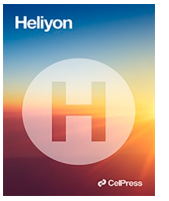
Heliyon 10 (2024) e27484

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon



Review article

Exploration of therapeutic models for psycho-cardiology: From cardiac to psychological rehabilitation

Yu Ren [a,1], Huilin Tang [b,1], Liwei Zhang [c,1], Chenfei Ying [c], Hua Luo [c,*]

[a] Department of Pharmacy, Taizhou Hospital of Zhejiang Province Affiliated to Wenzhou Medical University, Taizhou, 317000, Zhejiang, China
[b] Department of General Surgery, Taizhou Hospital of Zhejiang Province Affiliated to Wenzhou Medical University, Taizhou, 317000, Zhejiang, China
[c] Department of Orthopedic, Taizhou Hospital of Zhejiang Province Affiliated to Wenzhou Medical University, Taizhou, 317000, Zhejiang, China

ARTICLE INFO

Keywords:
Psycho-cardiology
Rehabilitation
Therapeutic
Cardiovascular diseases
Psychological

ABSTRACT

The prevalence and mortality of cardiovascular disease are relatively high. Currently, depression has been proven to be an independent risk factor for the occurrence and poor prognosis of cardiovascular disease. Psycho-cardiovascular comorbidity, as a reciprocal cause and effect, affects each other, leading to the deterioration of clinical prognosis and forming a vicious circle. Coronary artery disease comorbidity with depression is a common disease in psycho-cardiology medicine. This paper expounds on the exploration of the treatment model of psycho-cardiology from the aspects of epidemiological characteristics, comorbidity mechanism, screening, diagnosis, and treatment.

1. Introduction

Psycho-cardiology is the correlation between cardiovascular diseases (CAD) and psychological factors, such as white-coat hypertension. Some patients experience a rapid rise in blood pressure when they see a doctor in a white coat. What appears to be a problem of elevated blood pressure is hypertension caused by fear and mental tension under the action of the anxiety mechanism. Cardiovascular disease comorbidities with psychiatric disorders have a high prevalence and low diagnostic rate. The common symptoms such as chest tightness, chest pain, palpitation, shortness of breath, and dyspnea increase the difficulty of diagnosis, resulting in a high rate of misdiagnosis and missed diagnosis of psycho-cardiology diseases. High-cost and even invasive examinations and irrational drug use have caused a large consumption of medical resources, which has caused a heavy economic burden to society and individuals. Patients' unhealthy emotions, such as anxiety, depression, tension, and worry, seriously affect the quality of their lives.

Psycho-cardiology therapeutic models regard "psychological factors" as part of the "heart disease prevention and control system" to identify and intervene with similar CAD symptoms interfered by psychological factors or pure mental problems.

* Corresponding author. Department of Orthopedic, Taizhou Hospital of Zhejiang Province affiliated to Wenzhou Medical University, No. 150, Ximen Street, Linhai, Taizhou, Zhejiang, 317000, China.
E-mail address: 18732196660@163.com (H. Luo).
[1] Yu Ren, Huilin Tang, and Liwei Zhang contributed equally to this work.

https://doi.org/10.1016/j.heliyon.2024.e27484
Received 13 November 2022; Received in revised form 13 February 2024; Accepted 29 February 2024
Available online 15 March 2024

1.1. Epidemiological characteristics of psycho-cardiology diseases

Carney et al. observed in 1988 that depression doubles the risk of cardiac events in individuals newly diagnosed with heart disease [1]. Studies indicate that the prevalence of depression among patients with CAD ranges from 25% to 40%, significantly higher than that in the general population [2]. Depression stands as an independent risk factor substantially increasing the likelihood of both coronary artery disease and myocardial infarction [3–5]. O 'Neil et al. [6] proved that the association between depression and CAD prevalence surpasses that of traditional risk factors. Recent research published in JAMA Network Open adds weight to the mounting evidence, highlighting the detrimental impact of depression on health and longevity. Studies reveal that individuals with moderate to severe depressive symptoms face heightened risks of all-cause mortality, including cardiovascular and ischemic heart disease-related death [7]. More than a third of patients with coronary heart disease may experience myocardial ischemia under psychological stress conditions, a type known as mental stress-induced myocardial ischemia (MSIMI) [8]. Cohort studies in Chinese adults underscore a link between major depression and an increased risk of ischemic heart disease (IHD) independent of other major cardiovascular risk factors [9]. Additionally, depression is associated with high disability levels, significantly amplifying the incidence and mortality rates of heart disease, especially when it persists post-coronary events [10]. In 2014, the American Heart Association (AHA) issued a scientific statement reinforcing that depression post-acute coronary syndrome poses a risk factor for all-cause and cardiac deaths and composite endpoints, including mortality and non-fatal cardiac events [10]. Conversely, a considerable proportion of CAD patients grapple with depression, anxiety, and negative emotions, especially among those with poorer living conditions and lower education levels. These conditions exacerbate psychological issues, substantially diminish the quality of life, and eventually escalate into more serious depressive symptoms [4]. The intricate interplay between the two diseases significantly impacts a patient's quality of life and disease outcomes, perpetuating a detrimental cycle. Despite this, the treatment of mental health conditions was often excluded from the realm of cardiac rehabilitation practice.

