The literature search was done using the electronic databases PubMed/MEDLINE, Scopus, and Google Scholar. The priority was to include articles published between January 2015 and 2025, while landmark studies and essential guideline documents published before 2015 were included when considered essential for clinical relevance [10,11].

Recent guidelines issued by the European Society of Cardiology (ESC), American Psychiatric Association (APA), American Heart Association (AHA), British Heart Rhythm Society (BHRS), and other internationally recognized organizations were preferentially included to ensure that recommendations reflected current clinical practice. Priority was also given to systematic reviews, meta-analyses, randomized controlled trials, and large observational cohort studies published in peer-reviewed journals [12,13].

Studies were included if they evaluated CV outcomes associated with antipsychotic or antidepressant medications in adult populations or provided clinically relevant mechanistic, pharmacological, or monitoring data. Articles addressing QT interval prolongation, ventricular arrhythmias, myocarditis, cardiomyopathy, orthostatic hypotension, venous thromboembolism, metabolic complications, hypertension, heart failure, or CV mortality were included [14,15].

The retrieved literature was critically appraised according to study design, sample size, methodological quality, consistency of findings, and clinical applicability. When available, evidence from systematic reviews, meta-analyses, and international clinical guidelines was prioritized over individual observational studies. Areas in which evidence remained limited were added to show knowledge gaps and future research priorities [16].

Registration in the International Prospective Register of Systematic Reviews (PROSPERO) and reporting according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement were not required for this review design, because this review was conducted as a narrative review and therefore did not involve formal quantitative synthesis or meta-analysis [11,16].

3. Mechanisms of Cardiovascular Toxicity of Psychotropic Medications

Psychotropic medications exert CV effects through many mechanisms involving cardiac ion channels, autonomic nervous system modulation, inflammatory pathways, endothelial dysfunction, pharmacokinetic interactions, and metabolic alterations. These mechanisms differ among individual drug classes and even among agents within the same class, accounting for the heterogeneity of CV adverse effects observed in clinical practice. While some CV complications occur shortly after treatment initiation, others develop gradually during long-term therapy as a consequence of chronic myocardial injury or cumulative metabolic disturbances.

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Understanding these mechanisms is essential for identifying high-risk patients, selecting appropriate psychotropic agents, and implementing effective CV monitoring strategies [17,18].

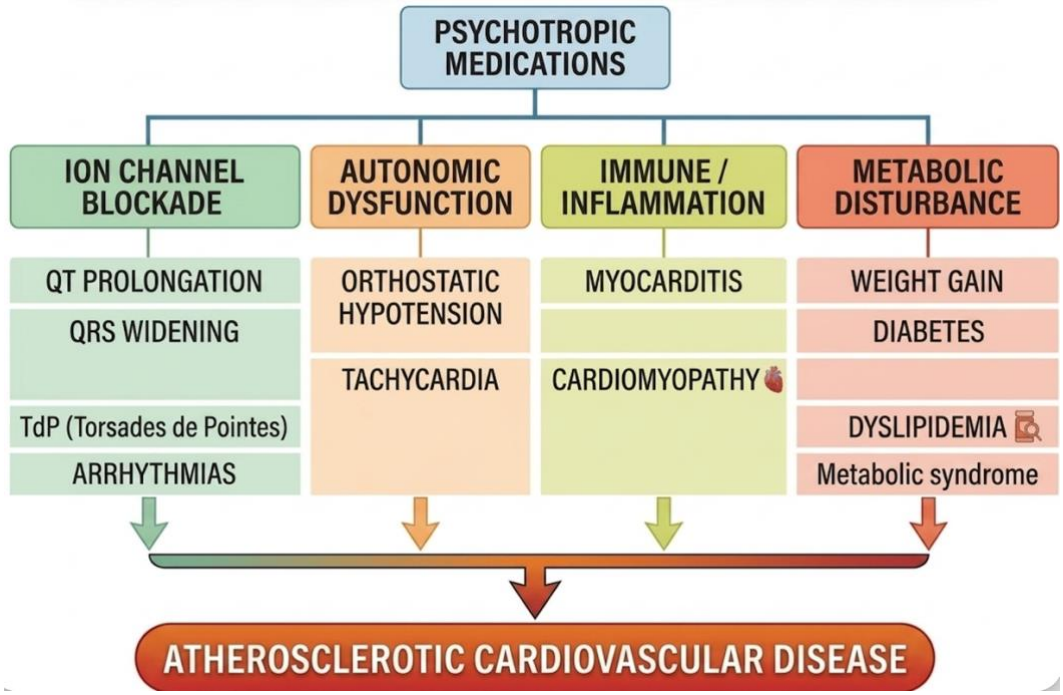

Figure 1: Mechanisms of Cardiovascular Toxicity of Psychotropic Medications.

3.1 Electrophysiological Mechanisms

Drug-induced cardiac arrhythmias represent one of the most clinically significant CV complications of psychotropic medications. The main electrophysiological mechanism involves inhibition of the rapid component of the delayed rectifier potassium current (IKr), mediated by blockade of the human ether-à-go-go-related gene (hERG) potassium channel. This potassium channel plays a critical role in ventricular repolarization, and its inhibition delays phase 3 of the cardiac action potential, resulting in prolongation of the corrected QT (QTc) interval on the electrocardiogram [19-21].

Marked QT prolongation increases susceptibility to early after-depolarizations, which may initiate torsades de pointes (TdP), a potentially fatal polymorphic ventricular tachycardia capable of degenerating into ventricular fibrillation and sudden cardiac death. Although TdP remains relatively uncommon, its occurrence is strongly influenced by other patient-related risk factors, including female sex, advanced age, electrolyte disturbances, bradycardia, structural heart disease, congenital long QT syndrome, and concomitant use of multiple QT-prolonging medications [22].

Not all psychotropic medications produce equivalent electrophysiological effects. Among antipsychotics, thioridazine, pimozide, ziprasidone, and intravenous haloperidol are associated with a greater risk of QT prolongation, whereas aripiprazole and lurasidone show minimal effects on ventricular repolarization. Among antidepressants, tricyclic antidepressants and high-dose citalopram have the greatest potential for QT prolongation, while sertraline and fluoxetine generally show favorable electrophysiological safety profiles [13,23,24].

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3.2 Autonomic Nervous System Dysfunction

Many psychotropic medications change CV function through their effects on autonomic neurotransmission. Antagonism of peripheral α_1-adrenergic receptors decrease systemic vascular resistance and may produce orthostatic hypotension, particularly during treatment initiation or rapid dose escalation. Patients may present with dizziness, presyncope, syncope, blurred vision or falls, especially older adults and those receiving antihypertensive medications [25].

Conversely, medications having significant anticholinergic activity inhibit parasympathetic cardiac regulation, resulting in sinus tachycardia and increased myocardial oxygen demand. Persistent tachycardia may contribute to reduced exercise tolerance and may occasionally precipitate myocardial ischemia in susceptible individuals with underlying coronary artery disease [26].

The severity of autonomic dysfunction varies significantly according to receptor-binding affinity. Clozapine, quetiapine, chlorpromazine and tricyclic antidepressants generally exhibit the greatest propensity to produce orthostatic hypotension, whereas aripiprazole and most SSRIs have relatively limited autonomic CV effects [26,27].

