

# The Process of Pharmacotherapy for Obese Patients

Alice Mc-Wane<sup>+</sup> and Timothy Wilson

### ABSTRACT

Obesity is a serious health issue with numerous comorbidities. Its incidence has risen drastically in recent decades, to the point where it is now considered an international epidemic. Obesity has been linked in large epidemiologic studies to cardiovascular disease and adverse effects in practically every organ system, implying that therapy is required. Several short- and long-term anti-obesity medications have been studied, but there has yet to be a "wonder pill" that can permanently cure or control obesity, despite the fact that a number of them have considerable medical benefits. Orlistat, sibutramine, and rimonabant are the most commonly prescribed anti-obesity medications in clinical practise, but newer, more promising options will be available in the coming years.

Keywords: Obesity; Comorbidity; Epidemiology

### Introduction

Obesity, formerly considered a symbol of power and money, is now the most frequent dietary issue and serious health condition, with numerous comorbidities. Its incidence has risen drastically over the last several decades over the world, to the point where it is no exaggeration to call it an international epidemic. It is estimated that around 315 million persons globally fit the WHO obesity criterion. Obesity is defined as a "disorder of body composition in which there is an abnormal, absolute or relative amount of body fat in relation to lean body mass, to the extent that health is affected," according to the World Health Organization. One of the primary causes of the rapid rise in obesity prevalence appears to be an increase in sedentary lifestyle.

Comfort eating, which is high in saturated fats and sweets, leads to an increase in average energy intake per person, which, when combined with reduced physical activity, leads to weight gain. Obesity is thought to be predisposed to by a person's genetic background due to the metabolic and endocrine problems it causes. However, because the obesity pandemic has spread too quickly to be explained solely by genetic alterations, it is obvious that environmental influences, particularly fat- and sugar-rich diets and decreased physical activity, are to blame for the current epidemic.

#### **Classification Obesity**

Obesity is recognised as a major risk factor for Cardiovascular Disease (CVD) in large epidemiologic studies, and it is also directly linked to increased long-term comorbidity and mortality from all causes. Obesity-related disorders include hypertension, hyperlipidemia, obstructive sleep apnea, and Type 2 diabetes, among others. Cancers such as breast, colon, prostate, and endometrial cancer are also more likely in obese people. According to the National Health and Nutrition Examination Survey (NHANES) III, morbidity and mortality rates are closely related to the degree of obesity, making weight status classification critical, as it allows health practitioners to stratify individuals' health risk and adjust intervention levels accordingly. In clinical practise, BMI and waist circumference are used to determine weight status, and the presence of obesity-related illnesses, such as hypertension and dyslipidemia, increases the **Received:** 3-Feb-2022, Manuscript No. M-54167;

**Editor assigned:** 5-Feb-2022, PreQC No. P-54167;

**Reviewed:** 17-Feb -2022, QC No. Q-54167;

Revised: 19-Feb-2022, Manuscript No. R-54167;

Published: 27-Feb-2022, DOI: 10.37532/1753-0431.2022.16(2).227

Editorial Office, International Journal of Clinical Skills, London, United Kingdom

<sup>&</sup>lt;sup>†</sup>Author for correspondence: Alice Mc-Wane, Editorial Office, International Journal of Clinical Skills, London, United Kingdom, Email: ijclinicalskill@journalres.com

total mortality risk [1]. BMI is calculated by multiplying the body weight (kg) by the square of the height (m). It is thought to be a fairly reliable indicator of total body fat mass. Individuals with a BMI of 18.5 kg/m<sup>2</sup> to 24.9 kg/m<sup>2</sup> are considered normal weight, whereas those with a BMI of 25 kg/m<sup>2</sup>-29.9 kg/m<sup>2</sup> are categorised as overweight. Obese Class I patients have a BMI of 30 kilogrammes per square metre-34.9 kilogrammes per square metre, while obese Class II patients have a BMI of 35 kilogrammes per square metre to 39.9 kilogrammes per square metre. Those who have a BMI of 40 kg/m<sup>2</sup> or higher are categorised as extremely obese Class III. The waist circumference is a measurement used in clinical practise to assess central obesity. Of course, waist circumference measures all adipose tissue (and everything else) located in the centre of the body, and ethnic and age-related adjustments may be required when interpreting its results; however, in clinical practise, it is widely regarded as a simple, accurate, and useful measurement to describe visceral adipose tissue accumulation. The distinction between visceral and peripheral fat is critical since it is regarded an independent risk factor for the development of cardiovascular and metabolic problems [1]. Most studies suggest that people with waist circumferences greater than 94 cm in men and 80 cm in women are more likely to develop obesity-related diseases, while more sophisticated cardiovascular risk charts, such as those from the Framingham study, can help identify people with high cardiovascular risk even if their BMI isn't yet significantly abnormal [2].

