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A case challenge: reducing metabolic syndrome associated risk through obesity management

Abstract

Obesity is a complex chronic condition that increases the risk for development of metabolic syndrome (MetS). MetS is a cluster of bi-directionally linked metabolic dysfunctions that include central obesity, dyslipidemia, hypertension, and impaired fasting glucose. Globally over 28% of adults and 18% of adolescents are obese, with one-third of the adult population meeting the diagnostic criteria for MetS.

Body mass index (BMI) is a common standard for categorizing and managing clinical obesity. There are more reliable measures for predicting obesity associated with risk. Among these measures is waist circumference, which is utilized as part of the diagnostic criteria for MetS. Additional reliable obesity measures include waist-to-hip ratio, magnetic resonance imaging, and computed tomography, but they have limited application due to time and cost factors.

Insulin resistance, alterations in cholesterol, systemic inflammation, hormonal dysregulation, visceral obesity, and hypertension are among the reversible obesity-induced MetS comorbidities that can be mitigated, reduced, or completely reversed with a sustained total body weight loss of 5–10%. According to clinical guidelines, a combination of dietary modifications, increased physical activity, and cognitive behavioral therapy are the primary recommendations for weight loss management. Additional weight loss benefit is potential with adjunct pharmacological therapies that include GLP-1 receptor agonist, oral anorexiants, phentermine-topiramate, naltrexone-bupropion, and orlistat. In addition, surgical interventions are an effective adjunct therapy for patients with health complications secondary to morbid obesity. A total body weight loss of 5–10% has the potential to reduce all MetS related comorbidities.

Keywords: obesity, metabolic syndrome, prevention, weight loss, management, BMI

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Abbreviations: AACE/ACE, American Association of Endocrinology and the American College of Endocrinology; ADA, American Diabetes Association; AE, aerobic exercise, BMI, body mass index, BMR, basal metabolic rate, CBT, cognitive behavioral therapy; CT, Computed Tomography; GLP-1, glucagon-like-peptide agonist; GIP, glucose-dependent insulinotropic polypeptides; HDL, high-density lipoproteins; HTN, hypertension; kg, kilogram; MetS, metabolic syndrome ; MHO, metabolically healthy obesity; MUO, metabolically unhealthy obesity; MRI, magnetic resonance imaging; NIH, National Institutes of Health; OMA, Obesity Medicine Association; RD, Registered dieticians; RT, resistance training; TDEE, total daily energy expenditure; USPTF, United States preventative task force.

Reducing metabolic syndrome associated risk through obesity management

Metabolic Syndrome (MetS) is a cluster of metabolic dysfunctions that are complicated by obesity. Clinical obesity is defined as excessive adipose tissue collection that increases the risk of an adverse health event resulting from an imbalance in caloric intake to metabolic caloric need.[1] According to the National Institutes of Health (NIH), approximately 40% of all adults are obese and one in three adults meet the diagnostic criteria for MetS. To diagnose MetS, three out of five of the following criteria must be present: central obesity, elevated triglycerides, low high-density lipoproteins, hypertension, or impaired fasting glucose.[1,2]

Case presentation

A 35-year-old Caucasian male presented to the clinic today for follow up after initiating primary care services two weeks ago. The patient had no history of known chronic disease or significant medical history. He has a family history of paternal hypertension, stating that his father started medication in his forties. He reports no medical history with his mother, siblings, or grandparents. The patient reported that he is an accountant and has a primarily sedentary lifestyle.

On his initial visit, the patient was afebrile with an elevated blood pressure of 144/89. He is 5' 8" tall and 217 pounds, giving him a BMI of 33 kg/m^2. He was asked to keep a home blood pressure log. In addition, the patient was counseled on the effects of obesity on his cardiovascular health and that it increases his risk of type 2 diabetes. He was also provided education on the benefits of weight loss management. The provider recommended that the patient be intentional about increasing his exercise to 150 minutes a week and recommended that he adopt a Mediterranean diet.

During the follow-up visit today, the patient indicated that he had not increased his exercise consistently at this point but had made plans to join a gym. He reports that he has made some changes to his diet but has not fully implemented the dietary recommendations. He provided his home blood pressure logs that indicated consistent blood pressure elevations in the 146–140/85–80 range. His in-office reading was 145/92 today.

