Obesity and metabolic syndrome pdf

Obesity is rampant in the United States and is becoming more prevalent worldwide. The increase in the prevalence of obesity is due to two main factors, abundant supplies of inexpensive foodstuffs and sedentary jobs. Both are controlled in not a small part of the technology. Thanks to technology, it is possible to produce a large number of cheap foodstuffs, and manual work is rapidly disappearing. In areas of the world where these advances have not been penetrated, obesity is not a significant public health problem. Obesity is thus a direct result of technological progress and poses a serious challenge to the technological society. Obesity should also be recognized as a product of a free society with many food options and employment opportunities. A public health approach to obesity that restricts choice would not be acceptable to a free society. This fact places an increased responsibility on the person to recognize the main causes of obesity and change behavior to reduce the personal burden of obesity. It is well known that obesity extracts social costs. The costs of physical health are less well recognized by the public. The main physical consequence of obesity is atherosclerotic cardiovascular disease (ASCVD) (1). Much of ASCVD is caused by obesity mediated by type 2 diabetes. But obesity is accompanied by a number of other risk factors for ASCVD. The amount of risk factors predisposing to ASCVD is called metabolic syndrome. Obesity is also accompanied by other medical complications besides ASCVD and diabetes; These include fatty liver, gallstone cholesterol, sleep apnea, osteoarthritis, and polycystic ovaries. These disorders are commonly found in people who carry metabolic syndrome. Obesity can be called the main risk factor for cardiovascular disease (ASCVD) (2). This is called this because it increases the risk to ASCVD through other risk factors. The latter include the main risk factors (hypercholesterolemia, hypertension, hyperglycemia) and emerging risk factors (atherogenic dyslipidemia, insulin resistance, prosposable state, protrombotic state). The relationship between obesity and major and emerging risk factors varies according to the genetic and acquired characteristics of people. Most obese people who develop ASCVD tend to have clustering of major and emerging risk factors (metabolic syndrome). The constellation of major and emerging risk factors that make up metabolic syndrome can be called metabolic risk factors (3). This article will first look at the variable characteristics of obesity; this will be followed by a study of the link between obesity and metabolic and finally, the link between metabolic syndrome and ASCVD will be considered. Obesity obesity categories can be defined as excess excess Fat. The surrogate marker of fat deposits is the body mass index (BMI), which is determined by the weight (kilograms) divided by height in a square (square meters). Clinically, a BMI of 25-29 kg/m2 is called overweight; higher BMI (≥30 kg/m2) are called obesity (4). The best way to determine obesity would be in terms of the percentage of total fat (4). This can be measured by several methods (skin thickness, bioelectric non-selection, underwater weighing). In terms of body fat percentage, obesity can be defined as 25% or more in men and 35% or more in women. Measuring the percentage of body fat is rarely used in clinical practice, however, due to inconvenience and cost. The best way to assess obesity in clinical practice is to measure waist circumference. This is because excess abdominal fat is most closely associated with metabolic risk factors. In the United States, abdominal obesity is defined as waist circumference in men 102 cm or more, and in women 88 cm or more (4). In other countries, a smaller increase in waist circumference was associated with metabolic risk factors, and other standards were used. Abdominal fat is located in two main compartments: sc and ip (visceral). The latter consists of fat mentum and mesenteria. Fatty acids released by visceral fat flow into the circulation of the portal. Some researchers (5) believe that excess visceral fat (visceral obesity) is more strongly associated with metabolic risk factors than any other fat compartment. Subcutaneous fat, however, is a much larger compartment than visceral fat. The latter is usually divided into gluteal and truncal sc of adipose tissue. Truncal fat is more closely associated with metabolic risk factors than gluteal fats (4). In addition, truncal sc fat may have a greater effect on risk factors than visceral fat due to its greater mass (6, 7). Several terms have been applied to excess fat in the trunk: abdominal obesity, truncal obesity, and upper body obesity. In fact, there is a strong correlation between waist circumference and upper body fat content. Hence because increased girth is most readily recognized clinically, the term abdominal obesity is useful and satisfactory (2, 4). Body Fat and Metabolic Syndrome Metabolic Syndrome is a constellation of metabolic risk factors that consist of the following (2): Atherogenic dyslipidemia serum height triglycerides, apolipoprotein B (apo B) and small low-density lipoproteins (LDL) particles plus low-density lipoproteins (HDL) cholesterol High blood pressure Elevated glucose associated with insulin resistanceProtrombotic stateMany stateMany these factors can be identified by special