Obesity, its Causes, and Effects on the General Health of Children and Adolescents

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Abstract— Obesity is defined as a medical condition in which excess body fat accumulates to the point of causing negative health effects, resulting in a reduced life expectancy and/or increased health problems. BMI, a measure corresponding to weight by height, defines overweight (pre-obese) individuals as individuals with a BMI between 25 kg/m² and 30 kg/m², and identifies obese individuals as having a BMI greater than 30 kg/m2.

Keywords— Health – Obesity – health effects – General Health - Adolescents

I. INTRODUCTION

Obesity increases the risk of many diseases associated with obesity, especially heart disease, type 2 diabetes, breathing difficulties during sleep, certain types of cancer, and osteoarthritis. Obesity usually results from a combination of extra calories, less physical activity, and genetic influences. That's even though a few cases are primarily caused by genes, endocrine disorders, medications, and mental illness. It should be noted that the evidence that obese individuals eat little but gain weight due to slow metabolism is low; on average, the energy loss of obese people is greater than that of their non-obese counterparts due to the energy requirement to maintain an increased body mass. The first treatment for obesity is diet and exercise. To support such activities, or in case of disappointment with this treatment, it may be possible to take slimming drugs to reduce appetite or prevent fat absorption. However, in advanced cases, surgery or a balloon is placed inside the stomach to reduce its size and/or the length of the intestine, which leads to early satiety and a reduced ability to absorb nutrients from food. Obesity is a leading preventable cause of death worldwide, and is increasingly common or prevalent among adults and children, and authorities consider obesity one of the most serious public health problems of the twenty-first century. Obesity is seen as a stigma in the modern world (particularly the Western world), although it was widely seen as a symbol of wealth and fertility in other eras throughout history, a view still prevalent in some parts of the world.

II.   THE EFFECTS OF OBESITY ON HEALTH

Excess body weight is linked to many diseases, especially heart disease, type 2 diabetes, obstructive sleep apnea, certain types of cancer, and osteoarthritis. This leads to a decrease in the life expectancy of the individual and/or to the occurrence of increased health problems.

Death rate

Obesity is one of the leading preventable causes of death worldwide. Large-scale studies in both the USA and Europe have found that the risk of death is lower if the BMI ranges between 22.5 and 25 kg/m² in non-smokers and if it is between 24 and 27 kg/m² in smokers, with an increased risk of developing Death in line with the change in both directions. A BMI greater than 32 was associated with a doubling of the death rate among women over a 16-year period. It is estimated that obesity in the United States is an underlying cause of the increase in death rates from 111,909 to 265,000 per year, while the cause of 1 million (7.7%) deaths in the European Union is due to being overweight. On average, obesity reduces life expectancy by approximately six to seven years: a mean BMI of 30-35 reduces life expectancy by two to four years, while severe obesity reduces life expectancy by ten years.

Morbidity

Obesity increases the risk of many physical and mental illnesses. The prevalence of such diseases can be observed in metabolic syndrome, which represents a mixture of disorders that include: type 2 diabetes, high blood pressure, high cholesterol, and high triglyceride levels.

Complications are caused either directly through obesity or indirectly through mechanisms of the same cause such as poor nutrition or a sedentary lifestyle. The strength of the relationship between obesity and specific conditions varies. One of the strongest of these is the relationship between obesity and type 2 diabetes. Excess weight accounts for 64% of diabetes cases in men and 77% of cases in women.

Health consequences fall into two main categories: those that are attributable to the effects of increased fat mass (such as osteoarthritis, obstructive sleep apnea, and social aversion) and those that occur as a result of an increased number of fat cells (diabetes, cancer, heart disease, and fatty liver disease). Non-alcoholic.Excess body fat changes the body's response to insulin, which may lead to insulin resistance.Excess fat also increases the risk of inflammation and blood clotting. Although the negative health consequences of obesity in all humans are well established by available evidence, in some subgroups, health outcomes improved with increasing body mass index, a phenomenon known as the Obesity Survival Paradox. This paradox was first described in 1999 in overweight and obese individuals with hemodialysis, and was subsequently observed in people with heart failure and exocrine arteropathy.

