OBESITY ETIOPATHOGENIC PARTICULARITIES

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Abstract: Obesity is the consequence of energy storage, as result of a disequilibrium between the energy intake and energy expenditure, which is developed gradually: overweight, moderate, severe and morbid obesity. The etiology of obesity is complex, causality factors are genetic and acquired: overeating, sedentary life, psychological and social factors, drugs and endocrine disorders.

Keywords: obesity, etiology, pathogenesis

Rezumat: Obezitatea este consecința unui cumul de energie, rezultat în urma unui dezechilibru dintre aportul și consumul de energie, ce are loc în etape care includ: supraponderalitatea, obezitatea moderată, severă și morbidă. Etiologia obezității este complexă: factorii cauzali sunt genetici și câștigați: supraalimentația, sedentarismul, factorii psihologici și sociali, anumite medicamente, afecțiuni endocrine asociate.

Cuvinte cheie: obezitate, etiologie, patogenie

INTRODUCTION

Obesity is characterised by the excessive accumulation of the adipose weight, which takes place in stages that include overweight, obesity (moderate, severe or morbid). Weight excess, through the included comorbidities, is placed among the first signs of a disease, invalidity and premature morbidity. The most used indicators in the assessment of obesity is the body mass index (BMI), expressed by the relation between the weight in kg and the height in square metres.

According to the BMI values, weight may be classified as follows:

- Normal weight (BMI 18,5-24,9);
- Overweight (BMI 25-29,9);
- I degree obesity (BMI 30-34,9);
- II degree obesity (BMI 35-39,9);
- Morbid obesity (BMI>40).

A body mass index higher than 28 is associated to an increased risk of comorbidities, such as the cerebral vascular accident, ischemic heart diseases or diabetes mellitus; it was proved that there is 3-4 times higher risk as against the general population. Obesity etiology is complex; the causal factors of obesity are genetic or acquired and most of the times, they are associated. Studies made on twins, adopted children, members of certain families, showed the role of the genetic factors in obesity occurrence. It was proved that 80% of the BMI variations are attributed to the genetic factors. It is appreciated that up to 30-40% of the factors, such as: distribution of the adipose tissue, physical activity, basal metabolism, alterations of energy consumption, certain aspects of food behaviour, food preferences, activity of the lipoprotein lipases, maximal synthesis of the glycerides stimulated by insulin and basal lipolysis are inherited. The inheritance of obesity with early debut is much higher than the obesity that starts at an adult age.

A recent study identified frequent mutations at the level of the FTO gene; heterozygotes registered 30% higher risks of obesity, while homozygotes registered a risk of 70%. Besides certain genetic syndromes, where obesity is present (Prader Willi syndrome, Bardet Biedel syndrome, Cohen syndrome), which are associated to other dysmorphic characteristics, obesity is probably determined in most of the cases, by the alterations of the interactions between the genetic factors and the environmental ones.

The main reason for the increase of obesity frequency in population is represented by the combination of an excessive intake of nutrients and a sedentary lifestyle, associated to the psychological and social factors.

Certain physical and psychical affections, such as drugs (steroids, certain antipsychotics) may lead to obesity.

The affections that raise the risk for obesity include some of the above-mentioned genetic symptoms, plus hypothyroidism, Cushing's syndrome, growth hormone deficit, polycystic ovary syndrome.

There is the question whether the endocrine changes occurred in obesity take place secondarily to obesity or, at least one of them is a factor that contributes to the maintenance and development of obesity.

Obesity is a frequent characteristic of the polycystic ovary syndrome (PCOS); approximately 50% of the women with PCOS are obese. PCOS women are also insulin resistant and present an increased risk for developing glucose and diabetes mellitus tolerance alteration. In hypothyroidism, obesity is usually moderate. There is also the possibility that, obesity of another etiology might coexist with hypothyroidism. In

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hypothyroidism, the increase of the body mass is mainly due to water and mucopolysaccharides retention.

In Cushing's syndrome, obesity is progressively installed; it is moderate and has facial, trunk and abdominal predominance.

Pathogenically, obesity is the consequence of energy storage, as a result of a disequilibrium between the energy intake and energy expenditure.

Regarding the regulation of the energetic equilibrium, a lot of factors intervene: insulin, cholecystokinin, other endocrine signs and peptides (glucocorticoids and glucagon that exercise their effects on the energetic intake, androgens, thyroid hormones and the growth hormone, which intervene in the energetic consumption), other intestinal peptides, such as gastrin releasing peptide, neuromedin B, enterostatin, amyline, leptin, leptin receptor, ghrelin, neuropeptide Y.

Leptin and ghrelin are considered to have complementary effects on the appetite; ghrelin modulates the control of the appetite on short term, leptin is produced by the adipose tissue in order to signal the reserves stored in the organism and mediates the control of the long term appetite.