His lab from 2 weeks ago revealed normal kidney and liver functions. He had a hemoglobin A1c of 5.9, LDL level of 180 mg/

dl high-density lipoprotein (HDL) level of 37 mg/dl, and triglyceride level of 175 mg/dl, with a total cholesterol level of 248 mg/dl. Clinically he has a basal metabolic index (BMI) of 33 kg/m^2 and a waist circumference of 42 inches. The patient was diagnosed with Stage 1 hypertension, prediabetes, dyslipidemia, and central obesity during the visit; in combination he met the diagnostic criteria for MetS (Table 1).

Table 1 National institute of health metabolic syndrome diagnostic criteria[1]

Metabolic syndrome risk factors requires 3 out or 5 criteria	Diagnostic parameters
Central Obesity	Waist Circumference >40 centimeters (men) >35 centimeters (women)
Hypertension	Systolic blood pressure > 130 Diastolic blood pressure > 85
Glucose Intolerance	Fasting glucose > 100 mg/dl
Elevated Triglycerides	> 150 mg/dl
Low High-Density Lipoproteins	< 40 in men < 50 in women

Initial management focused on re-educating the patient on the importance of obesity management to improve his blood pressure, cholesterol levels, and blood glucose levels to prevent progression of the disease processes and potential complications associated with MetS. The provider prescribed a weight loss plan that included initial exercise and dietary management, as well as recommendations for cognitive behavioral therapy (CBT). A referral was made for dietary counseling with a registered dietitian. The prescriptive management plan utilized the obesity clinical guidelines from the Obesity Medicine Association (OMA) and also incorporated clinical guidelines from the American Association of Endocrinology and the American Academy of Clinical Endocrinology (AACE/ACE), the American College of Cardiology/American Heart Association Joint Committee, and the American Diabetes Association (ADA) to guide management of the patient's other components of MetS. Semaglutide was used to target weight reduction and impaired glucose, lisinopril to target his hypertension, and atorvastatin to target HDL, triglyceride levels, and atherogenic changes.[3-6]

Case challenge questions

1. How is obesity diagnosed and categorized?
2. How does obesity affect the comorbidities of metabolic syndrome?
3. What are the screening recommendations related to metabolic syndrome comorbidities?
4. How can providers manage MetS through obesity management?
5. How effective is obesity management on improving comorbidities associated with MetS?

How is obesity diagnosed and categorized?

There are multiple acceptable ways to diagnose obesity. BMI is the most common screening tool used to assess obesity and its complications. BMI is the ratio between measurements of weight in kilograms divided by height in meters squared.[7] According to the WHO, obesity is diagnosed when an individual has a body mass index over 30.[8] Individuals of Asian descent are at risk of obesity-related complications due to lower levels of body fat, with obesity being diagnosed at a BMI of 25 or lower in those of Asian descent.[3]

In comparison to BMI, waist circumference, hip-to-waist ratio, magnetic resonance imaging (MRI), and computed tomography (CT) are recognized as more accurate methods to predict obesity-related complications. These techniques are better at differentiating subcutaneous fat from high levels of visceral fat, which increases the risk of obesity-related complications. Despite their increased accuracy, these tools are limited due to the time or cost associated with each technique.[9] The United States Preventative Task Force (USPTF) recommends BMI screening for all adult patients and recognizes that waist circumference should be obtained annually in individuals identified as obese through BMI to more accurately assess health risk.[10]

Once obesity is diagnosed, it is categorized into stages based on severity (Table 2).[3] It can further be categorized into metabolically healthy obesity (MHO) or metabolically unhealthy obesity (MUO). The categories are dependent on whether or not the patient has developed an obesity-induced co-morbidity. Individuals are classified as MHO if they have a BMI of 30.0 or greater and have not developed medical complications such as hypertension, impaired fasting glucose, or alterations in their triglycerides or high-density lipoprotein based on laboratory reference ranges. Individuals are at greater risk of becoming MUO if they have excess visceral adiposity.[2,11]

Table 2 Obesity classification based on BMI[3,12]