3.3 Myocardial Inflammation and Structural Cardiac Injury

Certain psychotropic medications are capable of inducing direct myocardial injury independent of their electrophysiological effects. Clozapine remains the agent most strongly associated with myocarditis and dilated cardiomyopathy, although these complications are uncommon. Most reported cases occur within the first month of therapy, suggesting an acute immune-mediated mechanism rather than cumulative dose-dependent toxicity [28,29].

The pathogenesis of clozapine-induced myocarditis has not been completely understood. Possible mechanisms include hypersensitivity reactions characterized by eosinophilic myocardial infiltration, excessive cytokine release, mitochondrial dysfunction, oxidative stress and catecholamine-mediated myocardial injury. Genetic susceptibility and inflammatory activation may further contribute to disease development [30].

Clinical manifestations are often nonspecific and may include persistent sinus tachycardia, fever, fatigue, flu-like symptoms chest pain and dyspnea. Rise in cardiac troponin and C-reactive protein frequently precede left ventricular dysfunction, highlighting the importance of early biochemical monitoring in patients receiving clozapine. Rapid diagnosis and immediate drug discontinuation substantially improve prognosis and reduce progression to irreversible heart failure [31].

3.4 Metabolic Mechanisms and Atherosclerotic Cardiovascular Disease

In addition to acute cardiac toxicity, several psychotropic medications contribute to CV disease by promoting adverse metabolic changes. Second-generation antipsychotics, particularly clozapine and olanzapine, have been strongly associated with central obesity, weight gain, insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus, dyslipidemia, and metabolic syndrome. These abnormalities substantially increase long-term CV risk through acceleration of atherosclerotic disease [32].

The mechanisms responsible for these metabolic disturbances are multifactorial and include antagonism of histamine H_1 receptors, muscarinic M_3 receptors, serotonin 5-HT_2C receptors, and dopamine receptors, resulting in increased appetite, reduced satiety, altered energy expenditure, and impaired insulin secretion. Chronic systemic inflammation and adipokine dysregulation may also contribute to endothelial dysfunction and vascular injury [33].

Routine monitoring of body weight, waist circumference, fasting plasma glucose (or HbA1c), lipid profile, and blood pressure is essential during long-term psychotropic therapy because CV mortality among patients with severe mental illness is largely attributable to preventable metabolic disease [34].

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4. Cardiovascular Effects of Antipsychotic Medications

Treatment for schizophrenia and other psychotic disorders is mainly based on antipsychotic medications, so, they are increasingly prescribed for bipolar disorder, treatment-resistant depression, autism spectrum disorders, delirium, behavioral disturbances associated with dementia, and many off-label indications. Although these agents have substantially improved psychiatric outcomes, their use has been associated with a wide range of CV side effects that lead to morbidity and mortality. The nature and severity of CV toxicity differ considerably among individual antipsychotic agents, emphasizing the importance of individualized treatment selection based on both psychiatric efficacy and CV risk profile [35].

4.1 QT Interval Prolongation and Torsades de Pointes

QT interval prolongation is the most extensively recognized CV adverse effect associated with antipsychotic therapy. The risk of drug-induced QT prolongation varies significantly among antipsychotic agents. Thioridazine and pimozide have consistently demonstrated the greatest arrhythmogenic potential and are therefore used infrequently in clinical practice. Intravenous haloperidol has also been associated with clinically significant QT prolongation, particularly in critically ill patients receiving high cumulative doses or concomitant QT-prolonging medications. Among second-generation antipsychotics, ziprasidone produces greater QT prolongation, whereas aripiprazole and lurasidone exhibit minimal effects on ventricular repolarization and are generally considered the preferred agents in patients at increased arrhythmic risk [36,37].

The clinical significance of QT prolongation depends on the administered drug and also on patient-related factors. Female sex, advanced age, electrolyte abnormalities, structural heart disease, renal impairment, bradycardia, congenital long QT syndrome, and concomitant administration of multiple QT-prolonging medications substantially increase the likelihood of dangerous ventricular arrhythmias. Consequently, assessment of these risk factors before initiating antipsychotic therapy is essential [23].

4.2 Sudden Cardiac Death

An increased incidence of sudden cardiac death among patients receiving antipsychotic medications compared with the general population was shown in many large epidemiological studies. This risk is multifactorial and reflects the combined effects of drug-induced ventricular arrhythmias, metabolic abnormalities, structural CV disease, cigarette smoking, diabetes mellitus, and lifestyle-related CV risk factors that are highly prevalent among individuals with severe mental illness.

Importantly, observational studies have demonstrated a dose-dependent relationship between antipsychotic exposure and sudden cardiac death, especially among patients receiving high-potency first-generation antipsychotics or medications associated with significant QT prolongation. But the absolute risk remains relatively low, and the benefits of antipsychotic therapy generally outweigh the CV risks when patients are appropriately selected and monitored [38,39].

4.3 Orthostatic Hypotension

Orthostatic hypotension is a common side effect of many antipsychotic medications and mainly results from antagonism of peripheral α_1-adrenergic receptors. The incidence is highest during treatment initiation and rapid dose escalation and may be increased by dehydration, advanced age, autonomic dysfunction, or concomitant antihypertensive therapy [25,40].

Clinically, orthostatic hypotension may manifest as dizziness, blurred vision, weakness, presyncope, syncope, or falls, potentially resulting in fractures and other traumatic injuries. Low-potency first-generation antipsychotics, clozapine, and quetiapine demonstrate the greatest propensity to produce symptomatic hypotension, whereas aripiprazole and lurasidone generally exhibit more favorable hemodynamic profiles. Gradual dose titration and careful monitoring during treatment initiation reduce the incidence of clinically significant hypotension [41].

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4.4 Tachycardia and Other Heart Rate Abnormalities

Persistent sinus tachycardia frequently occurs during treatment with antipsychotic medications having significant anticholinergic activity, especially clozapine. Although tachycardia is often benign and transient, persistent elevations in heart rate increase myocardial oxygen consumption and may increase myocardial ischemia in susceptible patients with underlying coronary artery disease or heart failure [42].

Less commonly, antipsychotic medications may contribute to clinically significant brady-arrhythmias through changes in conduction system effects or autonomic regulation, although these events remain considerably less frequent than tachyarrhythmias [35].

4.5 Clozapine-Associated Myocarditis and Cardiomyopathy

Clozapine has the strongest association with myocarditis and dilated cardiomyopathy among all currently available antipsychotics. Clozapine-induced myocarditis most commonly develops during the first two to four weeks of therapy and remains one of the most serious adverse reactions limiting its use despite its superior efficacy in schizophrenia resistant to treatment [28,29].

Clinical manifestations are often nonspecific and include persistent tachycardia, chest pain, fever, malaise, dyspnea and flu-like symptoms. Laboratory abnormalities, particularly elevated cardiac troponin and C-reactive protein concentrations, frequently precede overt ventricular dysfunction and facilitate early diagnosis. In more advanced cases, echocardiography typically demonstrates impaired left ventricular systolic function [30,43].

Early recognition and immediate discontinuation of clozapine remain the mainstay of management and are associated with favorable recovery in most patients. Delayed diagnosis may result in progressive heart failure, cardiogenic shock, or even death [30].

4.6 Venous Thromboembolism

Accumulating evidence indicates that antipsychotic therapy is associated with an increased risk of venous thromboembolism (VTE), including deep vein thrombosis and pulmonary embolism. Although the absolute incidence is relatively low, several observational studies and meta-analyses have consistently reported a higher thromboembolic risk among antipsychotic users compared to non-users [44].