Obesity is characterised through an increased level of the plasmatic insulin and resistance to insulin. Due to the fact that the serum level of leptin is increased in the obese patients in comparison with the normoweight patients, leptin resistance hypothesis was issued.

PURPOSE OF THE STUDY

Emphasizing the factors involved in obesity etiopathogeny.

MATERIAL AND METHOD

The factors involved in obesity etiopathogeny were studied on a group of patients selected aleatorily, out of those who come periodically to check ups. The subjects were the patients of C.I. Parhon Endocrinology Institute and suffered from obesity, hypothyroidism, PCOS, Cushing syndrome.

The analysis was made on a total number of 96 patients, aged between 20 and 69. Out of the total number, 88% were women and 12% were men.

The investigated parameters were: the anthropometric indices, obesity heredo-collateral incidents, data regarding their lifestyle (food behaviour, alcohol consumption, physical exercises), the moment of obesity debut.

The assessment was based on the clinical examination of the hormonal proofs (through FIA, RIA methods, chemiluminescence), computerized tomography, MRIEndocrine pathology consisted in hypothyroidism 30%, Cushing syndrome 40%, PCOS 30%.

RESULTS AND DISCUSSIONS

Taking into account the entire group and based on the patients' statements, the investigated parameters may be structured in the following way: 28% of the patients registered obesity heredo-collateral antecedents. The obesity occurred during childhood in 18% of the patients, after pregnancy in 12% of the patients, during post-menopause -2%, at the same time with the endocrine affection - 40\%, while 28% of the patients could not mention the moment when obesity occurred.

According to the BMI value, the classification of patients was made as follows: overweight (52 %), obesity (48 %), morbid obesity (BMI > 40 kg / m 2) 4%.

The physical activity they have declared in comparison with the other members of their families was reduced in 44% of the patients, the same as of the other members of the family in 48% of the patients and higher in 8%.

By analysing the lifestyle from the food customs point of view, the food survey evidenced the following characteristics: 30% of the patients mentioned 3 daily meals, 40% said that they did not use to take breakfast and 2% mentioned more than 3 meals per day.

From the point of view of food preferences, the food survey showed a moderate consumption of fat meat in 7% of the patients, sausages in 60%, cheese in 16%, concentrated sweets in 25%. Regarding the alcohol consumption, 2% of the patients confirmed the daily alcohol consumption, 5% reported a weekly consumption of alcohol and the rest of the patients – occasionally. An increased consumption of food associated to stress was mentioned in 20% of the patients. The assessment of the lifestyle showed the majority's preference for an unhealthy food, rich in hypercaloric foods (fats, meat, sweets) and no regular meals.

Family history of the investigated patients showed a quite increase prevalence of obesity (28%) in I degree relatives.

CONCLUSIONS

Obesity represents a major problem of public health, determined in most of the cases, by the association of the genetic component of the acquired factors, endocrine pathology and an unhealthy lifestyle.

Genetic predisposition, familial characteristics of lifestyle and the risk factors acquired through the development of an endocrine and metabolic pathology draw the attention upon this problem, which must be integrated in a complex therapeutic education programme.

Together with the clinical management of the endocrine pathology, the therapeutic education will be submitted to this one, and on long term, the control of the endocrine and metabolic-nutritional diseases may be accomplished through the collaboration between the primary, secondary and third medical assistance, for the improvement of the life quality and for the increase of life expectancy.

BIBLIOGRAPHY

- Anthony N Fabricatore PhD, Thomas A Wadden. Treatment of obesity: An Overview, Clinical Diabetes 21:67 – 72, 2003.
- John Orzano, MD, MPH, John G Scott, MD. Diagnosis and Treatment of Obesity in Adults: An Applied Evidence – Based Review, The Journal of

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the American Board of Family Practice, 2004, 17:359 – 369.

