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Anxiety and Risk of Cardiovascular Disease

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Abstract

Anxiety is a normal emotion that is owned by every individual. Pathologically increased anxiety can not only give rise to other anxiety disorders but can also lead to other mental illnesses. Anxiety is a type of mental disorder that is quite common in Europe, Switzerland, Iceland, Norway in 2010 it was found that the prevalence for 12 months was 14% and 61.5 million people were affected. According to WHO in the global population with anxiety disorders in 2015 the percentage was 3.6% and it was more common in women with a percentage of 4.6% compared to 2.6% for men. Individuals with symptoms of anxiety are likely to experience the effects of interference that can lead to metabolic syndrome. Metabolic syndrome leads to progressive and clustering of several metabolic risk factors that directly increase the risk of death from cardiovascular disease (CVD, type 2 diabetes mellitus). last year from 10% to 63.2%. Anxiety disorders appear to be caused by the interaction of bio-psychosocial factors. Genetic susceptibility interacts with stressful situations or the presence of trauma to produce a clinically significant syndrome. Treatment measures include pharmacological, psychological, and combination therapies. all are therapeutic measures for all anxiety disorders, selective serotonin is a first-line agent for several anxiety disorders, including panic disorder, social anxiety, a generalized anxiety disorder. Psychotherapy and pharmacotherapy should be given and both therapies are equally effective. The choice in determining the therapy given must be considered according to the severity of the disorder.

Keywords: Anxiety; cardiovascular disease; selective serotonin

Introduction

Anxiety is a normal emotion that every individual has. Pathologically increased anxiety can not only give rise to other anxiety disorders but can also lead to other mental illnesses. Anxiety can provide a signal that there is a potential danger in somatic diseases such as myocardial infarction or hypoglycemia in diabetic patients. Anxiety is a type of mental disorder that is quite common in Europe, Switzerland, Iceland, Norway in 2010 it was found that the prevalence for 12 months was 14% and 61.5 million people were affected (Str□hle et al., 2018). According to WHO in the global population with anxiety disorders in 2015 the percentage was 3.6% and it was more common in women with a percentage of 4.6% compared to 2.6% men (WHO 2017).

Currently, anxiety is becoming increasingly common in the community at a 1 year prevalence rate of anxiety is 18%. Individuals with symptoms of anxiety are likely to experience the effects of interference that can lead to metabolic syndrome. Metabolic syndrome leads to a progressive and clustering of several metabolic risk factors that directly increase the risk of death from cardiovascular disease (CVD, type 2 diabetes mellitus). in recent years from 10% to 63.2% (Akbari et al. 2019).

Research Methods

The method used uses a *papersystematic review* that comes from various types of sources from scientific journals. Search sources in this paper are done through the online portal of journal publications such as the National Center for Biotechnology Information (ncbi.nlm.nih.gov) and also Google Scholar (scholar.google.com). keywords that can be used are “Anxiety”, cardiovascular risk factor”

Results and Discussion

There are several reports related to anxiety and risk factors for cardiovascular disease.

Definition

Anxiety is a feeling that is owned by someone who feels afraid and worried that something bad will happen. Someone who experiences anxiety can be seen from unusual behavior, such as unreasonable panic, fear of an object or living conditions, performing repetitive actions that cannot be controlled properly, recollecting traumatic events.

Etiology

Anxiety disorders appear to be caused by the interaction of biopsychosocial factors. Genetic susceptibility interacts with stressful situations or the presence of trauma to produce a clinically significant syndrome. Anxiety can be caused by conditions such as (Chand et al., 2020):

- Herbal remedies
- Substance abuse
- Trauma
- Childhood experience
- Panic disorder
- Epidemiology

Anxiety disorders are a type of disorder that are quite common in the European Union, Switzerland, Iceland and Norway in 2010 the prevalence rate for 12 months was obtained with a percentage of 14% and approximately 61.5 million people were affected, in women it was higher for anxiety disorders to occur. three times that of men. Anxiety often begins in children or adolescents. This corresponds to specific phobias and social phobias. Selective mutism or a condition in a child where the child is communicative to a situation or place that makes the child choose not to interact or even not speak at all usually appears in the child's third year. In 2-3% of children separation anxiety will continue into preschool or school age (Ströhle et al., 2018)

According to data from WHO, the prevalence of anxiety disorders by region in America is found to be the highest in men with a percentage of 7.7% and women around 3.9% then in the western Pacific region which is in three continents, namely Asia, eastern Australia, and America. the western part found the number of men with a percentage of 3.5% and women with 2.1%. The global prevalence based on age and sex found that those aged 40-49 years were the highest number with a percentage of 5.5% and in women aged 15-19 years the number was the highest with a percentage of around 3.2%. Those aged 80 years and over are the lowest in women and men with a percentage of 3% in men and <2% in women (WHO 2017).

