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Mechanisms, risk factors and management of cardiovascular adverse effects of antidepressants and antipsychotics - a literature review

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ABSTRACT

Mental disorders represent a major burden to public health worldwide, while cardiovascular diseases are the leading cause of death among patients with these mental disorders. Antipsychotics and antidepressants, which are widely used in treatment, may produce a whole spectrum of cardiovascular adverse effects that cannot be overlooked. Antidepressants are mostly associated with arrhythmia, whereas antipsychotics are primarily associated with metabolic syndrome. All things considered, both medication classes show unique cardiotoxic patterns that need customized evaluation and observation. This review aims to summarise the mechanisms underlying these patterns and provide recommendations for the management of psychotropic cardiotoxicity.

Keywords: antidepressants, antipsychotics, cardiovascular adverse effects

1. INTRODUCTION

Over the last 30 years, mental disorders have remained consistently among the leading causes of disability worldwide with a growing number of disability-adjusted life years (DALYs). Furthermore, the proportion of DALYs due to mental diseases has risen from 3.1% in 1990 to 4.9% in 2019 (GBD 2019 Mental Disorders Collaborators, 2022). This results in the increased use of antidepressants and antipsychotics. Patients with mental illnesses have a two-fold higher cardiovascular mortality compared with the general population (Lambert et al., 2022). In this regard, although the benefits of psychotropic medications in curing mental disorders are substantial, their potential adverse effects and cardiotoxicity should always be taken into consideration. Despite rising recognition of psychotropic cardiotoxicity, data on this subject are still fragmented among drug classes. The aim of this review is to summarize the main mechanisms of cardiovascular toxicity and

its therapeutic care. Furthermore, this review seeks to provide an overview of the risk stratification and offer practical recommendations for clinical management.

2. REVIEW METHODS

We used Pubmed, Scopus and Google Scholar to search for articles published between January 2021 and January 2026. We also brought in papers that were published before 2021, when they gave useful background or explained long-known mechanisms of cardiotoxicity. The search used terms: "antidepressants", "antipsychotics", "SSRI", "TCA", "cardiotoxicity", "QT prolongation", "arrhythmia", "myocarditis", "metabolic syndrome", "cardiovascular adverse effects".

The inclusion criteria were as follows:

1. cardiovascular adverse effects of antidepressants and/or antipsychotics
2. full-text available in English
3. human study population

The exclusion criteria comprised:

1. studies not available in English.
2. studies without relevant clinical outcomes
3. studies involving irrelevant pharmacological agents

A total of 231 articles were identified through database search. After the removal of duplicates, we did titles and abstracts screening. Fifty-nine articles were eligible for detailed assessment, and we selected 30 for final inclusion. The selection process is shown in Figure 1.