- 3. Beck B, Burlet A, Nicholas JP, Burlet C. Hypothalamic neuropeptide Y (NPY) in obese Zucker rats: implications in feeding an sexual behavior, Phisiol Behav, 190, 47, 449-453.
- Beunen GP, Malina RM, Lefefre JA, Claessens Al, Reuson R, Venreusel B. Adiposity an biological maturity in girls 6 – 16 years of age. International Jurnal of Obesity, 1994, 18, 542 – 546.
- 5. Bray GA. The Syndrome of Obesity: An Endocrine Approach, in: DeGroot LJ (Ed): Endocrinology, 3rd Edition, Saunders, Philadelphia, 1995, 2624 2662.
- Bray GA. Obesity, in: Greenspan and Strewler GJ (Eds), Basical and clinical Endocrinology, 5th Edition, Prentince – Hall, London, 710 – 723.
- Bray GA. York DA. Leptin and clinical medicine: a new piece in the puzzle of obesity, J Clin Endocrinol Metab, 1997, 82 (9), 2771 – 2776.
- Bray GA. Obesity, Harrison's Textbook of Internal Medicine, 14th Ed., Mc.Graw Hill Book Co., 1998, 454 – 462.
- Brown A, Siahpush M. Centre for Behavioural Research in Cancer. Carlton. Australia Risk factors for overweight and obesity: result from the 2001, National Health Survey, Public Health, 12 Jun 2007.
- Considine RV, Sinha MK, Heiman ML, Kriauciunas A, Stephens TW, Nyce MR & others. Serum immunoreactive – leptin concentrations in normal weight and obese humans, N Engl J Med, 1998, 454 – 462.
- Couce ME, Bruguera B, Parisi JE, Jensen MD, Lloyd RV. Localization of leptin receptor in human brain, Neuroendocrinology, 1997, 66(3), 145 – 150.
- 12. Duncea I, Blendea MC. Leptina hormonul obezității? Sibiul Medical, VIII, 4, 1997, 205 206.
- Gerald M, Reaven. MD. Importance of Identifying the Overweight Patient Who Will Benefit the Most by Losing Weight, Annals of Internal Medicine, 4 mart 2003, vol. 132 fascicul 5, pg. 420 – 423.
- 14. Hainer V, Kunesova M. Thyroid and Obesity –The Thyroid Gland, 2 / 1997, 35 40.
- Hamann A, Matthaei S. Regulation of energy balance by leptin, Exp a Clin Endocrinol Diab, 1996, 104, 293 – 300.
- Hancu N, Danciu A. Abordarea practică a obezității, Ghid de practică medicală şi farmaceutică, Vol.1, Nr.1, iulie 1995, 1 – 8.
- Havel PJ, Kasim Karakas S, Mueller W, Johnson PR, Gingerich R, Stern JS. Relationship of plasma leptin to plasma insulin and adiposity in normal weight and overweight women: effects of dietary fat content and sustained weight loss. Journal of Clinical Endocrinology and Metabolism; 1996, 81(12), 4406 4413.
- Horton ES, Jeanrenaud B. Obesity and diabetes mellitus in: Rifkin H. And Porte D. (Eds), Diabetes Mellitus, 4th Edition, Elsevier, New York; 1990, 457 - 463.

- Hector F Escobar. Morreale Jose I, Botella Carretero, Francisco Alvarez Blasco, Jose Sancho and Jose L. San Milan. Departments of Endocrinology and Molecular Genetics, Hospital Ramon y Cajal, Madrid E – 28034, Spain. The Polychistic Ovary Syndrome associated with morbid obesity may resolve after weight loss induced by bariatric surgery. The Journal of Clinical Endocrinology & Metabolism; 2005, vol. 90, nr. 12 pg. 6364 – 6369.
- 20. Kopelman PG, Hormones and Metabolism in: Caterson ID (Ed): Baillere's Clinical Endocrinology and Metabolism, Vol. 8, No.3, July 1994, 549 – 575.
- Michael Rosenbaum, MD; Rudolph L. Leibel, MD; Jules Hirsch Obesity, The New England Journal of Medicine, 7 aug.1997, nr.6, vol 337:396 – 407.
- 22. Ogden CL, Yanovski SZ, Carroll MD, Flegal KM The epidemiology of obesity, PubMed, Gastroenetrology, May 2007, 132(6):2087 – 102.
- Portmann L, Giusti V Obesity and hypothyroidism: myth or reality? Rev Med Suisse, 4 Apr 2007, 3(105):859 – 62.
- Susan Z. Yanovski, MD; Jack A. Yanovski Obesity, The New England Journal of Medicine, 21 Feb 2002, nr.8, vol. 346:591 – 60.
- 25. Susan B, Racette, PhD, Susan S Deusinger PhD, Robert H Deusinger PT, PhD Obesity. Overview of Prevalence, Etiology and Treatment, Physical Therapy, vol.83, nr. 3, Mar. 2003, pg 276 – 288.
- 26. Thomas A Wadden, PHD, Robert I. Berkowitz, MD; Leslie G Womble, PhD; David Sarwer, PhD, Suzanne Phelan, PhD, Robert K Cato, MD; Louise A. Hensson, MSN; Suzette Y Osei, MD, PhD, Rosalind Kaplan, MD, Albert J Stunkard, MD Randomized Trial of Lifestyle Modification an Pharmacotherapy for Obesity, The New England Journal of Medicine, 17 Nov 2005, nr. 20, vol. 353:2111 – 2120.
- 27. Timoty John Aitman Genetic Medicine and Obesity, The New England Journal of Medicine, 22 May 2003, nr. 21, vol. 348:2138 – 2139.
- 28. Thorkild IA, Sorensen, Soren M Echwald, Jens Christian Holm Leptin in obesity, British Medical Journal, London, 10.0ct.1996, 953 – 954.
- 29. Yasser Ousman MD, Keneth D Burman, MD Chapter 12 - Endocrine function in obesity, Endotext.com, 21 Aug. 2002.
- 30. Wilson Williams Textbook of Endocrinology, 9th edition, 1998, Obesity, 1072 1081.