The mechanisms responsible for this association are likely multifactorial and include immobility, obesity, metabolic syndrome, endothelial dysfunction, platelet activation, and alterations in coagulation pathways. The highest risk appears to occur during the first three months after starting treatment, particularly among patients with additional predisposing factors such as malignancy, recent surgery, pregnancy, or inherited thrombophilia [45].

Clinicians should maintain a high index of suspicion for pulmonary embolism in patients receiving antipsychotic therapy who develop unexplained dyspnea, pleuritic chest pain, syncope, or unilateral lower-limb swelling [46].

4.7 Metabolic Syndrome and Long-Term Cardiovascular Risk

The introduction of second-generation antipsychotics has significantly reduced the incidence of extrapyramidal adverse effects but has increased concern regarding metabolic complications. Weight gain, insulin resistance, hypertension, dyslipidemia, and type 2 diabetes mellitus are now recognized as major contributors to the excess CV mortality shown among patients with severe mental illness [47,48].

Among currently available antipsychotics, clozapine and olanzapine consistently demonstrate the greatest metabolic liability, whereas aripiprazole, lurasidone, and ziprasidone show comparatively favorable metabolic profiles. These differences should be considered when selecting long-term therapy for patients with obesity, diabetes mellitus, or established CV disease [33,48].

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Routine monitoring of body weight, blood pressure, fasting plasma glucose (or HbA1c), and fasting lipid profile is recommended throughout treatment, particularly during the first year after starting second-generation antipsychotic therapy [49].

4.8 Comparative Cardiovascular Safety of Individual Antipsychotic Agents

The CV safety profiles of antipsychotic medications differ substantially and should influence therapeutic decision-making. Aripiprazole and lurasidone generally have relatively favorable CV profiles, although the specific risk depends on the endpoint considered and on patient characteristics. Conversely, clozapine remains indispensable for treatment-resistant schizophrenia despite its association with myocarditis, orthostatic hypotension and significant metabolic toxicity [37].

Olanzapine shows relatively modest electrophysiological toxicity but is associated with considerable weight gain and metabolic syndrome. Ziprasidone produces greater QT prolongation than most second-generation antipsychotics, while intravenous haloperidol requires careful electrocardiographic monitoring in hospitalized patients receiving high doses or multiple QT-prolonging medications [50].

A comparative summary of the principal CV adverse effects of individual antipsychotic medications is presented in Table 2.

Table 2: Comparative Cardiovascular Risk of Antipsychotic Medications*.

Drug	QT Prolongation	Orthostatic Hypotension	Myocarditis	Metabolic Risk	Overall Cardiovascular Profile
Clozapine	Moderate	High	High	Very high	High
Olanzapine	Low	Moderate	Rare	Very high	Moderate
Quetiapine	Moderate	Moderate	Rare	Moderate	Moderate
Ziprasidone	Higher	Low–moderate	Rare	Low	Moderate
Aripiprazole	Low	Low	Rare	Low	Favorable
Lurasidone	Low	Low	Rare	Low	Favorable
Haloperidol	Dose- and route-dependent	Low–moderate	Rare	Low	Requires risk-based monitoring

* Risk categories are qualitative and should not be interpreted as absolute rates or as a substitute for individual patient assessment. **Aripiprazole and lurasidone** generally have the most favorable cardiovascular profiles, whereas **clozapine** remains indispensable for treatment-resistant schizophrenia despite its higher cardiovascular and metabolic risks.

4.9 Clinical Implications

Selection of antipsychotic therapy should extend beyond psychiatric efficacy to include individualized CV risk assessment. Baseline evaluation should include a detailed CV history, medication review, assessment of CV risk factors, and electrocardiography when clinically indicated. During follow-up, clinicians should monitor blood pressure, heart rate, metabolic parameters, and electrocardiographic changes according to the CV risk profile of the patient and the pharmacological characteristics of the selected antipsychotic drug. Close collaboration between psychiatrists, cardiologists, internists, and primary care physicians is essential to optimize psychiatric treatment while minimizing CV complications [12,49].

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5. Cardiovascular Effects of Antidepressant Medications

Antidepressants are among the most frequently prescribed drugs worldwide and are indicated not only for major depressive disorder but also for anxiety disorders, obsessive-compulsive disorder, post-traumatic stress disorder, chronic pain syndromes, migraine prophylaxis, and several functional neurological disorders. Their CV safety differs considerably according to pharmacological class, receptor affinity, and patient-specific CV risk factors. While SSRIs generally have the most favorable CV safety profile among commonly used antidepressants, treatment should remain individualized in patients with CV disease, tricyclic antidepressants (TCAs) and certain serotonin-norepinephrine reuptake inhibitors (SNRIs) may produce clinically important CV adverse effects requiring careful monitoring [51-53].

5.1 Selective Serotonin Reuptake Inhibitors (SSRIs)

SSRIs are generally considered to have a favorable CV safety profile compared with older antidepressants. Their effects on cardiac conduction and blood pressure are usually limited, although individual agents differ and patient-specific risk factors remain important [53,54].

Among the SSRIs, sertraline has particularly strong clinical experience in patients with coronary artery disease. The SADHART trial found no excess CV safety signal with sertraline in patients with acute myocardial infarction or unstable angina, supporting its use when antidepressant treatment is indicated in this population [55].

Most SSRIs produce little or no clinically significant QT prolongation at therapeutic doses. However, citalopram and, to a lesser extent, escitalopram exhibit dose-dependent QT prolongation. Consequently, regulatory authorities recommend limiting the maximum daily dose of citalopram, particularly in older adults and patients with additional risk factors for QT prolongation [56].

SSRIs may also increase bleeding tendency through inhibition of platelet serotonin uptake, particularly when combined with antiplatelet or anticoagulant therapy. This interaction should be considered when assessing the overall risk-benefit profile in patients with CV disease [57].

5.2 Serotonin–Norepinephrine Reuptake Inhibitors (SNRIs)

SNRIs inhibit the reuptake of both serotonin and norepinephrine and may therefore produce greater noradrenergic cardiovascular effects than SSRIs, particularly changes in blood pressure and heart rate.

Among currently available SNRIs, venlafaxine has been consistently associated with dose-related increases in blood pressure, especially at higher doses. Periodic blood-pressure monitoring is therefore appropriate during treatment [35].

Duloxetine generally has a more limited blood-pressure effect than venlafaxine but should still be used cautiously in patients with uncontrolled hypertension. Overall, SNRIs remain appropriate options for many patients when CV risk factors are monitored [58].

5.3 Tricyclic Antidepressants (TCAs)

Despite their proven efficacy, TCAs have greater CV toxicity than most newer antidepressants and are no longer considered first-line therapy in most patients. Their CV effects include sodium-channel blockade, anticholinergic effects, and α_1-adrenergic antagonism, with important consequences for conduction, heart rate, and blood pressure [51].

Electrocardiographic abnormalities associated with TCAs include PR and QRS prolongation and QT abnormalities, with greater concern in patients with structural heart disease or overdose. Orthostatic hypotension and sinus tachycardia may also occur because of α_1-adrenergic blockade and anticholinergic effects [59].

