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OBESITY: FROM PHYSICAL HEALTH TO MENTAL HEALTH

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Abstract: Obesity is a multifactorial disease centered on the accumulation of fat that affects not only survival, but also the psychosocial issues of the individual, which is not restricted to a certain geographic space, constituting an epidemic. In this sense, it will be discussed about the various spheres that make up the plurality of this disease. The study divides the organic repercussions secondary to obesity correlating with damage to physical health: such as insulin resistance, dyslipidemia, heart, lung and gastrointestinal complications. And also with regard to social stigmas, which reflect on the quality of life and mental health of obese patients, leading to the appearance of disorders such as anxiety and depression, and eating disorders. This review article aims to associate obesity with its psychological and metabolic consequences, in order to optimize the therapy adopted for these individuals, leading to a greater perspective of the effects of this disease on them.
Keywords: Obesity; depression; Binge Eating Disorder; mental health

RESULTS AND DISCUSSION

PHYSIOLOGICAL CHANGES IN OBESITY

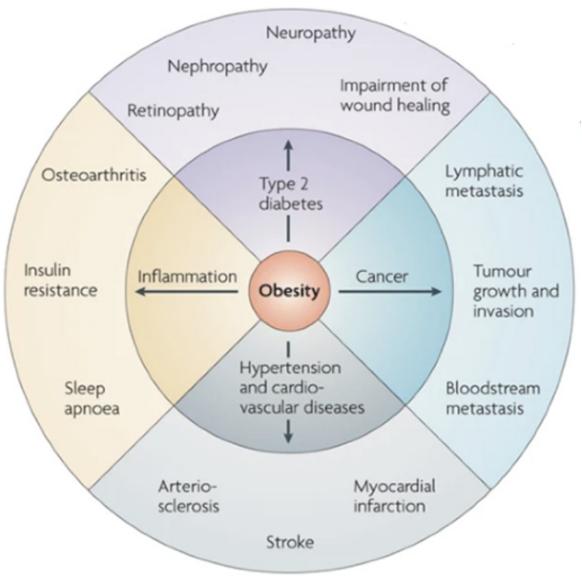
Obesity goes beyond the excessive accumulation of fat, and is associated with physiological disorders, mainly due to the phenomenon of lipotoxicity, through the excessive production of some adipokines and deregulation in the function of others. Adipokines are pro-inflammatory substances of lipid origin that cause cell dysfunction, associated with the production of reactive oxygen species (ROS), and the permanence of this leads to a chronic inflammatory state, predisposing to various metabolopathies[6].

In muscles, for example, adipokines slow down myokines, leading to muscle and bone weakness. In childhood, they progressively

delay neuropsychomotor and cognitive development, leading to delayed neurological development with repercussions on the immune system, making obese children more susceptible to mental and infectious diseases. On the other hand, leptin, increased in this state, is strongly associated with vascular phenomena. Ghrelin, responsible for regulating appetite in the hunger center, has its mechanisms enhanced, becoming even more capable of sensitizing appetite pathways and stimulating food consumption[6].

CONSEQUENCES OF OBESITY ON PHYSICAL HEALTH

The pro-inflammatory state associated with obesity (increased levels of adipokines and interleukins) affects the homeostasis of the body, which can lead to different pathologies, which will be discussed below [6].



Adipose tissue angiogenesis as a therapeutic target for obesity and metabolic diseases, 2010.

Insulin resistance:

The pathophysiology of insulin resistance is still not fully understood. The most accepted explanation is that it is a consequence of the inflammatory state, together with oxidative stress, with the release of EROs [3].

Such long exposure to oxidative stress leads to damage that can culminate in mitochondrial dysfunction, by reducing mitochondrial function and activity and biogenesis, through increased circulating lipids and greater deposition of ectopic fat, resulting in a vicious cycle of lipid accumulation and insulin resistance[6].

Hyperinsulinemia is associated with obesity due to the greater release of this hormone, proportionally to food intake, which, in the long term, overloads the beta-pancreatic cells, which can then culminate in a condition of diabetes[3].

