Obesity and weight management at menopause

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Background

Many women report gaining weight as they transition through menopause. For most, the weight gain is modest and can be reduced with a conscious effort to limit energy intake and increase energy expenditure. However, many women who are already overweight and obese will gain more weight as they approach menopause.

Objectives

The aims of this paper are to explain the reasons for menopausal weight gain and to detail a method for achieving and sustaining a substantial weight loss.

Discussion

Weight gain during menopause is predominantly due to a reduction in spontaneous activity. For women who are lean, advice about controlling energy intake and increasing physical activity may be all that is required to prevent weight gain. For women who are overweight and obese rapid weight loss is best achieved with the help of a very low energy diet. This must be followed by lifelong behaviour modification with or without the help of hunger-suppressing pharmacotherapy.

As menopause approaches, many women gain some weight. Studies have shown that the average weight gain is 2.1 kg ± 5.1.[1] Of concern is that all of the weight gained as a result of menopause is deposited in a central abdominal distribution, even in women who have a gynoid (hip and thigh) distribution of fat prior to menopause.

Why is there weight gain at the menopause?

A study by Ainslie et al[2] found that oestrogen deficiency induced by oophorectomy in rats resulted in moderate excess weight gain. This was due to a combination of transient hyperphagia that did not persist despite continuing oestrogen deficiency, and a significant reduction in spontaneous activity (fidgeting) that continued while there was oestrogen deficiency, but was corrected by replacing oestrogen.[2]

These findings were confirmed in humans by Lovejoy et al[3] who studied 103 Caucasian and 53 African-American women who were all premenopausal. Menopause was defined as no menstruation for one year and elevated levels of follicle-stimulating hormone (FSH). Lovejoy et al measured fat and lean mass using dual energy X-ray absorptiometry (DEXA), visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) using computed tomography (CT), dietary intake, serum sex hormones, physical activity (triaxial accelerometry), and 24-hour energy expenditure (whole room calorimeter).

Lovejoy et al[3] charted the changes over seven years of the journey through menopause (four years before menopause and two years after the first amenorrhoeic year). Oestradiol started to decline three years before menopause and dropped more significantly when amenorrhoea occurred. As expected, the reduction in oestradiol triggered an increase in FSH. There was a modest increase in body weight and total body fat as measured by DEXA. However, VAT and central abdominal fat increased more obviously. Lovejoy et al[3] were not able to detect a transient increase in energy intake, but the reduction in spontaneous activity was striking and, as in the rats, was persistent.

Managing weight gain in the perimenopausal years

Simkin-Silverman et al[4] have presented data from a five-year randomised clinical trial known as the Women's Healthy Lifestyle Project, conducted from 1992 to 1999. Participants were 535 healthy, premenopausal women aged 44–50 years at study entry. Participants were randomly assigned to either a lifestyle intervention group receiving a five-year behavioural dietary and physical activity program, or an assessment-only control group. The lifestyle intervention group was given modest weight loss goals (2.3–6.8 kg) to prevent subsequent gain above baseline weight by the end of the trial. To achieve weight loss, participants in the intervention group followed an eating pattern consisting of 1300 kcal/day and increased their physical activity expenditure (1000–1500 kcal/week).

At the end of the program, 55% (136/246) of participants in the intervention group were at or below baseline weight, compared with 26% (68/261) of participants in the control group after 4.5 years (P <0.001). The mean weight change in the intervention group was 0.1 kg below baseline (standard deviation [SD] = 5.2 kg), compared with an average gain of 2.4 kg (SD = 4.9 kg) observed in the control group.

Thus, lifestyle modification can prevent the modest weight gain that occurs as a result of declining oestrogen levels. However, overweight and obesity are now so prevalent that many women approach menopause already obese. What approach should be used to achieve weight loss if the patient is obese even before becoming menopausal?