1.2. Risk factors in psycho-cardiology diseases

Risk factors for CAD patients with depression encompass various elements, including genetic predispositions, personality traits, low socioeconomic status and social environment factors (such as childhood events, divorce, among others), somatic conditions (like diabetes, Parkinson's disease), and substance abuse. Among these factors, gender stands out as a critical risk determinant. Female patients with CAD demonstrate a higher susceptibility to mental stress-induced myocardial ischemia (MSIMI) compared to male patients [11–13]. This disparity might be attributed to the relatively thinner coronary arteries in female anatomy, making them more susceptible to microcirculation lesions [12,14].

1.3. Mechanisms of comorbidity in psycho-cardiology diseases

Persistent anxiety and depression significantly contribute to the occurrence and progression of CAD through multiple pathways.

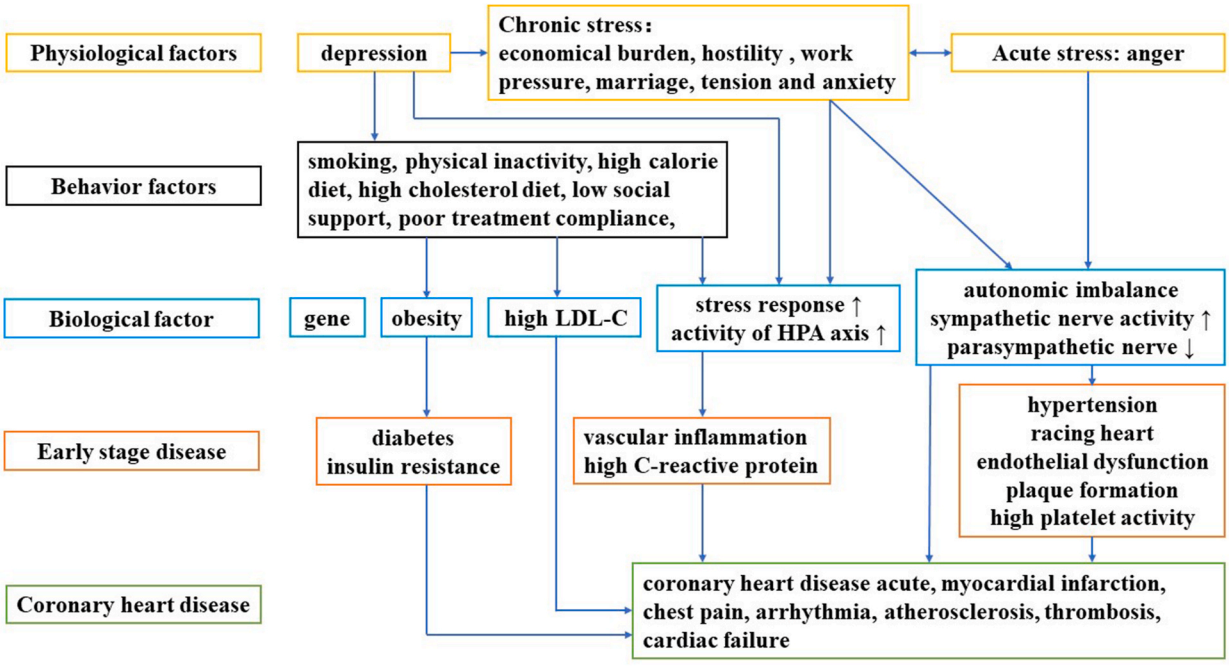


Fig. 1. Psycho-cardiology disease biological mechanism.