TCAs should generally be avoided in patients with significant structural heart disease or conduction disease Because of their CV risks, whenever safer therapeutic alternatives are available [60].

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5.4 Monoamine Oxidase Inhibitors (MAOIs)

Although MAOIs are now prescribed relatively infrequently, they can cause clinically important orthostatic hypotension and hypertensive reactions, particularly with dietary tyramine exposure or interacting sympathomimetic medications [61].

Patients receiving MAOIs require detailed education regarding dietary restrictions and potential drug interactions. Careful medication reconciliation is essential before initiating additional pharmacological therapy [62].

5.5 Atypical and Newer Antidepressants

Several newer antidepressants demonstrate favorable cardiovascular safety profiles compared with TCAs.

Bupropion generally has limited effects on cardiac conduction at therapeutic doses, although blood-pressure elevation can occur in susceptible patients; blood pressure should therefore be monitored when clinically appropriate [52].

Mirtazapine generally has limited CV effects at therapeutic doses, although weight gain and increased appetite are important longer-term adverse effects. Evidence regarding QT effects is limited; clinically important QT prolongation appears uncommon at usual doses [13].

Trazodone can cause orthostatic hypotension and should therefore be prescribed cautiously in older adults at increased risk of falls. Its cardiovascular effects should be considered in the context of dose, comorbidity, and concomitant QT-prolonging drugs [13].

Newer antidepressants such as vortioxetine and agomelatine appear to have relatively limited CV effects in clinical studies, although long-term CV outcome data remain limited [63].

5.6 Antidepressants in Patients with Established Cardiovascular Disease

Depression is highly prevalent among patients with coronary artery disease and is associated with adverse CV outcomes and reduced quality of life. Therefore, effective treatment of depression represents an important component of comprehensive CV care.

Sertraline has particularly strong clinical experience in patients with coronary artery disease, including the SADHART trial, and is generally a reasonable SSRI choice when antidepressant treatment is indicated in this population. TCAs should generally be avoided when safer alternatives are available because of their proarrhythmic and hemodynamic effects [64].

When antidepressant therapy is initiated in patients with significant CV disease, clinicians should evaluate baseline CV risk, review concomitant medications for potential interactions, and perform electrocardiographic monitoring when clinically indicated, particularly in patients receiving QT-prolonging agents [65].

5.7 Comparative Cardiovascular Safety of Antidepressant Classes

Overall, SSRIs generally have the most favorable CV safety profile, whereas SNRIs warrant attention to blood pressure and TCAs have greater electrophysiological and hemodynamic toxicity. MAOIs require particular attention to dietary and drug interactions, while several newer antidepressants have relatively limited CV effects. A comparison of the principal CV effects of antidepressant classes is presented in Table 3 [53,54].

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Table 3: Cardiovascular Effects of Antidepressant Classes.

Drug Class	QT Prolongation	Blood Pressure	Orthostatic Hypotension	Arrhythmia Concern	Use in Cardiovascular Disease
SSRIs	Generally low; citalopram/escitalopram dose-dependent	Usually limited	Uncommon	Generally low	Often appropriate; individualize
SNRIs	Generally low	Venlafaxine may increase BP	Uncommon	Generally low	Use with BP monitoring
TCAs	Higher	Variable	More common	Higher	Generally avoid when safer alternatives exist
MAOIs	Generally low	Hypertensive reactions possible	Clinically important	Interaction-dependent	Usually not first-line
Bupropion	Minimal at therapeutic doses	May increase BP	Uncommon	Generally low	Often appropriate with BP monitoring
Mirtazapine	Usually limited; evidence less extensive	Usually limited	Uncommon	Generally low	Often reasonable; monitor metabolic effects
Trazodone	Usually limited at usual doses	Usually limited	Can occur	Generally low at therapeutic doses	Individualize, particularly in older adults

5.8 Clinical Implications

Selection of antidepressant therapy should be individualized according to both psychiatric indication and CV risk profile. In patients with pre-existing CV disease, SSRIs—particularly sertraline—should generally be considered first-line therapy because of their favorable safety profile. Blood pressure monitoring is recommended for patients receiving SNRIs, whereas electrocardiographic surveillance should be considered for patients receiving TCAs or antidepressants associated with QT prolongation, particularly in the presence of additional risk factors. Clinicians should also evaluate potential pharmacokinetic interactions and avoid unnecessary combinations of QT-prolonging medications [13,53,60].

6. Clinical Recommendations for Cardiovascular Risk Assessment and Monitoring

The prescription of psychotropic medications should be based on an individualized benefit-risk assessment that considers both psychiatric efficacy and cardiovascular safety. Although most CV adverse effects are uncommon, they are often predictable and potentially preventable through appropriate patient selection, baseline CV evaluation, and structured follow-up. Close collaboration between psychiatrists, cardiologists, internists, primary care physicians, and clinical pharmacists is essential to optimize treatment outcomes while minimizing cardiovascular complications [12,65].

6.1 Baseline Cardiovascular Risk Assessment

Before initiating antipsychotic or antidepressant therapy, clinicians should perform a comprehensive CV assessment. This evaluation should include a detailed personal and family history of CV disease, previous ventricular arrhythmias, congenital long QT syndrome, unexplained syncope, sudden cardiac death, hypertension, diabetes mellitus, dyslipidemia, obesity, smoking status, chronic kidney disease, and concomitant CV medications. Physical examination should include measurement of blood pressure, heart rate, body weight, body mass index (BMI), and waist circumference [49,65].

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Laboratory evaluation should include fasting plasma glucose (or glycated hemoglobin [HbA1c]), fasting lipid profile, serum electrolytes, renal function, and liver function tests. Correction of reversible electrolyte abnormalities, particularly hypokalemia and hypomagnesemia, should be undertaken before initiating medications associated with QT interval prolongation [66].

Patients with structural heart disease, inherited arrhythmia syndromes, heart failure, previous myocardial infarction, or multiple cardiovascular risk factors should undergo careful risk stratification before selecting psychotropic therapy. Whenever clinically feasible, agents with lower CV risk profiles should be preferred in these populations [60].

6.2 Electrocardiographic Monitoring

Baseline electrocardiography (ECG) is recommended for patients receiving psychotropic medications associated with moderate or high arrhythmogenic potential, particularly those with pre-existing CV disease, previous ventricular arrhythmias, electrolyte abnormalities, or concomitant use of other QT-prolonging medications [67].

Repeat ECG assessment should be considered:

- After initiation of high-risk psychotropic medications;
- Following significant dose escalation;
- After the addition of another QT-prolonging medication;
- Following correction of electrolyte abnormalities; and
- Whenever patients develop syncope, presyncope, palpitations, or unexplained dizziness [68].

A corrected QT interval (QTc) of ≥500 ms or an increase of ≥60 ms from baseline should prompt reassessment of therapy, correction of reversible causes, discontinuation of non-essential QT-prolonging medications, and consideration of switching to an alternative psychotropic agent with a more favorable electrophysiological profile [60].

The proposed approach to cardiovascular assessment and ECG monitoring before initiating psychotropic medications is illustrated in Figure 2.