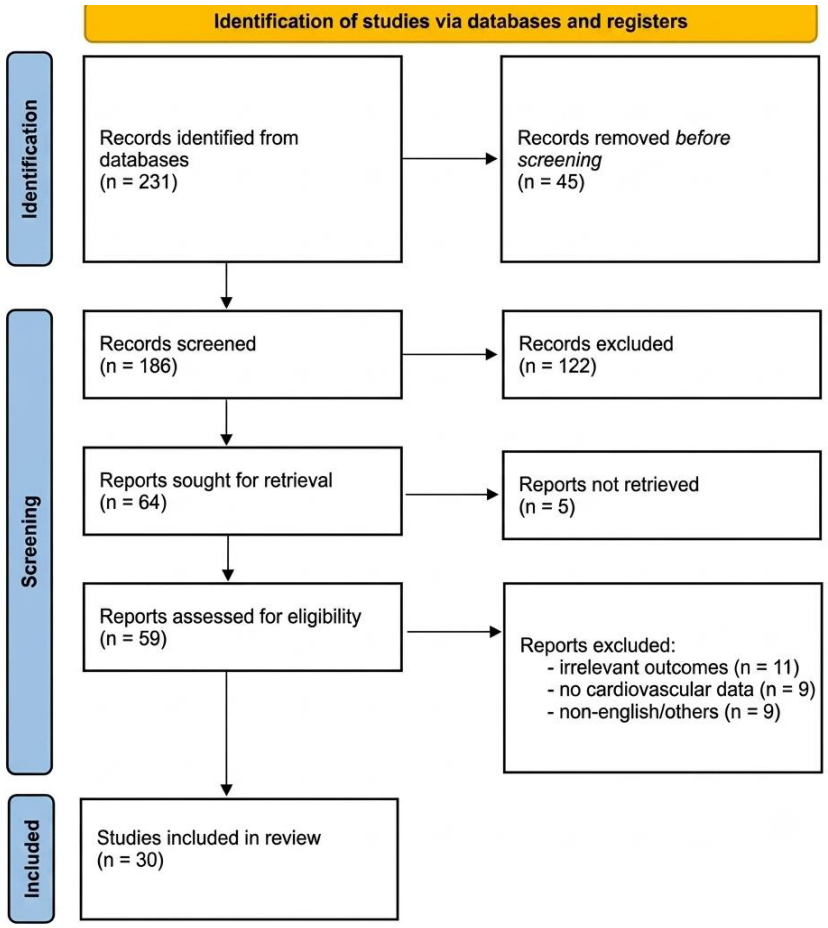

Figure 1. PRISMA flow diagram

3. RESULTS AND DISCUSSION

Antidepressant drugs

Mechanisms

Selective Serotonin Reuptake Inhibitors (SSRIs), to which belong paroxetine, sertraline, and fluoxetine, may exert cardiotoxic effects through several mechanisms. On the cellular level, they cause mitochondrial dysfunction by decreasing ATP production and mitochondrial respiration. Furthermore, mitochondrial dysfunction results in excess ROS production and oxidative stress. SSRIs induce mitochondrial fission, disruption of mitochondrial movement and cell apoptosis through PGAM5 protein. Production of this particular protein increases significantly especially after paroxetine exposure. Through another mechanism, SSRIs cause dysfunction of sarcomere structure, the fundamental contractile unit in cardiac muscle, which may impair cardiomyocyte structural integrity. As a result, the sarcomeric function of cardiac muscles is impaired and may contribute to the development of cardiomyopathy (Shen et al., 2025).

Citalopram and escitalopram also block the hERG K^+ channels. Citalopram and escitalopram inhibit the peak current of the Nav1.5 voltage-gated sodium channels. Both of these mechanisms affect QT and result in the QRS complex widening. However, this inhibition is dependent on concentration and can be reversible (Farhat et al., 2024). Citalopram and escitalopram also affect the function of calcium channels. In this mechanism occurs prolongation of Ca^{2+} transient duration and reducing L-type calcium current. As a result, intracellular levels of Ca^{2+} decrease which may contribute to electrical instability and arrhythmias (Li et al., 2025). Toxicity of tricyclic antidepressants (TCAs) is thought to be associated with multiway mechanisms, which include inhibition of norepinephrine reuptake and blockade of α-adrenergic receptors, anticholinergic activity, and lastly quinidine-like sodium channel blockade. This may contribute to abnormal conduction and, in consequence, arrhythmias (Taylor et al., 2024). Studies also suggest that venlafaxine and duloxetine can contribute to stress cardiomyopathy due to excessive amounts of catecholamines (Tan et al., 2024; Vasudev et al., 2016).

Clinical presentation

According to FDA labeling from 2011 with subsequent clarification about citalopram, that drug can cause dose-dependent QT prolongation and later lead to ventricular arrhythmias, like torsades de pointes (FDA, 2011). Among SSRIs, citalopram and escitalopram have the highest pro-arrhythmic potential; yet none of their metabolites are cardiotoxic. Importantly, the risk of arrhythmia increases with age due to age-related reduction of drug clearance (Al Nakhebi et al., 2025; Faraj et al., 2023; Farhat et al., 2024). In the young population treated with citalopram with no cardiovascular comorbidities, hypertension or diabetes, QTc prolongation and clinically significant arrhythmias appear to be modest. Yet the risk increases in patients with additional predisposing factors, which include higher doses, electrolyte disorders and polypharmacy with other QT prolonging drugs (Munivenkatappa et al., 2024).

