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DEPRESSION AND ANXIETY IN PATIENTS WITH CARDIOVASCULAR DISEASES: A NARRATIVE REVIEW OF PREVALENCE, MECHANISMS AND CLINICAL IMPLICATIONS

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ABSTRACT

Background: Cardiovascular diseases (CVD), including coronary artery disease, heart failure, and atrial fibrillation, remain major causes of morbidity and mortality worldwide. Depression and anxiety are highly prevalent in these populations and are associated with impaired quality of life, reduced functional status, and worse clinical outcomes.
Objective: This narrative review aimed to summarise current evidence on the prevalence, prognostic significance, and underlying mechanisms of depression and anxiety in patients with cardiovascular disease, and to discuss major diagnostic challenges, screening strategies, and treatment approaches relevant to clinical practice.
Methods: A narrative review of the literature was conducted using PubMed as the primary database, supplemented by searches in PubMed Central, Google Scholar, and selected open-access journals. Priority was given to meta-analyses, systematic reviews, clinical reviews, consensus statements, and key prospective or interventional studies published mainly between 2000 and early 2026, with a focus on coronary artery disease, heart failure, and atrial fibrillation. The review primarily emphasised studies published from 2020 onward, while earlier landmark papers were included when considered essential for contextual understanding.
Results: Depression and anxiety are common across major cardiovascular conditions and are associated with increased risks of incident cardiovascular disease, recurrent events, mortality, hospitalisation, and reduced health-related quality of life. Proposed mechanisms include autonomic and hypothalamic- pituitary-adrenal axis dysregulation, inflammation, endothelial dysfunction, platelet activation, and adverse health behaviours. Diagnostic overlap between cardiac and psychological symptoms contributes to underrecognition. Brief screening instruments such as the PHQ-9, GAD-7, and HADS appear feasible in routine care when linked to structured follow-up. Psychological interventions, selected antidepressants, and collaborative care models may improve mental health outcomes, although evidence for their effects on hard cardiovascular endpoints remains limited.
Conclusions: Depression and anxiety are clinically important comorbidities in cardiovascular disease and should be systematically addressed in cardiovascular care. Integrating mental health screening and multidisciplinary management into routine practice may enhance overall patient outcomes.

KEYWORDS

Cardiovascular Diseases, Coronary Artery Disease, Depression, Anxiety, Psychocardiology

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1. Introduction

Cardiovascular diseases (CVD), including coronary artery disease (CAD), heart failure (HF) and atrial fibrillation (AF), remain among the leading causes of morbidity and mortality worldwide [1,2]. At the same time, mental disorders, particularly depressive and anxiety disorders, are among the most common psychiatric conditions and have a substantial impact on functional status, quality of life and long-term prognosis [7]. Over the past two decades, accumulating epidemiological, clinical, and experimental evidence has shown that CVD and mood and anxiety disorders are closely inter-related, with associations that appear bidirectional in many studies. On the one hand, the presence of CVD increases the risk of developing depression and anxiety; on the other hand, depression and anxiety are associated with an increased risk of incident cardiovascular disease and with less favourable disease trajectories in those with established CVD [5,9].

Several meta-analyses and large observational studies indicate that depression is more prevalent in patients with CVD than in the general population, and that depressive symptoms frequently emerge or intensify in the context of acute cardiac events and chronic disease progression [5,6,7]. A recent meta-analysis including almost 40 studies reported that the overall prevalence of depression in patients with CVD is approximately 20%, with prevalence estimates of around 20% in CAD and about 25% in HF, while anxiety symptoms are present in nearly one quarter of CVD patients [6]. Similar figures were reported in a global systematic review and meta-analysis of depression, anxiety and stress in cardiac populations, which confirmed that affective and anxiety symptoms are highly prevalent across different cardiovascular diagnoses and care settings [7]. Consistent with these findings, narrative and systematic reviews emphasize that elevated anxiety and diagnosed anxiety disorders are markedly more frequent in individuals with CAD, HF and AF than in community samples [3,8,12,17].

Importantly, depression and anxiety in patients with CVD are not merely cooccurring conditions but key determinants of prognosis and disease trajectory [9,22]. Meta-analyses of prospective cohort studies have shown that depression is associated with an increased risk of developing CAD in initially healthy individuals and with a higher incidence of myocardial infarction and cardiac death over followup [10,23]. In HF, depressive symptoms following diagnosis are associated with substantially higher risks of allcause and cardiovascular mortality, more frequent hospitalisations and faster functional decline [11]. Anxiety has likewise been associated with an increased risk of incident CVD and adverse outcomes, although effect sizes vary between studies and depend on the type and severity of anxiety under investigation [12].