Pathogenesis

The neurochemical system is the most relevant in explaining how anxiety occurs. When we experience stress, our bodies activate neurotransmitters and neuropeptides. The result is a "fight or flight" response. Many areas of the brain are involved in this mechanism. The amygdala is an organ that has an important role. The amygdala has many subnuclei called the amygdala complex. Each of these sub-nuclei has its function. The lateral nucleus of the amygdala is related to the reception of sensory information in the form of visual, auditory, and somatic information from the thalamus and cortex. This connection is the center of fear and anxiety that can be aroused by a certain stimulus (Boland, Verdium, & Ruiz, 2021).

Once a stimulus enters the lateral nucleus, the pathway sends the stimulus to various other amygdala nuclei. These are then modulated by various systems including those that provide context from memory, or about homeostatic states in the body. Several other projections send impulses to the nucleus basalis (BA), which is involved in the formation of prolonged fear and anxiety. After much processing, including from other cortical regions, the amygdala afferent pathways regulate behavioral responses that reflect the amount of activity of many nuclei. The amygdala nucleus then sends stimuli to various motor, autonomic, and neuroendocrine systems involved in expressing anxiety. These include the hypothalamus, midbrain, and medulla. For example, the hypothalamus may then activate CRH to initiate a stress response to that stimulus. The hippocampus also has an important role in learning fear and assists in the development of emotional responses related to anxiety (Boland et al., 2021).

Anxiety triggers the activation of the human stress system through behavioral and physiological changes that enhance the organism's ability to adjust homeostasis and increase its chances of survival. This process affects autonomic and hormonal regulation, resulting in metabolic abnormalities, inflammation, insulin resistance and endothelial dysfunction. The most relevant underlying mechanism relies on the hypothesis that increased activation of the HPA axis may be pathophysiologically involved in the co-occurrence of typical MS risk factors and stressors. The HPA axis affects impaired glucocorticoid sensitivity accompanied by disturbances in systemic cortisol (Boland et al., 2021; Sardinha & Nardi, 2012). Hyperactivation of the HPA axis leads to accumulation of visceral fat through increased lipid storage and adipogenesis. This glucocorticoid-mediated effect results in increased expression of glucocorticoid receptors (high density of glucocorticoids) by abdominal adipose tissue. Hypercortisolemia induces lipolysis, release of fatty acids, and synthesis of very low density lipoprotein (VLDL) resulting in hypertriglyceridemia (Penninx & Lange, 2018).

White adipose tissue, especially in the abdominal region, is an endocrine organ that actively produces inflammatory cytokines and hormones (eg, leptin) and, therefore, is a major

contributor to the immunometabolic response of pathogens in the central nervous system, as well as throughout the bod. Peripherally produced cytokines can access the brain either directly across the blood-brain barrier via an active transport system or indirectly via microglia activation and result in decreased neurogenesis in brain structures that regulate emotion. Cytokines also catalyze the synthesis of kynurenine from tryptophan, resulting in reduced serotonin synthesis and increased synthesis of the tryptophan catabolite, which interferes with nerve transmission and causes nerve damage. Activation of the proinflammatory response stimulates the release of lipids in the bloodstream, resulting in a decrease in HDL-C and phospholipids and an increase in triglycerides. HPA axis stimulation and ongoing inflammatory response can affect insulin sensitivity and alter glucose metabolism which acts directly on pancreatic cells (Penninx & Lange, 2018).

Patients with anxiety disorders usually have more cortisol in the urine than individuals without psychiatric disorders, while there appears to be no difference in the excretion of catecholamines and serotonin. There is considerable evidence from clinical, cellular and molecular studies that elevated cortisol, especially when combined with secondary inhibition of sex steroids and growth hormone secretion, leads to fat accumulation in visceral adipose tissue as well as metabolic abnormalities (Epel, 2009; Sardinha & Nardi, 2012).). Glucocorticoid exposure is also followed by stress-induced overeating with increased food intake and leptin-resistant obesity, possibly upsetting the balance between leptin and neuropeptide Y. A study using a rat model of social stress found that consumption of a high-fat diet during social stress increased the effects of chronic stress on body composition, increasing the body's knowledge of the mechanisms responsible for the development of obesity, diabetes and ultimately metabolic disease (Sardinha & Nardi, 2012).

Chronic anxiety and psychosocial stress also produce a cascade of highly comorbid adverse health consequences, including emotional eating, affective disorders and MS. Consumption of high-calorie foods is thought to provide comfort in the face of persistent psychosocial stress, in a pattern observed in both humans. Anxiety disorders are also often accompanied by self-destructive and unhealthy behavior and non-adherence to medication and medication. Thus, modern genetic and environmental interactions may explain the explosive prevalence of MS and diabetes in psychologically stressed humans (Das & O'Keefe, 2006; Sardinha & Nardi, 2012).

