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GOOD CLINICAL PRACTICE RECOMMENDATIONS ON

OBESITY

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Dear Doctors,

It gives me immense pleasure to present this important publication focusing on Obesity in Women—a growing public health concern that significantly impacts the physical, reproductive, and emotional well-being of women across all age groups.

In recent years, obesity has emerged as one of the most pressing challenges in women's health. Changing lifestyles, urbanization, nutritional transitions, stress, and reduced physical activity have contributed to a steady rise in obesity among adolescents, women of reproductive age, and postmenopausal women alike.

As obstetricians and gynecologists, we are uniquely positioned to intervene across a woman's life course. Addressing obesity is not merely about weight reduction, but about empowering women to achieve sustainable health through lifestyle modification, evidence-based interventions, and psychosocial support.

FOGSI remains committed to advancing women's health through knowledge dissemination, capacity building, and community outreach.

Best wishes!

Dr. Sunita Tandulwadkar

President FOGSI

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Management of Obesity in Women:
Good Clinical Practice Recommendations

Obesity: An overview

Obesity has emerged as a rapidly increasing global health concern, currently affecting an estimated 1 billion adults aged 20 years and above. Its prevalence has risen significantly over the past three decades, with women being more susceptible to obesity than men across all age groups.[1]

According to the World Health Organization (WHO, 2022), approximately 2.5 billion adults are overweight and 890 million are obese worldwide. This represents 43% of adults ≥18 years (43% of men and 44% of women) being classified as overweight—an increase from 25% in 1990.[2] Globally, the prevalence of obesity in children and adolescents (aged 5–19 years) is projected to increase from 10% to 20% among boys and from 8% to 18% among girls between 2020 and 2035. This rising trend is closely associated with unhealthy behavioral patterns, including sedentary lifestyles, high consumption of junk foods, inadequate physical activity, and poor self-care practices, all of which contribute substantially to overweight and obesity. Furthermore, higher body mass index (BMI) is also associated with behavioral confounding factors such as unhealthy eating habits, excessive screen time, substance abuse, and mental health.[3,4] Looking ahead, current projections indicate that by 2035, approximately 23% of men and 27% of women aged ≥20 years worldwide will be living with obesity.[4]

Prevalence of obesity in India

Overweight and obesity, previously regarded as public health issues primarily affecting high-income countries, are now emerging as significant health concerns across all age groups, including children and adolescents in low- and middle-income countries (LMICs) such as India. Recent evidence indicates that both the prevalence and the health risks associated with overweight and obesity are increasing among Indian adolescents. A nationwide nutritional survey reported that more than 5% of adolescents are living with overweight or obesity.[3]

The burden of obesity in India has been underscored by findings from the Indian Council of Medical Research (ICMR)-India Diabetes (INDIAB) study, which reported that an estimated 254 million adults have generalized obesity, while 351 million adults exhibit abdominal obesity.[5]

Obesity and its impact on women's reproductive and general health

Obesity poses unique health risks for women across the reproductive lifespan, from menarche through menopause.[6] Overweight and obese young girls have an increased risk of developing polycystic ovary syndrome (PCOS) along with associated metabolic and reproductive health complications.[7] Obesity adversely affects fertility, pregnancy outcomes, and long-term maternal and child health.[8]

Adolescence

During adolescence, reproductive health is closely associated with metabolic homeostasis. Obesity in adolescence disrupts this equilibrium through multiple pathways, including hormonal dysregulation, chronic low-grade inflammation, and alterations in energy metabolism. These changes impair ovulation, reduce oocyte quality, and compromise endometrial receptivity, thereby impairing overall fertility.[7] Obesity in adolescence increases the risk of numerous chronic conditions later in life. These include hypertension, dyslipidemia, cardiovascular disease (CVD), fatty liver disease, type 2 diabetes mellitus (T2DM), and endocrine abnormalities such as insulin resistance. Additionally, obesity in adolescence is linked to hypogonadism in boys, as well as a higher risk of gallstone disease, stroke, and other long-term complications.[3]

Reproductive age

In women of reproductive age, obesity adversely affects reproductive function through hormonal and metabolic disturbances, leading to impaired

ovulation and reduced endometrial receptivity. It is also associated with prolonged time to conception and poorer outcomes with assisted reproductive technologies.[8]

Perimenopause

During the perimenopausal transition, hormonal changes together with reduced energy expenditure promote weight gain, characterized by increased abdominal/visceral adiposity and loss of lean body mass. These body composition changes are accompanied by adverse metabolic alterations and an increased risk of cardiometabolic disorders.[9]

Menopause

In menopausal women, the menopausal transition is closely associated with weight gain, driven by declining estrogen levels and a redistribution of fat from subcutaneous to visceral depots.[10]

Post-menopause

Post-menopausal women with obesity have an increased risk of cardiovascular disease and metabolic syndrome.[10]

Thus, there is a critical need for weight management strategies to optimize health outcomes. Structured lifestyle interventions, including targeted dietary modifications, regular physical activity, behavioral counselling, and pharmacotherapy should be the essential components of the care programs.[8]

Scope and methodology

These recommendations have been developed by a dedicated task force to provide healthcare professionals with evidence-based guidance for the evaluation and management of obesity. They are intended for use by gynecologists, reproductive health specialists, consulting physicians, and primary care physicians in routine outpatient clinical settings. This update enhances the clarity, credibility, and practical applicability of the guidance provided.