Beyond hard clinical endpoints, depression and anxiety influence intermediate outcomes that are crucial for longterm cardiovascular risk [9]. Patients with elevated depressive and anxiety symptoms are more likely to smoke, to be physically inactive and to maintain unhealthy dietary patterns, and they are less likely to adhere to pharmacological therapies, attend followup visits or participate in cardiac rehabilitation and secondary prevention programmes [13,30,31,41]. These behavioural patterns are likely to mediate part of the observed associations between mental disorders and cardiovascular events and may also contribute to poorer control of blood pressure, lipids and glycaemia [9,22]. As a result, targeting both psychological distress and health behaviours is increasingly recognised as a central component of comprehensive cardiovascular risk management [27,37].

There is growing evidence that these behavioural mechanisms interact with a range of biological pathways [22]. Proposed biological mechanisms include chronic activation of the hypothalamic-pituitary-adrenal axis, autonomic nervous system dysregulation with reduced heart rate variability, heightened sympathetic activity, systemic inflammation, platelet activation, procoagulant changes and endothelial dysfunction [26,28,29]. These alterations can promote atherogenesis, plaque instability, arrhythmogenesis and adverse remodelling, thereby providing plausible pathways through which depression and anxiety may influence both the onset and progression of CVD [26,27]. In patients with HF, CAD and AF, these mechanisms are superimposed on existing structural and haemodynamic abnormalities, which may amplify their impact on symptoms and outcomes [18,14,17].

From a clinical perspective, a major challenge is the substantial overlap between the symptoms of CVD and those of depressive and anxiety disorders, which complicates accurate recognition and diagnosis [3]. Dyspnoea, palpitations, chest tightness or pain, fatigue and dizziness may reflect myocardial ischaemia, HF or arrhythmias, but they may also occur in the context of anxiety disorders, including panic attacks and generalised anxiety disorder [3]. Similarly, symptoms such as low energy, sleep disturbances, reduced appetite and loss of interest may be interpreted as inevitable consequences of chronic somatic illness or ageing rather than as manifestations of a treatable depressive disorder [3]. These diagnostic complexities contribute to both

under and overdiagnosis of mental disorders in cardiology settings and underline the need for systematic, structured approaches to screening and further assessment [3].

Given the growing number of studies examining the relationships between depression, anxiety and cardiovascular diseases, there is a clear need for a focused narrative review that synthesises current evidence on the prevalence of depressive and anxiety disorders across major CVD entities, their impact on prognosis and treatment, and the available psychological and organisational interventions [9,22]. The aim of the present narrative review is to provide an uptodate overview of depression and anxiety in patients with CAD, HF and AF and to highlight key clinical implications for cardiovascular practice, including opportunities for systematic screening, integrated care and future research.

2. Methods of Literature Search

In this narrative review, we aimed to summarise current evidence on depression and anxiety in patients with cardiovascular diseases, with a particular focus on coronary artery disease (CAD), heart failure (HF), and atrial fibrillation (AF). The primary source of literature was the PubMed database, which provides broad coverage of biomedical and clinical research, and this search was supplemented by consultations of PubMed Central (a full-text repository) and Google Scholar, as well as selected open-access journals indexed in the Directory of Open Access Journals (DOAJ) and on the BioMed Central platform. In addition, relevant systematic reviews, meta-analyses, and clinical practice guidelines from the Cochrane Library and from major cardiology and psychiatry societies were examined to capture high-level evidence on the screening, prognosis, and management of depression and anxiety in cardiovascular populations.

The search strategy combined Medical Subject Headings (MeSH) and free-text keywords related to cardiovascular conditions and mood and anxiety disorders, including terms such as “cardiovascular diseases”, “coronary artery disease”, “heart failure”, “atrial fibrillation”, “depression”, “depressive disorder”, “anxiety”, “anxiety disorders”, “stress”, and “psychological distress”, together with methodological filters such as “meta-analysis”, “systematic review”, “review”, “prospective cohort”, “randomized controlled trial”, and “psychological intervention”. The core search primarily focused on articles published in English between January 2000 and early 2026; earlier seminal studies were additionally included when they provided historically important data on the associations between depression or anxiety and cardiovascular disease or on mechanistic pathways.

Priority was given to meta-analyses, systematic reviews, large narrative reviews, and clinical practice guidelines or consensus statements addressing depression, anxiety, and cardiovascular disease, reflecting the predominance of such sources in the contemporary psychocardiology literature. In addition, selected prospective cohort studies and randomised clinical trials were included when they provided relevant data on the associations between mental disorders and incident CVD or prognosis, or on the effectiveness of psychological, pharmacological, or collaborative care interventions in cardiovascular populations. Articles were selected based on thematic relevance and perceived clinical importance. A formal risk-of-bias assessment was not performed due to the narrative design of this review, and the most informative studies were used to synthesize evidence on epidemiology, prognosis, pathophysiological mechanisms, and treatment strategies across CAD, HF, and AF.

3. Epidemiology

Depression is among the most common co-occurring mental disorders in patients with cardiovascular diseases [22]. A recent meta-analysis of 39 studies including 63,444 patients with various forms of CVD found an overall prevalence of depression of 20.8%, with rates of 19.8% in coronary artery disease and 24.7% in heart failure, and an overall anxiety prevalence of 23.2% among patients with CVD [6]. A global systematic review and meta-analysis of cardiac patients reported even higher pooled estimates, with depression present in 31.3% and anxiety in 32.9% of patients, confirming that mood and anxiety symptoms are highly prevalent across cardiac populations [7].