The pathophysiology underlying CAD comorbid psychological issues primarily involves heightened immune inflammation, disturbances in the blood coagulation system, overactivity of the hypothalamus-pituitary-adrenal (HPA) axis, activation of the sympathoadrenal medullary system, autonomic nervous dysfunction, endothelial dysfunction, and disruption of the 5-hydroxytryptophan (5-HT) system. Depression, psychological stress, or negative emotions may stimulate the occurrence and development of cardiovascular diseases through the combined action of the HPA axis, 5-HT system, and other pathways, consequently impacting the prognosis of CAD patients. Moreover, individuals with CAD often exhibit elevated inflammation levels, abnormalities in the coagulation system, and impaired endothelial function, predisposing them to neuroendocrine-immune system dysfunctions that increase the likelihood of developing depression and anxiety. The intricate pathways intertwine depression, anxiety, acute and chronic stress on one end and the prognosis of CAD on the other (Fig. 1). Behaviors that affect CAD may be closely related to the mechanism of psycho-cardiology diseases, forming a complex disease network, prompting depression in patients with CAD, and increasing the risk of CAD in patients with depression [15].

Psycho-cardiology disease biological mechanism includes persistent dysregulation of the autonomic and HPA axis, vascular inflammation, endothelial dysfunction, and enhanced platelet aggregation (Fig. 1). Autonomic dysfunction is the key to the association between depression and CAD. Patients with depression usually have chronic increased sympathetic activity, especially cardiac sympathetic tone, which is one of the significant determinants of the onset and progression of CAD. Chronic sympathetic activation can increase abnormal oxidative stress, apoptosis and myocardial damage. Moreover, depression patients with lower heart rate variability and higher plasma catecholamine levels may accelerate the morbidity of CAD and increase the risk of cardiovascular death [16]. Depression, as a negative mental factor, leads to the over-expression of HPA axis function, resulting in neuro-humoral dysregulation, and eventually leads to abnormal glucose and lipid metabolism, obesity, and hyperlipidemia. Moreover, it may lead to increased blood pressure and heart rate, all risk factors for CAD [17,18].

The inflammatory process is associated with the progression of CAD and cardiac events, including myocardial infarction. Studies [19] have shown that triglyceride (TG), C-reactive protein (CRP), and interleukin-6 (IL-6), may be associated with depression. Depression and inflammation promote each other and affect the immune system, leading to excessive secretion of IL-6 cytokines and increased tumor necrosis factor-α (TNF-α), which are common in mood disorders and cardiovascular diseases [20]. Therefore, inflammation involving the immune system may be the shared mechanism of depression and CAD. Depression can activate platelets through two pathways: sympathetic excitation caused by excessive stress and the 5-HT system. Sympathetic nerve excitement can increase platelet activity, extend the activation time, and cause an increase in cardiovascular reactivity, leading to artery atheromatous plaque rupture thrombosis. The platelet factor 4 and β-globulin thrombus in patients with depression keeping a high level in plasma reflects the platelet aggregation, verifying that platelet activation was significantly increased when CAD comorbidity with depression [21].

2. Screening and diagnosis of psycho-cardiology patients

Timely identification and treatment of depression in patients with CHD are crucial for improving both physical and mental outcomes. Clinical manifestations in psycho-cardiology patients included symptoms of depression and CAD symptoms. CAD-related symptoms include chest tightness, pain, shortness of breath, wheezing, palpitations, nausea, sweating, and shivering. Manifestation of depression involves a persistent low mood; other indicators include loss of interest, the feelings of guilt, difficulty concentrating, insomnia, reduced appetite, and suicidal thoughts, alongside various cognitive, behavioral, and social dysfunctions. In patients with CAD comorbid depression, emotional states might concurrently heighten their first and second heart sounds. Emotional distress can trigger an increase in heart rate and blood pressure when chest pain occurs. Physical symptoms such as cold, clammy skin, sweating, and transient symptoms like third and fourth heart sounds and mitral insufficiency might also be observed. Specific psychological stress stimulation is recommended for individuals with a confirmed diagnosis of CAD to determine if they experience mental stress-induced myocardial ischemia (MSIMI) [22]. Employing cardiac ultrasound combined with myocardial perfusion effectively detects myocardial ischemia induced by mental stress [23,24]. Furthermore, inquiring about medical histories, including hypertension, diabetes, dyslipidemia, hyperuricemia, depression, and anxiety, as well as family history of cardiovascular disease and depression, aids in identifying such patients. Early screening and evaluation of psychological issues, according to relevant expert consensus [25] recommends a "three-question method" for preliminary screening: (1) whether sleep is poor; (2) whether irritable or lack of interest; (3) whether you feel unwell but have been negative many times. Suppose two or more positive responses are elicited, there is an approximately 80% probability of the patient experiencing psychological issues, necessitating further assessment using psychological scales such as the Patient Health Questionnaire-9 (PHQ-9) and Hospital Anxiety/Depression Scale (HADS). Additionally, self-rating questionnaires like the Baker Depression Scale (BDI), Zung Depression Scale (SDS), and Hamilton Depression Scale (HAMD) serve as preliminary tools for screening depression in CAD patients. Addressing diseases solely from a physiological or psychological standpoint is insufficient. Diagnosis should integrate clinical medical history, symptoms, electrocardiogram, cardiac markers, echocardiography or coronary angiography, and depression or anxiety-related scale test results to recognize and diagnose CAD in patients with depression.