Based on a comprehensive review of current evidence and clinical guidelines addressing the diagnosis and management of obesity, recommendations were developed. The clinicians involved convened to review the available evidence, deliberate on key issues, and curate the document through expert consensus. The task force employed a well-defined grading system (Table 1) for critical appraisal of evidence and for grading the strength of recommendations.

Table 1. Grading system based on level of evidence and class of recommendations

Level of evidence	Description
Level A	Data from multiple randomized trials, meta-analyses, or evidence-based clinical practice guidelines
Level B	Data from a single randomized trial or a large non-randomized trial
Level C	Expert consensus of small studies, retrospective studies or registries, or narrative/literature reviews
Level D	Data from clinical experience
Class of recommendations	
Class I	Strong evidence and/or general agreement that the treatment or procedure is beneficial, useful or effective. It is recommended
Class IIa	Evidence is in favor of efficacy/usefulness and should be considered
Class IIb	Efficacy/usefulness is less well established, and recommendations may be considered
Class III	Evidence and/or general agreement that an intervention is not beneficial, useful or effective, and in some cases may cause harm. Not recommended

Assessment of obesity

RECOMMENDATIONS

- BMI should be the primary screening tool for obesity. To enhance risk stratification in Indian women, Asian-specific cutoffs (overweight: 23–24.9 kg/m^2; obesity: ≥25 kg/m^2) can be considered to be applied. (Level A/ Class I)
- Adults diagnosed with obesity can undergo baseline cardiometabolic evaluation, including fasting glucose, postprandial glucose, HbA1c, lipid profile, liver enzymes, HOMA-IR, apolipoprotein, insulin resistance, and renal function tests, to enable early detection of associated metabolic comorbidities. (Level A/ Class I)
- Hormonal testing can be considered when clinical features suggest potential reproductive dysfunction (hypercortisolism, PCOS, menstrual irregularities). (Level A/ Class IIa)

Discussion

In clinical practice, key anthropometric measurements for women with obesity include height, weight, BMI, waist circumference, and waist-to-hip ratio.[8] BMI remains the most widely used screening tool for identifying obesity and is strongly correlated with cardiometabolic disease risk.[11,12] However, diagnostic cutoffs for overweight and obesity vary across racial and ethnic groups, necessitating region-specific criteria.[12,13] Among individuals of Asian descent, separate BMI cut-offs are used to define overweight and obesity (Table 2).[12] A high BMI is a well-established risk factor for several non-communicable diseases, including CVD, type 2 diabetes, musculoskeletal disorders, and multiple malignancies.[11]

Table 2. BMI cutoffs according to WHO classification and Asian-specific criteria[12,13,14]

BMI Category	WHO (kg/m^2)	Asian Population (kg/m^2)
Underweight	<18.5	<18.5 kg
Normal	18.50–24.99	18.5–22.9
Overweight	≥ 25	23–24.9
Obese	≥ 30	≥25

The Obesity Medicine Association (OMA) recommends a structured, multifactorial evaluation encompassing clinical history, physical examination, and relevant investigations. This includes assessment of vital signs (blood pressure and pulse rate) and a comprehensive anthropometric evaluation, including parameters such as waist-to-hip ratio and neck circumference.[15] Laboratory assessment should include both general and obesity-specific investigations.

Recommended baseline investigations include:[15]

- General tests: CBC, urinalysis, uric acid, urine protein/albumin-creatinine ratio
- Metabolic profile: Fasting glucose, HbA1c, lipid profile, apolipoprotein, and homeostatic model assessment for insulin resistance (HOMA-IR)
- Organ function: Liver function tests, renal function tests
- Endocrine markers: Thyroid-stimulating hormone (TSH)

Individualized testing may include assessment for insulin resistance, hypercortisolism, oligomenorrhea or amenorrhea, and hyperandrogenemia. Additional targeted assessments in patients with overweight/obesity may include imaging of the pituitary, like magnetic-resonance imaging (MRI) or computed tomography (CT), resting electrocardiogram, cardiac stress testing, echocardiography, coronary calcium scoring, ankle–brachial index, sleep studies, and liver imaging to assess for hepatic steatosis.[15]

Furthermore, the Endocrine Society of India (ESI) and OMA suggest that patients with obesity should be evaluated clinically and biochemically for associated comorbidities.[15,16] Assessment should include key metabolic conditions such as type 2 diabetes mellitus, hypertension, dyslipidemia, hyperuricemia, non-alcoholic fatty liver disease, cholelithiasis, and PCOS.

