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POSTMENOPAUSAL OBESITY AND HORMONAL IMBALANCE IN MIDDLE AGED WOMEN: STRATEGIES FOR MANAGEMENT

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ABSTRACT

The post-menopausal period causes a substantial change in middle-aged women's hormonal and metabolic profile, increasing the risk of obesity and related health concerns. Hormonal shifts during menopause include a drop in estrogen, which triggers various physiological changes, including gynoid fat distribution and metabolic modifications, such as insulin sensitivity, hunger changes, and inflammation. An age-related decrease in basal metabolic rate (BMR) leads to loss of muscle mass and a drop in total energy expenditure (TEE), which results in middle-aged women's weight gain. This obesity and estrogen loss causes many clinical implications like hypertension and cancer and increases the risk of cardiovascular diseases as it is associated with variations in the human ERα gene. Apart from physical health, it also affects mental health and self-esteem, which has to be taken care of. This review explains the relationship between obesity and hormonal imbalance in menopausal women and its effect on body composition, changes in energy expenditure, strategies to manage obesity, and hormonal imbalance to develop targeted interventions such as hormonal replacement therapy and lifestyle factors like behavioral, and dietary modifications, especially MED Diet and physical activity.

KEYWORDS: Menopause, Estrogen, Obesity, Postmenopausal obesity.

INTRODUCTION

According to traditional definitions, menopause is a time of transition marked by a gradual decline in estrogen levels as well as other symptoms.[1] The age at which menopause begins

and the length of time it lasts are two examples of individual co-factors that can intensify the severity of some menopausal symptoms, while gaining weight may lower one's quality of life and self-esteem.[2] Obesity and metabolic syndrome are serious concerns, with a prevalence of three times that of premenopausal women.[3] As a result, post-menopausal women are more likely to be obese (BMI >30 kg/m2) than premenopausal women, resulting in a complex process involving decreased energy expenditure due to muscular atrophy, a lower basal metabolic rate, and physical inactivity, which sometimes is exacerbated by depression.[4] It is unclear why menopausal women are becoming more obese. According to Certain researchers, a Major contributing factor to obesity may be the lack of estrogen. Estrogen deficiency exacerbates metabolic disorders.[4] Weight gain is not directly associated with menopause, but it does cause visceral adiposity, increasing total body fat and redistributing body fat from the periphery to the trunk.[3] Metabolic alterations, including insulin resistance, are attached to menopausal estrogen loss, elevated BMI, and higher body fat distribution (measured by the waist-to-hip ratio).[5] Today's epidemic of obesity is a clinical outcome of positive energy balance, which reflects the disparity in calorie intake between nations.[6] Obesity and hormone imbalances are common problems that influence health and quality of life after menopause. Changes in body composition and weight in midlife can lead to overweight and obesity, which raises the risk of cardiovascular disease (CVD) and type 2 diabetes overall.[7] These conditions have a significant impact on mental health in addition to physical health. Post-menopausal obesity has addressed both weight gain and increasing abdominal adiposity common in midlife women, Either alone or in combination.[8] Many factors can lead to changes in body composition and weight, including physical activity, a drop in hormone levels (or the ratio of estrogen to androgen), and dietary modifications.[9]

HORMONAL IMBALANCE AND OBESITY IN MENOPAUSE

Estrogens function in altering the mass distribution of adipose tissue and cardiovascular risk

Sex hormones have a dominant impact on adipocyte development and body fat distribution.[10] Estrogens variably impact adipocyte physiology, although it is debatable how important they are in the emergence of metabolic disorders during menopause. It's unclear what causes the rise in weight in women who are going through menopause.[11] Some researchers contend that a considerable contributing cause of obesity may be the lack of estrogen.[11] Clinical research has identified variables unique to women in the two physiological determinants of obesity: energy expenditure and absorption or eating.[12] For