BMI kg/m2	Stage of classification
18.5–24.9	Normal Weight
25–29.9	Stage 0 Obesity
30–34.9	Stage 1 Obesity
35–39.9	Stage 2 Obesity
>40	Stage 3 Obesity
Asian adjusted BMI, kg/m2	**Stage classification**
18.5–24.9	Normal Weight
>23	Overweight
>25	Obesity

MUO is defined as having a BMI of 30 or greater, with one or more comorbidities of hypertension (HTN), elevated glucose, elevated triglycerides, or low high-density lipoproteins (HDLs). The comorbidities associated with MUO are consistent with the diagnostic criteria of MetS. Each component of the diagnostic criteria for MetS has its own subset of comorbidities that increase the probability of developing chronic conditions such as stroke, coronary artery disease, peripheral artery disease, insulin resistance, type II diabetes, liver disease, or polycystic ovarian syndrome[2,11] (Table 3).

Table 3 Formulas for calculating caloric requirements[24,25]

Formulas for calculating caloric requirements		
Mifflin-St Jeor equation for BMR	Male	(10 x kilogram) + 6.25x height in cm – 4.92 x age +5
	Female	(10 x kilogram) + 6.25x height in cm – 4.92 x age -161
TDEE calculation	Sedentary	Basal metabolic rate x 1.2
	Lightly active (1–3 days/week)	Basal metabolic rate x 1.375
	Moderately active (3–5 days/week)	Basal metabolic rate x 1.55

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How does obesity affect the comorbidities of the syndrome?

Central obesity, hypertension, impaired fasting glucose, and alterations in cholesterol are interrelated components of MetS that exacerbate metabolic and cardiovascular risk factors through bidirectional mechanisms.[13] Central obesity, also referred to as visceral fat, is the primary driver and link to all the components of MetS. Visceral fat disrupts the normal hormonal balances of the endocrine system. Among these imbalances are an increase in the secretion of cytokine-like molecules called adipokines, pro-inflammatory cytokines, and free fatty acids that contribute to systemic inflammatory processes and metabolic dysfunction.[13-15]

Excessive accumulation of visceral fat leads to a decline in adiponectin levels, which exacerbates insulin and leptin resistance. Angiotensinogen levels increase because of decreased adiponectin. These hormonal imbalances lead to impaired glucose utilization, impaired lipid metabolism, and the stimulation of the sympathetic nervous system, which causes hypertension, elevated heart rate, endothelial dysfunction, vasoconstriction, and sodium retention.[11,13,16] Simultaneous stimulation of the renin-angiotensin-aldosterone-system further compounds sodium and water retention, compounding hypertension and vascular resistance.[13-15]

The insulin resistance that is affected by central obesity reduces lipoprotein lipase activity. This leads to atherogenic changes that result in elevated triglycerides, elevated very-low-density lipoproteins, and low levels of protective high-density lipoproteins. In addition, low-density lipoproteins become smaller and more permeable to the arterial vessel wall, leading to an increased risk of coronary artery disease, peripheral arterial disease, cerebrovascular injury, and hypertension.[13-15]

What are the screening recommendations related to metabolic syndrome comorbidities?

No organization has provided screening recommendations specifically for MetS. Screening schedules for the individual components of MetS do exist; the American Association of Endocrinology and the American College of Endocrinology (AACE/ACE) have published screening recommendations for obesity, while the United States Preventative Task Force (USPTF) makes recommendations for remaining of the components of MetS. The AACE/ACE guidelines recommend screening all adults annually for obesity and its related complications.[3] In contrast, the USPTF recommends that obesity screening start as early as 6 years of age.[18] The USPTF also recommends that screening for hypertension be provided at each office visit for all individuals greater than the age of 18.[19] As part of standard of care, it recommends that all individuals between the ages of 35–70 years are screened for impaired fasting glucose when their BMI is consistent with being overweight or obese.[20] In addition, it recommends that all individuals be referred for obesity interventions if they have a BMI greater than 30kg/m^2.[10] USPTF no longer recommends routine screening of cholesterol levels; it revised its recommendations in 2022 to focus on prevention of comorbidities associated with atherogenic dyslipidemia changes. As a result, it recommends initiation of statin therapy for all individuals 40 to 75 years who have one or more cardiovascular risk factors, regardless of cholesterol level.[21]

How can providers manage MetS through obesity management?