In heart failure, a classic metaanalytic review of 36 studies showed clinically significant depression in 21.5% of patients overall, with prevalence ranging from approximately 11% in NYHA class I to 42% in NYHA class IV, and with higher rates reported when questionnaire cutoffs rather than diagnostic interviews were used [18]. More recent reviews similarly report that around one fifth to over one third of patients with HF experience depressive symptoms, and they link depression to increased mortality, rehospitalisation, and poorer quality of life [11,19]. These findings indicate that depression is common across the spectrum of HF severity and is consistently linked to poorer quality of life and worse clinical outcomes [11].

Anxiety is also highly prevalent among patients with cardiovascular disease. A global systematic review and meta-analysis of adult cardiology outpatients, including 93 studies and 36,687 participants, found a pooled prevalence of anxiety symptoms or anxiety disorders of 28.9%, substantially higher than general population estimates of around 5-7% [20]. Subgroup analyses in that study showed particularly high rates among patients with hypertension, undifferentiated chest pain, and palpitations [20]. Another global meta-analysis focusing on cardiac patients likewise identified anxiety as common and highlighted the need to routinely assess anxiety alongside depression in cardiology settings [7].

In atrial fibrillation, a systematic review specifically examining depression and anxiety in AF concluded that both conditions are clearly overrepresented compared with the general population. Across the studies summarised in that review, anxiety symptoms commonly affected roughly one third of patients with AF, while depressive symptoms affected around one fifth, although estimates varied according to the psychometric instruments and thresholds applied [17].

Differences in reported prevalence rates across the literature partly reflect heterogeneity in samples, settings, and measurement methods. Meta-analyses of HF, CVD in general, and AF have noted that prevalence estimates are higher when self-report scales such as the Hospital Anxiety and Depression Scale (HADS) or the Beck Depression Inventory (BDI) are used with relatively low cut-offs, and lower when structured diagnostic interviews and stricter criteria are applied. Despite this variability, contemporary meta-analyses and systematic reviews consistently support the view that depressive and anxiety disorders, as well as subthreshold symptoms, are markedly more frequent in patients with cardiovascular diseases than in the general population and therefore represent a major clinical issue in cardiovascular care [6,7,17,18].

Depression and anxiety in patients with cardiovascular diseases have a significant adverse impact on clinical outcomes [22]. Meta-analyses of prospective cohort studies have shown that depression is independently associated with an increased risk of developing coronary heart disease and other cardiovascular events, even after adjustment for traditional risk factors [10,23]. Among patients with established coronary artery disease, the presence of depression is linked to higher cardiovascular and all-cause mortality and to an increased risk of recurrent coronary events [23].

4. Prognostic Impact

A large meta-analysis of 26 prospective cohort studies including nearly 2 million participants showed that major depression is associated with an increased risk of incident cardiovascular disease and worse outcomes in those with established CVD. In this analysis, depression was linked to higher risks of incident stroke, myocardial infarction, heart failure and any cardiovascular disease, as well as increased all-cause and cardiovascular mortality in individuals with CVD (with hazard ratios generally in the range of approximately 1.1–1.4) [22]. An updated meta-analysis of prospective cohort studies focusing specifically on coronary heart disease similarly demonstrated that depression is associated with about a 30% higher risk of myocardial infarction and coronary death [23].

In heart failure, depression is a consistent marker of poorer prognosis [11][18]. A meta-analysis of prospective studies showed that depressive symptoms are associated with a significantly increased risk of all-cause mortality in patients with heart failure, with stronger effects in older patients and in studies with shorter follow-up durations [11]. Cohort studies both hospitalised and community heart failure populations likewise report that patients with comorbid depression have substantially higher risks of death and heart failure–related hospitalisation over one to several years of follow-up compared with non-depressed patients, with hazard ratios typically in the range of about 1.3 to 2.0 after adjustment for clinical covariates [11][24]. Together, these data indicate that depression is not only common in heart failure but also an important prognostic factor that adds information beyond traditional clinical risk markers [11,18,24].

Recent work combining meta-analysis with Mendelian randomisation further suggests that the relationship between depression and cardiovascular disease may be partly causal, although residual confounding and methodological limitations cannot be fully excluded [6]. In a large study of patients with various CVD subtypes, depressive symptoms were associated with higher risks of all-cause mortality, and genetically informed analyses indicated that depression plays a critical role in the development and progression of specific cardiovascular conditions, including coronary artery disease, myocardial infarction, and heart failure [6]. These findings are in line with broader narrative reviews and meta-reviews that highlight depression as an independent risk factor for incident CVD and for adverse outcomes after cardiac events [6][22].