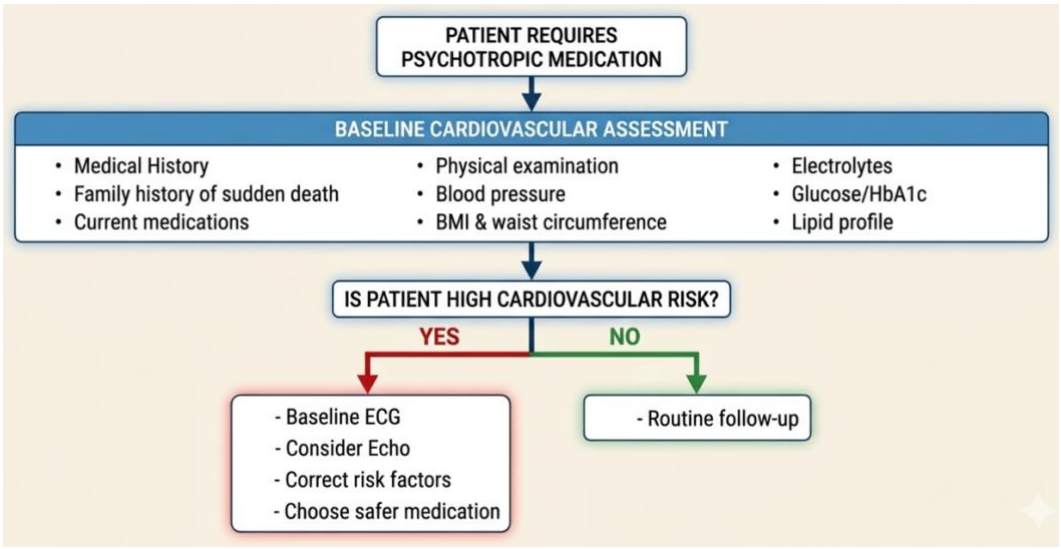

Figure 2: Cardiovascular Assessment Before Starting Psychotropic Therapy.

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6.3 Metabolic Monitoring

Patients receiving second-generation antipsychotics should undergo routine monitoring for metabolic complications because these abnormalities contribute substantially to long-term CV morbidity and mortality. Recommended monitoring includes body weight, BMI, waist circumference, blood pressure, fasting plasma glucose (or HbA1c), and fasting lipid profile. Monitoring should be most intensive during the first year of therapy, when metabolic changes are most likely to develop [12,49].

Patients receiving clozapine or olanzapine require particularly close surveillance because these agents exhibit the greatest propensity to induce weight gain, insulin resistance, dyslipidemia, and metabolic syndrome. Lifestyle interventions, including dietary modification, regular physical activity, smoking cessation, and weight management, should be encouraged throughout treatment [43].

Suggested monitoring intervals are summarized in Table 4.

Table 4: Recommended Cardiovascular Monitoring During Psychotropic Therapy.

Assessment	Baseline	Follow-up
Medical and cardiovascular history	Yes	As clinically indicated
Physical examination	Yes	Every visit / as indicated
Blood pressure	Yes	Periodically and after dose changes when relevant
ECG	For patients/drugs with relevant QT or conduction risk	After dose changes, new symptoms, or when clinically indicated
Weight/BMI	Yes	Frequently during early treatment, then periodically
Waist circumference	When metabolic risk is relevant	Periodically
HbA1c / fasting glucose	When metabolic risk is relevant	At approximately 3 months and periodically thereafter
Lipid profile	When metabolic risk is relevant	At approximately 3 months and periodically thereafter
Electrolytes	When QT risk, relevant comorbidity, or interacting therapy is present	As clinically indicated
Troponin / CRP with clozapine	According to local clozapine monitoring protocol	During early treatment and when symptoms/signals arise

Monitoring intervals should be individualized according to the medication, patient risk factors, local regulatory requirements, and clinical context.

6.4 Prevention and Management of QT Prolongation

The risk of drug-induced QT prolongation can often be minimized by identifying modifiable risk factors before treatment initiation. Clinicians should avoid unnecessary combinations of QT-prolonging medications whenever possible and should carefully review potential pharmacokinetic interactions involving cytochrome P450 inhibitors that may increase psychotropic drug concentrations [69].

When clinically appropriate, psychotropic medications with lower arrhythmogenic potential—such as aripiprazole or lurasidone among antipsychotics and sertraline among antidepressants—should be considered in patients at increased risk of ventricular arrhythmias [68].

Patients who develop torsades de pointes should be managed according to current advanced cardiac life support recommendations, including immediate discontinuation of the offending medication, intravenous magnesium sulfate, correction of electrolyte abnormalities, and temporary pacing or isoproterenol infusion when clinically indicated [70].

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A practical approach to the management of QT prolongation during psychotropic therapy is presented in Figure 3.

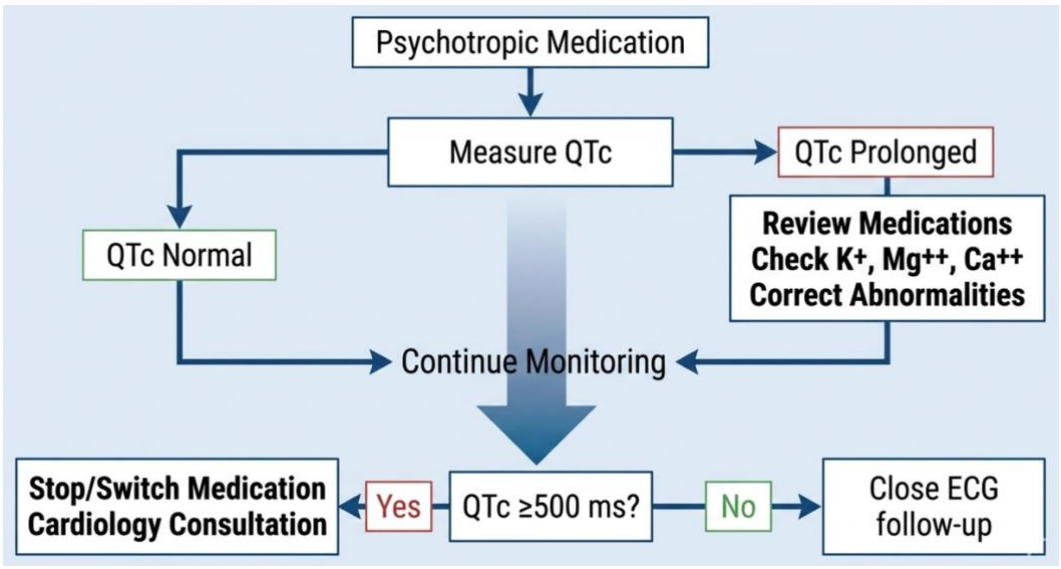

Figure 3: Management of QT Prolongation During Psychotropic Therapy.

6.5 Management of Orthostatic Hypotension

Orthostatic hypotension may frequently be minimized through gradual dose titration, maintenance of adequate hydration, patient education regarding slow positional changes, and review of concomitant antihypertensive medications. Persistent symptomatic hypotension may necessitate dose reduction or substitution with an alternative psychotropic agent exhibiting less α_1-adrenergic receptor antagonism [25].

Older adults require particular attention because orthostatic hypotension significantly increases the risk of falls, fractures, and hospitalization [25].

6.6 Monitoring for Clozapine-Induced Myocarditis

Patients initiated on clozapine should be closely monitored during the first four weeks of therapy, which represents the period of greatest myocarditis risk. Persistent tachycardia, fever, chest discomfort, dyspnea, or unexplained fatigue should prompt immediate clinical evaluation.