Dyslipidemia

Disturbances in the amount of circulating lipids manifest with hypertriglyceridemia, postprandial hyperlipidemia, HDL reduction and with small and dense LDL molecules. Increased lipid levels, decreased lipolysis and insulin resistance contribute to the pathogenesis of dyslipidemia, and there are three main mediators involved, namely: hormone-sensitive lipase (HSL), lipoprotein lipase (LPL) and hepatic lipase (LH)[6].

LHS acts on adipose tissue by stimulating the release of free fatty acids (FFA) and has its activity inhibited by insulin. In cases of insulin resistance, LHS is not inhibited, and continues to release FFA, which will reach the liver, serving as raw material for greater synthesis of lipids and VLDL cholesterol[3].

LPL, on the other hand, is responsible for hydrolyzing chylomicrons (QM) and VLDL, rich in triglycerides, reducing the level of circulating triglycerides, and its activity is stimulated by insulin. With insulin resistance, this mechanism does not occur, and this circulating level remains high[3].

Finally, LH is responsible for the formation of LDL cholesterol, "bad" cholesterol, and is activated by an increase in fatty acids in the liver. Therefore, its activity increases and more LDL is synthesized[6].

Cardiovascular Complications

Systemic arterial hypertension (SAH) is closely linked to dyslipidemia, and to other mechanisms such as inflammation and hyperglycemia, which were also related to an increased risk for congestive heart failure (CHF) and coronary disease, and are characteristics of obesity. The determining mechanisms of this association seem to be linked to increased activity of the sympathetic nervous system (SNS), renin-angiotensin system, hyperleptinemia, insulin resistance, inflammation, endothelial dysfunction and oxidative stress[6].

Leptin, for example, triggers coronary artery calcification, justifying the state of proatherosclerosis common in obese people. In addition to it, resistin increases the expression of intercellular adhesion molecules-1 and antivascular-1 in endothelial cells, intensifying atherogenic genesis, and increases the activity of the NFkB factor – signaling for adhesion of these molecules. Such changes in circulatory dynamics promote vascular dysfunction, which increases cardiovascular risks/phenomena (CVA and AMI)[3].

Pulmonary complications

The main complication for the respiratory system is the obstructive sleep apnea syndrome (OSAS). Increased fat deposition in the cervical region leads to airway collapse in the recumbent position. From this, a period of apnea arises, leading to hypoxemia, hypercapnia, pulmonary vasoconstriction, inflammation of the airways and stimulation of the SNS, which will cause systemic vasoconstriction and reflex arterial hypertension. To reverse the situation, the patient ends up waking up and breathing, and these events are repeated, impairing restful sleep and quality of life, causing irritation, memory loss and increasing risks for SAH, as it increases the oxidative stress of cells and the inflammation, causing endothelial dysfunction[5].

Reproduction and gynecological complications

Currently, research has been associating the pro-inflammatory state of obesity with male and female infertility. In women, the main cause comes from polycystic ovary syndrome (PCOS), and in men, the decrease in sperm count and motility, in addition to erectile dysfunction and reduced testosterone levels[2].

Gastrointestinal complications

Non-alcoholic hepatic steatosis is one of the main complications observed. Several mechanisms may contribute to its pathogenesis, such as: increased hepatic fat supply, increased hepatic lipogenesis and reduced lipid oxidation. Due to these events, hypertrophy and hyperproliferation of adipocytes occur, inducing secretion of pro-inflammatory substances such as interleukin 6 (IL-6) and tumor necrosis factor alpha (TNF-α). These conditions contribute to mitochondrial dysfunction and ROS production, which may aggravate hepatic steatosis leading to the process of fibrosis and cirrhosis[3].

Other complications are gastroesophageal reflux disease (GERD) and hiatus hernia, due to the increase in intra-abdominal pressure, due to the accumulation of local fat. Such pathologies contribute to damage to the esophageal mucosa, which may cause local metaplasia and even Barrett's esophagus, a pre-neoplastic lesion[3].