many women, obesity develops as a result of pregnancy.[13] Humans differ in how adipose tissue is distributed and metabolized; women have higher percentages of subcutaneous adipose tissue and body fat.[14] Women differ in the distribution and metabolism of adipose tissue; they have higher percentages of subcutaneous adipose tissue and more body fat overall.[15] Also, obesity has the utmost impact on how female reproductive hormones function.[13] After menopause, many women have changes in their body composition, including weight gain and an increase in central adiposity.[16] Weight gain significantly alters the activity of reproductive hormones.[13] By influencing other tissues that control energy expenditure or metabolism, estrogens indirectly help to control appetite and body fat.[17] Estrogens are known to have an essential effect on energy intake and expenditure, and the hypothalamus, the primary area of the brain that regulates energy balance, has an immense amount of estrogen receptors.[18] In women, visceral adipose tissue grows three to four years before menopause when blood FSH rises and circulating E2 falls.[18] Changes in the metabolism of adipose tissue are related to these modifications.[19] Estrogens promote lipolysis and affect the activity of adipose tissue lipoprotein lipase.[20] Either boosting the lipolytic effects of adrenaline or triggering the lipolytic enzyme hormone-sensitive lipase, estradiol might indirectly alter lipolysis.[21] However, a recent longitudinal study with a limited number of menopausal women revealed a general rise in total abdominal fat, including visceral and subcutaneous fat, without a dominance of visceral fat formation.[22] Studies have shown that adipose tissue and estrogens have a role in controlling the energy metabolic pathways from glycolysis to glucose transport.[18] Estrogens have anti-inflammatory and lipid-lowering properties that prevent atherosclerosis and produce vasodilation, suggesting that they directly affect vascular endothelial function.[23] Total Cholesterol, LDL, and triglycerides are the three lipids that peak between the end of the perimenopause and the post-menopausal period.[24] The transcription factors that control estrogen function are known as nuclear receptors because they are members of the nuclear receptor superfamily. Alpha (ERα) and beta (ERβ) are the two varieties of estrogen receptors (ER).[11] Only ERα mRNA has been found in brown adipose tissue, although human subcutaneous and visceral adipose tissues have the receptors ERα and ERβ. ERα significantly impacts sexual dimorphism of fat distribution and adipocyte activity.[11] Risk factors for cardiovascular disease are associated with variations in the human ERα gene. Both θ-adrenergic (lipolytic) and α2A-adrenergic (antilipolytic) receptors are the main regulators of lipolysis in humans.[25]

BODY COMPOSITION CHANGES AND ENERGY BALANCE IN OBESITY:

Effect of menopause on weight and weight distribution

The weight gain of approximately 0.5kg per year is due to age rather than menopause, according to cross-sectional and longitudinal studies.[26] Women typically go through menopause between the ages of 50 and 52. However, in developed nations, this age is 3–4 years earlier than average.[27] Women undergoing chemotherapy and have developed ovarian failure are more prone to weight gain than women who remain premenopausal. Likewise, weight has been distributed more around the trunk.[28]

Women undergo significant fluctuations in their weight and body composition. The decrease in estrogen levels in women plays a crucial role in body composition. It is also most important for managing weight, as variations happen in total and resting energy expenditure.[28] Follicle-stimulating hormone (FSH) plays a vital role in menopause as the levels of FSH are increased at menopause based on research (Wendy Cohort and her colleagues). Estrogen replacement therapy does not suppress the increased levels of FSH at the required levels.[29]

A study performed on mice by performing surgery oophorectomy exhibits results in obesity, adipocyte inflammation, and hepatic steatosis by providing supplemental estradiol to help protect the mice from various factors like oxidative stress, insulin resistance, and fatty liver. Mice's lack of the aromatase gene leads to gaining central weight.[30]

Gonadal steroids produce brown adipose tissue (BAT), a form of body fat that controls body temperature in response to cold temperatures. Estrogen keeps the body warm, while testosterone lowers it.[29] A significant risk factor for cardiovascular conditions, such as coronary artery disease, is the buildup of body fat. According to (SWAN) research on women nationwide, women going through menopause have higher levels of fat deposition around their hearts because their estrogen levels drop when they sleep, and insomnia causes weight gain.[31] Studies have shown a correlation between sleep and weight increase.[32]

In a specific study conducted over ten years, patients between the ages of 36 and 39 experienced the most weight gain of about 9 kg, while women between the ages of 36 and 79 experienced an increase of 5.4 kg. Furthermore, another Australian study of women between 18 and 23 found that they gained 8.6 kg over 13 years, with an average weight gain of 0.66 kg.[33]