Serial measurement of high-sensitivity cardiac troponin and C-reactive protein has been proposed as an effective strategy for early detection of clozapine-associated myocarditis. Echocardiography should be performed when clinical suspicion persists or cardiac biomarkers become elevated. Early recognition and prompt discontinuation of clozapine substantially improve clinical outcomes [28].

6.7 Drug–Drug Interactions

Drug interactions represent an important and potentially preventable cause of CV toxicity during psychotropic therapy. Clinicians should routinely evaluate all prescribed and over-the-counter medications before starting treatment. Particular caution is required when psychotropic medications are combined with Class IA or Class III antiarrhythmic drugs, macrolide antibiotics, fluoroquinolones, azole antifungal agents, methadone, or other medications known to prolong the QT interval.

Pharmacokinetic interactions involving CYP2D6, CYP3A4, CYP2C19, and CYP1A2 inhibitors should also be considered because increased psychotropic drug concentrations may increase CV toxicity [45,69,71].

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6.8 Special Populations

Older adults, patients with structural heart disease, inherited arrhythmia syndromes, chronic kidney disease, hepatic impairment, and individuals receiving multiple QT-prolonging medications require more intensive CV monitoring than the general population. Similarly, patients with obesity, diabetes mellitus, or established metabolic syndrome should preferentially receive psychotropic agents associated with lower metabolic risk whenever clinically appropriate [12,70].

6.9 Practical Clinical Pearls

Based on the available evidence, the following practical recommendations may assist clinicians in minimizing CV complications during psychotropic therapy:

- Perform an individualized CV risk assessment before starting psychotropic medications.
- Obtain a baseline ECG before prescribing psychotropic agents associated with moderate or high QT-prolonging potential.
- Correct electrolyte abnormalities before and during treatment.
- Monitor metabolic parameters regularly in patients receiving second-generation antipsychotics.
- Consider sertraline as the preferred antidepressant in patients with established coronary artery disease.
- Monitor carefully for myocarditis during the first month of clozapine therapy.
- Review concomitant medications routinely to minimize clinically significant drug–drug interactions.

7. Future Perspectives

Despite considerable advances in psychopharmacology, important knowledge gaps remain regarding the long-term CV safety of several newer psychotropic medications. Future prospective studies are needed to clarify the comparative CV risks of individual agents, particularly in patients already having CV disease and multiple comorbidities.

Emerging approaches such as pharmacogenomics, precision medicine, remote monitoring, and digital clinical decision support may eventually facilitate more individualized CV risk assessment in psychopharmacology. These areas should be regarded as promising research directions rather than established clinical standards.

Continued collaboration between psychiatry and cardiology will be essential for improving CV outcomes among patients receiving psychotropic medications and for developing evidence-based multidisciplinary care pathways.

Conclusion

Psychotropic medications remain indispensable for the treatment of numerous psychiatric disorders but are associated with a broad spectrum of CV side effects, including QT interval prolongation, ventricular arrhythmias, myocarditis, orthostatic hypotension, venous thromboembolism, and metabolic complications that contribute to long-term CV disease. Importantly, CV risk varies significantly among individual agents and should therefore be considered during therapeutic decision-making.

Careful baseline CV assessment, individualized selection of psychotropic medications, routine electrocardiographic and metabolic monitoring, and early recognition of CV adverse events are fundamental components of safe clinical practice. Close collaboration between psychiatrists, cardiologists, primary care physicians, and clinical pharmacists facilitates optimal psychiatric treatment while minimizing CV morbidity and mortality.

Although many newer psychotropic medications have favorable CV profiles compared with older agents, continued research is required to define their long-term CV outcomes and identify patients at greatest risk of adverse events. Advances in personalized medicine and multidisciplinary cardio-psychiatric care may further improve the safety of psychotropic therapy.

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Key Take-Home Messages

- CV risk assessment should be individualized before initiating psychotropic medications, with the intensity of assessment determined by the patient's CV risk and the known cardiac effects of the selected drug.
- ECG monitoring is recommended for patients receiving psychotropic agents associated with moderate or high risk of QT prolongation.
- Clozapine requires vigilant monitoring for myocarditis during the first month of therapy.
- Routine surveillance for metabolic complications is essential in patients receiving second-generation antipsychotics.
- Individualized prescribing and multidisciplinary collaboration remain the cornerstone of minimizing CV complications while maintaining optimal psychiatric outcomes.

REFERENCES

1. Manolis TA, Manolis AA, Manolis AS. Cardiovascular Safety of Psychiatric Agents: A Cautionary Tale. Angiology. 2019; 70(2): 103-129. doi:10.1177/0003319718780145.
2. Correll CU, Solmi M, Croatto G, et al. Mortality in People with Schizophrenia: A Systematic Review and Meta-analysis of Relative Risk and Aggravating or Attenuating Factors. World Psychiatry. 2022; 21(2): 248-271.
3. Lambiase PD, de Bono JP, Schilling RJ, et al. British Heart Rhythm Society Clinical Practice Guidelines on the Management of Patients Developing QT Prolongation on Antipsychotic Medication. Arrhythm Electrophysiol Rev. 2019; 8(3): 161-165. doi:10.15420/aer.2019.8.3.G1.
4. Abou-Setta AM, Mousavi SS, Spooner C, et al. First-Generation Versus Second-Generation Antipsychotics in Adults: Comparative Effectiveness. Comparative Effectiveness Review No. 63. Rockville (MD): Agency for Healthcare Research and Quality; 2012.
5. Cipriani A, Furukawa TA, Salanti G, et al. Comparative Efficacy and Acceptability of 21 Antidepressant Drugs for the Acute Treatment of Adults with Major Depressive Disorder: A Systematic Review and Network Meta-analysis. Lancet. 2018; 391(10128): 1357-1366.
6. De Hert M, Correll CU, Bobes J, et al. Physical Illness in Patients with Severe Mental Disorders. I. Prevalence, Impact of Medications and Disparities in Health Care. World Psychiatry. 2011; 10(1): 52-77.
7. De Hert M, Cohen D, Bobes J, et al. Guideline for Screening and Monitoring of Cardiometabolic Risk in Schizophrenia: Systematic Evaluation. World Psychiatry. 2011; 10(2): 138-151.
8. Galderisi S, De Hert M, Del Prato S, et al. Identification and Management of Cardiometabolic Risk in Subjects with Schizophrenia Spectrum Disorders: A Delphi Expert Consensus Study. Eur Psychiatry. 2021; 64(1): e7. doi:10.1192/j.eurpsy.2020.115.
9. Marano G, Traversi G, Romagnoli E, et al. Cardiologic Side Effects of Psychotropic Drugs. J Geriatr Cardiol. 2011; 8(4): 243-253.
10. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 Statement: An Updated Guideline for Reporting Systematic Reviews. BMJ. 2021; 372\: n71.
11. Ferrari R. Writing Narrative Style Literature Reviews. Acta Biomed. 2015; 86(2): 230-233.
12. Keepers GA, Fochtmann LJ, Anzia JM, et al. The American Psychiatric Association Practice Guideline for the Treatment of Patients with Schizophrenia. Am J Psychiatry. 2020; 177(9): 868-872.
13. Abdel-Qadir H, Roifman I, Wijeysundera HC, et al. Cardiovascular Toxicity of Psychotropic Medications: An Evidence-Based Review. Can J Cardiol. 2018; 34(7): 863-870.
14. Correll CU, Solmi M, Veronese N, et al. Prevalence, Incidence and Mortality From Cardiovascular Disease in Patients with Severe Mental Illness. World Psychiatry. 2017; 16(2): 163-180.