2.1. Psycho-cardiology treatment recommendations

In individuals with CAD and depression, the primary focus is the active treatment of coronary heart disease. Alleviating chest distress symptoms, such as chest pain, significantly impacts the patient's tolerance for activities, prognosis, and desire for an improved quality of life, which can notably alleviate depression. While prioritizing conventional coronary artery disease treatment, addressing

depression through intervention is equally crucial. Antidepressant treatment might not directly reduce all-cause mortality among coronary heart disease patients with depression. However, it plays a pivotal role in alleviating depressive symptoms and enhancing patients' quality of life. Hence, a range of clinical interventions are recommended to facilitate effective antidepressants.

2.2. Psychotherapy

The purpose of psychological treatment is to make patients understand CAD, identify the mental disorder symptoms, and

Table 1
The cardiovascular adverse effects of antidepressants.

Drug	Heart rate	blood pressure	QTc	arrhythmia	conduction disorder	After myocardial infarction	Note
Selective 5-HT reuptake inhibitors (SSRIs)							
fluoxetine	Mild bradycardia	Minimal impact	/	/	/	Use with caution, clinical experience is preferred	Evidence suggests that it is safe after MI
sertraline	Minimal impact	Minimal impact	/	/	/	first choice	It is safe for use after myocardial infarction and in patients with heart failure
fluvoxamine	Minimal impact	The systolic blood pressure decreased slightly	/	/	/	Use with caution	Limited changes in the ECG were found
paroxetine	Mild bradycardia	Minimal impact	/	/	/	usually used with caution in patients with heart disease	It may be safe after myocardial infarction
Citalopram	Mildly increased heart rate	mild decrease in systolic blood pressure	Dose-related QTc prolongation	Overdose has been reported to cause torsades de pointes ventricular tachycardia	/	Use with caution but evidence supports its safety in patients with cardiovascular disease	The secondary metabolites may be ↑QTc interval. There is no clear evidence of an increased risk of arrhythmia across the therapeutic dose range.
5-HT and norepinephrine uptake inhibitors (SNRIs)							
Venlafaxine	Mildly increased heart rate	Orthostatic blood pressure was elevated, and high doses resulted in elevated blood pressure	may be prolonged in overdose but is rare	Rare cardiac arrhythmias have been reported during overdose	Reports of rare conduction abnormalities	not been evaluated in post-MI patients and is avoided	It should be avoided in patients with a high risk of malignant arrhythmia and uncontrolled hypertension
Duloxetine	Mildly increased heart rate	Caution in High blood pressure	QTc prolongation was reported individually	Toxicity has been reported individually	Toxicity has been reported individually	Use with caution in patients with recent myocardial infarction	Limited clinical experience - not recommended
Other new antidepressants							
Bupropion	Mildly increased heart rate	mildly elevated, sometimes markedly elevated, and postural hypotension is rare	QTc is shortened, but may be prolonged in overdose	no effect, and overdose was rarely reported	/	Smoking cessation was well tolerated in patients after MI	Interactions were noted and blood pressure was monitored
mirtazapine	Minimal impact	Mild orthostatic hypotension was observed	The QTc was slightly prolonged	/	/	Use with caution in patients with recent myocardial infarction	it is safe to use after myocardial infarction and it is a good alternative to SSRIs
Agomelatine	No report	No report	QTc prolongation was reported in one case	No report	unknown	unknown	Limited data - not recommended
Trazodone	Slow down is common, but speed up can also occur	It can cause severe orthostatic hypotension	QTc can be prolonged	QTc prolongation and arrhythmia have been reported in several cases	unknown	used with caution in patients with severe heart disease	cause arrhythmias in patients with heart disease