In individuals with heart failure, those with a BMI between 30 and 34.9 had lower mortality than individuals of normal weight. This has been attributed to the fact that people often lose weight as their disease progresses. Similar results have been found in other types of heart disease. People with grade

1 obesity and heart disease do not have higher rates of heart problems than people of normal weight who also have heart disease. However, in individuals with higher levels of obesity, the likelihood of adverse consequences increases. Even after coronary bypass surgery, no increase in mortality was observed in overweight or obese individuals. One study found that the decrease in mortality could be explained by the more intensive treatment that obese patients receive after cardiac surgery. Another study found that if emphysema, or COPD, was taken into account in patients with EAD, obesity would have no beneficial effect afterwards.

III. OBESITY CAUSES

Annually, the US National Institutes of Health spends approximately $800 million on studies to understand the neurological, metabolic, and genetic underpinnings of obesity. During the research, the scientists uncovered complex biochemical pathways; feedback loops connect the brain to the digestive system; a new understanding of the regulating functions of adipose tissue; subtle genetic changes make some groups fatter than others; And the strong possibility that some foods and toxic substances when exposed to them modify some of these factors or mitigate their effects. Accordingly, we can divide the main causes behind obesity as follows:

Brain: Scientists have long known that the hypothalamus and the brain stem help regulate feelings of hunger and satiety. Over the past few years, researchers have found that the limbic system's reward centers and the evaluation functions of the prefrontal cortex are also deeply involved in those two processes: hunger and satiety. In fact, chronic overeating bears a biochemical similarity to drug addiction.

Metabolism: The ability to burn and store energy varies greatly from cell to cell. In 2009, three studies published in the New England Journal of Medicine demonstrated that some women and men continue into adulthood to benefit from small stores of brown fat. This fat is associated with thinness, unlike white fat. Brown fat helps generate heat and appears to be closer to muscle than white fat, whose primary purpose is to store excess energy.

Genes: Researchers have confirmed a difference in 20 abnormal genes that make their owners predisposed to gaining weight easily. But other research later showed that these effects are modest and not considered as a reason for this epidemic spread of obesity. However, it is possible that genes will continue to play a role, through environmental influence, turning it on or off. So far, these genetic keys to obesity have been identified in mice, although a few are candidates for action in humans.

It is believed that the combination of excessive caloric intake and lack of physical activity at the individual level explains most cases of obesity. While a small number of cases are attributed to genetic factors, medical reasons, or psychological diseases. On the other hand, the increase in obesity rates on a social level is due to easy-to-obtain delicacies, increased reliance on automobiles, and mechanized manufacturing.

A 2006 study identified ten other factors that have contributed to the current increase in obesity: insufficient sleep, endocrine disruptors (environmental pollutants that interfere with lipid metabolism), decreased ambient temperature, decreased smoking rates because smoking reduces appetite, use Increased medications that can cause weight gain (eg, atypical antipsychotics), relative increases in ethnic and age groups that tend to be overweight, later pregnancy (which may lead to susceptibility to obesity in newborns), risk factors Anatomy across different generations, natural selection for higher BMIs, and finally mixed interbreeding that leads to an increased concentration of factors leading to obesity (this will not necessarily increase the number of obese individuals, but it may increase the average weights of the population). Although there is significant evidence supporting the impact of these mechanisms on the increasing prevalence of obesity, this evidence is still inconclusive, and those who conducted the study acknowledge that these factors may be less influential than the factors discussed in the previous paragraph.

The share of dietary energy per capita varies significantly between regions and countries, and has also varied significantly over time. The average available calories per person (amount of food purchased) increased over the period from the beginning of the seventies in the twentieth century to the end of the nineties than in all parts of the world except Eastern Europe. In 1996, the United States achieved the highest rate of consumption, with 3,654 calories per person. However, this rate increased further during 2003 until it reached "3754". At the end of the nineties of the twentieth century, the Europeans were eating about "3394" calories per person, while in the developing regions of Asia, that amounted to "2648" calories per person, and in Black Africa it reached "2176" per person. It has also been found that total calorie consumption is related to obesity.