testing, but not measured in special testing, but not measured in Practice. Recently, the National Cholesterol Education Program for Adults Treatment Group III III III (2) suggested a simple scheme for routine diagnosis of metabolic syndrome. According to this scheme, the diagnosis of metabolic syndrome can be made if a person has three of the following five features: Increased waist circumference (≥102 cm in men and ≥ 88 cm in women) Elevated triglycerides (≥150 mg/dL) Reduced HDL cholesterol (LT;40 mg/dL) dl in men and lt; 50 mg/dL in women) High blood pressure (≥130/85 mmHg or in the treatment of hypertension) Increased glucose (≥100 mg/dL) When the waist circumference is 102 cm or more in men or 88 cm or more in women, the term abdominal obesity can be used. The advantage of measuring waist circumference is that excess abdominal fat is more closely correlated with the presence of metabolic risk factors than total fat. The cut points for determining abdominal obesity are arbitrary. For susceptible people, less abdominal fat accumulation can cause or exacerbate metabolic risk factors. This is especially true in some populations; for example, in Asian populations, waist-lengthity points have been identified to determine abdominal obesity. Patients with diabetes (rapid glucose ≥ 126 mg/dL) are said to have metabolic syndrome if two other features are present. If a person is eligible for metabolic syndrome in accordance with the adult Treatment Panel III criteria, measuring post-prandial glucose 2-h can reveal a diagnosis of diabetes (2-h glucose ≥ 200 mg/dL) or impaired glucose tolerance (IGT) (2-h glucose 140-199 mg/dL) (1). The presence of IGT indicates an increased risk of developing type 2 diabetes (8). Additional testing can provide confirmation of metabolic syndrome. Confirming biomarkers for this syndrome include high fasting insulin levels, 2-h post-predial insulin, apo B, an increase in small LDL particles, C-reactive protein (CRP), fibrinogen and plasminogen activator inhibitor (PAI)-1. The clinical usefulness of detecting these additional anomalies beyond confirmation of the syndrome is uncertain, although studies are underway to assess the potential usefulness. For example, the presence of elevated CRP may indicate a greater risk of developing acute coronary syndromes (9). The area of concern for obesity and metabolic syndrome concerns the role of insulin resistance. Most people with multiple metabolic risk factors are resistant to insulin. This observation led to the concept that insulin resistance is the cause of metabolic syndrome (10). This concept, in turn, gave rise to an alternative term for metabolic syndrome, namely insulin resistance syndrome (10). Various pathogenic patterns were proposed to explain the link between insulin resistance and metabolic risk factors. There is no doubt that insulin is a risk factor for IGT and type 2 diabetes. The cause-and-effect relationship between insulin resistance and other metabolic risk factors is less In addition, the interaction between obesity and defects in insulin signaling is so difficult that it is still impossible to separate the two. For example, obesity causes insulin resistance, while insulin resistance seems to exacerbate the adverse effects of obesity. A strong case can be done for the role of genetic forms of insulin resistance is a contribution to metabolic syndrome in the general population. On the other hand, there is no doubt that the increase in overweight/obesity prevalence is mainly responsible for the increase in the prevalence of metabolic syndrome in the United States and around the world (11). Our understanding of the link between obesity and metabolic risk factors is growing rapidly. This understanding is based on the discovery of several products released from adipocytes. In the presence of obesity, these products are released in abnormal quantities. Each of these products was involved in cause-and-effect metabolic risk factors. Below is a list of factors most implicated in the development of metabolic syndrome (12): Nonesterified fatty acids (NEFAs)Inflammatory cytokinesPAI-1AdiponectinLeptinResistinCurrent concept of communication of each of these products metabolic risk factors can be revised. Nave. Obese people release increased amounts of NEFAs into circulation (13). NEFA is obtained by either the triglycerides of the adipose tissue. The greater the amount of fat in the fat tissue, the greater the amount of NEFA released will be. This greater release of NEFA continues despite higher insulin levels that are present in obese people. Although high insulin levels suppress fat tissue lipolysis, they cannot reduce NEFA emissions to normal obesity. NEFA is the main source of nutrient energy in a state of fasting. In obesity, however, the NEFA flow exceeds the needs of tissues, and the protective mechanisms must come into play. The effects of these protective mechanisms undoubtedly contribute to metabolic risk factors. Excessive inflow of NEFA into the muscles leads to insulin resistance. The mechanisms by which the increase in fatty acids in the muscles cause insulin resistance have not been fully clarified. Randle et al. (14) has previously postulated that excess fatty acids inhibit glucose oxidation (glucose-fatty acid cycle). Recent studies (15) show that muscle levels of diacylglycerol are raised, which stimulates serin insulin receptor phosphorylation and