objectively evaluate the relationship between cardiovascular diseases and clinical symptoms. To make patients aware of the necessity of treating CAD comorbidity with depression and obtain the patient's active cooperation to treatment. The treatment of psycho-cardiology disease needs to pay attention to patients' mental health based on secondary prevention of coronary heart disease. According to the 2015 AHA recommendations for secondary prevention after coronary artery bypass grafting (CABG), it is reasonable for all patients after CABG to be screened for depression by a primary physician and mental health professional. For patients with depression after CABG, cognitive behavioral therapy or combination therapy may be considered to alleviate depression. Psycho-cardiology patients should be treated according to the severity of depression in a step-based order [26] to alleviate depressive symptoms, improve compliance, improve quality of life, and reduce recurrence and readmission rates.

2.3. Physical practice

Physical practice is one of the effective ways to treat psycho-cardiology disease [27]. In a randomized clinical trial [28], a 4-month trial group including aerobic exercise and sertraline treatment was superior to the placebo in reducing depressive symptoms. Studies [29] found that aerobic exercise and cardiac rehabilitation may relieve depressive symptoms and depression recurrence by regulating the parasympathetic nervous system and inflammatory mediators to reduce the prevalence and mortality of patients with psycho-cardiology disease. Milani and Lavie [30,31] followed up on 522 patients with coronary heart disease for 4 years. The results showed that exercise therapy could reduce the mortality of patients with CAD comorbidity depression by 73%. Meanwhile, it also suggested that only a slight improvement in the patient's cardiopulmonary function could reduce the incidence and mortality of such patients with depression.

2.4. Application of antidepressant medication

Expert consensus emphasizes the importance of antidepressants in treating individuals with CAD comorbid depression, particularly for those with moderate to severe depression [32]. Neurotrophic therapy is recommended for individuals with CAD and mild depression, whereas those with moderate or severe depression alongside CAD are advised to consider antidepressant treatment [33]. Antidepressants are typically initiated at low doses and gradually increased to the minimum effective dosage to mitigate adverse reactions. Usually, therapeutic effects manifest within approximately 2 weeks, with the effectiveness correlating with time. Combined psycho-cardiology therapy with conventional CAD treatment often involves integrating antidepressant therapy. However, it is important to note that antidepressants may lead to cardiovascular adverse reactions. Tricyclic antidepressants are discouraged due to their potential adverse effects, like cardiotoxicity in psycho-cardiological patients.

Selective serotonin reuptake inhibitors (SSRIs) such as sertraline and citalopram are the preferred first-line drugs for CAD patients with severe or recurring depressive syndrome [34,35]. SSRIs inhibit 5-HT reuptake and platelet activity while increasing 5-HT activity in interneurons. Given that platelet adhesion and aggregation are significant etiological factors for increased cardiovascular risks in depression, treating psycho-cardiology with SSRIs is beneficial [36]. However, SSRI might slightly prolong the adjusted QT interval at therapeutic levels, with higher risks observed at doses exceeding recommendation. These risks include notable QT interval prolongation, increased ventricular arrhythmias, and hypotension [36]. Beyond their antidepressant properties, SSRIs like sertraline can improve heart rate variability, while paroxetine can enhance vagal function.

Additionally, sertraline may exhibit anti-inflammatory effects by reducing CRP and IL-6 levels. Second-line antidepressants encompass various classes like 5-HT_1A receptor agonists (e.g., tandospirone citrate), 5-HT_2A receptor antagonists, 5-HT reuptake inhibitors (e.g., trazodone), NE and specific 5-HT receptor antagonists, dopamine and NE reuptake inhibitors, and 5-HT and norepinephrine uptake inhibitors (SNRI) (e.g., duloxetine)). SNRI augment cardiac sympathetic activity, potentially causing adverse effects such as tachycardia, hypertension, and arrhythmias, especially at high doses. Therefore, CAD patients on antihypertensive therapy should closely monitor their blood pressure if initiating SNRI treatment. Bupropion use in CAD patients with depression has been established. Acting through effects on norepinephrine and dopamine, bupropion serves as an antidepressant. Table 1 summarises the cardiovascular adverse effects of antidepressants [37].

Table 2
Cardiovascular drugs and antidepressants associated with the CYP450.