Providers can approach obesity management by applying evidence-based guidelines that target the client's individual stage of obesity, their co-morbidities, and their overall weight loss goals. The plan should be comprehensive and tailored towards the patient's individual goals for weight loss, with the primary focus being on decreasing the risk or complications associated with obesity-induced comorbidities.[22] The ACCE/ACE guidelines recommend that a 2.5% total body weight reduction be set for the first month of a weight-loss program, with an additional 5–10% reduction over the next 6 months.[3]

Obesity guidelines recommend a multidisciplinary approach to meet weight-loss targets.[3] The OMA recommends that weight management should include nutritional therapy, physical activity, behavioral modifications, and medical interventions as foundational components of any weight-loss plan. Targeting weight loss with one modality, such as caloric reduction alone, decreases long-term weight loss success and puts an individual at risk of rebound weight gain within 4–6 months.[23]

Providers should recommend dietary modifications that are guided by Registered Dieticians (RDs) or through healthcare professionals that are trained in providing medical obesity management.[22] RDs can help formulate a plan to estimate the total caloric needs for an individual that considers their chronic disease status, daily protein needs, fluid balance status, and target weight.[24] When consultation with an RD is not available, then primary care providers should use formulas such as the Mifflin-St Jeor equation to calculate an individual's basal metabolic rate (BMR). BMR reflects the daily caloric needs to maintain vital functions such as cellular repair, breathing, and blood circulation. The formula takes into consideration the age, current weight, and height to calculate minimum caloric needs. In addition, the total daily energy expenditure (TDEE) should be calculated using the BMR and the individual's current activity level to identify the average daily caloric needs to maintain their current weight. A caloric reduction between 300–500 calories per day below the TDEE is recommended for slow weight loss averaging approximately one pound per week.[24,25]

The OMA and AACE/ACE guidelines recommend an exercise prescription for all stages of obesity that considers current chronic disease and mobility status to decrease obesity-related complications and exacerbation of chronic disease. Guidelines recommend 150–180 minutes per week of aerobic exercise (AE), along with resistance training (RT).[3,22] AE targets overall body fat and visceral fat composition and is the most effective type when used alone for improving components of MetS. High-volume AE without RT results in a smaller reduction in overall body weight, with an average of up to 2 kg of weight loss. Weight-loss results are more effective when AE is combined with caloric restriction and RT. In combination, AE and RT provide the greatest overall weight loss benefit. RT alone improves core strength, muscle mass, and lean body mass more than AT, and as a result an individual is less likely to see weight reduction from RT alone. However, RT does improve the overall body composition because it increases the basal metabolic rate more effectively than AE alone, resulting in reduced visceral and overall fat composition. MetS improves as AE reduces systolic blood pressure by an average of 10 mm Hg and diastolic by an average of 5 mm Hg. In addition, research shows that HDL levels are very sensitive to AE and that high-intensity AE lowers triglyceride levels even without changes in diet within 6 months. Skeletal muscle contractions that occur during AE results in increased glucose uptake, while RT increases insulin sensitivity. The combination results in improved fasting glucose levels.[27]

Cognitive behavioral therapy (CBT) is an appropriate first-line intervention for individuals with all stages of obesity. When CBT is declined and not initiated with the initial weight-loss plan, the AACE/ACE guidelines recommend re-evaluation and initiation of CBT in all patients whose total body weight has not reduced by at least 2.5%

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within the first month of starting an individualized provider-prescribed dietary and physical activity plan.[3] CBT helps to identify and address underlying mental health conditions that contribute to poor lifestyle choices and eating patterns that potentiate obesity.[28] Research has demonstrated that patients with coronary artery disease were more likely have MetS when they also had a history of underlying anger, hostility, pessimism, or depressive symptoms. Research also found greater compliance to lifestyle modifications that promoted weight reduction, such as diet, exercise, and physical activity, when patients adhered to CBT as part of their first-line therapy. The results of lifestyle changes associated with CBT led to improvement in all the diagnostic components of MetS that were still prevalent up to 18 months post intervention.[29]