PATHOPHYSIOLOGY

Estradiol and FSH hormones in women are crucial for controlling energy balance and weight increase during the menopausal transition stage, which may affect the body's fat amount and composition, according to earlier research. Premenopausal women's insulin sensitivity, fat storage, metabolism, and the central nervous system's regulation of food intake and energy expenditure are all impacted by estradiol (E2).[34]

ENERGY BALANCE ON OBESITY

The weight gain results from an imbalance between calorie intake and total expenditure (TEE). Increased stored energy leads to weight gain.[35] The total energy expenditure (TEE) is determined using three variables: basal metabolic rate (BMR), resting metabolic rate (RMR), and active energy expenditure (EAT). 60-80% of the total energy expenditure (TEE) based on the basal metabolic rate (BMR).[33] Age causes a linear drop in BMR and RMR, which lowers TEE.[33] Muscle mass, or body lean mass, is a key component of BMR and the cause of the age-related decrease in BMR.[35] After age 30, muscle mass loss occurs at 3-8% per year.[33]
The enzyme lipoprotein lipase regulates the energy metabolism of plasma glycerides into fatty acids and glycerine.[36] Estrogens in the mitochondria produce around 90% of the ATP in cells. Mitochondrial activity significantly impacts apoptosis and cell survival, whereas the respiratory chain affects estrogen activity.[37]

White adipose tissue is metabolically less active than brown adipose tissue, and its distribution changes with age. In Adults, white adipose tissue found at the subcutaneous layer between muscle, dermis, heart, and kidney replaces the fat tissue found in the neck, thorax, and main blood arteries in newborns.[36] It's believed that brown adipose tissue is absent in mature adults. However, a new study indicates that (BAT) can be triggered and could have helped treat obesity.[38]

CLINICAL IMPLICATIONS

The accumulation of extra body fat causes the development of cardiometabolic issues, complications, tumors, changes in mental and cognitive function, and heightened menopausal symptoms. Midlife women are far more likely to suffer from obesity-related diseases, even if their prevalence can increase with age.[39]

CARDIOMETABOLIC RISK

HYPERTENSION

According to the well-established link between aging and high blood pressure, the prevalence of hypertension increases in midlife. Between 2017 and 2020, the rate of acquiring hypertension in the United States was 15% among those aged 20-34, 32% among those aged 35-54, and among those aged 55-64, it is 49% and, those aged 65-74 and 75 and above it is 75% and 81% respectively. Weight gain, in addition to age, is a symptom of hypertension; particularly, an earlier age at weight gain initiation, which encompasses early adulthood and early middle age, but late beginning weight growth, has been attributed to an increased risk of abrupt hypertension.[33]

METABOLIC DYSFUNCTION ASSOCIATED STEATOTIC LIVER DISEASE

MASLD, or non-alcoholic fatty liver disease, is a common illness associated with metabolic problems. In women under 50, it usually stays constant at 10% to 15% and progressively increases to 20% to 35% by the time they are 60.[40] MASLD's most prevalent consequences are cirrhosis, chronic liver disease, and liver cancer.[41]

DYSGLYCEMIA AND DYSLIPIDEMIA

The prevalence of diabetes and dyslipidemia varies throughout a woman's life. It gradually increases in early adulthood, then rises significantly between the ages of 40 and 55 before declining afterward.[42] As a result, the prevalence of these conditions is 3–4 times higher in midlife women than in women between the ages of 20 and 40, and it is slightly lower in midlife compared to women 65 and older.[33]

CARDIOVASCULAR DISEASE

The prevalence of cardiovascular disease (CVD) in women is estimated to increase with age, particularly after midlife, and 17% in women aged 20–39, 50% in women aged 30-59, 77% in 60–79 aged women, and in women aged 80 and older it is 90%. Menopause is a significant and independent risk factor for CVD, as evidenced by the inverse correlation between age at menopause onset and CVD risk. Weight gain and changes in body composition are related to a higher risk of death and cardiovascular disease (CVD) in midlife women.

