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Venlafaxine and the Cardiovascular System: A Review of Hemodynamic Changes, Arrhythmia Risk, and Drug Interactions

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Abstract

Background. Depression is one of the most common mental disorders worldwide and often coexists with cardiovascular diseases, posing significant clinical challenges. Venlafaxine is widely used to treat depression. Because of its mechanism of action, analyses of these potential cardiologic effects have attracted growing research interest.

Aim. The aim of this study was to analyze available data on the cardiovascular system effects of venlafaxine, including its impact on blood pressure, cardiac electrical activity, arrhythmia risk, and potential interactions with cardiovascular medications.

Material and methods. A literature review was conducted using the PubMed database, focusing on publications from the last ten years that addressed the effects of venlafaxine on hemodynamic, electrophysiological, metabolic parameters, and its interactions with other medications.

Results. Meta-analyses and clinical studies suggest that venlafaxine may increase blood pressure. Most studies did not demonstrate clinically significant QTc prolongation or other conduction abnormalities, though isolated reports indicate possible individual susceptibility. The drug shows a low potential for clinically relevant pharmacokinetic interactions. Acute cardiac events related to venlafaxine are very rare.

Conclusions. Venlafaxine is an effective and generally safe antidepressant in patients with cardiovascular disease; however, monitoring of blood pressure and cardiac function is advised, particularly in elderly or multimorbid patients. Further prospective studies are needed to assess its long-term cardiovascular effects.

Keywords: venlafaxine, antidepressants, cardiovascular system, blood pressure, QTc interval, cardiovascular safety

Introduction

Cardiovascular diseases are very common and are among the leading causes of death worldwide. They occur with a prevalence of 48.6% among adults aged 20 years and older in the United States [1]. Depression in patients with cardiovascular disease is a global problem that affects an increasing number of patients.

According to Li et al., up to 30% of patients develop depression after myocardial infarction, and among patients with coronary artery disease, depression occurs in 20–40% of cases [2].

Depression is not solely a disorder affecting a patient's mental health. It also impacts overall functioning and, consequently, influences the cardiovascular system [3]. It may increase mortality risk among cardiac patients and reduce treatment effectiveness. Therefore, a holistic

approach to patient care and effective treatment of psychiatric disorders in conjunction with cardiovascular therapy are essential [2].

One of the major challenges faced by clinicians is to identify an antidepressant that will be effective while exerting minimal influence on the cardiovascular system. One of the medications used in the treatment of this condition is venlafaxine, an SNRI, a serotonin-norepinephrine reuptake inhibitor.

Venlafaxine acts by inhibiting the reuptake of serotonin and norepinephrine. At lower doses, its mechanism primarily involves serotonergic pathways, whereas at higher doses, its noradrenergic effects become more pronounced. This dual mechanism enhances the efficacy of treatment for severe depressive episodes, anxiety disorders, and dysthymia. However, it may negatively affect other systems iassociated with sympathetic nervous system activity. Increased peripheral norepinephrine levels result in enhanced vascular tone and peripheral resistance, which may potentially translate into changes in blood pressure and cardiovascular function [4].

The drug is metabolized in the liver to the active metabolite O-desmethylvenlafaxine via CYP2D6. This metabolite also acts on serotonin and norepinephrine systems. While this prolongs the drug's action, it may simultaneously increase cardiovascular risk [5].

The purpose of this article is to evaluate the effects of venlafaxine on the cardiovascular system, with particular emphasis on both its therapeutic and adverse effects in patients with cardiovascular disease.

Antidepressants and Their Cardiovascular Effects

Antidepressant medications differ in their effects on the cardiovascular system, which results from their distinct pharmacological mechanisms of action. Among the drugs that most significantly affect this system are tricyclic antidepressants (TCAs). These medications may induce orthostatic hypotension, tachycardia, QT interval prolongation, and atrioventricular conduction abnormalities [6].

Selective serotonin reuptake inhibitors (SSRIs) are generally considered safer, although in the case of escitalopram and citalopram, instances of QT interval prolongation have been reported, particularly in patients with preexisting heart disease [7].

