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Relation between obesity and osteoarthritis

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Report Submitted to fulfill the requirements for Scientific Research Activity

Date of Submission:12/3/ 2020

Abstract

Obesity is currently considered a major public health issue in the world, already reaching epidemic characteristics. Excess weight is the major risk factor associated with various diseases, including osteoporosis osteoarthritis, type 2 diabetes mellitus, hypertension, dyslipidemia, and osteometabolic diseases. Osteoarthritis is the most severe rheumatic condition and the leading cause of the over 65-year-old population's physical disability and reduced quality of life. This includes mostly the weight-bearing joints-knees and hips. Nevertheless, its prevalence is growing along with the cases of obesity, as well as in other joints, such as hands. The effect of obesity on the production of (OA) is therefore believed to be beyond mechanical overload. The purpose of this report is to correlate the possible mechanisms underlying the genesis and development of these two diseases. Increased fat mass is directly proportional to excessive consumption of saturated fatty acids, resulting in systemic low-grade inflammatory condition and resistance to insulin and leptin. Leptin assumes inflammatory properties at high levels and functions in the articular cartilage, activating the inflammatory process, and altering homeostasis with consequent degeneration of this tissue. It had been concluded that obesity is a risk factor for osteoarthritis and that physical activity and dietary changes can reverse inflammatory and leptin resistance, decrease progression or prevent osteoarthritis from occurring.

Introduction

Obesity has already reached epidemic proportions, affecting more than one billion adults worldwide, according to the World Health Organization (WHO). It is the major risk factor associated with various diseases, such as resistance to insulin and type 2 diabetes mellitus (DM2), hypertension, dyslipidemia, and certain cancers. Osteoarthritis (OA) is the most prevalent osteometabolic rheumatic disease and the leading cause of physical incapacity and reduced quality of life for the over 65-year-old population. It is characterized by degradation of the articular cartilage.The relation between obesity and the development of OA was restricted to biomechanical changes in joints, caused by increased body weight, leading to the genesis of an inflammatory process in cartilages, and eventually in the development and progression of the condition. These changes preferably occur in joints with body weight bear such as knees and hip. However, in addition to the increased prevalence of obesity in the

world population, there has also been an increase in cases of OA in non-weight bearing joints such as hand and temporomandibular (TMJ) joints. In this way, the influence of obesity in OA development is thought to go beyond joint overload due to the increase body mass index (BMI).[1]

The aim of this report is to review the pathophysiology, the relation, and treatment of both of the two diseases.

Methods

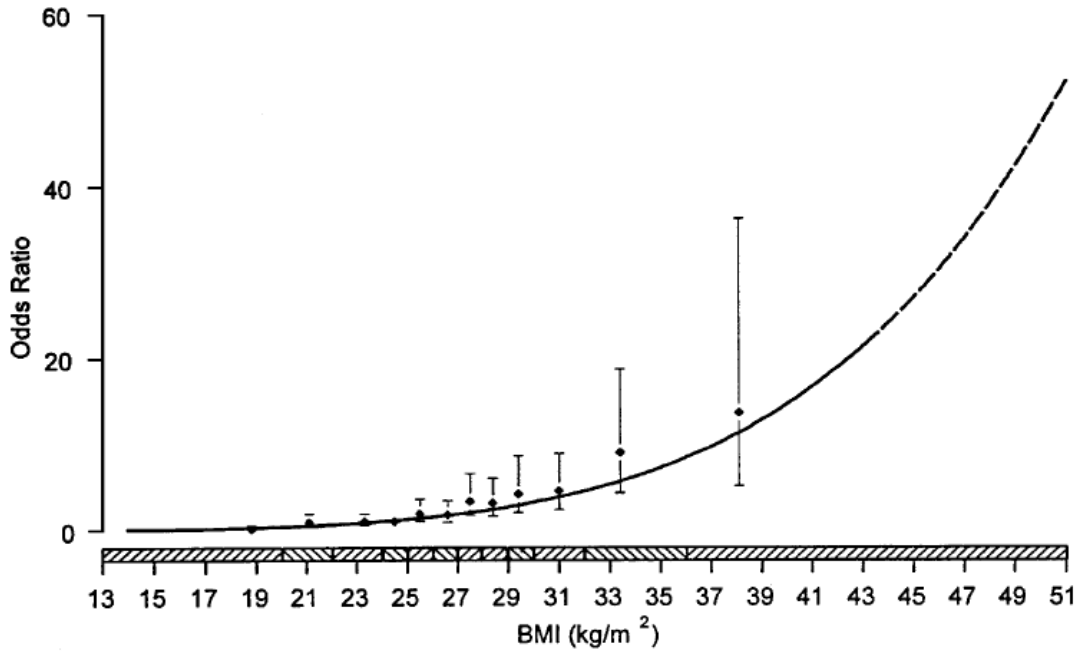
It has performed a population-based case control study in three health districts of England. A general of 525 women and men aged 45 y and over, consecutively indexed for surgical treatment of primary knee OA, were as compared with 525 controls matched via age, sex and own family practitioner. Each patient's case notes and radiographs had been reviewed to verify the analysis of osteoarthritis and to exclude those with underlying rhumatoid arthritis, ankylosing spondylitis, or injury to the knee inside the beyond 12 months. The radiographic severity of osteoarthritis inside the knee requiring surgical procedure become graded in line with the Kellgren and Lawrence classification. The height (using a portable stadiometer) and weight (using electronic scales). examined their hands for the presence of Heberden's nodes (as a marker for osteoarthritis at a different site).[2]