The wide availability of dietary guidelines "with a little" helps address the problems of overeating and poor food selection. From 1971 to 2000, obesity rates in the United States increased from 14.5% to 30.9%. During the same period, there was an increase in average calories consumed. For women, the average increase was 335 calories per day (1542 calories in 1971 and 1877 calories in 2004), while the average increase for men was 168 calories per day (2450 calories in 1971 and 2,618 calories in 2004). Most of these extra calories came from increased consumption of carbohydrates, not fats. The main source of these carbohydrates is sweetened beverages, which alone provide approximately 25% of daily calories in adults in the United States these days. Here it is believed that the consumption of sweetened beverages contributes to the increase in obesity rates.

IV. HEREDITY, PHYSICAL AND PSYCHOLOGICAL DISEASES

Like many other medical conditions, obesity is the result of the interaction of genetic and environmental factors together. The polymorphism of genes that control appetite and metabolism makes an individual more likely to develop obesity if he has enough calories. Since 2006, more than 41

of these different genetic variants have been linked to obesity in an appropriate environment. The incidence of obesity, which can be attributed to genetic factors, varies according to the individuals under study, from 6% to 85%. Obesity is a hallmark of many syndromes such as Prader-Willi syndrome, Bardet-Biedl syndrome, Cohen syndrome, and Momo syndrome. (Sometimes, the term "non-syndromic obesity" is used to exclude such cases.) In early-stage severely obese individuals (diagnosed at age 10 and having a BMI of 3 standard deviations from the normal), only 7% had a single DNA mutation.

Studies that focused on inheritance patterns rather than specific genes found that 80% of the offspring of any obese parents were also obese, and on the other hand, less than 10% of the offspring of any parents of normal weight were obese.

The Thrifty Gene Hypothesis makes the assumption that certain ethnic groups may be more likely to be obese in equal environments. Their ability to take advantage of the short periods of food availability by storing energy as fat is of great benefit in times of low food availability, as individuals with more fat are more likely to survive during periods of famine. However, this tendency to store fat may not be appropriate in societies with permanent food sources. This is supposed to be the reason why the Pima Indians, who grew up in a desert environment, had the highest rates of obesity when exposed to the Western lifestyle.

Physical and Mental Illnesses

Some mental and physical illnesses, as well as certain medications, can increase the likelihood of obesity. Physical diseases that increase the risk of obesity include rare genetic syndromes, as well as some genetic or acquired conditions, including: hypothyroidism, hyperadrenocorticism, also called "Cushing's syndrome", growth hormone deficiency, and eating disorders: bulimia nervosa, eating syndrome nocturnal; However, obesity is not considered a mental disorder, so it is not listed in the Diagnostic and Statistical Manual of Mental Disorders. The risk of obesity and obesity increases among patients who suffer from mental disorders, rather than those who do not suffer from them.

Some drugs may also cause weight gain or changes in body composition, and these drugs include insulin, sulfonylurea, thiazolidinediones, atypical antipsychotics, antidepressants, stimulants, in addition to some antibiotics, including (phenytoin and valproate), as well as pizotifen and some forms of contraceptives. Hormonal pregnancy;

Social determinants

While genetic influences play an important role in understanding obesity, they cannot explain the current significant increase observed in specific countries or worldwide. Although it is accepted that a higher intake of calories than is consumed in the human body leads to obesity at the individual level, the reason for these shifts between intake and consumption factors at the societal level is still a matter of debate and controversy. There are a

number of theories about this cause, while most specialists think that the cause is a combination of various factors.

The association between social class and BMI varies globally. A study conducted in 1989 concluded that women are less likely to be obese in developed countries. While no significant differences were observed between men in different social classes. In the developing world, obesity affects more women, men, and children in the higher social classes. However, this study was updated again in 2007, and found the same relationships, but they were weaker. He believed that the decrease in the strength of the link was due to the effects of globalization. A correlation has also emerged between obesity levels in adults and the proportion of adolescents, in developed countries, who suffer from obesity with income disparity. A similar relationship has been found between 'different states' in the US: more adults - even in the highest social strata - are found to be obese in states with unequal incomes.