Intensive behavioral interventions for managing obesity

RECOMMENDATIONS

- Behavior-based clinical interventions may be considered as a foundational component of a comprehensive multimodal therapeutic approach, to help amplify and sustain the therapeutic benefits of pharmacotherapy. (Level A/ Class I)
- Clinicians may consider educating patients about biological mechanisms such as elevated ghrelin during caloric restriction, leptin resistance, and metabolic adaptation, which can contribute to resistance to weight loss in women. (Level C/ Class IIa)

Discussion

Behavior-based clinical interventions for obesity management are based on structured lifestyle modification that integrates dietary intervention, physical activity, and behavioral strategies to support weight loss.[17]

Dietary modification is central to weight loss, as reduction in body weight requires the establishment of a negative energy balance through caloric restriction tailored to individual needs.[17,18]

Low-energy diets in women typically prescribe a consistent caloric deficit, with commonly

recommended daily calorie targets of approximately 1200–1500 kcal/day, adjusted according to baseline body weight and activity levels to promote sustainable weight reduction.[17]

Different dietary patterns, including low-fat, low-carbohydrate, Mediterranean-style, and balanced macronutrient diets, can result in comparable weight loss outcomes when adherence to calorie reduction is maintained over time.[17,18]

Physical activity complements dietary intervention by improving cardiometabolic health and supporting long-term weight maintenance, even though exercise alone produces minimal short-term weight loss in the absence of calorie restriction.[17,19]

Behavioral strategies such as goal setting, self-monitoring of dietary intake and physical activity, and structured counselling are core components of lifestyle intervention that support adherence and help sustain clinically meaningful weight loss.[17]

Table 3. Physiological barriers to behavioral weight loss interventions

Metabolic adaptation during weight loss	Behavioral changes like eating low-fat foods and exercising can trigger early weight loss, but the body eventually adapts and reaches a new plateau. To maintain and further reduce weight, these strategies must be sustained and intensified.[20]
Elevated Ghrelin / Leptin Resistance	Ghrelin increases during fasting and drops within an hour of eating. Its levels rise after weight loss. Exercise does not significantly alter appetite hormones in obese adults compared with lean individuals, and may reduce fullness perception, leading to compensatory eating (hedonic activity) offsetting weight loss.[21,22] Obese individuals often have high circulating leptin levels due to leptin resistance, where the brain becomes less responsive to leptin's appetite-suppressing effects. This reduced sensitivity promotes increased food intake and further weight gain. Physical activity programs have only a limited impact on lowering leptin levels.[22,23]
Hormonal changes promote visceral adiposity in perimenopausal/ menopausal women	Menopause-related physiologic and metabolic changes arise from estrogen deficiency, which affects lipid metabolism, energy use, insulin resistance, and body fat distribution. Reduced estrogen promotes abdominal and visceral fat accumulation.[24]

Behavioral interventions alone for weight loss: An unmet need

Although several guidelines support the benefits of behavioral interventions, physiology (metabolic adaptation, appetite hormones, menopause biology, and insulin resistance) often limits results (Table 3).

Therefore, although behavioral interventions offer potential benefits, they generally result in temporary changes that are difficult to sustain and often fail to achieve the modest 5–10% weight loss required to reduce comorbidities and improve quality of life.[25]

Pharmacotherapy-based weight loss and weight maintenance interventions

RECOMMENDATIONS

- Clinicians should recognize obesity as a chronic, relapsing and remitting disease requiring long-term, multimodal management. Anti-obesity medications (AOMs) may be initiated as part of a chronic care model rather than short-term, episodic treatment. (Level C/ Class I)
- Pharmacotherapy may be considered as an adjunct to lifestyle intervention in adults with BMI ≥30 kg/m^2, or BMI 27–30 kg/m^2 with at least one obesity-related comorbidity. In reproductive-aged women, AOMs may also be considered when BMI ≥27 kg/m^2, after assessing individual metabolic, reproductive, and fertility considerations. (Level A/ Class I)
- When lifestyle therapy alone yields inadequate weight reduction, long-term AOMs may be considered, based on efficacy, safety, comorbidities, and patient-centered goals. (Level A/ Class I)

Discussion

Multiple scientific organizations, including the American Medical Association and the National Institutes of Health, have emphasized that obesity requires a long-term management approach, which may include the use of anti-obesity medications (AOMs).[22] The emergence of newer, highly effective

agents has positioned pharmacologic therapy at the forefront of obesity management, with increasing patient demand for physician guidance regarding these treatments.[26]

As per the Endocrine Society of India (ESI) 2025 update, adults with obesity, particularly those with BMI ≥30 kg/m² or BMI 27–30 kg/m² with obesity-related comorbidities such as diabetes mellitus, hypertension, dyslipidemia, coronary artery disease, or stroke, may benefit from the addition of pharmacotherapy when lifestyle measures alone do not achieve adequate weight reduction.[27,28] Appropriate prevention and treatment strategies can significantly reduce morbidity and mortality and improve overall quality of life. As per Central Drugs Standard Control Organisation (CDSCO), pharmacological agents used in clinical practice include orlistat for weight management and incretin-based therapies such as Semaglutide and Tirzepatide for glycemic control in individuals with type 2 diabetes mellitus.[29] Beyond weight reduction, these agents have demonstrated benefits across multiple health parameters, including blood pressure, lipid profiles, and glycemic control.[26]