Therapeutic Management

With therapy, many patients show improvement in their anxiety symptoms. Therapeutic actions with pharmacological, psychological, and combination therapy are all therapeutic measures for all anxiety disorders. The meta-analysis suggests pharmacological

therapy because it has a fairly large effect size from several available options. But depending on the anxiety disorder, there are many pharmacological, psychotherapeutic, and combined options available.

Selective serotonin is a first-line agent for several anxiety disorders, including panic disorder, social anxiety, a generalized anxiety disorder. Several classes of non-SSRI drugs can be given such as venlafaxine for panic disorder, social anxiety. Tricyclic antidepressants can be used in panic disorder as well, although they are not very popular because they have some side effects. Benzodiazepines are some of the drugs that are quite often used in anxiety disorders. Several guidelines or guidelines recommend considering the long-term use of benzodiazepines for patients who cannot tolerate SSRIs because of the potential for dependence and cognitive and other side effects. Most conservative approaches to anxiety disorders begin with SSRIs. Benzodiazepines are useful when patients require rapid control with severe symptoms of anxiety (Boland, Verdium, and Ruiz 2021).

Psychotherapy and pharmacotherapy should be given and both therapies have the same effectiveness. The choice in determining the therapy given must be considered according to the severity of the disorder, patient preferences in the form of latency, and endurance according to the effect of the drug.

TABLE 2

The pharmacotherapy of anxiety disorders, according to the German guidelines (20)

Evidence level/ recommendation grade [a]	Active substance class	Drug	Daily dose
Social phobia			
Ia; A	SSRI	Escitalopram	10–20 mg
		Paroxetine	20–50 mg
		Sertraline	50–150 mg
Ia; A	SNRI	Venlafaxine	75–225 mg
CCP	MAO inhibitors	Moclobemide	300–600 mg
Panic disorder			
Ia; A	SSRI	Citalopram	20–40 mg
		Escitalopram	10–20 mg
		Paroxetine	20–50 mg
		Sertraline	50–150 mg
Ia; A	SNRI	Venlafaxine	75–225 mg
Ia; B	TCA	Clomipramine	75–250 mg
Generalized anxiety disorder			
Ia; A	SSRI	Escitalopram	10–20 mg
		Paroxetine	20–50 mg
Ia; A	SNRI	Venlafaxine	75–225 mg
		Duloxetine	60–120 mg
Ia; B[b]	Anticonvulsants	Pregabalin	150–600 mg
Ib; 0	Anxiolytic drugs (tricyclic)	Opipramol	50–300 mg
Ib; 0	Azapirones	Buspirone	15–60 mg

[a] according to the German guidelines

[b] Case studies indicate that pregabalin has a potential for abuse, mainly in patients with substance-related disorders and above all in opiate-dependent patients (26). It is accordingly recommended in the German guidelines that patients with substance-related disorders, and particularly those who are addicted to multiple drugs, should not be treated with pregabalin. If a patient does not respond to a drug or cannot tolerate it, the next step may be a switch from one standard drug to another (e.g., from an SSRI to an SSNRI; in generalized anxiety disorder, from an SSRI to pregabalin) or a switch to a nonstandard drug, such as one with a lower evidence level or recommendation grade (e.g., moclobemide in social phobia), or to one that is not approved for the treatment of anxiety disorders, but has nonetheless been reported to be clinically effective (e.g., quetiapine, agomelatine, lavender oil, and, in some cases, mirtazapine).

CCP = clinical consensus point; SSRI = selective serotonin reuptake inhibitors; SNRI = selective serotonin and norepinephrine reuptake inhibitors; MAO = monoamine oxidase; TCA = tricyclic antidepressants

Source Image : (Ströhle et al., 2018)

Conclusion

Anxiety is a feeling that everyone has when they are afraid and worried that something bad will happen. A person can be said to experience anxiety seen from unusual behavioral changes, such as unreasonable panic, fear of an object, or living conditions. Several factors can cause anxiety, one of which is biopsychosocial factors where a person is in a stressful situation or a trauma that produces a clinically significant syndrome. Individuals with symptoms of anxiety are likely to experience the effects of interference that can lead to metabolic syndrome. Metabolic syndrome is progressive and leads to clustering of metabolic risk factors that directly increases the risk of cardiovascular disease mortality

Anxiety triggers the activation of the human stress system through behavioral and physiological changes that increase the ability of the organism to adjust to homeostasis and increase the chances of survival. These processes affect autonomic and hormonal regulation, resulting in metabolic abnormalities, inflammation, insulin resistance, and endothelial dysfunction. With therapy, many patients show improvement in their anxiety symptoms. Therapeutic actions with pharmacological, psychological, and combination therapy are all therapeutic measures for all anxiety disorders. But depending on the anxiety disorder, there are many pharmacological, psychotherapeutic, and combined options available. Selective serotonin is a first-line agent for several anxiety disorders, including panic disorder, social anxiety, a generalized anxiety disorder. Several classes of non-SSRI drugs can be given, for example, venlafaxine for panic disorder, social anxiety

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