thus suppresses normal insulin signaling. Other mechanisms (16) have been proposed and may play a role. The resulting insulin resistance in the muscles predisposes to hyperglycemia; The latter becomes clinically manifested in these individuals to acquire a defect in the secret ability of insulin. Inflow of excess NEFA liver increases the content of triglycerides in the liver (fat liver) (17). The accumulation of fat in the liver would seem to be insulin resistance, as in the muscles. Reducing the action of insulin in the liver allows to increase glyconeogenesis and increase the output of hepatic glucose; this will highlight hyperglycemia in those patients who have reduced insulin secretory capacity. Increased fat in the liver also contributes to the development of atherogenic dyslipidemia. This provides an incentive to increase the formation and secretion of very LDL (VLDL) particles. The result is higher levels of triglycerides in serum, apo B and small particles of LDL. High serum triglycerides reduce the concentration of HDL-cholesterol by exchanging VLDL triglycerides with HDL cholesterol esters. The decrease in HDL-cholesterol is highlighted by the increase in the synthesis of hepatic lipase, which occurs in obese people induced by fatty liver; lipase degrades HDL particles by turning large HDL into a small HDL. An important but unresolved question is whether high levels of NEFA contribute to increased blood pressure or pro-life. Hypotheses have been developed that link higher levels of NEFA to higher blood pressure (18). Whether the link is a cause-and-effect relationship remains to be determined. In addition, the accumulation of fat in the liver is reported to be associated with an increase in hepatic synthesis of PAI-1, fibrinogen and inflammatory cytokines, key mediators of prothrombothic and pro-inflammatory conditions (19). Inflammatory cytokines. Fat tissue synthesizes and secretes TNFH, IL-6 and other cytokines. Production of these cytokines is increasing in obese people. This increased synthesis may interfere with the action of insulin to suppress lipolysis; if so, it will represent insulin resistance to adipose tissue. In addition, obese people have elevated levels of circulating cytokines; it is not yet clear whether these circulating cytokines have systemic effects, i.e. contribute to insulin resistance in the muscles (15), increased synthesis of acute-phase reactionary drugs in the liver (CRP and fibrinogen), or activation of macrophages in atheromat plaques (20). Perhaps the increased release of acute phase response from the liver may be the result of the complete accumulation of lipids in this organ. PAY-1. Fat tissue synthesizes PAI-1, too. Reports show that abdominal adipose tissue is more active in the synthesis of PAI-1 than the lower body fat tissue (21). Another source of PAI-1 may be fatty liver. The high level of PAI-1 in obese people, together with the high content of plasma fibrinogen observed in such individuals, contributes to prothrombotic condition. Other foods of adipose tissue. Several other foods of adipose tissue can affect the development of metabolic syndrome. However, their exact role has yet to be fully determined. Adiponectin is one of the potentially important products (22). The substance is reported to have and anti-atherogenic properties. Obese people tend to have low levels of adiponectin and therefore may devoid of protective effects from metabolic syndrome. Leptin can also play a systemic role, in addition to being a fatty tissue derived from appetite suppressant. Discussions are under way as to whether the systemic effects of leptin are direct or secondary to its actions for the central nervous system. Despite this, this hormone is reported to have beneficial effects on the liver to protect against fatty liver (23). Its mechanism may be to increase the oxidation of fatty acids in the liver. Finally, resyfin is a fatty tissue-derived hormone that seemingly opposes the action of insulin

(24). Whether it has a physiological role in the human body has not yet been determined. Obesity-induced metabolic syndrome as a multidimensional risk factor for ASCVD and type 2 diabetes Several recent reports (25-28) show that the presence of metabolic syndrome is associated with an increased risk for both ASCVD and type 2 diabetes. Individuals with metabolic syndrome have at least a 2-fold increased risk for ASCVD, compared to those without (1). The risk of developing type 2 diabetes in men and women increases by about 5 times (1). The risk of developing diabetes is highest in people with fasting glucose disorders or IGT. Once a patient develops type 2 diabetes, the risk for ASCVD increases. Not only does the relative risk of coronary heart disease (ICD) increase 2-3 times, but as soon as ICD is manifested in a patient with diabetes, the prognosis for survival is significantly reduced (2). In addition, diabetes is accompanied by a microvascular disease, which is a common cause of chronic renal failure. The relationship between metabolic risk factors and the development of ASCVD is complex and certainly not well understood. However, a brief overview of hypothetical mechanisms may be of interest. Atherogenic dyslipidemia. This condition is characterized by an increase in elevated triglycerides (and an increase in the number of VLDL particles), an increase in small LDL particles, and low HDL cholesterol (2). It is