Pharmacologic therapy is a first-line management option that should be used as an adjunct to strict lifestyle modifications and CBT for the management of both MUO or MHO individuals.[3] There are several drug classes that have clinical indications by the United States Food and Drug Administration (FDA) for obesity management, as well as FDA-approved medications for other disease processes that are used for their side-effect profiles, including the promotion of weight loss. Each medication has its benefits and risk profiles, with varying levels of efficacy for assisting with weight-loss management (Table 4). Prescribers should consider an individual's comorbidities, the medications' mechanisms of action, the dual indications of the medications, and the duration of therapy needed to maximize weight loss. Medications with short-term indications should be considered in individuals who have MHO in Stage 0 or Stage 1. Because research shows that discontinuation of weight-loss medications typically results in rebound weight gain, it is important that patients who have MUO or chronic stage 2 or greater obesity be prescribed long-term therapy regimens for sustained weight loss.[30]

Table 4 Efficacy of obesity prevention and management options[15,38,45]

Efficacy of obesity prevention and management options	
Lifestyle modification first line adjunct therapy for all levels of obesity dietary prescriptive exercise, & cognitive behavioral therapy dietary prescriptive Exercise, & cognitive behavioral therapy	
BMI >27 with 1 Co-Morbidity or BMI > 30	
Medication adjunct to lifestyle modifications	Expected Weight Loss
Semaglutide	15–17%
Liraglutide	5–10%
Tirzepatide	8–22%
Phentermine	3–8%
Topiramate (off label)	5–10%
Phentermine-topiramate	5–10%
Naltrexone-bupropion ER	5–11%
Diethylpropion	5–10%
Orlistat	5–8%
BMI > 35	
Medication adjunct to lifestyle modifications	Expected Weight Loss
Vertical Sleeve Gastrectomy	50–75%
Laparoscopic Adjustable Gastric Banding	30–50%
Roux-en-gastric bypass	55–75%

When pharmacologic therapy is prescribed, the American Gastroenterology Association recommends the use of medications such as Glucagon-like-peptide agonist (GLP-1), glucose-dependent insulinotropic polypeptides (GIP), phentermine (alone or in combination with topiramate), naltrexone-bupropion, orlistat, or diethylpropion. GLP-1 and GIP are first-line pharmaceutical agents with FDA-approved indications for long-term obesity management.[30] They target and greatly reduce visceral fat associated with MetS.[31] Their mechanism of action is to stimulate insulin production in response to a glucose load. This results in regulated blood-glucose levels, improved cholesterol levels, and endothelial function. Glucagon-like peptide receptors are also located in areas such as the hypothalamus, brainstem, carotid body, kidneys, and vasculature structures that control blood pressure. These receptors are altered and become dysfunctional in hypertension. As a result, the agonistic effects of GLP-1s aid in improving hypertension.[32] GLP-1s also increase glucose intake in the muscle, reduce production of glucose in the liver, and increase satiety, leading to weight loss.[33] As a result of reduced visceral fat, triglyceride and HDL levels are normalized as hormonal imbalances are regulated, leading to improvement in all components of MetS.[31]

Phentermine-topiramate is a combination medication that has been approved by the FDA for long-term weight-loss management. Individually, topiramate is FDA approved for partial seizure control or migraine prevention. It is used off label as a long-term therapy for weight loss because of its side-effect profile. It is inexpensive, does not create a seizure risk in individuals without a seizure history, and can provide dual benefit for prevention of migraine. The side-effect profile of the medication provides appetite suppression and early satiety, and can also cause dysgeusia, which decreases the desire to eat and promotes weight loss.[34] Phentermine is FDA approved as monotherapy for the treatment of obesity. It is one of the most common medications prescribed for weight loss.[35] Phentermine is a sympathomimetic amine that induces appetite suppression. Phentermine has the potential to raise blood pressure and resting heart rate. When used in monotherapy, it should be limited to short-term use averaging 12 weeks, in order to avoid development or exacerbation of previously controlled hypertension and other cardiovascular disease.[30] When the medications are prescribed in combination under the brand name of Qsymia, the individual medications are provided in smaller doses that allows for a long-term clinical indication. The long-term effects of the medication contribute to the reduction of overall adipose tissue as well as reduction of the effects of MetS.