Neurological complications

They are related to the harmful effects of excess leptin on the nervous system, since hyperinsulinemia promotes greater expression of leptin receptors, and obesity leads to an increase in circulating leptin, generating a positive feedback loop, with

a progressive increase in leptin oxidation. nervous tissue, triggering irreversible inflammatory changes. In this scenario, another adipokine stands out, the acylation-stimulating protein, which does not have its actions fully defined, but in hyperlipidic environments it is capable of stimulating the Toll-like receptor 4 (TLR4) activation pathway, activating apoptosis mechanisms of hippocampal neurons, causing damage to the neurocognitive areas, leading to a higher risk of early dementia[5].

Cancer

Several types of cancer have been linked to obesity, such as: esophageal adenocarcinoma, colorectal cancer, breast cancer and endometrial cancer. With less definitive evidence, there are: hepatocarcinoma, pancreas and thyroid cancer. This relationship is complex and poorly understood, but many hormones and markers are involved, such as insulin, insulin-like growth factor 1 (IGF-1), leptin, adiponectin and inflammatory markers[2].

The hyperinsulinemic state of obesity contributes through the anabolic effects of insulin, in addition to mitogenic and anti-apoptotic effects, by stimulating beta-catenin (prevents the process of cell apoptosis). This state increases the circulating levels of IGF-1, whose oncogenic action occurs directly, through its IGF-1R receptor, which has increased expression in several tumors, and also indirectly, acting on other cancer-related molecules, such as p53 hormone suppressor. Several studies show that increasing IGF-1 in cell culture media enhances the growth of cancer cell lines. Thus, tumor growth may occur due to the anabolic and anti-apoptotic actions of insulin, mediated by the insulin receptor (IR) and the IGF-1R receptor[6].

OBESITY AFFECTS ON MENTAL HEALTH

Obesity can significantly affect the individual's emotional state, resulting in self-depreciation, low self-esteem, impairing their daily activities. In addition, social pressure to lose weight can be stressful and make it difficult to adhere to obesity treatment. Children and adolescents with obesity can face behavioral disorders, low self-esteem and various social and psychological impairments, including anxiety, depression and suicide risk. Body image begins to form since childhood, and family habits can influence food preferences and the practice of physical activities[6].

For a long time, it was thought that obesity was solely a reflection of psychological and psychiatric disorders. Today, we see that both are related; many obese patients have behavioral eating disorders, psychiatrically classified as eating disorders (ED). By the criteria of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), there is an association of compulsive eating disorder in approximately 30% of cases of obesity, and psychiatric disorders in 60% of them.[6]. In addition, an important link was found between an unhealthy Western dietary pattern - featuring ultra-processed foods - and the development of depression (high glycemic index diets were also associated with a risk factor for the disease). Otherwise, the intake of natural foods, a balance of micronutrients and macronutrients, and an adequate energy balance for the goals and needs of the patients proved to be a protective factor. In short, the relationship with food plays a crucial role in the appearance of symptoms of depression and anxiety, validating the view of obesity and types of eating disorders in mental health[4].

Binge eating disorder (BED) comprises the most prevalent type among obese patients, and, when present, given obesity, it favors evolution to psychiatric comorbidities, including depressive disorder, when compared to a group of obese people without BED [1].

EATING DISORDERS RESULTING FROM MENTAL HEALTH IMPAIRMENT

It is clear that subgroups of obese individuals have abnormal eating patterns. Among them, the three types of ED most described in obese patients are: binge eating disorder (BED), night eating syndrome (NCS) and bulimia nervosa[6].

Periodic binge eating disorder:

It is characterized by episodes of unrestrained consumption of large amounts of food not followed by any inappropriate compensatory behavior, even when the patient is not hungry[6].

From this lack of control, together with the low quality, nutritional and caloric value of the foods usually consumed, a feeling of guilt, shame, disgust begins, which can also unfold into a bulimic condition[6].

Diagnostic criteria suggested by the DSM-IV-TR
A. Recurrent episodes of binge eating (overeating + loss of control)
B. Behaviors associated with binge eating (at least 3):
1. To eat quickly
2. To eat until you feel full
3. To eat large amounts of food even when not hungry
4. To eat alone out of embarrassment from the amount of food
5. Feeling disgusted with yourself, depressed, or too much guilt after bingeing.
C. Marked distress from binge eating
D. Frequency and duration of binge eating: average of 2 days/week for 6 months
E. It does not use inappropriate compensatory methods (eg, purging)

Present in the diagnosis of BED are recurrent episodes of binge eating associated with overeating (for the duration of ingestion) and loss of control[6].