usually present in obese people. The increase in the number of VLDL and LDL particles explains the elevated level of total apo B commonly observed with atherogenic dyslipidemia. The atherogenic potential of each lipoprotein anomaly has long been a topic of great interest, but which is not fully solved. For many years triglycerides of rich lipoprotein (TGRLPs) were considered non-atherogenic. However, there is growing evidence that smaller TGRLP (residual lipoproteins) is actually atherogenic (29). This is evidenced by studies of laboratory animals, patients with genetic disorders that cause residue accumulation, meta-analysis of epidemiological studies and clinical trials (1). TGRLPs as a class is a mixture of lipoproteins, and it was difficult However, there is a growing consensus among investigators that TGRLP TGRLP is growing definitely contains atherogenic lipoproteins. LDL particles associated with metabolic syndrome and atherogenic dyslipidemia are usually small and dense. The theory is widespread in that smaller LDL particles are more atherogenic than larger LDL (30). Smaller LDLs can filter more readily into the arterial wall. They may then be more prone to atherogenic modification. Despite this, not all studies are convinced that small PARTICLES of LDL are unusually atherogenic, compared to other apo B-containing lipoproteins. However, when small LDLs are present, the total number of lipoprotein particles in the LDL fraction usually increases (31). Most researchers would agree that the greater the number of LDL particles present, the higher the atherogenic potential. In other words, small particles of LDL are often a surrogate for an increase in the number of LDL particles (31). A simple strategy for estimating the amount of atherogenic particles is to measure LDL-VLDL cholesterol (cholesterol, not HDL) or general apo B (2). In people with metabolic syndrome and atherogenic dyslipidemia, both LDL-VLDL cholesterol and general apo B are usually elevated. These measurements should increasingly be used in both risk assessment and therapy targets in people with metabolic syndrome (32). Low HDL is another characteristic of atherogenic dyslipidemia (2). As a predictor of risk, low HDL rivals elevated common apo B (or VLDL-LDL cholesterol). This fact has led to the concept that HDL is closely involved in the atherogenic process. Theories abound regarding the mechanisms by which HDL is anti-atherogenic, such as the expansion of reverse cholesterol transport, anti-inflammatory properties, the ability to protect against LDL modification, among others. Although HDL may actually be directly anti-atherogenic, it is also a marker of other lipid and non-lipid risk factors. Obesity alone reduces HDL (4) levels, and obese patients with metabolic syndrome and atherogenic dyslipidemia almost always have low HDL levels. Thus, the link between low hdL and ASCVD risk is complex (2), and the different components of this association are difficult to differentiate. Regardless of the mechanism, however, the presence of a low HDL carries a strong predictive power for the development of ASCVD. High blood pressure. Obese people have a higher prevalence of high blood pressure than thin people. In addition, higher blood pressure is a strong risk factor for cardiovascular disease (CDC) (33). Known complications of hypertension are ISP, stroke, left ventricular hypertrophy, heart failure and chronic renal failure. However, some reports (34, 35) indicate that elevated blood accompanying obesity is less likely to produce CCC than when it occurs in lean people. It is implied that obesity-induced hypertension is not particularly dangerous for cardiovascular system. This concept is generally not accepted by the hypertension community, and it has not been supported by the Framingham Heart Study (36). Increased plasma glucose. There is no doubt that people with diabetes are at increased risk for ASCVD. In epidemiological studies, the onset of diabetes is accompanied by an increased risk for ASCVD, suggesting that hyperglycemia as such is atherogenic. There is limited evidence directly related to the question of whether hyperglycemia accelerates the development of atherosclerosis. However, one recent study (37) showed that intensive diabetes therapy for type 1 diabetes is accompanied by a decrease in the thickness of the carotid arteries. Although this finding is consistent with epidemiology, it has generally been not possible to demonstrate the atherogenic potential of hyperglycemia in animal models. Also, whether hyperglycemia of type 1 diabetes contributes to atherosclerosis was uncertain. The leading cause of death for people with type 1 diabetes is THE CSE; despite this, it is possible that most atherosclerotic diseases develop later in the course of the disease after the development of chronic renal failure and hypertension. Various mechanisms have been proposed under which hyperglycemia can contribute to the development of atherosclerosis (38). Examples include noncimatic glycosylation of lipids and proteins, pathogenic effects of advanced glycation products, increased oxidative stress, activation of protein kinase C and microvascular diseases of coronary artery vazorum. All these potential mechanisms are of interest, but so far none of them have played a direct role in atherogenesa; most likely, everyone is involved in one way or another. But the fundamental question remains