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Naltrexone-bupropion is an FDA-approved medication for weight reduction. Naltrexone is an opioid receptor antagonist, and bupropion is a norepinephrine-dopamine reuptake inhibitor. Naltrexone is FDA approved for the treatment of opioid- and alcohol-use disorders, while Bupropion is approved for the treatment of depressive disorders and smoking cessation. In combination, they work to suppress appetite and the reward centers of the brain, thus decreasing food cravings and the brain's recognition of food as a reward, resulting in weight reduction.[3] Naltrexone-bupropion improves MetS because of its synergistic effects and its effectiveness at reducing central obesity.[37]

Diethylpropion is a medication with similar properties and indications to phentermine. It gained its FDA indication for short-term management of weight loss in 1959, and is a monotherapy medication that is used in conjunction with lifestyle modifications such as diet, exercise, and behavioral interventions.[30] Diethylpropion is a sympathomimetic amine and anorectic that has a lower risk of adverse effects on the cardiovascular system.[38] Monitoring blood pressure and heart rate are recommended while a patient is taking diethylpropion, and it should be avoided in patients with a history of cardiovascular disease. In comparison to other FDA-approved medications, newer studies have found varying reliability for consistent weight-loss benefit among patients, which limits its use.[30]

Orlistat is another FDA-approved medication for the treatment of obesity. It is a pancreatic lipase inhibitor that aids in weight reduction by preventing absorption of dietary fat up to 30%.[31] The decrease in dietary fat absorption results in decreased energy intake, resulting in weight loss. It works by temporarily inhibiting pancreatic and gastric lipases in the gut. Inhibition of the lipases allows triglycerides to be broken down into absorbable forms of monoglycerides and free fatty acids. Orlistat provides overall improvement in total cholesterol levels, including triglyceride and LDL levels. The medication also improves central obesity, as evidenced through lower waist circumference and BMI. Because the medication works by reducing absorption of dietary fat, monitoring and supplementation of fat-soluble vitamins is recommended. The medication has off-label indications for reducing non-alcoholic fatty liver disease and non-alcohol steatohepatitis, and aids in the management of congestive heart failure patients. The medication should be avoided in patients with chronic malabsorption, cholestasis, or pregnancy.[39] This medication is not often used as a first line therapy because the side effects of the gastrointestinal effects result in steatorrhea and fecal incontinence.[40]

The NIH set guidelines in 1991 that continue to guide practice for surgical intervention as a preferred management option for obesity management. These recommendations have indications for endoscopic or bariatric surgery when BMI exceeds 40 or when comorbidities are present with a BMI of 35 kg/m2 or greater.[41] The American Society for Metabolic and Bariatric Surgery recognizes that surgical intervention is superior for sustained weight loss and reversal of associated comorbidities in comparison to diet, exercise, and other weight-loss therapies over a 20-year period. As a result, it also recommends surgical intervention for those with metabolic disease and BMI of 30 or greater who have continued obesity-associated comorbidities despite previous intervention with diet, exercise, behavioral, and/or pharmaceutical therapies. In addition, it recognizes that individuals with severe morbid obesity with BMI > 50 kg/m2 are at increased surgical risk for complications. Surgical risk versus risk of mortality from other comorbidities must be weighed. Bariatric surgical procedures have the highest risk when is greater than BMI kg/m^2, but research indicates that it is still a viable option when weighing the benefit to risk profile.[41]

How effective is obesity management on improving comorbidities associated with MetS?

All components of MetS can effectively be treated with weight loss reduction. A 5–10% total body-weight reduction can significantly reduce waist circumference, the synergistic effects of central obesity, and comorbidities associated with MetS.[8] ACCE/ACE recognizes that a 5% reduction in total body weight can prevent the development of new obesity-related complications. Amelioration of current risk associated with Stage 1 and Stage 2 obesity occurs with a 10% reduction in total body weight.