New frontiers in obesity management: The role of GLP-1 receptor agonists

RECOMMENDATIONS

- GLP-1 receptor agonists may be considered as pharmacologic therapy for adults with obesity who do not achieve adequate weight loss with lifestyle interventions alone. (Level A/Class I)
- Pharmacological therapy may be considered using approved agents such as orlistat for weight management and GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists for approved indications, based on individual clinical profile. (Level A/Class I)
- Clinicians should evaluate metabolic health, glycaemic status, gastrointestinal tolerance, and comorbidities prior to GLP-1 receptor agonist initiation and monitor response thereafter. (Level A/Class I)

Discussion

Given that obesity contributes to a wide range of metabolic and organ-specific complications including type 2 diabetes, CVD and liver disease, there is a growing emphasis on therapies that target both excess weight and the associated metabolic burden.[30]

In this context, the ICMR describes obesity as a metabolic disorder resulting from chronic energy imbalance and recognizes its association with cardiometabolic complications, underscoring the chronic nature of the condition.[31] Indian consensus guidelines further highlight that Indians develop metabolic and cardiovascular complications at lower body mass index and waist circumference thresholds, with abdominal obesity and insulin resistance being highly prevalent, thereby warranting earlier identification and intervention.[32] The Obesity and Lifestyle Modification guidelines highlight that obesity is associated with multiple systemic comorbidities, including cardiovascular, metabolic, respiratory, musculoskeletal, and psychological disorders, underscoring its chronic, multifactorial nature and the need for sustained, long-term management.[33]

In clinical practice, pharmacological management of obesity extends beyond weight reduction alone and includes agents approved for metabolic disease management. Orlistat is approved for obesity management, including weight loss and weight maintenance in individuals with a BMI ≥30 kg/m² or ≥27 kg/m² with associated risk factors.[34] In addition, Glucagon-like peptide (GLP)-1 receptor agonists such as exenatide, liraglutide and Semaglutide, and the dual GIP/GLP-1 receptor agonist Tirzepatide, are approved for the management of type 2 diabetes mellitus and are used in clinical practice among individuals with obesity and related metabolic comorbidities.[35] The ESI guideline (2025 update) suggests considering the use of both Tirzepatide and injectable Semaglutide, in obese individuals with at least one associated comorbid condition, particularly when a weight loss of 10–15% is targeted.[28]

GLP-1 and dual GIP/GLP-1 receptor agonists in obesity treatment

Glucagon-like peptide (GLP)-1 receptor agonists have emerged as promising agents for obesity management.[36] Among available anti-obesity medications, GLP-1 receptor agonists achieve the largest and most consistent weight loss with additional cardiometabolic benefits demonstrated across clinical trials.[37,36] This class includes liraglutide, dulaglutide and Semaglutide, as well as the dual GIP/GLP-1 receptor co-agonist Tirzepatide, which are used in the management of obesity.[37] These drugs act on physiologic pathways that regulate appetite, satiety, and energy intake.[36] Among these agents, Semaglutide has been evaluated in multiple clinical studies, with regulatory approvals obtained between 2017 and 2020,[38] compared to Tirzepatide which has received regulatory approval in 2022. Therefore, the availability of obesity-specific data is comparatively more with Semaglutide.[30,37,39]

GLP-1 receptor agonists differ in their dosing regimens and titration requirements across agents; therefore, dosing and up-titration should be followed as per the specific drug's approved product information for precise dosing instructions.[40]

Semaglutide is widely studied for its metabolic and appetite-regulating effects and reduces hunger, enhances fullness, and lowers overall energy consumption through coordinated central and peripheral actions. These include modulation of hypothalamic appetite pathways, delayed gastric emptying, and glucose-dependent regulation of insulin and glucagon secretion, supporting sustained weight management.[37,38]

The American Gastroenterological Association (AGA) supports the use of Semaglutide 2.4 mg for adults who do not achieve adequate weight loss with lifestyle intervention alone. Semaglutide 2.4 mg may be prioritized over other approved AOMs for the long-term treatment of obesity for most patients. Furthermore, Semaglutide provides glucoregulatory benefits and is also approved for the treatment of T2DM. Since Semaglutide may delay gastric emptying with adverse effects of nausea and vomiting, the AGA recommends gradual dose titration to mitigate these adverse effects.[41]

In obesity management, Semaglutide is administered as a once-weekly subcutaneous injection, initiated at a low dose and gradually escalated to a maintenance dose of 2.4 mg based on clinical response and tolerability.[42]

Exploratory studies suggest broader metabolic effects through modulation of oxidative stress, inflammation, mitochondrial biogenesis, and adipose-tissue signaling in preclinical models.[33] Incretin-based therapies also differ in receptor specificity; for example, Tirzepatide activates both GIP and GLP-1 receptors, whereas Semaglutide acts selectively on the GLP-1 receptor, converging on shared pathways that regulate appetite and metabolic homeostasis.[43]