Studies show that TCAs can also cause cardiovascular toxicities such as arrhythmia characterized by QT prolongation and QRS widening. There are no significant differences in type of TCAs but occurs frequently after TCAs overdose, particularly amitriptyline. Importantly, ECG abnormalities were significantly associated with intensive care unit admission suggesting an association between conduction disturbances and poisoning severity (Javadipour et al., 2024). Research indicates that treatment with amitriptyline dothiepin and lofepramine pose an increased risk for myocardial infarction (Taylor et al., 2024). Compared to SSRIs described above, which are associated with QT prolongation, SNRIs such as venlafaxine may also induce hypertension, postural hypotension, syncope, ventricular fibrillation, and ventricular tachycardia including torsade de pointes.

Both of these drug classes are reported to cause cardiomyopathy, mostly associated with venlafaxine and fluoxetine, which occurs primarily in females between the ages of 45 and 65. Among others, the most severe and important symptom is acute cardiac dysfunction, which frequently happens soon after drug exposure and has a median time to onset of about 20 days. Moreover, these symptoms are reversible after a reduction in venlafaxine. In any case, when the patient is taking venlafaxine and presents any signs of cardiotoxicity, these symptoms should be considered potentially related to this treatment (Batusha Sopi et al., 2025; Tan et al., 2024). Venlafaxine is generally associated with elevated blood pressure, however there were also reported rare cases of patients with orthostatic hypotension after venlafaxine. This shows that symptoms caused by venlafaxine are heterogeneous and may manifest in patients without typical risk factors (Agrawal et al., 2025).

Orthostatic hypotension is associated with treatment with trazodone and prevalent especially in older adults which tend to have a greater blood pressure drop. This drop can predispose them to an increased risk of syncope and falls. These effects are primarily attributed to α 1-adrenergic receptor blockade, which impairs vascular compensatory responses during standing. However, researchers point out that when adjusting for dementia diagnosis the association was not confirmed (Rivasi et al., 2025).

Risk factors

The risk of cardiotoxicity induced by antidepressants depends on the class of the drug and factors specific to the patient. Therefore, it is essential to stratify risk for identifying individuals who may require closer monitoring during treatment (Prasitlumkum et al., 2021). Assessment of QT prolongation risk during antidepressant therapy depends on non–drug-related factors. It is widely recognized that higher age and female sex are independent risk factors and are often accompanied by cardiovascular comorbidities such as arterial hypertension, heart disease (myocardial infarction, heart failure, valvopathy or cardiomyopathy), kidney or liver failure, and metabolic diseases (diabetes, obesity).

Furthermore, as clinically recognizable risk factors are mentioned also electrolyte imbalances, especially hypokalemia, hypomagnesemia, and hypocalcemia, as well as genetic predispositions (Gotta & Donner, 2025). Pharmacologic medications from the Arizona Center for Education and Research on Therapeutics (AzCERT) classification significantly increase the accumulated risk. However, there is no clarity whether polypharmacy represents a risk factor (Schulze Westhoff et al., 2023).

In a study by Wang et al., (2024) the authors give some predictors of severe outcomes, with advanced age (particularly ≥75 years) being the most important, male sex, and the presence of comorbid cardiovascular disorders, neurological diseases, and infections. Another study also includes cardiac sympathetic stimulation, high venlafaxine doses, and prolonged treatment for potential risk factors. However, in this case, cardiotoxicity has also been observed in standard therapeutic doses, putting at risk also patients during the first period of treatment and those without additional risk factors (Batusha Sopi et al., 2025). Patients that are at the highest risk of orthostatic hypotension receiving trazodone belong to the elderly and demented, with additional risk among patients receiving multiple medications, especially antihypertensive therapy, and those with occurring orthostatic hypotension or frailty before trazodone (Rivasi et al., 2025).

Clinical Management

Firstly, studies highlight the importance of assessing existing cardiovascular risk factors. Moreover, before initiating treatment with a QT-prolonging drug, it is recommended to perform an ECG and monitor blood pressure. That cardiovascular screening should also be continued throughout the whole duration of therapy (Al Nakhebi et al., 2025; Bulatova et al., 2021; Javadipour et al., 2024). During prolonged therapy and in primary care settings, drug concentration in plasma should be monitored at regular intervals (Batusha Sopi et al., 2025). In clinical practice, risk scores should be calculated, such as the Tisdale Risk Score, which is based on easily acquired variables and can aid in guiding monitoring and treatment decisions (Tisdale et al., 2013). When prescribing trazodone, it is neccessary to systematicaly measure orthostatic blood pressure and assess fall risk (Rivasi et al., 2025).