Results

Tabel 1 Proportions of knee osteoarthritis cases that might be avoided

by reducing obesity**[2]**

Reduction in obesity	*Proportion of cases (95% CI) that might be eliminated*
All overweight and obese reduce weight by 2 kg or until their BMI is in the recommended normal range, whichever is less weight loss	10.9% (8.8–12.8%)
All overweight and obese reduce weight by 5 kg or until their BMI is in the recommended normal range, whichever is less weight loss	23.6% (19.4–27.4%)
All overweight and obese reduce weight until their BMI is in the recommended normal range	57.1% (46.7–64.2%)



Figuer 1 Odds ratios and 95% confidence intervals for 12 categories of BMI. The boundaries of these categories are indicated by the shading on the horizontal axis, and the odds ratios are depicted at points corresponding to the median BMI in each category. Also shown is the fitted curve,[(2)]

Tabel 2 Prevalence of obesity and association with knee osteoarthritis [(2)]

BMI (kg/m^2)	Cases		Controls		
	n	%	n	%	OR (95% CI)
< 20.0	5	1.0	42	8.0	0.1 (0.0–0.5)
20.0–24.9	96	18.3	201	38.3	1
25.0–29.9	247	47.1	221	42.1	2.5 (1.8–3.6)
≥ 30.0	177	33.7	61	11.6	6.8 (4.4–10.5)

Odds ratios derived from matched, unadjusted analysis.

Discussion

To try to understand the mechanisms involved in the relationship between obesity and OA pathogenesis. In obesity, fat hypertrophy increases the expression, and release of so-called adipocytokines such as interleukin (IL) 1β,IL-6, leptin, and tumor necrosis factor alpha (TNF-α), among other functions necessary for cell differentiation and hematopoiesis. These proteins reach the bloodstream and then the hypothalamus in the central nervous system, interfering in the fine regulation it exerts, by controlling hunger and energy expenditure. This disturbance makes it mainly resistant to peripheral hormones insulin and leptin. In this way, a person will have excessive satiety, increase food intake, and eventually increase in weight and fat, which in effect releases more leptin, reaching the level of hyperleptinemia. at high concentrations, leptin becomes inflammatory.

Leptin, through its receptor ObR and the interleukin 6 pathway, via its receptor GP-130, activates the transcription factor STAT3, which within the nucleus, transcribes the gene of SOCS3 that suppresses the leptin signaling pathways. However, it also activates the transcription of metalloproteinases and aggrecanases, cartilage degradation proteins. Signaling pathways of inflammatory cytokines: tumor necrosis factor alpha through the tumor necrosis factor receptor, interleukin 1 β by its interleukin receptor 1 β, and the saturated fatty acids, that are recognized by the Toll-like 4 receptors. These pathways culminate in activation of the transcription factors NF-k β and AP-1, that transcribe the genes of inflammatory proteins (interleukin 1 β , interleukin 6, tumor necrosis factor alpha, induced nitric oxide synthase among others). There is a relation between obesity and systemic

inflammatory status that, in turn,provides increased expression of MMPs and aggrecanases to articular chondrocytes, resulting in reduced expression of collagen and proteoglycans. Thus, there is a homeostatic imbalance of cartilage, favoring the initial osteoarthritic inflammation. And the composition of the fat rich diet, regardless of increase in weight and consequent mechanical overload on joints, predisposes OA to emerge.

Some studies showed that exercising may impair the cartilage degradation process due to the increase in mechanical overload, but, on the other hand, evidence has shown that there are no correlations between exercise and OA, or even that exercise is effective for its treatment. OA does not have cure perspectives yet, and modern studies using stem-cells for replacing cartilage tissue are, for now, distant from being plausible. The treatment has been based on pain control, and on the dysfunction and control of the speed of the cartilage destruction process mainly by taking medication and by exercising.

Hypothalamic sensitivity to leptin also improves with exercising. This is because skeletal muscle releases IL-6 during physical activity and, at the central level, IL-6 regulates energy expenditure, appetite and body composition, decreasing inflammation and increasing sensitivity to leptin.

Non-saturated fatty acids, mainly omega 3 and omega 9 have been proven to have anti-inflammatory effects. [(1)]

Conculation

Obesity is a risk factor for osteoarthritis and the growth in fat mass is directly proportional to exaggerated consumption of nutrients, especially saturated fatty acids, responsible for low grade inflammation and central resistance to insulin and to leptin. At high levels, leptin becomes inflammatory and can trigger an inflammatory process in articular cartilage, changing the homeostasis of this tissue. Exercising and changing diet composition, such as replacing fat by non-saturated fatty acids, can revert the inflammatory process and resistance to leptin, reduce the speed of progression or preventing the development of osteoarthritis.

Refrenses

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(2) Zhou, Z., Liu, Y., Chen, H., & Liu, F. (2014). Body mass index and knee osteoarthritis risk: A dose-response meta-analysis. *Obesity*, *22*(10), 2180-2185. doi: 10.1002/oby.20835