It should be noted that newer-generation agents such as mirtazapine and bupropion are characterized by a more favorable cardiac safety profile. However, isolated cases of arrhythmias have also been described in patients treated with these medicines [6].

Serotonin-norepinephrine reuptake inhibitors constitute a widely used class of antidepressants. These drugs may cause cardiac rhythm disturbances and changes in blood pressure and may

also influence pharmacokinetic interactions with cardiovascular medications [8, 9]. Among them, venlafaxine warrants particular attention, as its complex effects on the cardiovascular system and its potential interactions with cardiac drugs require separate discussion.

Effect on Arterial Hypertension

Arterial hypertension is a disease that constitutes a serious problem in contemporary medicine. It is a major risk factor for cardiovascular morbidity and mortality, affecting approximately 1.28 billion adults worldwide. Therefore, an important aspect of treatment is evaluating the effect of administered medications on blood pressure [10].

In one study, the effect of venlafaxine on hemodynamic parameters was analyzed. The study included 429 individuals over the age of 60 with a current episode of major depression. Patients received extended-release venlafaxine at doses of up to 300 mg per 24 hours for a period of 8–16 weeks. The results were as follows: 6.5% of patients with previously normal blood pressure developed hypertension, while 20.1% developed orthostatic hypotension. An increase in blood pressure was observed mainly in patients receiving higher doses of the drug (>225 mg/day) however, due to the study design, this association does not allow for definitive confirmation of a dose-dependent effect [11].

The study by Kivrak et al. focuses on the case of a 23-year-old male who developed arterial hypertension during treatment with venlafaxine. The patient suffered from a depressive disorder. He was initially treated with an antidepressant at a dose of 75 mg/day, which was later increased to 150 mg/day due to a lack of clinical improvement at the lower dose. Before initiating therapy, the patient had normal blood pressure values. During treatment, after 10 months of using the higher dose, patients presented to the hospital with symptoms of hypertension and a blood pressure of 210/170 mmHg. Secondary causes of hypertension were excluded. After discontinuation of venlafaxine, his blood pressure values decreased, suggesting a possible association between the medication and the elevation in blood pressure [12].

In a series of five cases described by Sopi et al., three patients developed arterial hypertension during treatment with venlafaxine despite previously normal blood pressure values. Similarly to the studies cited above, blood pressure returned to normal values after discontinuation of the medication [13].

A large meta-analysis conducted by Thase and colleagues is mentioned in the literature review, in which several randomized clinical trials were analyzed, including a total of 3,744 patients with depression receiving venlafaxine at various doses. According to the authors, the drug was

associated with an increase in supine diastolic blood pressure in a dose-related manner. In patients receiving doses up to 150 mg/day, the increase was mild and considered not clinically significant. At higher dose ranges, elevated blood pressure values occurred more frequently than in the control group. These findings indicate a gradual dose–response relationship; however, the applied dose categorization (e.g., >300 mg/day) results from the analytical methodology and does not necessarily reflect the existence of a distinct clinical threshold [14, 15].

These studies clearly suggest an association between venlafaxine use and increased blood pressure. Recent findings indicate that although elevations are most commonly observed at higher doses, in isolated cases they may also occur at lower doses, which supports the need for blood pressure monitoring in all patients receiving this medication. It should also be noted that cases of orthostatic hypotension during venlafaxine treatment have been reported. Therefore, regular blood pressure assessment in both the seated and standing positions should be considered, particularly in elderly patients treated with this drug.

Effect on the QT interval

One of the important cardiological aspects is the effect of venlafaxine on cardiac arrhythmias and the QT interval. This is significant because many medications, including antidepressants, prolong this interval, which in turn may increase the risk of potentially life-threatening arrhythmias, such as torsades de pointes (TdP) [16].

In one study, the effect of venlafaxine on changes in electrocardiographic parameters was evaluated. The study included 169 elderly patients aged 60 years and older with severe depressive disorder. Participants received the medication for up to 14 weeks at doses of up to 300 mg/day. During the study, the QTc interval, atrioventricular conduction time, QRS complex duration, and heart rate were regularly monitored, and the results were compared with serum concentrations of venlafaxine and its active metabolite, O-desmethylvenlafaxine. According to the analysis, treatment with this drug did not result in clinically significant QTc prolongation and was, in fact, associated with a slight shortening of the interval. The administered dose did not correlate with changes in the QT interval. The results were also not influenced by sex, the presence of heart disease, or remission status of depression. The drug did not affect QRS complex duration [17].