Research shows that a 1.05 mmHg reduction in systolic pressure and 0.92 mmHg reduction in diastolic blood pressure is associated with 1 kilogram (kg) of weight loss.[42] In addition to this improvement in blood pressure, cholesterol levels also improve. Research has found that with 1 kg of weight loss, HDL levels increased by 0.42 mg/dl, while triglyceride and LDL levels reduced by 4.0 mg/dl and 0.33 mg/dl respectively.[43]

Evidence indicates that glucose metabolism improves with the first 2.5–5% initial reduction of total body weight.[22] The ADA endorses that a 5–7% total weight reduction will reduce cardiovascular risk and glycemic control. Progression from pre-diabetes to diabetes is halted when individuals lose 5–7% of their total body weight. The ADA recommends setting an initial total body weight loss goal of 7% to target impaired fasting glucose, recognizing that at least 43% of individuals will have normalized blood sugars at this level of weight loss. It endorses that with 10% total body weight loss, patients will also have improvement in conditions such as systemic inflammatory processes associated with adiposity, metabolic dysfunction-associated steatohepatitis, and sleep apnea[44] (Table 4).

Case study outcomes

The patient followed up with the provider at 4 and 12 weeks after starting the prescribed weight loss plan and prescriptive regimen to address the hypertension, dyslipidemia, and impaired glucose associated with his MetS. The patient reported compliance lifestyle modifications that included a Mediterranean diet, two days a week of a 75-minute physical activity plan that included both RT and AT, and cognitive behavioral therapy. He reported adherence to prescribed lisinopril, atorvastatin, and semaglutide, which were prescribed in alignment with evidence-based practices for his newly diagnosed chronic conditions of hypertension, hypertriglyceridemia, low HDL, and impaired fasting glucose, all consistent with pre-diabetes.[3-6]

The patient's blood pressure stabilized within 4 weeks into the normal range after he started lifestyle medications and lisinopril. His in-office reading was 130/78. At 12 weeks his labs were rechecked. The semaglutide was effective, as it helped the patient lose greater than the 2.5% total weight loss goal for his first month, and his glucose impairment resolved. He weighed in at 206 pounds, making his BMI 31.3 kg/m^2, and his A1c was below the pre-diabetes threshold with a reading of 5.5. Lifestyle changes in combination with atorvastatin were also effective at improving his dyslipidemia. The total cholesterol level was 229 mg/dl, HDL of 50 mg/dl, LDL of 150 mg/dl, and triglyceride level of 145 mg/dl. Although his total cholesterol and LDL were above ideal ranges, his HDLs and triglycerides were no longer in ranges meeting criteria for MetS. Encouragement, reiteration, and reassurance that continued strict adherence to the lifestyle changes and prescriptive plan would result in continued weight loss and improvement in metabolic health were provided, with the recommendation to follow up in 3 months.

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Conclusion

Three out of 5 components related to central obesity, hypertension, impaired glucose, elevated triglycerides, and low high-density protein levels are required for an individual to be diagnosed with MetS.[1] Obesity is a common factor among all of the diagnostic components of MetS. Targeting obesity is a primary factor that should be used in conjunction with other treatment protocols in the management of obesity-induced complications associated with MetS. For individuals with stage 1 or 2 obesity, modest weight loss of up to 10% of the total body weight can reduce cardiovascular associated risk and impaired glucose metabolism and can also improve triglyceride levels.[8]

Decreasing overall body weight has been shown to have similar effects on reductions in excess visceral tissue. This reduction in visceral fat helps to disrupt the effects of hormonal imbalances that lead to impaired glucose metabolism and alterations in cholesterol levels. Weight-loss regimens should include dietary management, prescriptive physical activity, cognitive behavioral therapy, and adjunctive pharmaceutical or procedural care based on the patient's baseline BMI.[22] GLP-1s have a clinical indication in stage 1 and stage 2 obesity due to their effects on glucose metabolism, cholesterol levels, and hypertension.[30,32] Surgical procedures are a first-line option for individuals who have not responded to pharmaceutical treatment or that have chronic obesity with a BMI of 35 or greater.[41]

Clinical guidelines should be implemented with the addition of statin therapy to prevent the development of atherogenic disease in individuals with MetS who have normal cholesterol, per USPTF recommendations.[21] AACE/ACE and ADA protocols for hypertension and impaired glucose metabolism should be utilized to manage obesity-induced hypertension altered glucose until sustained weight loss can be achieved.[44]

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Conflicts of interest

In compliance with national ethical guidelines, the authors have no relationship with business or industry that would pose a conflict of interest.

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