CLINICAL CASE 1

CLINICAL HISTORY

This clinical case involves a 50-year old woman, who started weight gain at 20 years old. Dieting since 21 years old, with slight weight loss and rapid regaing of lost weight.

During her first pregnancy she gain 30 Kg (95 kg) and 20 Kg during the second pregancy. The maximum weight reached was 121 Kg (in year 2006).

In 1994 she suffered two heart attacks within 6 months time, requiring revascularization and application of 3 Stent. She presented HTA and dislipidemia and starting DM2. Since then, she is in permanent leave and follows cardiac rehabilitation. Medication: metformine, plavix, atenolol, enalapril and zarator.

Additionally she presented undiagnosed and untreated OSA (Obstructive sleep apnea syndrome), as well as multiple joint pain.

1st consultation (Nov 2008): she consulted a specialist because she couldn't loose weight by combining a 1,250 kcal/day low-calory diet (leaving her feeling hungry) and exercise (2 hours walk/day).

Weight: 108,7 kg

Height: 169 cm

BMI (Body Mass Index): 38,4

Impedanciometry: Fat: 50,7 (55,7 Kg); FFM (fat-free mass): 54,1 Kg; water 39,6 Kg.

Central obesity: waist perimeter: 137 cm.

GENETIC STUDY

Performed by DNA extraction from oral mucosa sample on swab, followed by DNA amplification by PCR and DNA analysis with molecular biology techniques. 17 genes were studied. Genetic results are presented in table 1.

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GENE	THRIFTY POLYMORPHISM	ASSOCIATED WITH:
LEP	Negative	No
MC4R	Negative	No
POMC	Negative	No
FTO	Heterozygous	Reduced satiety
UCP1	Homozygous	Decreased thermogenesis
UCP3	Heterozygous	Decreased thermogenesis
ADRB2	Negative	No
ADRB3	Heterozygous	Adrenergic stimulation of adipocytes. Metabolic syndrome
PPARG	Heterozygous	Insulin resistance
IL-1B	Negative	No
IL-1RN	Negative	No
TNFa	Heterozygous	Insulin resistance. Chronic inflammation
ADIPOQ	Negative	No
FABP2	Homozygous	Increased absorption and oxidation of fatty acids.
ACE	Heterozygous	Transformation of pre-adipocytes into adipocytes. Control blood pressure
GNB3	Homozygous	Progression of Arterial Hypertension
APOA5	Heterozygous	Hypertriglyceridemia

Table 1. Patient's genetic test results

RESULTS

Result: a very high genetic load

Based on the results of the genetic study, personalized diet and pharmacological treatment were established, according to the guidelines listed below:

1. Respect the circadian rhythm: calories and protein intake should decrease throughout the day (more in the morning and less at the end of the day).
2. Favor satiety, eating a high-protein breakfast and following a very fractionated diet (in order to increase the thermogenic effect of food).
3. Take thyroid hormone, due to peripheral resistance to this hormone.
4. Avoid chronic stress. Associate anxiolytic treatment.

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5. Anti-inflammatory diet. Associate ASA (acetylsalicylic acid).
6. Replace Metformin by Pioglitazone (insulin resistance by PPARG transcription factor).
7. Follow low-fat diet associated with supplementation of omega 3 and 6, soluble fiber and fibrates.
8. Follow a strict low-sodium diet.

EVOLUTION

After 2 years (November 2010) the patient had lost 31,4 kg (of which 80% was fat mass).

Last consultation (November 2012): patient maintains lost weight and has not presented any complication attributable to diet or associated medication. SAOS symptoms and osteoarticular pain have disappeared. Her biochemistry is within normal limits, including thyroid hormones.

CONCLUSION

This case exemplifies how the molecular study of safety genes allow adapting medical treatment for a better control of both obesity and its comorbidities.

In addition, it is a predictive study because it allows selecting population at risk and applying preventive measures in the group at risk, increasing efficiency and reducing costs.

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