Management of obesity

To achieve weight loss, it is necessary to reduce energy intake below energy expenditure. Although there are many ways of doing this, the best method to successfully lose weight is to do so rapidly and with the help of mild ketosis. In a recently published study, 200 volunteers who were obese were randomised to either slow or rapid weight-loss groups. The results clearly showed that losing weight rapidly achieved the best results; 78.4% of the rapid group successfully reached the target weight of 15% weight loss, compared with 52.4% in the gradual group. The volunteers were followed for three years and it was found that the rate of weight loss did not have an impact on the rate of weight regain.[5]

Thus, the best way to lose weight is as follows. Assess if the patient is ready for a period of changed lifestyle to achieve a large weight loss. If so, ask the patient to select a day when she is going to start. Before starting, she needs to purchase a very low energy diet (VLED). There are many of these products on the market, but not all are suitable. Suitability can be assessed by a qualified dietitian who can check that all required micronutrients are included at the appropriate levels. These VLEDs serve two purposes. These provide the micronutrients needed, such as vitamins (eg vitamins A, B, C) and minerals (eg iron, calcium, selenium, zinc). This is very important because during this diet, the patient will not be consuming enough food to obtain all of these nutrients. Secondly, VLEDs replace two meals, usually breakfast and lunch. VLEDs come as powders that are mixed with water to make a shake, or as preformed bars for convenience. In addition, advise the patient to have available a roasted chicken or similar protein source, such as tofu, in her refrigerator.

On Day one, the patient takes the first shake or bar and has her usual morning sugarless drink. Nothing else must be eaten. At morning tea, she can have another drink, but again, nothing else must be eaten. However, on this day, if the patient is very hungry, she can nibble the roast chicken or other protein source that was prepared before starting. Lunch consists of a second shake or bar, and another sugarless drink. An afternoon sugarless drink is allowed, but nothing else must be eaten (with the exception of the chicken or other protein source in the fridge). In the evening, the patient has a large dinner, but this must not include any carbohydrates. Dinner is made up of proteins (eg meat, fish, eggs, tofu), three non-starchy vegetables (only those chosen from Appendix 1 of reference 6)[6] and a salad. Advise the patient to dress the salad or vegetables with oil, preferably olive oil, as it is essential to have a small amount of fat daily to empty the gall bladder. This will reduce the risk of developing gallstones.

The routine for day two is the same as day one. Day three is the same as for day one, but the patient will notice fewer tendencies to go to the fridge to top up with roast chicken or other protein because she is less hungry than she was on the first two days. From day four, the patient continues with the regime of replacing two meals a day with a VLED and having a carbohydrate-free dinner until she has achieved her weight-loss goals.

In terms of quantities, the protein is the size of the patient's hand and the vegetables and the salad need to fit comfortably in a normal-sized dinner plate.

Why does hunger disappear after two days?

The reason this diet stresses the avoidance of carbohydrate is that if adhered to, hunger is suppressed after two days. Why?

On the first day of this diet, the patient takes in only 3200 kJ (800 kcal), but her body must burn about 9430 kJ (2300 kcal), so it searches for the missing calories in stored energy. The body burns glucose in preference to fat, so it looks for stored glucose (glycogen). The same happens on day two. On day three, however, there is no glycogen left, as humans can only store two days of glycogen. Thus, on day three, the body moves to burn its next favourite fuel, fat.

When the body burns a lot of fat, the liver releases some of the partly burned fat molecules containing the last four carbon atoms into the blood; we call these 'ketones', which suppress hunger. It has been demonstrated that ketosis prevents the rise in ghrelin[7] that occurs after weight loss, and also increases the secretion of the hunger-suppressing hormone cholecystokinin.[8] There are additional nutrient changes that contribute to hunger

suppression.[7] The heart and brain are the only two organs that can burn ketones, so the other possibility, not yet proven, is that ketones, like other nutrients such as glucose and fatty acids, suppress hunger directly by working on the brain. The point is that during the weight-loss phase, the patient can manufacture her own appetite suppressant. The downside to being mildly ketotic is that the ketones are volatile and, thus, can be breathed out, so the patient may develop ketotic breath. Chewing sugarless gum may help.

If the patient has diabetes and is taking insulin or a sulphonylurea, the dose of these medications must be reduced on starting the diet to avoid hypoglycaemia. Individuals taking warfarin or who have heart, liver or kidney diseases also need more medical supervision when commencing VLEDs.

What if the patient cannot tolerate VLEDs?

Some people cannot tolerate VLEDs because of their taste or texture, or because they may develop symptoms such as diarrhoea. What should they do? Weight will be lost provided energy intake is lower than expenditure. However, if VLEDs cannot be used, the minimum energy intake cannot be less than 4920 kJ (1200 kcal). It takes at least that amount of 'ordinary' food to have enough variety to ensure an adequate intake of the large range of micronutrients. Other options to reduce energy intake are noted below.