Safety, contraindications, and precautions of GLP-1 receptor agonists

GLP-1 receptor agonists are contraindicated in women with a personal or family history of medullary thyroid carcinoma or multiple endocrine neoplasia type 2 and should not be used in those with prior hypersensitivity to these agents. GLP-1 receptor agonists should be used carefully in women with a history of pancreatitis, gallbladder disease, or gastrointestinal motility disorders such as gastroparesis. Baseline evaluation of glycemic status, renal function, and diabetic retinopathy (in women with diabetes) is recommended before initiation.[44]

Worsening of diabetic retinopathy has been observed predominantly in patients with pre-existing retinopathy and is considered to be related to the magnitude and rapidity of glycemic improvement, rather than a direct drug-specific effect.[46]

During GLP-1 receptor agonist therapy, patients should be counselled to ensure adequate protein intake and to incorporate resistance or strength training to help preserve lean body mass during weight loss. Patients should also be counselled regarding gastrointestinal adverse effects and practical dietary measures to help mitigate them.[40]

As adverse event profiles, contraindications, and dose interruption or missed-dose management

strategies vary across individual anti-obesity medications, clinicians should refer to the individual drug's approved product information for detailed safety and dosing guidance.[40]

For women planning pregnancy, a washout period of about two months is recommended before conception, as described for injectable Semaglutide. Women of reproductive age receiving GLP-1 receptor agonists should be advised to use effective contraception during treatment and to discontinue therapy well in advance of a planned pregnancy.[44]

Pregnancy and lactation considerations

GLP-1 receptor agonists should be discontinued prior to a planned pregnancy, as recommended in clinical guidance.[46] Preclinical studies have reported adverse effects on fetal growth and development following exposure to GLP-1 receptor agonists.[46] Available evidence indicates that incretin-based anti-obesity medications, including GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists, are contraindicated during pregnancy and are best avoided in women who are pregnant or planning pregnancy.[47] Use of GLP-1 receptor agonists during lactation should also be avoided due to limited human safety data.[46,47]

Comparative studies and clinical evidence of GLP-1-based therapies in obesity

Key safety outcomes and comparative tolerability of GLP-1–based therapies are summarized in Table 4.

Across the STEP clinical trial program, including STEP 2, STEP-4, STEP 5 (104-week phase 3 trial), and STEP-UP, once-weekly Semaglutide 2.4 mg consistently produced clinically meaningful mean weight loss in the range of approximately 15–18%, with greater reductions of approximately 19–21% observed with the 7.2 mg dose in STEP-UP.[50,51,53,54]

The FDA recommends that health providers monitor patients receiving Tirzepatide with kidney disease, diabetic retinopathy and depression or suicidal behaviors or thoughts.[59] Semaglutide is currently the more established therapy with robust long-term data in obesity management.[58]

Table 4. Safety outcomes of GLP-1 and dual GIP/GLP-1 receptor agonists in clinical studies

Study	Intervention and Population	Results	Outcome
Systematic review and meta-analysis (11 RCTs)[48]	1328 non-diabetic overweight or obese adults treated with liraglutide 3.0 mg once daily subcutaneously versus placebo.	Compared with placebo, use of liraglutide led to an overall mean difference of −4.59 kg in body weight, -3.22 cm in waist circumference, and −1.71 kg/m^2 in BMI; all being statistically significant (p<0.00001).	Liraglutide 3.0 mg results in a greater reductions in body weight, waist circumference, and BMI versus placebo in non-diabetic overweight or obese adults.
STEP-1 (Randomized, double-blind, placebo-controlled)[49]	Adults with overweight or obesity without diabetes (n=1961) were randomized in a 2:1 ratio to receive once-weekly subcutaneous Semaglutide 2.4 mg (n=1306) or placebo (n=655), both in combination with lifestyle intervention, for 68 weeks.	At week 68, mean body weight decreased by 14.9% with Semaglutide compared with 2.4% with placebo (p<0.001). A higher proportion of patients in the Semaglutide group achieved clinically meaningful weight loss compared with placebo, including ≥5% weight loss (86.4% vs. 31.5%), ≥10% (69.1% vs. 12.0%), and ≥15% (50.5% vs. 4.9%) (all p<0.001).	Once-weekly Semaglutide 2.4 mg in combination with lifestyle intervention resulted in substantial and sustained clinically meaningful weight loss compared with lifestyle intervention alone.
STEP-2 (Phase 3, randomized, double-blind, placebo-controlled trial)[50]	T2DM overweight/obese adults (n=1210) received once-weekly subcutaneous Semaglutide 2.4 mg (n=404), Semaglutide 1.0 mg (n=403), or placebo (n=403) for 68 weeks.	From baseline to week 68, Semaglutide resulted in significantly greater mean body weight reduction vs. placebo (−9.6% with 2.4 mg and −7.0% with 1.0 mg vs. −3.4% with placebo; p<0.0001).	Semaglutide 2.4 mg led to significantly greater reduction in weight than placebo over 68 weeks in T2DM overweight/obese adults.
STEP-4 randomized, double-blind, placebo-controlled withdrawal trial[51]	Adults with overweight or obesity who completed a 20-week run-in with once-weekly subcutaneous Semaglutide 2.4 mg were randomized to continue Semaglutide 2.4 mg (n=535) or switch to placebo (n=268) for 48 additional weeks.	From week 20 to week 68, mean weight change was −7.9% with continued Semaglutide 2.4 mg and +6.9% with placebo; this difference was statistically significant (p<0.001).	Continuing Semaglutide maintains weight loss, while discontinuation results in weight regain