An important study addressing this issue is the analysis conducted by Abbas et al. The study included 54 healthy volunteers who, in three consecutive periods, received extended-release venlafaxine at a dose of 450 mg/day, placebo, and moxifloxacin 400 mg as an active control to confirm assay sensitivity. The results indicated that venlafaxine did not result in clinically significant QTc prolongation. The maximum difference between the drug and placebo was 5.8 ms (upper bound of the 90% confidence interval), and no value exceeded 10 ms. No correlation was found between drug concentration and QTc duration, suggesting that venlafaxine does not meaningfully affect cardiac repolarization in healthy individuals. However, it should be noted that the study did not include patients with pre-existing cardiovascular disease [18].

In one study, changes in heart rate and the QTc interval were analyzed in relation to two medications: amitriptyline and venlafaxine. In patients treated with venlafaxine, no significant changes were observed in these parameters, whereas treatment with amitriptyline was associated with a significant increase in heart rate and QTc prolongation, which may suggest that venlafaxine has a more favorable cardiac profile than tricyclic antidepressants [19].

In the study by Bavle, a case was described involving a patient who developed QTc prolongation during treatment with venlafaxine at a dose of 300 mg/day. The patient had no history of heart disease and was not taking other medications that could have affected cardiac repolarization. After discontinuation of the drug, the QT interval returned to normal values. Upon reintroduction of venlafaxine at a lower dose of 150 mg/day, the problem did not recur [20].

The analysis described by Aronow and Shamliyan refers to a post-marketing study conducted by the pharmaceutical company after the approval of venlafaxine, in which QT interval prolongation was observed in one patient following an overdose [21, 22].

An analysis of the available data suggests that venlafaxine rarely results in clinically significant changes in the QTc interval. In the majority of studies, such abnormalities were not observed even at higher doses of the drug. Additionally, venlafaxine appears to have a more favorable cardiac profile compared with tricyclic antidepressants. Isolated case reports suggesting possible QTc prolongation may be related to individual patient susceptibility, comorbid conditions, or interactions with other medications.

Effect on the Development of Atrial Fibrillation

Atrial fibrillation (AF) is the most common arrhythmia in humans, and its prevalence continues to rise. It is associated with a higher risk of mortality, stroke, and thromboembolic events. [23] Evidence regarding the potential association between venlafaxine and AF remains limited, with only a few case reports and small case series published to date.

Sopi et al. described a series of 5 patients (three men and two women aged 51–87 years). In three patients from this series, atrial fibrillation developed during treatment with venlafaxine. After discontinuing the medication, the arrhythmias were resolved. The authors noted a possible temporal association between therapy and the onset of arrhythmia and proposed a hypothesis involving increased sympathetic activity [13]. A similar case was described in a 76-year-old patient who developed an acute episode of atrial fibrillation during treatment with venlafaxine in combination with electroconvulsive therapy. Therefore, from a clinical perspective, vigilance may be warranted when prescribing venlafaxine to patients at increased risk of this arrhythmia [24].

Effect of Venlafaxine on Metabolic Parameters and the Risk of Metabolic Syndrome

Metabolic syndrome is a clinically significant condition that increases the risk of atherosclerosis, cardiovascular diseases, prediabetic states, and diabetes. It is a common problem among patients receiving psychiatric treatment. Its prevalence in this population is estimated to range between 30% and 50%. One of the key components of this syndrome is lipid abnormalities [25].

In one study, the metabolic effects of antidepressant medications, including venlafaxine, were analyzed. During this analysis, seven patients receiving venlafaxine 75 mg/day, later increased to 150 mg/day, and were observed over an eight-week period. Due to the small sample size, these findings should be considered preliminary and require confirmation in studies involving larger populations [26].