Metabolic pathways	Antidepressants	Cardiovascular drugs
CYP1A2	SSRI, SNRI, TCA：mirtazapine	antiplatelet/anticoagulation: warfarin; antiarrhythmic: mexiletine, propranolol, verapamil
CYP2B6	amfebutamone	prasugrel, clopidogrel
CYP2C9	SSRI: fluoxetine, sertraline; TCA: amitriptyline; vortioxetine	warfarin, lovastatin, fluvastatin, valsartan, diuretic
CYP2C19	SSRI: citalopram, sertraline; TCA: amitriptyline, clomipramine	irbesartan, losartan, practolol; warfarin; clopidogrel
CYP3A4	SSRI: citalopram, fluoxetine, paroxetine, sertraline; TCA: mirtazapine、imipramine	antiplatelet/anticoagulation: apixaban, edoxaban, prasugrel; atorvastatin, simvastatin, felodipine, verapamil

3. Interactions between psychiatric drugs and cardiovascular drugs

The study found that 37% of cardiovascular patients were prescribed psychotropic drugs. About half of the patients in cardiovascular medicine were using diuretics, and nearly half were using angiotensin-converting enzyme inhibitors. Therefore, clinicians must be aware of drug-drug interactions, especially in elderly patients with cardiovascular disease. Psychotropic drugs approved for antidepression and cardiovascular drugs classified by CYP interaction type are listed in Table 2. This table lists partial standard drugs. When clinicians have to prescribe both psychotropic and cardiac medications, seeking the advice of a psychiatrist or pharmaceutical specialist is advised.

For patients with comorbid CAD and depression, treatment should integrate conventional coronary artery disease protocols alongside dietary adjustment, exercise guidance, appropriate psychological therapy selection, and tailored interventions such as nutritional support for the nerves and antidepressant treatment aligned with the severity of depression. Psycho-cardiology conditions are often linked to adverse lifestyle habits, psychological well-being, and physical factors, necessitating comprehensive intervention and systematic management. Upon discharge, health professionals, including doctors, nurses, and pharmacists, should establish health records and conduct structured health education. Regular follow-ups should be implemented, evaluating both the patient's psychological and cardiac conditions to devise personalized intervention plans. It is crucial to involve family members in the patient's treatment process, fostering a supportive family network to facilitate their self-care and overall health management. In today's technological era, telephone and internet-based follow-ups have emerged as viable methods to maintain contact and provide ongoing support [38,39].

4. Conclusion

Depression has been identified as an independent risk factor contributing to the onset and poor prognosis of cardiovascular diseases. The coexistence of psychological and cardiac conditions forms a reciprocal relationship, impacting each other and leading to a decline in clinical outcomes, establishing a harmful cycle. Treating depression in individuals with comorbid coronary heart disease not only improves their mental health but also positively impacts the treatment and prognosis of heart disease. A comprehensive treatment approach addressing both mental and physical health concerns provides comprehensive support and management. We strongly advocate for psychological and cardiology interventions for CAD patients who are predisposed to depression. Comprehensive understanding through effective communication regarding the patient's medical history is crucial. Simultaneous physical and psychological screenings allow for tailored intervention based on the severity of depression. Medication therapy in cases of heart disease complicated by psychological issues should adhere to precise diagnosis, personalized prescription, attention to potential drug interactions and adverse reactions, and consistent medication monitoring. SSRIs have demonstrated safety and efficacy in treating depression alongside coronary heart disease. Collaborative efforts between doctors and patients are vital in coping with the condition. While active treatment for coronary heart disease remains imperative, interventions targeting depression are equally essential to achieve holistic physical and psychological healing.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability statement

The authors do not have permission to share data.

CRediT authorship contribution statement

Yu Ren: Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Huilin Tang:** Validation, Supervision, Methodology. **Liwei Zhang:** Supervision, Software, Project administration, Methodology. **Chenfei Ying:** Resources. **Hua Luo:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Formal analysis, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgment

None.