Several studies have established a strong link between central obesity and higher cardiovascular disease mortality in middle-aged women.[33]

OSTEOARTHRITIS

A loss of bone strength that can lead to fractures is a hallmark of osteoporosis, a systemic skeletal disorder. Fractures of the vertebrae, hip, wrist, pelvis, sacrum, ribs, sternum, clavicle, and humerus are most commonly associated with osteoporosis. In addition to being associated with increased rates of morbidity and mortality, osteoporotic fractures significantly affect pain and disability.[43] Every year, at least one-third of women suffer fractures brought on by osteoporosis.[44] According to scientific research, a higher body fat percentage is an early risk factor for osteoarthritis.[45]

CANCER

Overweight can lead to several cancers, including those of the esophagus, stomach, colon, liver, gallbladder, pancreatic, breast, endometrial, ovarian, and kidney.[46] A study conducted by Chadid et al. found that midlife women who gain more than 0.45 kg (1 pound) of weight per year have a 30% increased risk of obesity-related cancers.

Furthermore, combining weight gain with metabolic dysfunction (such as prediabetes, type 2 diabetes, dyslipidemia, and hypertension) increased this risk by 77%. This study found that women who became overweight in their middle years after having a normal BMI had a 60% increased risk of fat-related cancer. These findings are in line with evidence that weight gain raises the risk of breast cancer, the most common cancer in women.[47]

MANAGEMENT OF OBESITY AND HORMONAL IMBALANCE

HORMONAL REPLACEMENT THERAPY

Menopausal hormone therapy (MHT), also known as hormone replacement therapy, is the most efficient therapy for post-menopausal symptoms in women.[7,48] Perimenopausal women who are obese are more likely to be symptomatic, necessitating MHT.[49] Evidence suggests that using it in young, healthy menopausal women under 60 or within 10 years of menopause is beneficial and outweighs the hazards.[50] It shouldn't be encouraged that older women begin MHT for preventive purposes.[43,50,51]

In premenopausal women, estrogen may contribute to the gynoid distribution of body fat. However, in post-menopausal women, estrogen insufficiency may result in a more central, 'male-patterned' distribution of body fat. Hassager and Christiansen demonstrated that estrogen replacement inhibited total body fat growth.[48]

Additionally, estrogen/progestin-based Hormone replacement therapy reduces visceral adiposity, insulin levels, fasting serum glucose, and cardiovascular risk factors associated with menopause in healthy individuals.[36,51] Therefore, Estrogen therapy may decrease total cholesterol and relative LDL levels, improving health outcomes. Transdermal patches of 17-β-estradiol with medroxyprogesterone acetate resulted in higher fat reduction in obese and nonobese menopausal women.[36]

MHT use comes with an increased risk of venous thromboembolism, CVS problems, and breast and endometrial malignancies, particularly in obese women. As a result, obese women who require MHT should undergo a thorough risk-benefit assessment. For brief periods, utilize the lowest estrogen dose and micronized progesterone patches.[7]

A meta-analysis of over 100 randomized trials in menopausal women examined the impact of hormonal replacement therapy on metabolic syndrome components. The authors conclude that oral and transdermal estrogen, with or without progestin, increases lean body mass, reduces abdominal fat, improves insulin resistance, lowers LDL/HDL cholesterol ratio, and lowers blood pressure in women without diabetes.[36,52]

Overweight women who use oral MHT (combined or estrogen-only) have a higher risk of TED. Still, the absolute risk is minimal, especially for women under 60 and those with healthy weight. Transdermal MHT (with or without progesterone) is not associated with an elevated risk, making it the preferable method for individuals with a risk or history of TED. As a result, MHT prescriptions must now be individualized to the person, considering risks and benefits.[50]

FORMULATION	ROUTE AVAILABLE	DOSE (mg/dl)
Conjugated equine estrogen	Oral	0.3, 0.45, 0.625, 0.9, 1.25
17β-Estradiol	Oral	0.5, 1.0, 2.0
Estradiol Acetate	Oral	0.45, 0.9, 1.8
Estropipate	Oral	
17β-estradiol matrix patch	Transdermal	0.025, 0.0375 0.05, 0.075, 0.1 twice/week.
17β-estradiol reservoir patch	Transdermal	0.05, 0.1 twice/week.
17β-estradiol transdermal gel (Estrogel)	Transdermal	0.035/d
17β-estradiol topical emulsion	Transdermal	0.05/d (2 packets)
17β-estradiol transdermal spray	Transdermal	0.021/90 µL/d (up to 1.5/90 µL/d)