In contrast, a large population-based study conducted as part of the UK Biobank project, which included nearly 470,000 participants, examined the effects of antidepressants on lipid profiles. The analysis indicated that venlafaxine was associated with the most pronounced unfavorable changes in lipid parameters. Treatment with this drug was associated with significant increases in total cholesterol, LDL cholesterol, and triglycerides, as well as a decrease in HDL cholesterol. After adjustment for confounding factors, the mean difference in total cholesterol concentration

was approximately +0.21 mmol/L (95% CI: 0.17–0.26), which was considered clinically significant [27].

The available results are inconclusive and based on a limited number of studies. Although short-term therapy with venlafaxine appears to be safe according to smaller observational data, findings from a large population-based analysis suggest a potentially unfavorable lipid profile. Therefore, caution is advised when prescribing venlafaxine to patients with metabolic risk factors, and regular monitoring of lipid parameters during treatment should be considered.

Interactions with Cardiovascular Medications

Statins

Drug interactions constitute a highly important issue in the management of cardiovascular patients. Statins, which are HMG-CoA reductase inhibitors, are among the most prescribed medications for treating dyslipidemia [8]. Their concomitant use with drugs from the SSRI and SNRI classes is common. Therefore, it is important to consider potential interactions between these agents [28, 29].

In the review by Palleria et al., possible pharmacological interactions between these groups of medications were discussed. Venlafaxine was presented as one of the safest antidepressants in terms of interactions with statins. The authors noted that venlafaxine is metabolized through two metabolic pathways, CYP2D6 and CYP3A4, and exhibits weak inhibitory activity toward CYP2D6. It has minimal or no effect on the activity of CYP1A2, CYP2C9, CYP2C19, and CYP3A4 isoenzymes. O-desvenlafaxine, the active metabolite of venlafaxine, does not interact with CYP isoforms. Therefore, venlafaxine rarely results in clinically significant interactions with most statins metabolized through these pathways. However, the authors emphasize that particular attention should be paid to potential interactions between these two drug classes in elderly patients [9].

Beta-blockers

Beta-blockers are antagonists of beta-adrenergic receptors. They attenuate the effects of catecholamines on beta-receptors. This reduces heart rate and myocardial contractility. Beta-blockers slow conduction through the sinoatrial and atrioventricular nodes. They also suppress ectopic pacemaker activity [30].

In a study conducted by Woroń et al., which described 66 cases of adverse events resulting from the concomitant use of antidepressants and cardiovascular medications, venlafaxine was also included. In the analyzed group, 7 cases (10.6%) of abnormal interactions involved venlafaxine. The observed events included bradycardia and dizziness (in combination with propafenone, n = 2); increased muscle tone and urinary urgency (following the addition of propranolol, n = 3); and bleeding from the genital tract and nose (in combination with clopidogrel, n = 3). The authors noted that when combination therapy with an antidepressant and a beta-blocker is required, the most favorable option appears to be the use of metoprolol or propranolol in combination with venlafaxine or sertraline [31].

Cyclosporine

Cyclosporine is an immunosuppressive drug used in the treatment of various autoimmune diseases. A case report involving a patient with systemic lupus erythematosus suggests a protentional interaction between venlafaxine and cyclosporine. In the reported patient, who was receiving cyclosporine, bradycardia developed after the initiation of venlafaxine therapy. The patient had no prior history of cardiac disorders, and following discontinuation of the antidepressant, heart rhythm returned to normal values [32].

Venlafaxine is characterized by a relatively favorable safety profile with regard to interactions with cardiovascular medications. Authors emphasize that it is a weak inhibitor of the CYP2D6 isoenzyme and is also metabolized through the alternative CYP3A4 pathway, which contributes to a low risk of clinically significant drug interactions. However, analyses suggest that particular caution should be exercised when combining venlafaxine with other medications in elderly patients and in those with multimorbidity.

Acute Cardiac Events Associated with Venlafaxine Use

An important aspect in the context of the safe use of venlafaxine is the identification of its potential adverse effects that may lead to acute cardiac and vascular dysfunction.