References

[1] A. Mindel, O. Carney, Acyclovir malabsorption, Br. Med. J. 296 (6636) (1988) 1605.
[2] T.S. Jørgensen, I.K. Wium-Andersen, M.K. Wium-Andersen, et al., Incidence of depression after Stroke, and associated risk factors and mortality outcomes, in a large cohort of Danish patients, JAMA Psychiatr. 73 (10) (2016) 1032–1040.
[3] Q. Wu, J.M. Kling, Depression and the risk of myocardial infarction and coronary death: a meta-analysis of prospective cohort studies, Medicine (Baltim.) 95 (6) (2016) e2815.
[4] E. Wiernik, P. Meneton, J.P. Empana, et al., Cardiovascular risk goes up as your mood goes down: interaction of depression and socioeconomic status in determination of cardiovascular risk in the CONSTANCES cohort, Int. J. Cardiol. 262 (2018) 99–105.
[5] A. Newhouse, W. Jiang, Heart failure and depression, Heart Fail. Clin. 10 (2) (2014) 295–304.
[6] A. O'Neil, A.J. Fisher, K.J. Kibbey, et al., Depression is a risk factor for incident coronary heart disease in women: an 18-year longitudinal study, J. Affect. Disord. 196 (2016) 117–124.
[7] Z. Zhang, S.L. Jackson, C. Gillespie, R. Merritt, Q. Yang, Depressive symptoms and mortality among US adults, JAMA Netw. Open 6 (10) (2023) e2337011.
[8] J.L. Sun, S.H. Boyle, Z. Samad, et al., Mental stress-induced left ventricular dysfunction and adverse outcome in ischemic heart disease patients, Eur J Prev Cardiol 24 (6) (2017) 591–599.
[9] N. Liu, X.F. Pan, C. Yu, et al., Association of major depression with risk of ischemic heart disease in a mega-cohort of Chinese adults: the China kadoorie biobank study, J. Am. Heart Assoc. 5 (12) (2016).
[10] J.H. Lichtman, E.S. Froelicher, J.A. Blumenthal, et al., Depression as a risk factor for poor prognosis among patients with acute coronary syndrome: systematic review and recommendations: a scientific statement from the American Heart Association, Circulation 129 (12) (2014) 1350–1369.
[11] W. Jiang, Z. Samad, S. Boyle, et al., Prevalence and clinical characteristics of mental stress-induced myocardial ischemia in patients with coronary heart disease, J. Am. Coll. Cardiol. 61 (7) (2013) 714–722.
[12] V. Vaccarino, A.J. Shah, C. Rooks, et al., Sex differences in mental stress-induced myocardial ischemia in young survivors of an acute myocardial infarction, Psychosom. Med. 76 (3) (2014) 171–180.
[13] A. Peix, A. Trápaga, L. Asen, et al., Mental stress-induced myocardial ischemia in women with angina and normal coronary angiograms, J. Nucl. Cardiol. 13 (4) (2006) 507–513.
[14] H. Ma, L. Guo, H. Fei, et al., Assessing mental stress on myocardial perfusion and myocardial blood flow in women without obstructive coronary disease: protocol for a mechanistic clinical trial, BMJ Open 10 (12) (2020) e038362.
[15] Y. Ge, T. Chao, J. Sun, W. Liu, Y. Chen, C. Wang, Frontiers and hotspots evolution in psycho-cardiology: a bibliometric analysis from 2004 to 2022, Curr. Probl. Cardiol. 47 (12) (2022) 101361.
[16] J.P. Headrick, J.N. Peart, B.P. Budiono, D.H.K. Shum, D.L. Neumann, N.J.C. Stapelberg, The heartbreak of depression: 'Psycho-cardiac' coupling in myocardial infarction, J. Mol. Cell. Cardiol. 106 (2017) 14–28.
[17] A.H. Kemp, D.S. Quintana, M.A. Gray, K.L. Felmingham, K. Brown, J.M. Gatt, Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis, Biol. Psychiatr. 67 (11) (2010) 1067–1074.
[18] H. Luo, Z.L. Jiang, Y. Ren, Therapy management of metabolic disorder comorbidity with depression, Front. Psychol. 12 (2021) 683320.
[19] G.M. Khandaker, V. Zuber, J.M.B. Rees, et al., Shared mechanisms between coronary heart disease and depression: findings from a large UK general population-based cohort, Mol. Psychiatr. 25 (7) (2020) 1477–1486.
[20] A. Fotopoulos, P. Petrikis, C. Sioka, Depression and coronary artery disease, Psychiatr. Danub. 33 (1) (2021) 73.
[21] R. Delle Chiaie, E. Capra, M. Salviati, et al., Persistence of subsyndromal residual symptoms after remission of major depression in patients without cardiovascular disease may condition maintenance of elevated platelet factor 4 and β-thromboglobulin plasma levels, J. Affect. Disord. 150 (2) (2013) 664–667.