Anxiety has also been linked to unfavourable prognosis in cardiovascular populations, although the evidence is more heterogeneous than for depression [8]. In a metaanalysis of 16 studies including 9,373

patients after myocardial infarction, elevated anxiety was associated with an increased risk of overall poor clinical outcomes, including higher rates of shortterm complications and longterm adverse prognosis, such as mortality and major adverse cardiac events, compared with patients without high anxiety [25]. In a broader metaanalysis of cohort studies, anxiety disorders and clinically significant anxiety symptoms were associated with an increased risk of cardiovascular disease overall (with summary risk estimates in the range of approximately 1.3–1.4) and with higher risks of coronary heart disease, stroke, and heart failure, whereas associations with atrial fibrillation and composite major cardiovascular events were weaker and less consistent [8]. Narrative reviews integrating these metaanalytic findings conclude that anxiety frequently cooccurs with depression in cardiovascular patients and may contribute to increased event rates and mortality, but also note that the independent effect of anxiety varies between studies depending on how anxiety and depression are defined, measured, and statistically adjusted [8][22].

Overall, contemporary metaanalyses and highquality reviews provide converging evidence that depression is a consistent, albeit modest, predictor of incident cardiovascular disease, recurrent events, and mortality across coronary artery disease, heart failure, and broader CVD populations [9,22], while anxiety is also associated with poorer prognosis, particularly after myocardial infarction and in highrisk groups [8,25]. These findings support the systematic assessment and management of depressive and anxiety symptoms as an integral part of cardiovascular risk stratification and secondary prevention [22,37].

5. Biological and Behavioural Mechanisms

Multiple, interacting biological and behavioural mechanisms are thought to underlie the association between depression, anxiety, and cardiovascular disease [9,22]. Contemporary reviews emphasise that neuroendocrine dysregulation, activation of inflammatory pathways, autonomic imbalance, endothelial dysfunction, platelet activation, and adverse health behaviours form a network of partly overlapping pathways linking mood and anxiety disorders with atherosclerosis, coronary events, and heart failure [16,27,29].

Neuroendocrine alterations include hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis and elevated cortisol levels, which promote central obesity, insulin resistance, dyslipidaemia, and a metabolicsyndrome-like state, thereby increasing cardiovascular risk [26,28]. Chronic stress and persistent depressive symptoms have also been associated with higher circulating concentrations of proinflammatory markers such as Creactive protein and interleukin6, and these inflammatory changes are implicated in the development and progression of atherosclerosis, plaque instability, and thrombosis [26,28,29]. At the same time, autonomic nervous system dysfunction, characterised by increased sympathetic activity and reduced parasympathetic tone, can lead to elevated heart rate, reduced heart rate variability, and a greater propensity to arrhythmias and myocardial ischaemia [26,29].

Endothelial dysfunction and platelet activation represent additional vascular pathways [26,27]. Depression has been linked to impaired endothelial nitric oxide bioavailability and increased markers of endothelial activation, which may facilitate vasoconstriction, impaired coronary flow, and microvascular dysfunction [26,29]. Several reviews also highlight enhanced platelet reactivity and aggregation in patients with depression, providing a plausible mechanism for an increased risk of coronary thrombosis and acute coronary syndromes [26,27]. Genetic and molecular studies suggest shared pathways between depression and cardiovascular disease involving altered brainderived neurotrophic factor signalling, oxidative stress, and gut-brain-heart axis regulation, but this evidence remains preliminary [26][28].

Behavioural mechanisms play a crucial complementary role [9]. Depressed and anxious patients with cardiovascular disease are more likely to smoke, be physically inactive, follow unhealthy diets, and show poorer adherence to cardioprotective medications and cardiac rehabilitation programmes [13,30,41]. Prospective and interventional studies indicate that persistent depressive symptoms are consistently associated with poorer adherence to antiplatelet agents, beta-blockers, statins, and lifestyle recommendations, which in turn is linked to increased rehospitalisation and mortality [30,31]. Analyses from the UK Biobank prospective cohort study further showed that lifestyle factors such as current smoking and central obesity, together with metabolic mediators including elevated systolic blood pressure and C-reactive protein, each explained around 10–17% of the excess cardiovascular risk associated with depression and anxiety disorders, indicating that these factors partially mediate the pathway from mental disorders to incident cardiovascular disease [32].

Finally, several recent reviews stress that these biological and behavioural mechanisms do not operate in isolation but are tightly interconnected [9,22,26]. HPA-axis hyperactivity can amplify sympathetic activation and inflammatory responses; inflammation can further impair endothelial function and alter central nervous system circuits involved in mood regulation; and maladaptive health behaviours can exacerbate cardiometabolic risk, contributing to a self-perpetuating cycle between psychological distress and cardiovascular disease [26,28,29,32].