The balanced, reduced energy diet

The balanced, reduced energy diet is the typical diet long used to lose weight, and best undertaken with the help and supervision of a dietitian. The strategy is to calculate the subject's energy expenditure and then to prescribe a diet that is reduced by about 2500 kJ (600 kcal). This will result in 0.5 kg weight loss per week. The help of a dietitian is needed to ensure the reduced diet is balanced so that the patient has all of the required micronutrients. The problem with the balanced, reduced energy diet is that hunger develops as weight starts to reduce. To control hunger, there are two options available. Start an appetite-suppressing drug when hunger starts or weight loss stalls, or instead of using a balanced energy reduced diet, induce ketosis by avoiding carbohydrates.

Maintaining weight loss

Weight loss is only the first step of the treatment and, in many ways, the easier of the two phases. The next phase is maintenance of the weight loss. Maintenance will remain a lifelong effort as powerful physiological mechanisms are triggered following weight loss that result in increased hunger because of changes in hunger-controlling hormones[9,10] and a reduction in energy expenditure.[11,12] It has now been confirmed that these changes are long-lasting.[13,14] The patient may need pharmacological assistance and advice to make a conscious effort to restrict calorie intake and increase energy expenditure by regular exercise.

In Australia, two drugs, phentermine (15, 30 and 40 mg) and liraglutide (3 mg),[15] have been approved for hunger suppression. In addition, topiramate can be used off-label, either on its own or in combination either with phentermine or liraglutide. As increased hunger persists for a long time, obesity must be considered a chronic disease, which means that as with other chronic illnesses, medication must be lifelong.

In summary, most women experience modest weight gain during menopause, predominantly because of a reduction in spontaneous activity. Women who are lean and transitioning through menopause can prevent weight gain by paying attention to lifestyle. Women who are overweight and obese may require more vigorous energy restriction, and some may also require long-term pharmacotherapy to suppress hunger.

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References

1. Guthrie JR, Dennerstein L, Dudley EC. Weight gain and the menopause: A 5-year prospective study. Climacteric 1999;2:205–11.
2. Ainslie DA, Morris MJ, Wittert G, Turnbull H, Proietto J, Thorburn AW. Estrogen deficiency causes central leptin insensitivity and increased hypothalamic neuropeptide Y. Int J Obes Relat Metab Disord 2001;25(11):1680–88.
3. Lovejoy JC, Champagne CM, de Jonge L, Xie H, Smith SR. Increased visceral fat and decreased energy expenditure during the menopausal transition. Int J Obes (Lond) 2008;32(6):949–58.
4. Simkin-Silverman LR, Wing RR, Boraz MA, Kuller LH. Lifestyle intervention can prevent weight gain during menopause: Results from a 5-year randomized clinical trial. Ann Behav Med 2003;26(3):212–20.
5. Purcell K, Sumithran P, Prendergast LA, Bouniu CJ, Delbridge E, Proietto J. The effect of rate of weight loss on long-term weight management: A randomised controlled trial. Lancet Diabetes Endocrinol 2014;2(12):954–62.
6. Proietto J. Body weight regulation: Essential knowledge to lose weight and keep it off. 2016.
7. Sumithran P, Prendergast LA, Delbridge E, et al. Ketosis and appetite-mediating nutrients and hormones after weight loss. Eur J Clin Nutr 2013;67:759–64.
8. Chearskul S, Delbridge E, Shulkes A, Proietto J, Kriketos A. Effect of weight loss and ketosis on postprandial cholecystokinin and free fatty acid concentrations. Am J Clin Nutr 2008;87(5):1238–46.
9. Geldszus R, Mayr B, Horn R, Geisthövel F, von zur Mühlen A, Brabant G. Serum leptin and weight reduction in female obesity. Eur J Endocrinol 1996;135(6):659–62.
10. Cummings DE, Weigle DS, Frayo RS, et al. Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. New Engl J Med 2002;346:1623–30.
11. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. N Engl J Med 1995;332:621–28.
12. Levine JA, Eberhardt NL, Jensen MD. Role of non-exercise activity thermogenesis in resistance to fat gain in humans. Science 1999;283:212–14.
13. Sumithran P, Prendergast LA, Delbridge E, et al. Long-term persistence of hormonal adaptations to weight loss. N Engl J Med 2011;365(17):1597–604.
14. Fothergill E, Guo J, Howard L, et al. Persistent metabolic adaptation 6 years after "The Biggest Loser" competition. Obesity (Silver Spring) 2016;24(8):1612–19.
15. Neoh SL, Sumithran P, Haywood CJ, et al Combination phentermine and topiramate for weight maintenance: The first Australian experience. Med J Aust 2014;201:224–26.

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