CE (E)+ MPA (P)	Oral Continuous-Cyclic	0.625 mg E+ 5.0 mg P (E alone x days 1-14, E+P days 15-28)
17β-estradiol (E)+ progesterone (P)	Oral Continuous-Combined	1 mg E +100 mg P
17β-estradiol (E)+ norgestimate (P)	Oral Intermittent-Combined	1 mg E + 0.09 mg P E alone x 3 days, E+P x 3 days, repeat
17β-estradiol (E)+ NETA (P)	Transdermal Continuous-Combined	0.05 mg E + 0.14 mg P (9 cm^2 patch, twice/week)
17β-estradiol (E)+ LVG (P)	Transdermal Continuous-Combined	0.045 mg E + 0.015 mg P (22 cm^2 patch, once/week

MPA, Medroxyprogesterone acetate; E, Estrogen; P, Progestogen; CE, Conjugated estrogens; NETA, Norethindrone acetate; LVG, Levonorgestrel.

PHARMACOTHERAPY

DRUG	**Mechanism of action**	**dosage**	**EFFICACY**
Orlistat	Orlistat is a GI Lipase inhibitor that decreases calorie uptake by inhibiting pancreatic lipase activity.	120 mg BD/ TID	In 1-year and longer trials, it has exhibited 30-40% attrition rates and 5-10% weight reduction.
Liraglutide	It is a GLP-1 receptor agonist that slows gastric emptying, reduces hunger, and improves insulin sensitivity.	Initially, start with 0.6 mg OD daily for one week and 1.2mg for the second week, followed by 1.8mg for the third week, 2.4 mg for the fourth week, and 3mg from the fifth week onwards.	It has been demonstrated to reduce body weight by more than 5% over a 12-month period.
Tirzepatide	GLP-1 and GIP Receptor dual agonists; activation of these receptors decreases appetite while improving glycaemic control.	15 mg	In Phase III clinical studies, body weight was reduced by 20%
Phentermine	It suppresses appetite.	15-37.5 mg /day OD/BD	Over 36 weeks, the mean weight loss was 12–13 kg as opposed to 4.8 kg with a placebo.
Topiramate	Suppresses appetite by increasing	150 mg /day	Efficiently drops about 5% of body

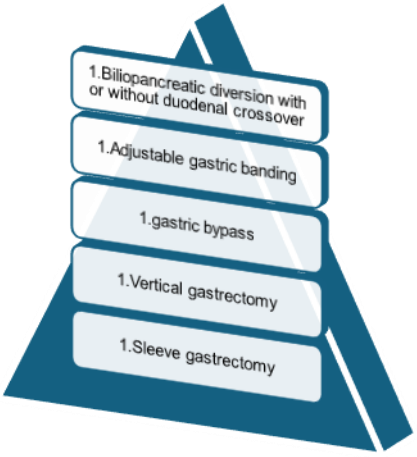
	appetite.		weight in obese people and 6.58 kg in studies of > 2weeks
Metformin	It is beneficial for people who are at risk of developing diabetes mellitus and tackles insulin resistance.	150 mg/ daily	It is reported to reduce weight by 1-2 kg.
Bupropion/ Naltrexone	Nor epinephrine and dopamine reuptake inhibitor and regulates food intake.	90mg OD/BD, 8mg OD/BD	Co-administration has shown efficacy in weight loss in clinical trials.
Semaglutide	It is a GLP-1 receptor agonist that reduces hunger and delays gastric emptying.	2.4mg	In a study, women showed a 16.1% reduction in weight at week 68 compared to men who experienced an 11.4% reduction.

BARIATRIC SURGERY

Bariatric surgery is a successful strategy to treat obesity. It is the most preferable economic intervention that provides weight loss for moderate to severely obese people compared to lifestyle changes and medication.[30,53] It is advisable in patients:

- BMI ≥ 40 kg/m2
- BMI ≥ 35 kg/m2 with severe comorbidities.
- BMI - 30- 34.9 kg/m2 with inadequately managed Type 2 Diabetes.