to be answered, namely whether hyperglycemia is directly atherogenic. Another possibility is that insulin resistance per se is independently atherogenic. In prospective studies, insulin resistance is associated with an increased risk of ASCVD (39). But in people with insulin resistance mixed by other known risk factors makes it difficult to be sure that insulin resistance (or as a result of hyperinsulinemia) is directly atherogenic (39). If so, the mechanisms of such an effect are now completely speculative. Prothrombotic state. Obesity is accompanied by a large amount of coagulation and fibrinolitic abnormalities (40). This suggests that obesity causes a prothrombotic state. At present, it is not known how the prothrombotic state will either contribute to the development of atherosclerosis or participate in the development of acute events of the ACCVD. Perhaps the most attractive candidate for increasing atherogenity associated with coagulation and fibrinolitic anomaly is Dysfunction. Many workers believe that endothelial dysfunction is somehow involved in atherogenic atherogenic Several avenues have been suggested; however, so far none of them have been substantiated. Perhaps more likely, obesity-induced procoagulant and antifibrinolytic factors contribute to the deterioration of acute coronary syndromes. Thrombosis occurring with ruptured plaques or erosion is a key element in determining the severity of the syndrome. If normal coagulation and fibrinolism are disturbed during plaque rupture or erosion, a larger blood clot should be formed. An attractive hypothesis is that acute plaque disorder is common, but only when a large thrombosis has a significant acute coronary syndrome. If this is the case, this may make the presence of a prothrombotic condition important for determining a clinical outcome. It's a pro-life state. The cardiovascular field has recently shown great interest in the role of inflammation in the development of ASCVD. The basic concept is that atherosclerosis is a condition of chronic inflammation. It is characterized by a lipid-induced injury that initiates the invasion of macrophages followed by the spread of smooth muscle cells. All these processes are classic features of chronic inflammation, although they occur very slowly. The conclusion that the height of CRP serum carry a predictive force for the development of major cardiovascular events led to the concept that advanced and unstable atherosclerotic plaques are in an even higher state of inflammation than stable plaques (9). Of interest is the fact that obese individuals (42) and especially those with metabolic syndrome (43) also have elevated levels of CRP. This finding showed that obesity is a pro-life condition and is somehow associated with the development of unstable atherosclerotic plaques. So far, however, a mechanistic connection has not been made. The association is suggestive, but how the height of CRP associated with obesity may contribute to or exacerbate major cardiovascular events is not clear. The absence of the identified mechanism does not exclude causality. But so far the connection has not been disclosed. Summary Obesity is a major major risk factor for ASCVD. This is due to several risk factors asSCVD, and is also a risk factor for the development of type 2 diabetes. Diabetes itself is a cardiovascular risk factor. Despite the strong link between obesity and ASCVD, the mechanisms underlying this relationship are not well understood. Our understanding of the link between obesity and vascular disease is complicated by many possibilities. Obesity affects so many metabolic pathways, producing so many potential risk factors that it is virtually impossible to distinguish between the more important and the less important. The possibilities for confusion of variables are enormous. This complexity is a big problem fundamental and clinical research. It also increases the possibility of new targets metabolic syndrome therapy. The main problem, however, is how to intervene at the public health level to reduce the high prevalence of obesity in the general population. This approach offers the greatest opportunity to reduce the cardiovascular risk that accompanies obesity. Abbreviations: atherosclerotic cardiovascular disease; high-density lipoprotein; impaired glucose tolerance; Unsterified fatty acids; Plasminogen activator inhibitor; triglycerides of rich lipoproteins; Links 1, , , Kang RA; American Heart Association; National Heart, Lung and Blood Institute; American Diabetes Association Clinical Metabolic Syndrome Management: Report by the American Heart Association/National Heart, Lung and Blood Institute/American Diabetes Association Conference on Scientific Issues Related to Management. :-2National Cholesterol Education Program (NCEP) Expert Group on the Detection, Evaluation and Treatment of High Blood Cholesterol In Adults (Adult Treatment Group III) Third Report of the National Cholesterol Education Program (NCEP) Expert Group on The Detection, Evaluation and Treatment of High Blood Cholesterol In Adults (Adult Treatment Group III) final report. :-3, ,, Lenfant C; National Heart, Lung and Blood Institute; American Heart Association Definition of Metabolic Syndrome: Report by the National Heart, Lung and Blood Institute/American Heart Association Conference on Scientific Issues Related to Definition. 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