### **Clinical Management**

While in the old, HHCY is basically brought about by lack of vitamin B, it isn't notable whether these nutrients assume a huge part in bone wellbeing. Considering the instruments, past examinations proposed a decrease in osteoblast movement in relationship with low vitamin B12 focuses [3,4]. Goerss et al. seen that in patients with malicious weakness (brought about by lack of vitamin B12), the dangers of proximal femur, vertebral and lower arm cracks were 1.9, 1.8 and 2.8 times more than controls, individually [5]. In one planned preliminary on 600 patients with osteopenia and osteoporosis, the significant job of B nutrients in bone wellbeing was examined. Sato et al. treated the

patients with 5 mg of folic corrosive and 1500 µg of vitamin B12 or fake treatment for quite some time. They noticed an around 75% abatement in the frequency of cracks in the treatment bunch, which was practically identical with that of alendronate. Considering the different folate fixations in various compartments of the body, Golbahar et al. recommended RBC folate as a preferred indicator of BMD over plasma folate, for which lack might be related with the pathogenesis of osteoporosis in postmenopausal ladies [6]. Roughly 1 year after the fact, Gjesdal et al. played out one more review on 5338 older patients to look at the relationship between hip BMD and plasma levels of HCY, folate, vitamin B12 and the Methylenetetrahydrofolate Reductase (MTHFR) polymorphism. They presumed that raised HCY and low folate levels were related with decreased BMD in ladies yet not in men. In another new review, Green et al. researched 276 solid more seasoned subjects who were haphazardly appointed to get either day by day supplement of folate, vitamin B12 and vitamin B6 or fake treatment for a long time. By estimating bonespecific soluble phosphatase and bone-inferred collagen parts at gauge and the finish of study, they presumed that supplementation with folate and vitamin B6 and B12 can bring down plasma HCY however has no impact on bone turnover. Concerning the meaning of folic corrosive, many gatherings concentrated on the impact of the C677T MTHFR polymorphism on bone. An increment of break occurrence was recognized with each Tallele, particularly in patients with low folate levels. There are a few problematic outcomes in this issue. Li et al. revealed no relationship between MTHFR (C677T) and the BMD of Chinese men or ladies. They played out the future science bunch study on postmenopausal ladies, old ladies and older men. High folate and vitamin B admission in the review populace, added to the low number of patients and low pervasiveness of the TT genotype, ought to be considered. Abrahamsen et al. affirmed that in the most minimal quartile of riboflavin, B12, B6 and folate admission, BMD in MTHFR TT genotype is just altogether diminished, basically at the hour of menopause, and vitamin B supplementation would simply be expected to influence BMD in around 2% of the populace, for example, those with the TT genotype and low vitamin B consumption. They likewise noticed critical skeletal impacts of

# **Mini Review**

this normal polymorphism at lumbar spine in men at 25 years old years [7]. Hong et al. gotten comparable outcomes. They included 1899 Chinese postmenopausal ladies to confirm the relationship of the MTHFR polymorphism with BMD and cracks. They showed that the MTHFR C677T polymorphism is an autonomous indicator of break hazard, despite the fact that it just weakly affected BMD. Other than higher break hazard, low flowing degrees of vitamin B12 and folic corrosive are additionally connected with low BMD, which is in concurrence with the connection among HCY and BMD. Baines et al. concentrated on the connection between plasma HCY, its determinant folate, vitamin B12, vitamin B6, MTHFR genotype and BMD in 328 postmenopausal ladies. As indicated by the standard BMD, the subjects were doled out to three gatherings of control, osteopenic and osteoporotic. The osteoporotic patients showed an altogether lower serum folate and a higher frequency of late crack. All in all, they observed that low serum folate is a significant gamble factor for osteoporosis, with HCY level having a lesser significance. The two nutrients B12 and B6, by influencing HCY, may likewise affect the skeleton, albeit a more fragile one than folate