The physiological intervention mechanism separates bariatric surgery approaches into malabsorptive and restrictive treatments. Malabsorptive techniques shrink the small intestine's functional area, reducing nutrition absorption. Restrictive surgeries decrease stomach storage capacity, resulting in lower calorie and food intake due to earlier satiety.[54] The most performed surgeries in Bariatric surgery are in ascending way.[53]



In two cohort trials of bariatric surgery that reported the outcome at the end of two years, considerable weight loss ranging from 16% to 28.6% was observed compared to the non-surgical group.[30] Weight loss maintenance must be a lifelong commitment since it causes varied hormonal changes that drive appetite and long-term reductions in energy expenditure. Hence, maintain a complete post-surgery care plan, including dietary supplementation and regular health monitoring.[28]

Beyond weight loss, it also improves metabolic health and enhances the quality of life and psychological well-being. It also seems to lessen the risk of obesity-related breast and endometrial cancers. This drop is due to the improvement in insulin sensitivity and hormonal balance that happens with crucial weight loss.[53]

PHYSICAL ACTIVITY

Menopause generally alters body bulk and composition.[55] Women acquire weight, especially in the abdomen, which causes profuse health issues. Regular physical activity is crucial for both weight loss and maintenance.[56]

Exercise improves body composition by lowering visceral adiposity and preventing excessive lean mass loss. Exercise is vital for midlife women going through menopause.[57]

Menopause also causes vasomotor, urogenital, and cognitive alterations in women. Hot flashes affect 86% of menopausal women, along with joint pains, exhaustion, depression, anxiety, vaginal dryness, and other sexual issues due to decreasing estrogen levels during menopause. Research suggests that physically active climacteric women experience fewer

vasomotor symptoms, mood fluctuations, muscle soreness, and sleep issues as physical activity boosts endorphin levels. It also aids in the regulation of body mass.[55]

Enhancing physical activity is also an excellent way to guard against metabolic dysfunction post-ovarian hormone loss; thus, physical activity is a key modifiable risk factor that could prevent or attenuate harmful metabolic alterations after menopause, such as hypertension, dyslipidemia, stroke, Type 2 diabetes, gallbladder disease, cancer, and osteoarthritis.[7,58]

Scientific data on physical exercise have shown that a well-structured program combining aerobic and resistance training is optimal, which helps reach a healthier weight, total body fat percentage, and waist circumference while increasing strength, muscle mass, bone density, and physical ability and reducing inflammatory indicators, sarcopenia, and osteoporosis.[53]

Physical activity may not entirely prevent weight increase with age, but it can reduce the risk of obesity. A one-unit increase in physical activity score leads to a reduction of 4 cm 2 of intra-abdominal fat.[59] Aerobic exercise, including walking, jogging, swimming, cycling, and aerobic dancing, is ideal for weight loss.[56] According to recommended guidelines, adults should engage in at least 150-300 minutes of moderate-intensity or 75-150 minutes of vigorous-intensity aerobic activity each week, along with resistance or muscle-strengthening activities on two or more days per week, which preserves lean mass during weight loss, leading to higher basal metabolic rate and energy expenditure.[28,52] Increased activity is also recommended for overweight and obese adults to prevent muscle loss.[28]

BEHAVIOURAL THERAPY

Behavior plays a crucial influence in maintaining weight loss. Changing daily behaviors aims to reduce food consumption and boost energy expenditure through physical activity.[56] Behavioral change therapies assist patients in achieving health goals such as nutrition, physical exercise, sleep, stress reduction, and enhanced quality of life (e.g., obesity management). Obesity management programs provide typical behavioral treatment, including goal setting, self-monitoring, stimulus control, problem-solving, cognitive restructuring, and relapse prevention.[53] Psychiatric conditions like anxiety and sadness have to be taken care of because they might make it harder for patients to maintain a healthy lifestyle.[52]

Behavioral treatment focuses on conscious satiety/hunger regulation, which promotes better control of anxiety, portion management, speed, and obesity biology.[53] Individual or group

counseling sessions can be provided, depending on the patient's needs, preferences, and available resources.[52]

DIET

Dieting is effective for initial weight loss but not long-term maintenance.[56] No diet is superior to the other, whether it is low fat, low carbohydrates, or high protein, in virtue of achieving sustained weight loss.[28]