Initial studies addressing associated behavioral measures assess that compulsive eating leads to greater food intake than obese people without this disorder, even in situations where obese people were instructed to eat compulsively. In addition, individuals who had BED reported an earlier onset of obesity, associated with a greater number of diets than obese people without BED, causing a decrease in self-esteem and greater concern with body weight and physical shape. As a result, patients with BED had higher rates of depression and personality disorders[6].

Thus, BED is characterized by several possible etiological triggers and maintenance mechanisms, obtaining a range of therapeutic interventions (such as group or individual psychotherapy, prescription of psychotropic drugs, nutritional guidance...). Therefore, the work of a multidisciplinary team with nutritionists, psychologists, psychiatrists, endocrinologists, general practitioners and physical activity educators is necessary. These professionals must pay attention to treating: binge eating, psychopathological symptoms and overweight[6].

Regarding pharmacological treatment, with Sibutramine and Topiramate it was possible to reduce episodes of binge eating (48.7% versus 28.5%) and weight loss. This treatment associated with psychological interventions increases the efficiency of weight loss through Cognitive Behavioral Therapy (CBT) and behavioral treatments for weight loss[6].

Night Eating Syndrome:

According to Stunkard, SCN happens due to a circadian stress with a special response, mainly in obese people. Studies analyze that

the prevalence among patients of clinics for obesity is 10% and among patients of surgery for obesity is 27%. In addition, approximately 15% of people with binge eating also have CNS[6].

Diagnostic criteria by Birketvedt et al.
A. Morning anorexia
B. Intake ≥ 50% of the daily caloric value after 7 pm
C. Waking up to eat at least once/night in the last 3 months, with awareness of the act
D. Frequent consumption of high-calorie snacks when waking up at night
E. Absence of criteria for bulimia nervosa or binge eating disorder

In their study, Birketvedt et al. examined "night eaters" and control subjects. Night eaters consumed more daily calories, with 56% of their caloric intake between 10 pm and 6 am, with controls only consuming 15% within this range. In addition, night eaters woke more often at night and reported lower mean mood than controls (evidence of a continuing decline in the late afternoon) at 24h. This is due to lower levels of melatonin from mid-afternoon to mid-night[6].

Furthermore, this picture was associated with a stress disorder due to higher plasma levels of cortisol during the day, in addition to not showing increased levels of leptin during the night (which is an appetite suppressant, generating inhibition of impulses of the nocturnal hunger responsible for awakening)[6].

In Brazil, Harb et al. recently translated and validated an SCN questionnaire with satisfactory internal consistency and a general Cronbach's alpha coefficient equal to 0.78. This instrument may be suitable for screening SCN[6].

The treatment of SCN is still in the initial evaluation phase. The approach is characterized by psychoeducation associated with nutritional counseling and pharmacological interventions. As for these, sertraline was shown to be effective in a randomized placebo-controlled study[6].

Nervous bulimia:

Among eating disorders, we have bulimia nervosa as one of the best known, characterized by the use of elimination strategies by purgative compensatory methods - vomiting or diarrhea, induced or not by medication - and non-purgative - prolonged fasting and intensive physical exercise - after episodes of binge eating, in order to avoid weight gain, aiming at an unattainable beauty standard[6].

Diagnostic criteria for bulimia nervosa, DSM V.
A. Recurrent episodes of compulsive eating, characterized by:
• Eating, within a restricted period of time (eg, 2 hours), an amount of food that is noticeably larger than what another person would eat in the same period of time and under the same circumstances.
• Feeling of lack of control over food intake.
• Feeling of lack of control over food intake.
B. Inappropriate and recurrent compensatory behaviors with the intention of not gaining weight, such as self-induced vomiting, misuse of laxatives, diuretics, enemas or other medications, fasting or abusive exercise.
C. Both the binge eating episodes and the inappropriate compensatory behaviors occurred, on average, at least once a week over the past 3 months.
D. Self-evaluation is excessively linked to body shape and weight.
E. The condition does not occur exclusively during episodes of anorexia nervosa.
Specify remission: partial or complete.
Specify severity: mild (1 to 3 compensatory episodes per week), moderate (4 to 7 compensatory episodes per week), severe (8 to 13 compensatory episodes per week), or extreme (greater than 14 compensatory episodes per week).