Many explanations have been formulated for the associations between BMI and social class. It is believed that the rich, in developed countries, can get more nutritious foods, but social pressure pushes them to keep in shape, and they have more opportunities along with higher expectations of access to physical fitness. In developing countries, the ability to purchase food, the higher energy consumption associated with physically demanding work, and cultural values favoring larger body sizes are thought to contribute to the patterns observed. Also, the attitudes towards body mass espoused by individuals may play a major role in

causing an individual to become obese. A correlation was found in BMI changes over time among friends, children, husbands, and wives. Stress and perceived low social rank also appear to increase the risk of obesity.

Smoking also plays an important role in influencing an individual's weight. Men who stop smoking gain an average of 4.4 kilograms (9.7 pounds), and women gain 5.00 kilograms (11.01 pounds) over ten years. However, changing smoking rates has little effect on overall obesity rates. The number of children in the same family, in the United States, is associated with the risk of obesity. A woman's risk of obesity increases by 7% with each child, while the risk of a man increases by 4% with each child. This may be explained in part by the fact that having dependent children results in less physical exertion by parents in the Western world. In the developing world, urbanization plays a role in increasing the incidence of obesity. In China, the overall rates of obesity are less than 5%, although in some cities the rates of obesity are more than 20%. It is also believed that malnutrition plays a role in the high rates of obesity, at a young age, in the developing world. The change in endocrine secretions that occurs during periods of malnutrition may support fat storage once more calories are available.

Infectious agents

The study of the effect of infectious agents on metabolism is still in its early stages. Where it was found that intestinal parasites differ between obese and thin individuals. There is evidence that stomach parasites in both obese and thin people affect the metabolic capacity of individuals. It is

believed that this apparent alteration of metabolic capacity is carried out with the aim of conferring a greater capacity for energy, which contributes to obesity. It is not yet clear whether these differences are a direct cause of obesity or a consequence of it. He also found a link between viruses and obesity in humans and many different animal species. The extent to which these associations contribute to the rising incidence of obesity is now to be determined.

V. PATHOLOGICAL PHYSIOLOGY

Flair summarizes several pathophysiological mechanisms that have been involved in the development and persistence of obesity. Here it is noted that this field of research was not the subject of research and investigation until the discovery of leptin in 1994. Since this discovery, many other hormonal mechanisms that contribute to the regulation of appetite and food intake, and in adipose tissue storage patterns, as well as the development of resistance against insulin have been identified. . Also, since the discovery of leptin, ghrelin, insulin, orexin, polypeptide 3-36, cholecytokinin, and adiponectin, in addition to many other mediators, have been studied. Where adiponectin represents the group of mediators that are produced by adipose tissue; it is believed to modify many diseases associated with obesity.

Leptin and ghrelin play a complementary role in influencing appetite, as the stomach produces ghrelin to regulate appetite in the short term (meaning eating is done when the stomach is empty and stopped when the stomach is stretched). While adipose tissue secretes leptin as an indicator of body fat stores, it interferes with appetite control

in the long run (meaning eating more when fat stores are low and eating less when fat stores are large). Although leptin administration may be effective in a small subset of obese and leptin-deficient patients, it is believed that most obese individuals are resistant to leptin, and have been found to have higher levels of it. This resistance is believed to partly explain the reason why leptin is not effective in reducing appetite in most obese people.

While ghrelin and leptin are produced decently, they control appetite through their effects on the central nervous system. They and other appetite-related hormones, in particular, affect the hypothalamus, an area of the brain that is instrumental in regulating food intake and energy consumption. There are many pathways within the hypothalamus that are involved in its role in appetite regulation, perhaps the most obvious being the melanocortin pathway. This pathway begins with a portion of the hypothalamus, the arcuate nucleus, which excretes its secretions into the lateral hypothalamus and the medial hypothalamus, which are the brain's feeding and satiety centers, respectively.

