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THE PRESENT AND FUTURE

JACC STATE-OF-THE-ART REVIEW

Psychopharmacology and Cardiovascular Disease

Ileana L. Piña, MD, MPH,[a] Katherine E. Di Palo, PharmD,[a] Hector O. Ventura, MD[b,c]

ABSTRACT

This review discusses common mental health disorders and their associations with cardiovascular disease risks. Commonly found mental health disorders include depression, anxiety, and personality types. The link between depression and cardiovascular disease mortality has been established. Depression is also common in patients with heart failure. In addition to discussing psychological disorders, a review of psychotropic drugs is also included. Drugs are described for therapy for depression and anxiety, as well as associations with cardiovascular drug-drug interactions. Drug-drug interactions are more common and potentially dangerous in elderly patients, in whom the conditions often coexist. The most common drug-drug interactions involve the P450 system of enzymes. (J Am Coll Cardiol 2018;71:2346–59) © 2018 by the American College of Cardiology Foundation.

In recent years, psychological factors have been reported to be tightly linked to cardiovascular (CV) disease. The link is bidirectional in that psychological factors may be common in certain CV diseases (CVDs) and portend worse outcomes, or psychological conditions may pre-exist and precede CVD. Furthermore, treatment for mental health disorders can present CV risk due to side effects of the drugs or interactions with other medications. Reports are also consistent that patients with significant psychological disorders, such as depression, have higher morbidity and mortality compared with a general population (1). A recent meta-analysis of 203 publications from 29 countries on 6 continents found a relative risk for mortality of 2.22 for those with mental disorders compared with the comparison populations and estimated that approximately 8 million deaths worldwide (14.3%) can be associated with mental disorders (2).

For clinicians, it is important to detect psychological disorders in patients with CVD that may affect overall outcomes and choose effective therapeutic agents that do not aggravate the underlying CV disorder but address mental health. Alternatively, early and appropriate referral to mental health providers can aid in decision making for pharmacological therapy that is safe and compatible with CV drugs.

In this review we focus on the more common mental health disorders in patients with CVD, such as depression and anxiety, and review current information on treatment alternatives. In addition, we briefly describe CV side effects of psychological drug therapy and potential drug-drug interactions, some of which could be lethal. This review is not meant to be a comprehensive list of all pharmacotherapy but to highlight the agents that have reported CV interactions.

EPIDEMIOLOGY

CVD is the leading cause of morbidity and mortality and a major public health burden in industrialized countries, including the United States and European nations (3). Data from prospective cohort studies have

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From the [a]Albert Einstein College of Medicine and Montefiore Medical Center, Bronx, New York; [b]John Ochsner Heart and Vascular Institute, Ochsner Clinical School, New Orleans, Louisiana; and [c]The University of Queensland School of Medicine, St. Lucia, Queensland, Australia. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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found that CVD risk factors such as cigarette smoking, abnormal blood lipid levels, hypertension, diabetes, abdominal obesity, physical inactivity, low daily fruit and vegetable consumption, alcohol overconsumption, and psychological factors play a major role in the development of coronary heart disease (CHD) (4).

PSYCHOLOGICAL RISK FACTORS AND CVD: PATHOPHYSIOLOGY AND POTENTIAL MECHANISMS

Several psychological factors, such as depression, anxiety, hostility, and type A personality have been shown not only to be risk factors for CVD but also to affect clinical outcomes. Depression may have both behavioral and physiological backgrounds. Patients who are depressed may be more likely to engage in unhealthy behaviors and be less compliant with therapies. However, there are also physiological changes, such as hypercortisolemia and a lower than expected response to corticotropin-releasing factor administration (5,6). Platelet function has also been reported to be abnormal, with high reactivity and release of factor 4 and beta-thyroglobulin (7). Both of these physiological changes could be atherogenic. In addition, depressed patients have also been found to have abnormal heart rate variability, which could lead to arrhythmias (8).

Acute stress has been among the best studied psychological states, with its physiological effects on the CV system. The **Central Illustration** depicts various aspects of acute stress on the sympathetic nervous system resulting in arrhythmias, endothelial function, and platelet activation, among others (9). Some of the effects are mediated by heart rate and acute blood pressure increases, which may be exaggerated and could activate the mechanisms that lead to atherosclerosis. This hypothesis has been tested in the Kuopio Ischemic Heart Disease Study, in which mental stress in hyperreactors was linked to intima-media thickness in carotid arteries with diastolic blood pressure responses (10). Other mechanisms of coronary blood flow may be mediated by neuronal nitric oxide synthase pathways (11).

Finally, associations between inflammatory mechanisms have been implicated in both coronary plaque lesions and in depressed subjects. These cytokines include C-reactive protein, tumor necrosis factor-alpha, and interleukin-6, among others (12). Therefore, there are multiple pathways that link CV mechanisms and mental health in a bidirectional fashion. Clinicians need to consider the psychological factors that modulate or even interact with their patients' CVD.

DEPRESSION

Presentation of patients with depression can range from subclinical depressive symptoms to a full major depressive disorder. It is important to point out that to characterize a patient with a major depressive disorder, the presence of symptoms must interfere with normal life (1).

The prevalence of depression in patients with CVD is 3-fold higher that in the general population and has been studied extensively. The American Heart Association recommends that depression should be recognized as a major risk factor for coronary artery disease (CAD) (13). Subjects with depression have an 80% increase in the risk for developing new or worsening CVD as well as death from CVD (14). In addition, depression is commonly present in patients with angina and can increase the development of myocardial infarction (MI), stroke, sudden death, and atrial fibrillation (13,15–18). Thus, the relationship between depression and CVD is bidirectional. Depression increases the risk for developing CVD, and CVD can increase the risk for developing depression. Recognizing depression is therefore critically important, given that depression after an MI is present in 17% to 45% of patients and considered major in 15% to 18%, and some may remain depressed for months following the event (19).

Early observational studies have shown that depression, diagnosed during hospitalization for CHD, is associated with higher mortality 6 to 18 months after hospitalization (20,21). Interestingly, the more severe the depression, as measured using the Beck Depression Inventory (BDI), the more severe the CHD risk. Patients with acute MI have a 3-fold higher prevalence of depression compared with those without MI. The risk for sudden cardiac death is 3-fold higher in patients with moderate and severe depression (22).

A meta-analysis of all prospective studies examining depression and risk for CHD showed that depression was associated with significantly increased risk for CVD (23). Other studies, including those with longer follow-up, have shown similar results, including the deleterious association between depression and cerebrovascular disease (24–27).

The presence of moderate to severe depression before coronary artery bypass grafting surgery and persistent depression following the procedure places

ABBREVIATIONS AND ACRONYMS

ASA = aspirin

BDI = Beck Depression Inventory

BZD = benzodiazepine

CAD = coronary artery disease

CHD = coronary heart disease

CV = cardiovascular

CVD = cardiovascular disease

CYP = cytochrome P450

FDA = U.S. Food and Drug Administration

ICD = implantable cardioverter-defibrillator

MI = myocardial infarction

SNRI = serotonin-norepinephrine reuptake inhibitor

SSRI = selective serotonin reuptake inhibitors

TCA = tricyclic antidepressant

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