### **Pharmacotherapy of Obesity**

The idea of treating obesity is simple: create a negative energy balance. However, the reality is rather different. Because of a powerful internal biological system based on survival that tends to maintain and restore fuel stores and return body weight to baseline, any weight loss triggers a series of neuroendocrine effects that resist further weight loss, resulting in the failure of most obesity treatments relying solely on lifestyle changes. This is precisely the point at which a pharmaceutical intervention can overcome any internal biopsychologic hurdles, resulting in successful weight-loss efforts. For many years, the use of medications to help people lose weight has piqued people's interest. Because of their potential for abuse or the development of major side effects, the recommended medications were only licenced for short-term use until recently. Fenfluramine and its isomer dexfenfluramine are the most recent anti-obesity drugs to be taken off the market after being linked to the development of primary pulmonary hypertension and valvular heart disease. Obesity has recently been

reclassified as a dangerous chronic relapsing disease that necessitates long-term or possibly lifelong therapy, prompting researchers to look at anti-obesity therapies that could meet these new criteria. In general, the optimal antiobesity medication should be both effective and safe. Most appetite suppressants (such as sibutramine and phentermine) work by altering the CNS's monoamine and neuropeptide pathways, whereas rimonabant is the only one on the market that affects the endocannabinoid system. The use of anti-obesity medications in combination is not yet licenced, as additional side effects and no advantages for weight loss have been documented [8,9]. According to NICE guidelines, pharmacother-apy is indicated in patients with a BMI of 30 kg/m<sup>2</sup> or higher, as well as those with a BMI of 27 kg/m<sup>2</sup> or higher, if they have an established obesity-related comorbidity (CVD, Type 2 diabetes mellitus, or sleep apnea) or three or more cardiovascular riskfactors (such as smoking, hypertension, dyslipidemia, and so on). In any event, medication should only be used in conjunction with lifestyle changes if those changes have failed to produce the targeted weight loss.

## Conclusion

Obesity is not a new condition, but the obesity epidemic is. Obesity is prevalent in modern human cultures, as well as undeveloped ones, with an estimated 315 million people globally meeting WHO obesity standards. Obesity is a chronic disease that requires long-term or perhaps lifelong therapy because it is linked to multiple potentially life-threatening cardiovascular and metabolic problems, as well as considerable morbidity, mortality, and diminished quality of life. Moderate, consistent weight loss, on the other hand, has major health benefits. According to NICE calculations, the cost-effectiveness of current antiobesity medications is between £15,000 and 30,000 per quality-adjusted life year gained. Diet, exercise, and behaviour therapy remain the gold standard of weight-loss regimens, although pharmaceutical medication is suggested in select patients who have failed to lose weight through lifestyle changes. Although there is still some ambiguity about the safety and effectiveness of current medication therapy, long-term pharmacological approaches appear to be promise not only for weight loss but also

for weight management. Orlistat, sibutramine, and rimonabant are the only anti-obesity drugs currently approved for long-term use, and they have been shown to improve several surrogate cardiovascular markers in addition to weight loss; however, interpretation is limited due to high attrition rates (30%-40%) and a lack of long-term outcome data.

# **Mini Review**

#### References

- 1. American Obesity Association: Treatment of obesity (2005).
- Despres JP, Moorjani S, Lupien PJ, et al. Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. Arteriosclerosis 10, 497-511 (1990).
- Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 364, 937-952 (2004).
- 4. Benoit SC, Schwartz MW, Lachey JL, et al. A novel selective melanocortin-4

receptor agonist reduces food intake in rats and mice without producing aversive consequences. J Neurosci. 20, 3442-3448 (2000).

- Valsamakis G, Anwar A, Tomlinson JW. 11β-hydroxysteroid dehydrogenase type 1 activity in lean and obese males with Type 2 diabetes mellitus. J Clin Endocrinol. Metab. 89(9), 4755-4761 (2004).
- Fineman MS, Bicsak TA, Shen LZ, et al. Effect on glycemic control of Exenatide (synthetic exendin-4) additive to existing metformin and/or sulfonylurea treatment in patients with Type 2 diabetes. Diabetes Care. 26, 2370-2377 (2003).
- Després JP, Golay A, Sjostrom L: Effects of rimonabant on metabolic risk factors in overweight patients with dyslipidemia. N Engl J Med 353(20), 2121-2134 (2005).

7.

- National Institute for Health and Clinical Excellence (NICE): CG 43 Obesity Guidance on the Prevention, Identification,
  Assessment and Management of Overweight and Obesity in Adults and Children. NICE, London, UK (2006).
- 9. Tomer Y, Davies TF. Searching for the Autoimmune Thyroid Disease Susceptibility Genes: From Gene Mapping to Gene Function. Endocrine Reviews 24(5), 694-717 (2003).