6. Clinical Challenges and Diagnostic Difficulties

In clinical practice, a major challenge is the substantial overlap between the somatic symptoms of cardiovascular disease and those of anxiety and depressive disorders, which makes accurate diagnosis difficult [3]. Symptoms such as palpitations, chest pain, dyspnoea, dizziness, nausea, and sweating can arise both in the context of acute coronary syndromes, arrhythmias, or heart failure and during panic attacks or generalised anxiety, so that many typical panic symptoms may also reflect cardiac pathology [3]. Similar diagnostic ambiguity exists for core depressive features such as fatigue, low energy, poor concentration, and sleep disturbances, which are very common in patients with cardiac disease particularly heart failure and are easily attributed to the underlying cardiovascular condition rather than to a potentially treatable mood disorder [3,18,19].

An additional illustration of these diagnostic and communication challenges comes from the emergency department setting, where many patients present with chest pain but have a low estimated risk of acute coronary syndrome [34]. In a survey of emergency medicine providers, clinicians estimated that roughly 30% of patients with low-risk chest pain have anxiety or panic as the primary cause of their symptoms, even though objective cardiac pathology is usually absent in this group [34]. However, the same study showed that providers directly communicate their suspicion of anxiety to only about 42% of these patients and document a specific anxiety diagnosis in fewer than one third of cases, while tailored written discharge instructions addressing anxiety are provided in less than half [34]. Moreover, only a minority of respondents felt that their hospital has adequate resources or structured pathways for follow-up of patients with presumed anxiety-related chest pain, and the vast majority expressed a need for dedicated clinics or clear referral options for further assessment [34]. These findings suggest a marked gap between clinicians' recognition of anxiety as a driver of recurrent, low-risk chest pain and the actual implementation of diagnostic labelling, patient-centred communication, and appropriate mental health referral, which may contribute to repeated emergency department utilisation and persistent symptom burden [34].

Beyond the acute setting, broader cardiology cohorts show that depression and anxiety in cardiac patients remain highly prevalent, under-recognised, and insufficiently screened for in routine care [33,36,38]. A recent review on diagnosing and screening depression and anxiety in cardiac disease emphasises that roughly 20–40% of patients with coronary artery disease or heart failure have elevated depressive symptoms and that anxiety may be at least as common, yet systematic, guideline-based screening is rarely implemented [33,37]. Many cardiology services do not routinely use validated instruments such as the PHQ-9 (Patient Health Questionnaire-9) or HADS, and mental health assessment is often triggered only by overt distress rather than embedded into standard workflows, so clinically significant depression or anxiety is frequently missed when somatic symptoms can plausibly be attributed to underlying cardiovascular disease [33,36,37,38].

In patients with atrial fibrillation, Lomper et al. found that anxiety and depressive symptoms are strongly related to how patients perceive their arrhythmia and to their quality of life [35]. In a cohort of 116 hospitalised AF patients assessed with the HADS, about one in five had markedly elevated anxiety and almost one in ten had markedly elevated depressive symptoms, while most showed at least some psychological distress [35]. Higher anxiety and depression scores were significantly associated with worse AF-specific quality of life on the ASTA questionnaire, with moderate correlations indicating greater physical and psychological limitations and lower overall well-being [35]. The authors conclude that overlapping AF symptoms (such as palpitations, dyspnoea, and fatigue) and affective symptoms substantially impair quality of life, underscoring the need for routine assessment and targeted management of anxiety and depression in AF care [35].

Taken together, evidence from emergency departments, general cardiology populations, atrial fibrillation cohorts, and heart failure studies indicates that clinical challenges around depression and anxiety in cardiovascular disease extend well beyond simple symptom overlap [18,19,17,34]. Anxiety can drive recurrent lowrisk chest pain presentations without adequate diagnostic labelling or referral, depression and anxiety remain underdetected in routine cardiology despite high prevalence, and both disorders strongly influence symptom perception, quality of life, and patterns of helpseeking [18,19,33,34]. Addressing these challenges requires routine use of brief, validated screening tools, clearer communication about suspected anxietyrelated presentations, and explicit referral pathways to mental health care within cardiovascular services [33,36,37,38].

7. Screening and Assessment Tools

Given the high prevalence and prognostic importance of depression and anxiety in cardiovascular disease, brief standardised questionnaires have emerged as key tools for case finding in cardiology settings [33,37]. Current evidence indicates that instruments such as the PHQ9 and GAD7 (Generalized Anxiety Disorder-7) can be integrated into routine workflows with high completion rates and minimal time burden, while still identifying a substantial proportion of patients with clinically relevant psychological distress who might otherwise go unnoticed [33,36,37].

In studies of cardiac inpatients, systematic use of the PHQ-9 for depressive symptoms and the GAD-7 for anxiety has shown that a meaningful minority of patients screen above clinical cut-offs, supporting the notion that routine, rather than symptom-triggered, screening is necessary to capture the true burden of mood and anxiety disorders in this population [36,38]. Expert reviews and position papers further argue that a stepped strategy using very brief initial questions followed by more detailed instruments like the PHQ-9 for depression and the GAD-7 for anxiety offers a practical balance between feasibility and diagnostic accuracy in busy cardiovascular services [33,37]. Importantly, these sources emphasise that the effectiveness of screening depends not only on the choice of questionnaire but also on clearly defined referral pathways and access to psychocardiology or mental health support, so that positive screening results consistently lead to more comprehensive assessment and appropriate intervention rather than remaining an isolated administrative exercise [33,36,37].