From the diagnostic criteria mentioned in the table above, an intimate relationship between BED and bulimia nervosa is observed, with the main difference being the non-use of compensatory mechanisms in the first[6].

CONSEQUENCES OF EATING DISORDERS ON PHYSICAL HEALTH

From the consequences of eating disorders, in addition to psychosocial injuries, one can address the consequences on physical health and clinical complications that can help in the diagnostic investigation. Among these, we mention: dehydration, hydroelectrolytic changes, headache, hypotension, fainting, fatigue, edema. In the case of bulimia, we can find the *Russell's sign,* marked by callosity on the back of the hand due to tooth abrasion, in addition to erosion of dental enamel, caries, hypertrophy of parotid glands. As gastrointestinal symptoms, the patient may complain of abdominal pain, gastritis, in addition to presenting lesions in the pharynx, Mallory-Weiss syndrome, hiatal hernia, esophageal spasms, gastroesophageal reflux disease, among others. There are also hypovitaminosis, due to impaired intestinal absorption of nutrients, recommending the dosage of vitamins, especially B12. In the long term, they may be associated with a higher incidence in these patients of kidney problems, alopecia, cardiac arrhythmia, heart failure, esophageal rupture, systemic arterial hypertension, diabetes, among others[6].

CONCLUSION

From this study, it is observed the wide correlation of obesity not only as a physical disorder, but with major psychosocial impairments. Obese patients have a higher rate of depression, anxiety, in addition to other repercussions on quality of life. Along with this aspect of mental health, eating disorders are associated with a higher incidence in these individuals, whether as a cause or consequence of the metabolic condition. The importance of psychotherapeutic follow-up for these patients is significant and positive, therefore, it must be more valued as part of the therapeutic treatment.

REFERENCES

1. *CALDAS, N. R. et al.* ***Binge eating disorder, frequency of depression, and systemic inflammatory state in individuals with obesity – A cross sectional study****. Archives of Endocrinology and Metabolism, São Paulo, v. 65, n. 1, p. 26-32, Feb. 2021. DOI: https://doi.org/10.20945/2359-3997000000311.*

2. *CAO, Yihai (org.).* ***Adipose tissue angiogenesis as a therapeutic target for obesity and metabolic diseases****. 2010. Disponível em: https://www.nature.com/articles/nrd3055. Acesso em: 05 ago. 2022.*

3. *DEL CAMPO JA, et al.* ***Role of inflammatory response in liver diseases: Therapeutic strategies****. World Journal of Hepatology, 2018; 10 (1): 1-7.*

4. *LANG, U. E.; BEGLINGER, C.; SCHWEINFURTH, N.; WALTER, M.; BORGWARDT, S.* ***Nutritional Aspects of Depression. Cellular Physiology and Biochemistry****, v. 37, n. 3, p. 1029-1043, 2015. DOI: 10.1159/000430231.*

5. *LIMA, Ronaldo César Aguiar; CARNEVALI JÚNIOR, Luiz Carlos; FERREIRA, Layana Liss Rodrigues; BEZERRA, Layanny Teles Linhares; BEZERRA, Tayanny Teles Linhares; LIMA, Bruna da Conceição.* ***PRINCIPAIS ALTERAÇÕES FISIOLÓGICAS DECORRENTES DA OBESIDADE: UM ESTUDO TEÓRICO.*** *2018. Disponível em: https://www.researchgate.net/publication/337356892_PRINCIPAIS_ALTERACOES_FISIOLOGICAS_DECORRENTES_DA_OBESIDADE_UM_ESTUDO_TEORICO. Acesso em: 05 ago.. 2022*

6. ***Tratado de obesidade*** */ Marcio C. Mancini [et. al]. - 2. ed. - Rio de Janeiro : Guanabara Koogan, 2015. il.*