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15. Rado J, Janicak PG. Cardiovascular Complications Associated with Psychotropic Medications. Psychiatr Clin North Am. 2016; 39(3): 391-404.
16. Green BN, Johnson CD, Adams A. Writing Narrative Literature Reviews for Peer-reviewed Journals: Secrets of the trade. J Chiropr Med. 2006; 5(3): 101-117.
17. Androshchuk V, et al. Adverse Cardiovascular Effects of Psychotropic Medications. Br J Hosp Med (Lond). 2025; 86(4): 01-18. doi:10.12968/hmed.2024.0773.
18. Leung JY, Barr AM, Procyshyn RM, et al. Cardiovascular Side-effects of Antipsychotic Drugs: The Role of the Autonomic Nervous System. Pharmacol Ther. 2012; 135(2): 113-122. doi:10.1016/j.pharmthera.2012.04.003.
19. Schwartz PJ, Woosley RL. Predicting the Unpredictable: Drug-induced QT Prolongation and Torsades de Pointes. J Am Coll Cardiol. 2016; 67(13): 1639-1650.
20. Vandenberg JI, Walker BD, Campbell TJ. hERG K+ Channels: Structure, Function, and Clinical Significance. Physiol Rev. 2012; 92(3): 1393-1478. doi:10.1152/physrev.00036.2011.
21. Crumb WJ Jr, Ekins S, Sarazan RD, et al. Effects of Antipsychotic Drugs on I(to), I(Na), I(sus), I(K1), and hERG: QT Prolongation, Structure Activity Relationship, and Network Analysis. Pharm Res. 2006; 23(6): 1133-1143. doi:10.1007/s11095-006-0070-7.
22. Zeltser D, Justo D, Halkin A, et al. Torsade de Pointes Due to Noncardiac Drugs: Most Patients have easily Identifiable Risk Factors. Medicine (Baltimore). 2003; 82(4): 282-290. doi:10.1097/01.md.0000085057.63483.9b.
23. Funk MC, Beach SR, Bostwick JR, et al. APA Resource Document on QTc Prolongation and Psychotropic Medications. Am J Psychiatry. 2018.
24. Beach SR, Celano CM, Noseworthy PA, et al. QTc Prolongation, Torsades de Pointes, and Psychotropic Medications: A 5-year Update. Psychosomatics. 2018; 59(2): 105-122. doi:10.1016/j.psym.2017.10.009.
25. Rubenfire M, Brook RD. Orthostatic Hypotension: Pathophysiology and Clinical Management. Circulation. 2022; 145: 1687-1699.
26. Heal DJ, Gosden J, Smith SL, et al. The Affinity and Selectivity of α-adrenoceptor Antagonists, Antidepressants, and Antipsychotics for the Human α1A, α1B, and α1D-adrenoceptors. Pharmacol Res Perspect. 2020; 8(4): e00601. doi:10.1002/prp2.601.
27. Fiani D, Campbell H, Solmi M, et al. Impact of Antidepressant use on the Autonomic Nervous System: A Meta-analysis and Systematic Review. Eur Neuropsychopharmacol. 2023; 71: 75-95. doi:10.1016/j.euroneuro.2023.03.013.
28. Ronaldson KJ, Fitzgerald PB, Taylor AJ, et al. Rapid Clozapine Titration and Incidence of Myocarditis. Aust N Z J Psychiatry. 2012; 46: 458-465.
29. Ronaldson KJ, Fitzgerald PB, Taylor AJ, et al. Diagnostic Characteristics of Clozapine-induced Myocarditis Identified by an Analysis of 75 cases. J Clin Psychiatry. 2010; 71: 976-981.
30. Patel RK, Moore AM, Piper S, et al. Clozapine and Cardiotoxicity: A Guide for Clinicians. BJPsych Adv. 2019; 25: 175-183.
31. Knoph KN, Morgan RJ, Palmer BA. Clozapine-induced Cardiomyopathy and Myocarditis Monitoring: Current Evidence and Clinical Recommendations. Expert Rev Clin Pharmacol. 2018; 11: 1111-1119.
32. Correll CU, Frederickson AM, Kane JM, et al. Does Antipsychotic Polypharmacy Increase the Risk for Metabolic Syndrome? Schizophr Res. 2007; 89(1-3): 91-100.
33. Correll CU, Lencz T, Malhotra AK. Antipsychotic Drugs and Obesity. Trends Mol Med. 2011; 17(2): 97-107.

Citation: Abuosa A, Abdel-Monem Afifi H. What Should a Cardiologist Know About Psychiatric Medications? A Narrative Review of Cardiovascular Safety, Monitoring, and Clinical Management. Case Rep Case Ser Cardiol J. April-June 2026; 02(02): 59-77. **DOI:** doi.org/10.64874/crcscj.v2i2.2026.028.

34. Cooper SJ, Reynolds GP, Barnes TRE, et al. BAP Guidelines on the Management of Weight Gain, Metabolic Disturbances and Cardiovascular Risk Associated with Psychosis and Antipsychotic Drug Treatment. J Psychopharmacol. 2016; 30(8): 717-748. doi:10.1177/0269881116645254.
35. 2025 ESC Clinical Consensus Statement on Mental Health and Cardiovascular Disease: Developed Under the Auspices of the ESC Clinical Practice Guidelines Committee. Eur Heart J. 2025; 46(41): 4156-4213.
36. Taipale H, Tanskanen A, Mehtälä J, et al. Twenty-year Follow-up Study of Antipsychotic Treatment and Mortality in Schizophrenia. Am J Psychiatry. 2020; 177(8): 765-773.
37. Correll CU, Rubio JM, Inczedy-Farkas G, et al. Efficacy and Safety of 32 Oral Antipsychotics in Schizophrenia: Systematic Review and Network Meta-analysis. World Psychiatry. 2022; 21(2): 273-311.
38. Lichtman JH, Bigger JT Jr, Blumenthal JA, et al. Depression and Coronary Heart Disease: Recommendations for Screening, Referral, and Treatment. Circulation. 2008; 118(17): 1768-1775.
39. Ray WA, Chung CP, Murray KT, et al. Atypical Antipsychotic Drugs and the Risk of Sudden Cardiac Death. N Engl J Med. 2009; 360(3): 225-235. doi:10.1056/NEJMoa0806994.
40. Freeman R, Wieling W, Axelrod FB, et al. Consensus Statement on the Definition of Orthostatic Hypotension. Clin Auton Res. 2011; 21: 69-72.
41. Rivasi G, Rafanelli M, Mossello E, et al. Drug-related Orthostatic Hypotension: Beyond Anti-hypertensive Medications. Drugs Aging. 2020; 37(10): 725-738. doi:10.1007/s40266-020-00796-5.
42. Better Reference: Gee S, Taylor D. The Effect of Clozapine on the Heart: A Systematic Review of Cardiovascular Side Effects and Autonomic Dysfunction. CNS Drugs. 2018.
43. Nielsen J, Correll CU, Manu P, et al. Termination of Clozapine Treatment Due to Medical Reasons: When is it Warranted and how can it be avoided? J Clin Psychiatry. 2013; 74(6): 603-613.
44. Parker C, Coupland C, Hippisley-Cox J. Antipsychotic Drugs and Risk of Venous Thromboembolism: Nested Case-control Study. BMJ. 2010; 341: c4245.
45. Barbui C, Conti V, Cipriani A. Antipsychotic Drug Exposure and Risk of Venous Thromboembolism: Systematic Review and Meta-analysis. Drug Saf. 2014; 37(2): 79-90.
46. Liu Y, Xu J, Fang K, et al. Current Antipsychotic Agent use and Risk of Venous Thromboembolism and Pulmonary Embolism: A Systematic Review and Meta-analysis of Observational Studies. Ther Adv Psychopharmacol. 2021; 11: 2045125320982720. doi:10.1177/2045125320982720.
47. Mitchell AJ, Vancampfort D, De Herdt A, et al. Is the Prevalence of Metabolic Syndrome and Metabolic Abnormalities Increased in early Schizophrenia? A Comparative Meta-analysis. Schizophr Bull. 2013; 39(2): 295-305.
48. American Diabetes Association, American Psychiatric Association, American Association of Clinical Endocrinologists, North American Association for the Study of Obesity. Consensus Development Conference on Antipsychotic Drugs and Obesity and Diabetes. Diabetes Care. 2004; 27(2): 596-601.
49. De Hert M, Yu W, Detraux J, et al. Guideline Recommendations for Monitoring Metabolic Adverse Effects of Second-Generation Antipsychotics: A Systematic Review. World Psychiatry. 2021; 20: 373-374.
50. Spertus J, Horvitz-Lennon M, Abing H, et al. Risk of Weight Gain for Specific Antipsychotic Drugs: A Meta-analysis. NPJ Schizophr. 2018; 4: 12.
51. Roose SP, Glassman AH, Giardina EG. Tricyclic Antidepressants in Depressed Patients with Cardiac Conduction Disease. Arch Gen Psychiatry. 1987; 44(3): 273-275.
52. Glassman AH, Bigger JT Jr. Antidepressants and Cardiovascular Risk. Am J Psychiatry. 2001; 158(1): 11-17.