This Norwegian cardiology-department study adds a few important, very practical points to the assessment-tools section [38]. The authors showed that using ultra-brief PHQ-2 and GAD-2 questions (plus one panic-attack item from the PHQ-SADS) in routine outpatient and post-discharge visits identified depressive or anxiety symptoms in about one quarter of cardiac patients, confirming that clinically relevant distress is common even outside the acute phase [38]. Most patients who screened positive on the PHQ-2/GAD-2 also scored above the cut-off on the HADS, which supports the use of a stepped screening approach with short initial questions followed by a more detailed instrument that minimises overlap with somatic cardiac symptoms [38]. At the same time, the study highlights typical barriers to implementation reported by clinicians, including time constraints, uncertainty about benefits, and lack of clear follow-up procedures, suggesting that successful screening depends as much on organisational pathways and access to psychological follow-up as on the choice of questionnaire itself [38].

Taken together, these findings suggest that no single instrument is sufficient on its own; rather, a layered strategy that combines ultra-brief initial questions with more detailed, validated scales such as the PHQ-9, GAD-7, or HADS is most appropriate for cardiology settings [33,37,38]. Such an approach recognises the high burden and clinical relevance of depression and anxiety, while remaining feasible in busy services, and it creates a structured entry point from simple screening to more comprehensive psychocardiological assessment and tailored intervention when needed [33,36,37,38].

8. Treatment Strategies

Management of depression and anxiety in patients with cardiovascular diseases should combine pharmacological and psychological approaches with lifestyle modification, taking cardiovascular safety into account [22,27,37]. Systematic reviews and meta-analyses indicate that psychological interventions, particularly cognitive-behavioural therapy (CBT), psychoeducation, and stress-management programmes, effectively reduce depressive and anxiety symptoms in patients with coronary artery disease, heart failure, and atrial fibrillation, and improve health-related quality of life [4,39,40]. Cardiac rehabilitation programmes that integrate exercise training, education, and psychological support can further alleviate depressive and anxiety symptoms following acute coronary syndromes [4,39,41].

Antidepressant pharmacotherapy should be chosen with explicit consideration of cardiovascular risk profiles and potential drug-disease and drug-drug interactions [22,27,37]. Across multiple trials and reviews in patients with coronary heart disease, selective serotonin reuptake inhibitors (SSRIs) have emerged as first-line options, showing efficacy in improving depressive symptoms with generally favourable cardiovascular safety [21,22,42]. Data from pivotal studies such as SADHART and ENRICHD support the use of sertraline and other SSRIs in post-myocardial infarction and stable coronary heart disease populations with no major adverse impact on left ventricular function or arrhythmic risk and with inconsistent signals regarding morbidity and cardiovascular outcomes [21,43]. In contrast, antidepressants associated with a greater propensity for QT prolongation or proarrhythmic effects warrant particular caution in patients with atrial

fibrillation or other rhythm disorders, and treatment decisions in complex cases with severe depression, suicidality, or multimorbidity are best made collaboratively with psychiatry. [22,27,37].

Collaborative care has been proposed as a structured treatment model for depression in patients with coronary heart disease, particularly when care is fragmented across specialties [15,37]. In this model, treatment is delivered by a multidisciplinary team and includes a structured management plan, scheduled follow-up, and enhanced interprofessional communication [15,47]. In a meta-analysis of six randomized controlled trials involving 1,284 patients, collaborative care was associated with a significant short-term reduction in depressive symptoms and a higher likelihood of depression remission compared with usual care [15]. However, although short- to medium-term reductions in major adverse cardiac events were observed, these effects were not sustained over longer follow-up [15].

Psychological treatment remains an important component of care in patients with cardiovascular disease, particularly in those with heart failure, where psychosocial distress is common and closely linked to poorer quality of life [18,19]. A systematic review and metaanalysis of psychosocial interventions in heart failure showed that cognitive behavioural therapy and stressmanagement programs significantly reduced both depressive and anxiety symptoms compared with control conditions [4,40]. The same analysis found that CBT was also associated with improved quality of life, whereas evidence for effects on hospitalization and mortality remained limited and less consistent [4,18]. These findings suggest that psychological interventions are most strongly supported as a means of improving mental health outcomes and patientreported wellbeing, rather than as a proven strategy for reducing hard cardiovascular endpoints [4,18,41]

9. Discussion

This narrative review highlights that depression and anxiety are not peripheral comorbidities in cardiovascular medicine but clinically meaningful factors that influence symptom burden, quality of life, treatment adherence, and longterm prognosis [22]. Across coronary artery disease, heart failure, and atrial fibrillation, the literature consistently shows that depressive and anxiety symptoms are substantially more common than in the general population and are associated with poorer patient-reported outcomes as well as less favourable clinical trajectories [7,20]. Taken together, these findings support the view that mental health should be considered part of the core clinical profile of cardiac patients rather than a secondary or optional domain of assessment.