If a patient develops cardiotoxic effects, it should be considered reducing dosage, discontinuation of the therapy with this drug, or switching to a safer alternative. More severe side effects require stabilization of vital signs, gastrointestinal decontamination, and treatment of cardiac complications. In the most severe cases may be required therapy with sodium bicarbonate, extracorporeal membrane oxygenation (ECMO) or targeted temperature management (TTM) (Al Nakhebi et al., 2025).

Antipsychotic Drugs' Cardiovascular Adverse Events

Impact on the QTc Interval

Antipsychotic drugs are known to increase the risk of sudden cardiac death (SCD) through a mechanism of serious ventricular arrhythmias. The mechanism of its induction was described several decades ago (Kongsamut et al., 2002; Glassman & Bigger, 2001). The therapeutic effects of the antipsychotics are a result of binding dopamine or serotonin receptors localized in the central nervous system. However, these are not the only binding molecular targets of these medications. The drugs in question have affinity to potassium channels known as human ether-a-go-go-related gene (HERG) channels, which are expressed in cardiomyocytes (Kongsamut et al., 2002).

A consequence of such an affinity is prolongation of cardiomyocytes' repolarisation phase, which is a cause of QTc interval variability on the electrocardiogram (ECG). This influence is not uniform for all antipsychotic drugs due to the heterogeneity of this group of medications. A classic example of a molecule with a significant influence on potassium channels is thioridazine, which has been associated with an increased risk of sudden cardiac death (Glassman & Bigger, 2001). A more recent study documented that quetiapine increases SCD risk, particularly in patients with advanced age, other cardiovascular diseases, hypokalemia, and use of other drugs that impact on QTc (Wang et al., 2024).

Antipsychotic drugs are not restricted only to psychiatrists, but they are also used by other specialists in emergency departments (EDs) or intensive care units (ICUs) to help patients with severe agitation, nausea, abdominal pain, or headache (Ernst et al., 2024). In such clinical conditions, the long-term adverse effects of these drugs do not play a crucial part. Much more important are the immediate effects that may worsen an already complicated patient's condition. Among the most feared cardiovascular adverse effects are QTc prolongation and ventricular arrhythmias, including torsade de pointes. Interestingly, the arrhythmias mentioned above do not seem to be provoked by the intake of the antipsychotic drugs during comparatively short time spent on ED or ICU (Stollings et al., 2024; Ernst et al., 2024). In another study neither haloperidol or ziprasidone significantly affected QTc length in ECGs of the ICU patients with delirium. The researchers examined a comparatively large group of 566 patients who were randomized either to a placebo or to one of the two mentioned above antipsychotic cohorts. The randomized design and relatively large sample size enhance the reliability and generalizability of the findings (Stollings et al., 2024). It was also observed that despite the prolongation of the QTc interval on average for 29.9 ms after the administration of droperidol, no serious arrhythmia was induced in a group of 68 patients (Hernández-Rodríguez et al., 2022). This study, however, conducted with the participation of only a limited number of patients in the ED, repeats the conclusions about the safety of antipsychotics stated in the article mentioned above (Stollings et al., 2024).

Cardiometabolic Diseases

Another adverse event of antipsychotic drugs is a disequilibrium of metabolic homeostasis, including the occurrence of diabetes mellitus (DM), obesity, and dyslipidemia. The global burden of antipsychotics-induced obesity is considerable. It is estimated that the problem of obesity induced by antipsychotics concerns about 20 million patients worldwide (He et al., 2022). Their life expectancy is estimated to be reduced by 16-20 years due to metabolic reasons (Siskind et al., 2025). Consequently, the prevention and treatment of metabolic complications is one of the priorities in contemporary psychiatric care. The problem in question, however common, has not been fully investigated yet, and the molecular mechanisms leading to the obesity caused by the antipsychotics are unknown. The proposed ones include sympathetic nervous system overactivation, leading to the augmentation of appetite and difficulties in achieving satiety (Manta et al., 2025). As a result, patients may consume excessive amounts of food, leading to progressive weight gain over time.