Study	Intervention and Population	Results	Outcome
STEP-5 (Phase 3, randomized, double-blind, placebo-controlled trial)[52]	Non diabetic overweight or obese adults (n=304) were randomized to once-weekly subcutaneous Semaglutide 2.4 mg (n=152) or placebo (n=152) for 104 weeks.	At week 104, mean body weight change from baseline was −15.2% with Semaglutide 2.4 mg and −2.6% with placebo; this difference was statistically significant (p<0.0001). The proportions of patients achieving ≥10%, ≥15%, and ≥20% weight loss were significantly higher with Semaglutide than with placebo: 61.8% vs. 13.3%, 52.1% vs. 7.0%, and 36.1% vs. 2.3%, respectively.	Semaglutide demonstrated substantial and sustained weight loss over 104 weeks compared with placebo, with a high proportion of patients achieving (≥10%, ≥15%, and ≥20%) weight reduction and associated improvements in cardiometabolic risk markers.
A large, propensity matched, cohort study[53]	Overweight or obese adults (n=18,386) treated with once-weekly subcutaneous Semaglutide (target dose 0.5 mg) or once-weekly subcutaneous Tirzepatide (target dose 5 mg) with on-treatment follow-up of up to 12 months.	The mean on-treatment change in body weight was −5.9% vs. −3.6% at 3 months, −10.1% vs. −5.8% at 6 months, and −15.3% vs. −8.3% at 12 months with Tirzepatide and Semaglutide, respectively.	Both Semaglutide and Tirzepatide have demonstrated weight reduction with differences in magnitude varying across study designs and populations.
STEP-UP (Phase 3b, randomized, double-blind, placebo- and active-controlled trial)[54]	Adults with obesity (BMI ≥30 kg/m^2) without diabetes, randomized to once-weekly subcutaneous Semaglutide 7.2 mg, Semaglutide 2.4 mg, or placebo, in addition to lifestyle intervention, for 72 weeks.	At week 72, mean body weight change was −18.7% with Semaglutide 7.2 mg, −15.6% with Semaglutide 2.4 mg, and −3.9% with placebo (p<0.0001).	Semaglutide 7.2 mg led to greater reduction in body weight than Semaglutide 2.4 mg and placebo over 72 weeks in adults with obesity.
STEP 3 (Randomized, double-blind, placebo-controlled phase 3 trial)[55]	Adults with overweight or obesity without diabetes (n−611) were randomized to receive once-weekly subcutaneous Semaglutide 2.4 mg (n−407) or placebo (n−204), both in combination with intensive behavioral therapy and an initial low-calorie diet, for 68 weeks.	At week 68, mean body weight decreased by 16.0% with Semaglutide versus 5.7% with placebo, and the difference was statistically significant (p<0.001). A significantly greater proportion of patients receiving Semaglutide achieved ≥5%, ≥10%, and ≥15% weight loss (86.6% vs. 47.6%, 75.3% vs. 27.0%, and 55.8% vs. 13.2%, respectively) compared with placebo (p<0.001).	Semaglutide 2.4 mg, when combined with intensive behavioral therapy and a low-calorie diet, produced significantly greater and clinically meaningful weight loss than placebo.
Systematic review and meta-analysis (14 RCTs)[56]	Adults with obesity without diabetes (n=10,638) received semaglutide (n=2,581), liraglutide (n=3,147), tirzepatide (n=630), exenatide (n114), or orforglipron (n=53), or placebo (n=4,113).	GLP-1 receptor agonists produced significant weight loss (MD −8.77 kg; p<0.01) and also significantly improved systolic and diastolic blood pressure and lipid parameters (triglycerides, total cholesterol, VLDL, LDL), with a significant increase in HDL (p<0.01).	GLP-1 receptor agonists reduce body weight and improve blood pressure and lipid parameters in adults with obesity.
Real-world retrospective cohort[57]	Women (n=1882) with overweight or obesity.	With Semaglutide, the median BMI decreased by 3.18 kg/m^2 and the mean body weight decreased by 10.73% from baseline. The proportions of patients achieving ≥5%, ≥10%, and ≥15% weight loss were 82.36%, 52.07%, and 23.22%, respectively.	These findings supported the use of Semaglutide for achieving clinically meaningful reductions in body weight and BMI in women with overweight or obesity.
Real-world data analysis[58]	Adults (2771) with obesity treated with Semaglutide.	The average change from baseline were reductions of 1.54 kg/m^2 in BMI, 4.65 kg in body weight, and 0.75% in HbA1c, following treatment with Semaglutide.	Semaglutide was associated with greater reductions in BMI, body weight, and HbA1c in adults with obesity and/or T2DM.