A central message is that the relationship between cardiovascular disease and depression or anxiety is bidirectional and multifactorial: cardiac illness can precipitate psychological distress, while depression and anxiety may contribute to disease progression through behavioural and biological mechanisms already outlined above.

Another major theme is the persistent diagnostic challenge created by the overlap between psychiatric and cardiovascular symptoms [33,36]. Symptoms such as dyspnoea, fatigue, dizziness, chest discomfort, and palpitations may reflect myocardial ischaemia, arrhythmia, heart failure progression, or panic and generalized anxiety, making interpretation difficult in both acute and chronic settings [34]. Similarly, reduced energy, sleep disturbance, poor concentration, and loss of interest may be attributed to chronic somatic illness, older age, or deconditioning rather than recognised as possible manifestations of depression. The practical consequence is a dual risk: some patients may have organic disease insufficiently investigated because symptoms are labelled as “functional,” whereas others with established cardiovascular disease may have clinically relevant anxiety or depression that remains undetected and untreated [36,37].

The reviewed evidence also suggests that depression and anxiety modify how patients perceive and report their cardiovascular symptoms, which further complicates clinical care [30,31]. Anxiety may heighten vigilance toward bodily sensations and increase the likelihood that benign or nonspecific symptoms are interpreted as dangerous, contributing to repeated presentations and higher healthcare utilisation. Conversely, depressive symptoms may reduce motivation, blunt symptom reporting, and delay help-seeking, particularly in conditions such as heart failure where self-monitoring and timely response to deterioration are crucial. This means that the clinical relevance of depression and anxiety extends beyond psychiatric diagnosis itself and directly affects the diagnostic process, patient behaviour, and the timing of intervention.

Available data indicate that brief screening for depression and anxiety in cardiology is feasible and can uncover substantial unrecognised distress. However, questionnaires are clinically useful only when embedded in structured pathways that link positive results to diagnostic clarification and appropriate psychological or psychiatric care, rather than functioning as stand-alone administrative tasks.

The therapeutic literature reviewed here also points to a nuanced but clinically useful message. Psychological interventions, particularly cognitive behavioural therapy and broader psychosocial programmes, are supported by the most consistent evidence for improving depressive symptoms, anxiety, and health-related quality of life in cardiac populations. Pharmacological treatment, especially with SSRIs, appears generally acceptable from a cardiovascular safety perspective in selected populations, but the evidence for major benefits on hard cardiovascular endpoints remains less convincing than the evidence for improvement in mental health symptoms. Similarly, collaborative care and integrated models appear promising because they address the reality that cardiac patients often need coordinated management across specialties, yet even here the strongest benefits are seen in symptom reduction and care processes rather than durable reductions in mortality or recurrent cardiovascular events [15,43].

Overall, the most balanced interpretation of the current literature is that depression and anxiety in cardiovascular disease should be treated as clinically significant, modifiable contributors to illness burden, but not as isolated targets whose treatment alone can be expected to transform cardiovascular prognosis. Their management is most likely to be effective when incorporated into broader, patient-centred cardiovascular care that also addresses risk factors, rehabilitation, adherence, communication, and long-term follow-up.

10.Clinical Implications

From a clinical perspective, the evidence supports a more deliberate integration of mental health assessment into routine cardiology practice [37]. In practical terms, this means that patients with CAD, HF, or AF particularly those with persistent symptom burden, reduced adherence, repeated admissions, or poor quality of life should be considered for structured screening for depression and anxiety using brief validated tools, where feasible within local resources and workflows [33]. It also means that clinicians should remain alert to the possibility that worsening cardiac symptoms may coexist with psychological distress rather than assuming that the presentation is exclusively organic or exclusively psychiatric [37].

Equally important, positive screening results should not be viewed as a diagnosis in themselves, but as a signal for more detailed assessment and, where appropriate, referral for psychological or psychiatric evaluation [37]. A collaborative model involving cardiologists, primary care clinicians, psychologists, and psychiatrists may be particularly helpful in patients with recurrent admissions, multimorbidity, or persistent depression and anxiety despite standard cardiac treatment [15]. Such an approach aligns with the broader movement in cardiovascular medicine toward more holistic, person-centred care.

11.Limitations

This review has several limitations that should be acknowledged. First, as a narrative review, it does not aim to provide a fully systematic or exhaustive synthesis of all available studies, and some degree of selection based on thematic relevance and clinical importance is unavoidable. Second, the underlying literature is highly heterogeneous with regard to cardiovascular populations, methods of psychiatric assessment, cut-off values for screening instruments, and clinical endpoints, which limits direct comparison across studies and may partly explain differences in reported prevalence or treatment effects. Third, a substantial part of the evidence base remains observational, particularly in the areas of prognosis, behavioural mediators, and implementation of screening, which constrains causal inference. Fourth, we did not perform a formal, toolbased assessment of study quality or risk of bias, which limits the ability to weigh individual findings according to methodological rigour.