Citation: Abuosa A, Abdel-Monem Afifi H. What Should a Cardiologist Know About Psychiatric Medications? A Narrative Review of Cardiovascular Safety, Monitoring, and Clinical Management. Case Rep Case Ser Cardiol J. April-June 2026; 02(02): 59-77. **DOI:** doi.org/10.64874/crcscj.v2i2.2026.028.

53. Roose SP, Glassman AH. Selective Serotonin Reuptake Inhibitors and Cardiovascular Disease: Has the Story been told? J Clin Psychiatry. 2007; 68(8): e22.
54. Pacher P, Ungvari Z. Selective Serotonin Reuptake Inhibitors and Cardiovascular Effects: Friend or foe? Am J Cardiovasc Drugs. 2001; 1(3): 201-208.
55. Glassman AH, O'Connor CM, Califf RM, et al. Sertraline Antidepressant Heart Attack Randomized Trial (SADHART) Group. Sertraline Treatment of Major Depression in Patients with Acute MI or Unstable Angina. JAMA. 2002; 288(6): 701-709. doi:10.1001/jama.288.6.701.
56. Beach SR, Kostis WJ, Celano CM, et al. Meta-analysis of Selective Serotonin Reuptake Inhibitor-associated QTc Prolongation. J Clin Psychiatry. 2014; 75(5): e441-e449. doi:10.4088/JCP.13r08672.
57. Nochaiwong S, Ruengorn C, Awiphan R, et al. Use of Serotonin Reuptake Inhibitor Antidepressants and the Risk of Bleeding Complications in Patients on Anticoagulant or Antiplatelet Agents: A Systematic Review and Meta-analysis. Ann Med. 2022; 54(1): 1040-1055. doi:10.1080/07853890.2021.2017474.
58. Thase ME. Effects of Venlafaxine on Blood Pressure: A Meta-analysis of Original Data From 3744 Depressed Patients. J Clin Psychiatry. 1998; 59(10): 502-508. doi:10.4088/JCP.v59n1002.
59. Vieweg WVR, Wood MA. Tricyclic Antidepressants, QT Prolongation and Torsade de Pointes. Psychosomatics. 2004; 45(5): 371-377.
60. Bueno H, Deaton C, Farrero M, et al. 2025 ESC Clinical Consensus Statement on Mental Health and Cardiovascular Disease: Developed Under the Auspices of the ESC Clinical Practice Guidelines Committee. Eur Heart J. 2025; 46(41): 4156-4225. doi:10.1093/eurheartj/ehaf191.
61. Taylor D, Paton C, Kapur S. *The Maudsley Prescribing Guidelines in Psychiatry.*14th ed. Wiley-Blackwell; 2021.
62. Werneke U, Horn O, Maryon-Davis A, et al. Clinically Relevant Drug Interactions with Monoamine Oxidase Inhibitors. CNS Spectr. 2022; 27(6): 720-731.
63. Baldwin DS, Florea I, Jacobsen PL, et al. A Meta-analysis of the Efficacy of Vortioxetine in Patients with Major Depressive Disorder. Int Clin Psychopharmacol. 2016; 31(6): 360-370.
64. Glassman AH, O'Connor CM, Califf RM, et al. Sertraline Treatment of Major Depression in Patients with Acute Myocardial Infarction or Unstable Angina. JAMA. 2002; 288(6): 701-709.
65. Food and Drug Administration. FDA Drug Safety Communication: Revised Recommendations for Citalopram Related to QT Interval Prolongation. U.S. Food and Drug Administration; 2012.
66. Keepers GA, Fochtmann LJ, Anzia JM, et al. The American Psychiatric Association Practice Guideline for the Treatment of Patients With Schizophrenia. Am J Psychiatry. 2020; 177(9): 868-872. doi:10.1176/appi.ajp.2020.177901.
67. Funk MC, Beach SR, Bostwick JR, et al. APA Resource Document on QTc Prolongation and Psychotropic Medications. American Psychiatric Association; 2018.
68. Lambiase PD, de Bono JP, Schilling RJ, et al. British Heart Rhythm Society Clinical Practice Guidelines on the Management of Patients Developing QT Prolongation on Antipsychotic Medication. Arrhythm Electrophysiol Rev. 2019; 8(3): 161-165. doi:10.15420/aer.2019.8.3.G1.
69. Spina E, de Leon J. Clinically Relevant Interactions between newer Antidepressants and Second-generation Antipsychotics. Expert Opin Drug Metab Toxicol. 2014; 10(5): 721-746.
70. Visseren FLJ, Mach F, Smulders YM, et al. ESC Guidelines on Cardiovascular Disease Prevention in Clinical Practice. Eur Heart J. 2021; 42(34): 3227-3337.
71. Roden DM, Harrington RA, Poppas A, et al. Considerations for Drug Interactions on QTc Interval in Clinical Practice. Circulation. 2019; 140: e906-e917.

Citation: Abuosa A, Abdel-Monem Afifi H. What Should a Cardiologist Know About Psychiatric Medications? A Narrative Review of Cardiovascular Safety, Monitoring, and Clinical Management. Case Rep Case Ser Cardiol J. April-June 2026; 02(02): 59-77. **DOI:** doi.org/10.64874/crcscj.v2i2.2026.028.