RCT: Randomised controlled trial, T2DM: Type 2 diabetes mellitus, VLDL: Very low-density lipoprotein, LDL: Low density lipoprotein, HDL: High density lipoprotein, MD: Mean difference

An evidence-based lifecycle approach to obesity management in women

Managing obesity in adolescents (≥12 years)

RECOMMENDATIONS

- When initiating AOMs in adolescents, preference may be given to agents with demonstrated benefits in BMI reduction and cardiometabolic risk improvement, and with acceptable safety profiles. (Level A/ Class I)
- In adolescents aged 12–18 years with BMI ≥95th percentile or ≥85th percentile with at least one obesity-related comorbidity, adjunctive pharmacotherapy such as once-weekly Semaglutide 2.4 mg subcutaneous injection may be considered when lifestyle interventions alone are insufficient. (Level A/ Class I)
- Semaglutide, administered using an approved dose escalation approach, may be considered for long-term obesity management in adolescents. (Level A/ Class I)

Discussion

Adolescent obesity is rising rapidly and posing serious metabolic, physiological, and psychological risks. Early identification using growth charts and a multidisciplinary, individualized approach to care is essential. Effective tools for screening, prevention, diagnosis, and treatment support better outcomes.[60]

According to the Indian Academy of Pediatrics guidelines, pharmacotherapy may be considered in adolescents aged ≥12 years only after failure of structured lifestyle interventions or in the presence of obesity-related comorbidities.[61]

Additionally, weight-modifying medications may help reduce hunger, cravings, and improve regulation of eating behaviors. This can be particularly helpful for adolescents with special healthcare needs (SHCN), who often experience increased hunger or reduced satiety. GLP-1 receptor agonists are commonly used pharmacologic options in this population.[62]

The STEP-TEENs phase 3a trial (n=201) showed that once-weekly subcutaneous Semaglutide 2.4 mg in adolescents aged 12–18 years, alongside lifestyle intervention resulted in greater decrease in absolute BMI, in BMI as a percentage of the 95th percentile, in absolute body weight, and in percentage body weight at week 68 compared with placebo. At week 68, 76%, 62%, 53%, and 37% of adolescents receiving Semaglutide demonstrated a ≥5%, ≥10%, ≥15%, and ≥20% reduction in BMI, compared with 23%, 8%, 5%, and 3% in the placebo group, respectively. Over the trial period, treatment was generally well tolerated, with mostly mild gastrointestinal adverse effects, and no adverse effects on growth or pubertal development.[63]

Semaglutide exerts cardiometabolic benefits, including reductions in waist circumference, glycated haemoglobin, alanine aminotransferase, and unfavourable lipid parameters (total cholesterol, LDL-C, VLDL, and triglycerides). Once-weekly subcutaneous Semaglutide 2.4 mg, combined with lifestyle changes, significantly reduced BMI in adolescents with obesity and improved cardiometabolic markers. It is generally well tolerated, with mild gastrointestinal side effects and no impact on growth or puberty.[63,64]

Managing obesity in reproductive aged women

Preconception and pregnancy

RECOMMENDATIONS

- Women planning pregnancy should ideally avoid AOMs and rely solely on lifestyle interventions. GLP-1 RAs including Semaglutide may be discontinued prior to conception, with a minimum washout of 2 months when clinically appropriate. (Level A/ Class I)
- For women who conceive accidentally while receiving GLP-1 receptor agonists, routine obstetric care with appropriate monitoring maybe considered. (Level C/ Class I)

Discussion

Across the STEP clinical trial program evaluating Semaglutide for obesity, the pivotal trials consistently

enrolled a predominantly female population. For example, women comprised 81.0% of participants in STEP 3, 79% in STEP 4, and 77.6% in STEP 5, supporting the relevance of the Semaglutide evidence base for obesity management in women.[55,51,52]

Impact of obesity on fertility

- Excess adiposity contributes to anovulation through a complex endocrine milieu characterized by insulin resistance, compensatory hyperinsulinemia, low sex hormone binding globulin, elevated androgens, increased aromatization, higher free insulin-like growth factor -1, and elevated leptin. These alterations disrupt hypothalamic–pituitary signalling, impair folliculogenesis, reduce luteal progesterone, and lead to menstrual irregularities that worsen with increasing BMI. Obesity also reduces natural fertility even in ovulatory women, prolonging the time to conception.[65]
- Obesity has also been linked with reduced sexual desire, sexual dysfunction (including erectile dysfunction in male partners), and decreased frequency of intercourse, potentially confounding fertility outcomes.[65]
- Obesity may exert adverse effects on endometrial receptivity. Altered endometrial gene expression has been observed during the implantation window in natural cycles among obese women, and reduced implantation and clinical pregnancy rates have been reported in obese donor-oocyte recipients.[65]
- Obesity induces hormonal alterations that significantly affect endometrial function, impair embryo implantation, and promote abnormal endometrial proliferation, potentially progressing to endometrial hyperplasia.[66]