The arcuate nucleus contains two distinct groups of neurons. One is neuropeptides and agouti-associated peptide which have excitatory inputs traveling into the lateral hypothalamus and inhibitory inputs towards the medial hypothalamic region. The second group expresses the initiating hormone "propiomelanocortin" and the regulating transcript of cocaine and amphetamine, which have tonic inputs to the middle hypothalamic region and inhibitory inputs to the lateral hypothalamus. Thus, neurons of NPP

and agouti-associated peptide stimulate feeding and inhibit the feeling of satiety, while PAP neurons stimulate the process of satiety and inhibit food intake or feeding. These two groups of neurons of the arcuate nucleus are partially regulated by leptin, wherein leptin inhibits the neuropeptide group and the agouti-related peptide, while stimulating the proopiomelanocortin group. Ingestion of food may be responsible for some hereditary and acquired forms of obesity.

VI.   BARIATRIC TREATMENT

The main treatment for obesity consists of slimming or diet and exercise. Diet programs may lead to weight loss in the short term, but maintaining this weight loss can be a problem, and this usually requires that physical exercise and diet, containing fewer calories, become a constant part of one's lifestyle. Noting that the success rates in maintaining weight after its loss are low and range between 2 and 20%. However, in settings where the required care is available, about 67% of individuals who lost more than 10% of their weight retained this weight loss, or continued to lose weight for one year. The average of those individuals who lost more than 3kg of their total body weight might keep doing so for five years.

Some studies have found significant benefits associated with weight loss and its relationship to mortality in some societies. A prospective study of obese women who suffer from diseases related to being overweight found that decreasing the intended weight and by any amount was associated with a 20% reduction in mortality. In obese women without obesity-related diseases, a loss of 9 kg (20 lb) was associated with a 25% reduction in mortality. While a recent study concluded that certain subgroups, such as those with type 2 diabetes and women, benefit in the long run, which reduces rates in those subgroups for all reasons, while the results for men did not show an improvement with weight loss. A subsequent study concluded that the reduction in mortality was due to the intended weight loss in obese individuals. Bariatric surgery is the most effective bariatric treatment. However, due to its cost and risk of complications, researchers are looking for other effective and less dangerous treatments.

VII.   CONCLUSION

Bariatric surgery (weight loss surgery) is the use of surgical intervention to treat obesity. Since each surgery has its own complications, the surgery is only recommended for people who are obese (BMI >40) and have failed to lose weight by changing their diet and taking drugs. Here, weight loss surgery is based on several principles: the two most popular methods are (i) reducing the size of the stomach (eg modified gastric banding and cosmetic vertical gastric banding), which results in an early sense of satiety, and reducing the length of the intestine exposed to food) this is a method that directly reduces absorption. Gastric banding surgery can return the patient to his first condition, while intestinal shortening surgery is not possible with it. In addition, some surgical procedures can be performed through the use of the laparoscope. However, complications from weight loss surgery are multiple and frequent.

While surgery to eliminate severe obesity is associated with long-term weight loss and lower mortality in general. One study found a weight loss of between 14% and 25% over 10 years (depending on the type of procedure performed), and a 29% reduction in all-cause mortality when compared to standard weight loss procedures. It also found a significant reduction in the risk of diabetes, heart disease and cancer after bariatric surgery. There is a significant decrease in weight during the first months after surgery, and this decrease will continue in the long term. While in one study there was an incomprehensible rise in deaths from accidents and suicide, this did not equate to the benefit of preventing disease. When comparing the two previous methods, it is noted that the gastric bypass method leads to a weight loss of about 30% more than gastric bands one year after surgery.

The effectiveness of liposuction in combating obesity has not been well defined yet. Some studies report benefits to this method, while other studies have not demonstrated any benefit from these procedures. One of the most promising surgeries in the field is the treatment that involves placing a balloon inside the stomach through the use of gastroscopies. One type of balloon results in a weight loss of 5.7 BMI units in six months, or 14.7 kilograms (or 32.4 pounds). However, it is common to gain that lost weight after removing this balloon, while 4.2% of people could not tolerate using it.

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