What is more, the insulin-glucagon balance also seems to be interrupted in a way that stimulates gluconeogenesis in the liver by the augmentation of glucagon and glucagon-like peptide 1 (GLP-1) secretion (Manta et al., 2025). The latter mechanism constitutes a potential target for pharmacotherapy. Semaglutide, a GLP-1 receptor antagonist, was evaluated in the obesity therapy of patients with schizophrenia and obesity, treated with olanzapine. In this randomized controlled trial, the participants received either semaglutide or a placebo for 36 weeks. In the semaglutide group, weight loss of 13,88% was noted. However, the study group was relatively small, including only 31 participants (Siskind et al., 2025).

The GLP-1 receptor antagonists are promising in the treatment of antipsychotic-induced obesity. Still, due to their comparatively short presence in the treatment of diabetes mellitus, they should be evaluated more carefully. However, there exists a drug, metformin, with strong evidence of efficacy in the treatment of the disorder in question. It has been stated that it reduces the weight of patients receiving antipsychotics by 4.03 kg, while also being effective in the prevention of weight gain (Carolan et al., 2025). What is more, it is comparatively cheap, safe, and easily accessible. The summary of the main adverse effects, their mechanisms and risk factors are shown in Table 1.

Table 1. Summary of the main cardiovascular adverse effects of antipsychotics and antidepressants

	Drug/Examples	**Mechanisms**	**Main risk factors**
QT-prolongation and arrhythmias	SSRI, TCA, first-generation antipsychotics	inhibition of HERG potassium channels, inhibition of peak current of the Nav1.5 sodium channels, dysfunction of calcium channels	age, female sex, cardiovascular comorbidities, kidney failure, liver failure, diabetes, obesity, hypokalemia, hypomagnesemia and hypocalcemia
Hypertension	SNRIs	Increased sympathetic stimulation	age, male sex, cardiovascular disorders, neurological diseases, infections, high doses

Orthostatic hypotension	venlafaxine, trazodone	α1-adrenergic receptor antagonism	age (≥75 years), polypharmacology, existing orthostatic hypotension, cardiovascular disease
Cardiomyopathy	venlafaxine, duloxetine	excessive catecholamine stimulation	age (45-65yo), female sex polypharmacy
Sudden cardiac death	thioridazine, quetiapine	inhibition of HERG potassium channels	age, cardiovascular diseases, hypokalemia, polypharmacology
Obesity	antipsychotics	increased appetite, insulin resistance	class of the drug, high doses, treatment duration

4. CONCLUSION

Psychotropic medications can cause a broad spectrum of cardiovascular complications, yet comparative studies evaluating both antidepressants and antipsychotics are limited. As mentioned, patients receiving treatment for mental illnesses belong to a population with elevated risk for cardiovascular morbidity and require integrated care from both cardiologists and psychiatrists. Given that and the heterogeneity of adverse effects across psychotropic medications, physicians should individually assess risk and implement continuous monitoring. This includes thorough clinical evaluation, regular ECG monitoring, assessment of metabolic parameters, and efforts to minimize polypharmacy and unnecessary dose escalation of psychotropic drugs.

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Authors' Contributions

Przemysław Paduch: Literature Search, Study Selection, Data Interpretation, Writing – Original Draft, Supervision.
Kaja Zdrojewska: Data Curation, Data Synthesis, Writing – Original Draft, Writing – Review & Editing.
Klaudia Kapelka: Formal Analysis, Data Interpretation, Writing – Original Draft.
Klaudia Czech: Data Curation, Formal Analysis, Validation, Writing – Review & Editing.
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Dawid Kłos: Study Selection, Methodology, Writing – Review & Editing.
Stanisław Malik: Writing – Review & Editing, Validation, Final Approval of the Manuscript.
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Ethical approval

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Conflict of interest

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Data and materials availability

All data associated with this study will be available based on the reasonable request to corresponding author.

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