[22] F. Alenezi, B.H. Brummett, S.H. Boyle, et al., Usefulness of myocardial annular velocity change during mental stress to predict cardiovascular outcome in patients with coronary artery disease (from the responses of mental stress-induced myocardial ischemia to escitalopram treatment trial), Am. J. Cardiol. 120 (9) (2017) 1495–1500.
[23] J.A. Nieuwsma, J.W. Williams Jr., N. Namdari, et al., Diagnostic accuracy of screening tests and treatment for post-acute coronary syndrome depression: a systematic review, Ann. Intern. Med. 167 (10) (2017) 725–735.
[24] Y. Liu, B. Kong, H. Ma, et al., Consistency of positron emission tomography and myocardial contrast echocardiography in diagnosing mental stress-induced myocardial ischemia: study protocol of a prospective study-background, design and method, Ultrasound Med. Biol. 46 (12) (2020) 3200–3209.
[25] D. Hu, China expert consensus on psychological prescription for patients with cardiovascular disease (2020), Chin. J. Intern. Med. 59 (2020) 764–771.
[26] M.K. Jha, A. Qamar, M. Vaduganathan, D.S. Charney, J.W. Murrough, Screening and management of depression in patients with cardiovascular disease: JACC state-of-the-art review, J. Am. Coll. Cardiol. 73 (14) (2019) 1827–1845.
[27] H.M. Malian, P.J. Smith, B. Hoffman, et al., Effects of acute exercise on anxiety ratings in patients with coronary heart disease and elevated anxiety, J Cardiopulm Rehabil Prev 41 (4) (2021) 277–281.
[28] J.A. Blumenthal, A. Sherwood, M.A. Babyak, et al., Exercise and pharmacological treatment of depressive symptoms in patients with coronary heart disease: results from the UPBEAT (understanding the prognostic benefits of exercise and antidepressant therapy) study, J. Am. Coll. Cardiol. 60 (12) (2012) 1053–1063.
[29] F. Doyle, K.E. Freedland, R.M. Carney, et al., Hybrid systematic review and network meta-analysis of randomized controlled trials of interventions for depressive symptoms in patients with coronary artery disease, Psychosom. Med. 83 (5) (2021) 423–431.
[30] C.J. Lavie, R.V. Milani, Adverse psychological and coronary risk profiles in young patients with coronary artery disease and benefits of formal cardiac rehabilitation, Arch. Intern. Med. 166 (17) (2006) 1878–1883.
[31] R.V. Milani, C.J. Lavie, Impact of cardiac rehabilitation on depression and its associated mortality, Am. J. Med. 120 (9) (2007) 799–806.
[32] Corrigendum to: depression and coronary heart disease: 2018 position paper of the ESC working group on coronary pathophysiology and microcirculation developed under the auspices of the ESC Committee for Practice Guidelines, Eur. Heart J. 41 (17) (2020) 1696.
[33] G.L. Xiong, K. Prybol, S.H. Boyle, et al., Inflammation markers and major depressive disorder in patients with chronic heart failure: results from the sertraline against depression and heart disease in chronic heart failure study, Psychosom. Med. 77 (7) (2015) 808–815.
[34] T. Rutledge, L.S. Redwine, S.E. Linke, P.J. Mills, A meta-analysis of mental health treatments and cardiac rehabilitation for improving clinical outcomes and depression among patients with coronary heart disease, Psychosom. Med. 75 (4) (2013) 335–349.
[35] W. Jiang, J.R. Davidson, Antidepressant therapy in patients with ischemic heart disease, Am. Heart J. 150 (5) (2005) 871–881.
[36] S. Kasper, Choosing among second-generation antidepressant treatments for depressed patients with cardiac diseases, Int. J. Psychiatr. Clin. Pract. 23 (2) (2019) 134–148.
[37] J. Woroń, M. Siwek, A. Gorostowicz, Adverse effects of interactions between antidepressants and medications used in treatment of cardiovascular disorders, Psychiatr. Pol. 53 (5) (2019) 977–995.
[38] C. Helmark, R. Ahm, C.M. Andersen, et al., Internet-based treatment of anxiety and depression in patients with ischaemic heart disease attending cardiac rehabilitation: a feasibility study (eMindYourHeart), Eur Heart J Digit Health 2 (2) (2021) 323–335.
[39] Y. Zhang, X. Lv, W. Jiang, et al., Effectiveness of a telephone-delivered psycho-behavioural intervention on depression in elderly with chronic heart failure: rationale and design of a randomized controlled trial, BMC Psychiatr. 19 (1) (2019) 161.