The American College of Cardiology/American Heart Association Task Force advised a daily calorie decrease of 500-750 kcal, resulting in a mean weight reduction of 0.5-0.75 kg/week for most women.[28] The recommended body weight reduction is limited to 4 kg per month or 0.5-1 kg per week.[60]

The dietary regimen delivers 1500 calories with 60% carbohydrates, 30% fat, and 10% protein.[3] A proven protein supply of at least 1.2g/kg improves body composition after a weight loss intervention in middle-aged women.[57] The current suggested calorie intake for individuals is 0.8g/kg/day of protein.[61] Fiber intake should be 20-30 g/day, with soluble fiber accounting for 25%.[60] While on the diet, drink two liters of still water and Limit table salt intake to 5g /day.[62]

Patients can lose weight if they stick to the dietary plan with various hypocaloric diets, independent of macronutrient composition. Dietary constraints can include a low fat-low calorie diet, a moderate fat-low calorie diet, a balanced low-calorie diet, a low carbohydrate diet, or a Mediterranean diet. A low-fat diet improves LDL cholesterol levels. A low-carbohydrate diet reduces triglyceride and high-density lipoprotein cholesterol levels more effectively.[52]

If a low carbohydrate diet is preferred, it should include healthy fat (mono and polyunsaturated), and protein options, including fish, nuts, lentils, and poultry, should be included. In the case of a low-fat diet, consumption of healthy carbohydrates like fish, veggies, and whole grains is necessary.[59]

A direct investigation analyzed the nutritional content of three main dietary methods and a study by Sacks et al. including 811 overweight adults, as well as a similar randomized trial involving 160 overweight adults aged 22-27 years with known hypertension and dyslipidemia to four diet groups, carried out for two years. The above studies reveal that although initial

carbohydrate restriction diets offer a substantial weight reduction, at the end of two years, all diets appear to achieve equivalent weight loss while rising patient compliance is significant in producing results.[54] Adherence to the Mediterranean diet tends to lessen the risk of weight gain after menopause while improving cardiometabolic risk factors and decreasing metabolic symptoms.[52]

MED DIET	SOURCE	SERVINGS
Legumes and Nuts	Lentils, Black beans, Kidney beans, Almonds, Walnuts, Hazelnuts	>2 serves weekly & 1-2 servings daily
Vegetables	Leafy greens, Bell pepper, Broccoli, Carrot.	2 serves every meal or 5 servings / day
Fruits	Apple, Grapes, Citrus fruits, Berries.	5 servings / day or 1-2 serves every meal
Healthy Fats	Extra virgin olive oil	Every meal
Eggs and Poultry	Eggs, Chicken, Turkey	2-4 serves per week
Dairy foods	Milk, Cheese, Yogurt	1-2 serves per day
Herbs and Spices	Turmeric, Cinnamon, Garlic	in moderation
Beverages	Water Red wine	2 liters per day In moderation
Sweets	Dark Chocolate	< 2serves/ week
Red meats	Beef, Lamb	<2 serves/ week
Seafood	Salmon, Tuna, Shrimp	2serves weekly
Salt	-	5g per day

CONCLUSION

Post-menopausal women are more likely to experience obesity and metabolic implications. Women's midlife weight gain is due to the decline in estrogen levels and an increase in FSH, triggered by a linear drop in BMR and alterations in body composition such as decreased TEE, muscle mass loss, and an increase in central adiposity. The hormonal imbalance and extra body fat accumulation increase the risk of metabolic ramifications like hypertension, osteoarthritis, cancer, genitourinary atrophy, CVD, steatotic liver disease, Dysglycemia, and Dyslipidemia, resulting in higher mortality and morbidity rates. As the predominance of obesity in ladies rises amid and after midlife, it is of most extreme significance to execute key mediations to avoid weight pick up and manage corpulence in this populace. Endeavors should center on behavioral and way-of-life changes, notably decreasing caloric intake, adherence to the MED diet, and expanding physical action, with second-level anti-obesity medicines used when required. Hormone replacement therapy will always present a mixed bag of benefits and risks. Transdermal MHT is not associated with an enhanced risk, making

it the preferable approach to oral MHT. Another successful strategy to treat obesity is bariatric surgery. Therefore, suitable treatment is provided based on individual needs.

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