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Editorial

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Obesity: Key Factor for Cardiometabolic Risk

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Obesity is a chronic multifactorial desease whose increasing prevalence is associated with several comorbidities. Diabetes, cardiovascular diseases, musculoskeletal diseases, mental health problems and even certain cancers are more common among obese patients. According to World Health Organization (WHO), obesity is one of the ten responsible for more than a third of deaths, being the second cause of premature avoidable mortality after smoke.

Metabolic syndrome (MS) is a clinical entity characterized by the presence of insulin resistance, compensatory hyperinsulinism, glucose intolerance, hypertension, dyslipidaemia and obesity, which increases cardiovascular risk. The prevalence of this clinical condition has increased in the last decade in the western world, being very common in Europe and in the United States (it may reach around 25% in people over 20-years-old and 50% in people over 60-years-old).[1] Unlike the WHO or European Group for the study of Insulin Resistance (EGIR) group criteria, abdominal obesity/waist circumference (men >102 cm and women >88 cm) gains importance in the Adult Treatment Panel III (ATPIII) which is recognised as similar as other main criteria; not being necessary the presence of insulin resistance, diabetes or hydrocarbon intolerance. These convenience standards make useful the anthropometric and laboratory accessible tests in primary and hospital care. Overall, there are three kinds of risk factors (underlying, major and emerging) in the MS, some of them have proved relation to the obesity.[2] Firstly, obesity (especially visceral or abdominal obesity) is considered one of the main underlying risk factors, along with a lack of physical activity and an atherogenic diet. Secondly, elevated blood pressure is strongly associated with obesity, more frequently with insulin resistance people, as a metabolic risk factor. Another emerging risk factor to consider is the frequent proinflammatory state in the MS, which is clinically detected by an increase of C-reactive protein (CRP) levels. One of the principal cause among the multiple mechanisms underlying of this is the obesity due to the excess adipose tissue releases inflammatory cytokines that can produce high-levels of CRP.

Obesity, along with others adipose tissue disorders, insulin resistance and diverse independent factors; is one of the potential etiopathogenic categories in this MS.[3] "Epidemic obesity" was established by ATPIII as the main responsible for the increasing prevalence of MS. It is proved that obesity contributes to the development of hypertension, elevated cholesterol serum, low HDL-cholesterol serum and hyperglycaemia, thus contributing to an increase of cardiovascular risk. Notably, this 'metabolic obesity' has demonstrated straight relation with several metabolic risk factors. Excessive adipose tissue releases certain products that directly increase these risk factors. These include non-esterified fatty acids, cytokines, plasminogen activator inhibitor-1 (PAI-1) and adiponectin. Elevated plasma levels of non-esterified fatty acids (NEFA) produce a muscular and hepatic lipid overload that increases insulin resistance. Elevated levels of CRP are owned to either proinflammatory status or excess of cytokines. Elevated levels of PAI-1 contribute to the prothrombotic state and low-levels of adiponectin associated to obesity are frequently related to the metabolic risk factors. The connection between obesity, especially abdominal obesity, and metabolic risk factors happens to be so important that adult treatment panel III (ATP III) defined MS mainly as a group of metabolic complications of obesity.[3]

According to the International Diabetes Federation (IDF), MS is centred on visceral

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obesity (defined by a men waist circumference >94 cm and women waist circumference >80 cm) along with others additional factors.[4] The management of insulin resistance patients is complicated by its association with obesity. Insulin resistance is generally elevated as a function of increasing body fat, although a wide range of insulin sensitivity indices can be possible at any body fat percentage.[5] Many individuals with categorical obesity (body mass index (BMI) ≥30 kg/m^2) present postprandial hyperinsulinemia and have relatively low insulin sensitivity.[6] However, there is an insulin sensitivity variation, even among the obese population.[5] Overweight people (BMI between 25 to 29.9 kg/m^2) show a maintained spectrum of insulin sensitivity, so that it is demonstrated an hereditary component into insulin resistance. Besides, in some populations (e.g., South Asian), it is common an insulin resistance even if BMI is <25 kg/m^2, which may contribute to an increase of prevalence type 2 diabetes and premature cardiovascular disease. As a result of it, individuals who manifest insulin resistance and overweight, even though low or moderate grade, have a primary resistance to insulin. Although, primary insulin resistance is present, a weight gain seems to increase an insulin resistance and consequently the development of MS. Therefore, it is difficult to separate obesity and primary resistance to insulin.

Another issue to examine is anthropometric indices, such as waist circumference, which may predict seven cardiovascular risk factors frequently associated to obesity: hypertension, hypertriglyceridemia, LDL-c, non-HDL-c, low-HDL-c, glycaemia basal or their associations.[7] In accordance with recent findings in the Framingham study, the measurement of visceral and subcutaneous abdominal fat, either by volumetric method or by fatty area in tomographic cut at different levels (particularly at L3-L4), is directly related to the next metabolic risk factors: fasting blood glucose, triglycerides, HDL-c and systolic blood pressure.[8] Accordingly, there has been a requirement to find biological markers in the daily clinical practice to associate anthropometric and metabolic alterations with the prevalent syndrome metabolic in obese patients. Peculiarly, the combination of increased waist circumference and high triglyceride concentration may determinate a subgroup with high risk of developing MS. In particular, it has been proved that the well-known 'hypertriglyceridemia waist' may be an accurate marker not only of obesity visceral but also of others metabolic disorders of the MS.[9] According to hypertriglyceridemia waist researches in different populations, this phenotypic alteration is strongly linked to the risk of diabetes, probably as a consequence of its association with obesity.[10,11]

Apart from the increased morbidity and mortality associated with overweight and obesity, there is evidence in the increased cardiovascular risk of metabolic origin associated to a visceral adiposity. In fact, visceral adipose tissue correlates strikingly with proatherogenic, prodiabetrogenic, prothrombogenic, and proinflammatory phenomena. Although, waist circumference has demonstrated to be a useful marker for assessing obesity beyond the BMI, abdominal fat accumulation may be the result of an increase in visceral or subcutaneous adipose tissue.[12] Nevertheless, the accumulation of visceral fat is directly associated with an increased risk of coronary heart disease or type 2 diabetes.[13,14] In addition, aside from the isolated increase in the waist circumference as a clinical data may be only an anthropometric marker, the accompanying metabolic consequences to this alteration can be detected by other biochemical markers which express the inaccurate management of fat storage in the adipose tissue.[15] In Spain, a study has recently been published which demonstrates that waist circumference, along with other markers, is a practical predictor of metabolic disorders in overweight-obesity phenotype patients.[16] Furthermore, the measurement of waist circumference could be used, not only as a surrogate marker of accumulation of visceral adipose tissue, but also of insulinemia.[17]

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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