Future research should move beyond documenting the high prevalence of depression and anxiety in cardiovascular disease and focus more directly on how mental health assessment and intervention can be integrated into routine cardiac care in a practical and effective way. Large prospective studies are still needed to identify which cardiovascular subgroups are at the greatest risk of persistent psychological distress and to determine whether the prognostic impact of depression and anxiety differs according to disease phenotype, symptom burden, age, sex, or frailty.

Another major priority is to refine screening pathways. Although instruments such as the PHQ-9, GAD-7, and HADS are widely used, further research should clarify the optimal timing and frequency of screening, compare single-time-point versus repeated assessment, and determine whether different cardiac populations require different tools or thresholds [37]. Equally important, future studies should evaluate integrated psychocardiology models in which screening is directly linked to referral, collaborative care, and follow-up, rather than functioning as an isolated administrative step [15].

Intervention research should also become more targeted. Randomized trials are needed to compare different treatment approaches, including CBT, stress-management programmes, rehabilitation-based

interventions, and digital or hybrid models of care, and to determine whether these strategies improve not only psychological symptoms and quality of life but also adherence, hospitalizations, and other clinically meaningful outcomes [4]. Greater standardization of outcome measures and follow-up intervals would further strengthen the evidence base and improve translation of research findings into routine cardiovascular practice.

12.Future Directions

Future directions in psychocardiology should prioritise moving beyond descriptive prevalence data toward interventions that can be implemented at scale in routine cardiovascular care [7, 22]. Large prospective cohort studies are needed to clarify how depression and anxiety differentially affect prognosis across specific cardiovascular phenotypes, including coronary artery disease, heart failure, and atrial fibrillation, and to identify subgroups at highest risk according to age, sex, symptom burden, and multimorbidity [6, 32]. Such work should pay particular attention to the trajectories of psychological distress over time, examining which patients develop persistent, recurrent, or late-onset symptoms and how these patterns relate to adverse clinical outcomes [7, 9].

Future research should refine screening strategies by clarifying the optimal timing, frequency, and setting for brief instruments in different cardiac populations, and by testing integrated pathways in which positive screens trigger structured assessment and followup. Comparative studies are needed to assess whether single-time-point screening, repeated assessment, or triggered screening based on clinical events (for example, hospitalisation or device therapy) is most effective in identifying clinically meaningful distress and guiding management [36, 38]. In parallel, implementation research should evaluate integrated screening pathways in which positive results are directly linked to structured diagnostic assessment, clear referral routes, and access to psychological or psychiatric care rather than remaining isolated administrative steps [15, 37].

Intervention studies represent another key priority. Randomised controlled trials should compare different treatment approaches including cognitive behavioural therapy, broader psychosocial programmes, and rehabilitation-based interventions to determine which strategies most effectively improve depressive and anxiety symptoms, health-related quality of life, and adherence in specific cardiovascular conditions [4, 39, 41]. Greater emphasis is also needed on evaluating hard outcomes such as mortality, myocardial infarction, heart failure hospitalisation, stroke, and recurrent arrhythmia, as current evidence suggests more consistent benefits for symptoms and quality of life than for major adverse cardiovascular events [4, 15]. Digital and hybrid models of care, including ehealth and blended collaborative approaches, warrant further investigation as potentially scalable options, although robust outcome data in cardiac populations are still limited [15, 37].

Finally, future work should deepen understanding of the behavioural and biological mechanisms linking depression and anxiety with cardiovascular risk, with a view to identifying more precise therapeutic targets. Studies examining autonomic dysregulation, hypothalamic–pituitary–adrenal axis activation, inflammation, and platelet and endothelial dysfunction, alongside health behaviours such as medication adherence, physical activity, and smoking, may help explain why psychological distress confers excess cardiovascular risk beyond traditional factors [26, 27, 29, 30]. Integrating mechanistic insights with large-scale epidemiological data could support a more personalised approach, in which screening, treatment, and follow-up strategies are tailored to individual risk profiles rather than applied uniformly to all patients with cardiovascular disease [6, 22, 32].

13.Conclusions

Depression and anxiety are common, clinically relevant, and still insufficiently recognised comorbidities in patients with cardiovascular disease. Their importance extends beyond emotional distress alone, as they influence symptom perception, healthcare-seeking behaviour, adherence, quality of life, and, at least in part, cardiovascular prognosis. Taken together, these effects mean that depression and anxiety should be regarded as integral components of the clinical profile of cardiac patients rather than optional or peripheral concerns.

The available evidence supports routine consideration of mental health in cardiology, including the use of brief validated screening tools and the development of integrated care pathways that connect detection with meaningful intervention. Such pathways are most effective when they offer clear routes to psychological or psychiatric assessment and when they are embedded within multidisciplinary, patient-centred models of cardiovascular care. Although further research is needed to clarify whether treating depression and anxiety improves hard cardiovascular outcomes, incorporating psychocardiological assessment and management into standard practice already appears justified on clinical and patientcentred grounds.

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