A case report described a 45-year-old man who developed acute heart failure during treatment with venlafaxine. Due to a relapse of severe depression, the patient was initially treated with the following antidepressant medications: amitriptyline, chlordiazepoxide, and olanzapine. Owing to a lack of therapeutic efficacy, venlafaxine was introduced at a dose of 75 mg/day. After seven days of treatment, the patient developed acute heart failure accompanied by renal

and hepatic failure. Echocardiographic examination revealed global hypokinesia and severe left ventricular dysfunction. Clinical improvement was observed after discontinuation of venlafaxine. The authors suggested that venlafaxine may have contributed to the development of this condition; however, they also noted that a concurrent stressful life situation could have played a role [33].

Born et al. described a case of a 32-year-old patient who attempted suicide and ingested approximately 12 g of extended-release venlafaxine. As a result of the overdose, he developed acute heart failure and cardiogenic shock. Extracorporeal membrane oxygenation (ECMO) was initiated to maintain circulatory perfusion. After three months of treatment, cardiac function improved [34].

The available reports suggest that, in rare cases, venlafaxine use may be associated with the development of acute cardiovascular events. However, due to the limited number of observations and the presence of potential confounding factors, these findings do not allow for the establishment of a causal relationship; nevertheless, they justify particular caution in patients with predisposing risk factors.

Discussion

An analysis of the available data regarding the effects of venlafaxine on the cardiovascular system indicates a complex and inconclusive pattern of action. Differences among the findings of clinical trials, meta-analyses, and case reports suggest that the cardiovascular response to this medication may depend on the dose administered, as well as on individual patient susceptibility, comorbid conditions, and the duration of therapy.

The most frequently described adverse cardiovascular effect of venlafaxine is its impact on blood pressure. Although clinical studies have demonstrated a dose-related pattern, case reports indicate that hypertension may occur even at lower doses of the drug. These discrepancies may result from differences in diagnostic criteria for hypertension, variations in follow-up duration, failure to account for comorbidities in the studied populations, or interindividual variability in drug metabolism.

The effect of venlafaxine on cardiac electrophysiology remains inconclusive. The absence of significant QTc interval changes in most prospective studies contrasts with isolated clinical reports describing QTc prolongation. It should be noted that case reports often involve patients

with additional risk factors that are not always fully controlled or reported. This limits the ability to definitively attribute the observed changes directly to venlafaxine.

Similar interpretative challenges apply to reports of atrial fibrillation occurring during therapy. The available observations are descriptive in nature and involve small patient groups, which precludes determination of the true incidence of this phenomenon. Furthermore, patients treated with venlafaxine frequently present with coexisting risk factors for arrhythmia, such as advanced age, hypertension, or structural heart disease, which substantially complicates assessment of the drug's potential contribution.

Data regarding the effect of venlafaxine on metabolic parameters also remain inconclusive. Discrepancies between short-term studies and population-based analyses may indicate the importance of treatment duration, as well as the role of environmental and lifestyle factors that are not always accounted for in analyses.

The evaluation of venlafaxine interactions with cardiovascular medications is based primarily on pharmacokinetic data and isolated case reports. Although metabolic mechanisms suggest a low interaction potential, the actual clinical relevance of these findings remains difficult to estimate, particularly in elderly patients and those receiving multiple medications.

Conclusions

Venlafaxine is an effective antidepressant with an overall favorable cardiologic safety profile. Available data indicate that it may increase blood pressure not only at high doses but also at moderate doses. Therefore, regular blood pressure monitoring during therapy is important, regardless of baseline values. Although clinically significant electrophysiological disturbances are rarely observed during venlafaxine treatment, follow-up electrocardiographic assessment may be justified in patients with comorbid conditions or in those receiving higher doses of the medication. Isolated reports of atrial fibrillation and other arrhythmias are anecdotal in nature and most likely reflect individual susceptibility rather than a direct pharmacological effect of the drug. Venlafaxine is characterized by a relatively low potential for pharmacokinetic drug interactions. Its concomitant use with medications commonly prescribed in cardiology is generally considered safe; however, given the limited number of studies addressing this issue, caution should be exercised when combining venlafaxine with other agents. Further prospective studies involving cardiovascular patient populations are needed to more precisely evaluate the long-term cardiovascular and metabolic effects of treatment with this medication.

Disclosure Section

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