Obesity and PCOS are prevalent metabolic disorders associated with subfertility. Metabolic disorders can impair endometrial receptivity and embryo implantation, contributing to increased miscarriage rates. Since obesity affects reproductive function at both ovarian and endometrial levels, comprehensive reproductive counselling is recommended for couples experiencing fertility challenges related to excess weight. In particular, young women with obesity should be strongly encouraged to pursue weight reduction, especially when planning or undergoing assisted conception.[67]

A recently published 2026 systematic review evaluating semaglutide exposure and fetal–neonatal outcomes found that, across included studies, there was no significant increase in the risk of major congenital malformations associated with exposure to semaglutide within two months prior to conception or during pregnancy.[68]

However, due to limited human data, GLP-1 receptor agonists are advised be stopped at least 2 months before planned conception and discontinued if pregnancy occurs.[69]

In studies where short-acting GLP-1 RAs were discontinued and insulin initiated within 15 weeks of exposure, no fetal malformations, comorbidities, or maternal complications were reported, and neonatal weights were appropriate for gestational age. In Semaglutide registration trials (SUSTAIN 1–6 and STEP 1–5 and 8), pregnancies were exposed briefly until detection; all Semaglutide-exposed women delivered healthy infants, with no reported pregnancy loss or confirmed teratogenic effects.[70]

Since the body takes a longer time to metabolize Semaglutide, approximately 6 weeks in healthy non-pregnant women, product labelling (PL) mentions discontinuing the drug when pregnancy is confirmed.[71,72] As per a 2025 real world cohort and Semaglutide PL, while no increase in major congenital malformations were observed, higher rates of preterm birth, large for gestational age infants, neonatal hypoglycemia, and jaundice have been reported. Hence, it has been suggested that Semaglutide be discontinued at least 2 months prior to the planned conception.[73]

Obesity and infertility: Impact and management

RECOMMENDATIONS

- In women presenting with infertility, clinicians may consider evaluating the reproductive impact of excess adiposity. Central adiposity may also be assessed, as it is strongly correlated with anovulation independent of BMI. (Level A/ Class I)
- In women with obesity, including those undergoing ART, structured counselling emphasizing weight reduction may be considered to improve ovulatory function and oocyte quality, endometrial receptivity, implantation rates, and pregnancy outcomes. (Level B/ Class IIa)
- Clinicians should inform patients that higher BMI is associated with reduced ovarian response to ovulation induction and ART, including increased gonadotropin requirements, longer stimulation duration, lower follicular yield, and higher risk of cycle cancellation. (Level A/ Class I)
- In women with obesity seeking fertility treatment, weight reduction before ovulation induction or IVF may be considered. Structured weight-management interventions may also be considered early in fertility care pathways. (Level B/ Class I)

Discussion

Impact of obesity on fertility treatments

Overweight and obese women show reduced responsiveness to ovulation induction, need higher gonadotropin doses and longer treatment duration, and produce fewer oocytes, leading to more frequent cycle cancellations.[74]

Altered responsiveness to ovarian stimulation

Reports have shown that in normogonadotropic anovulatory women, higher BMI and abdominal obesity reduce ovulatory response to clomiphene citrate.[75] A prospective cohort study from North India showed Grade II obesity independently predicted poor response to letrozole ovulation induction in PCOS women.[76] Obesity is linked to higher doses of medications to induce ovulation or stimulate the ovaries for IVF.[77] Several large retrospective ART datasets (1,721–8,145 women) confirm that obesity impairs ovarian responsiveness to gonadotropin stimulation (increased duration, amount of gonadotropin administered, increased cycle cancellation; fewer oocytes retrieved).[75]

The American Society of Anesthesiologists (2024) recommends stopping long-acting GLP-1 and/or GIP receptor agonists 7 days before any procedure, regardless of the indication, type of procedure, or planned anesthesia (including both intravenous sedation and general anesthesia). This guideline is based on concerns about delayed gastric emptying and the associated increased risk of aspiration.[70]

Impaired oocyte quality

Table 5. Impact of obesity on reproduction[68]

Condition	Associated risks
Menstruation and menorrhagia	Menstrual dysfunction: amenorrhea, oligomenorrhea
Functional changes in the Hypothalamo-Pituitary-Ovarian (HPO) axis	Lower natural pregnancy rates. Exogenous gonadotropins indicated to counter the risk
Lower adiponectin serum levels and reduced adiponectin synthesis	Possible negative effect on ovulation control, despite still inconclusive data; obesity-related ovarian dysfunction.
Impaired stromal decidualization in obese women	Placental abnormalities, stillbirth and preeclampsia.
Infertility treatment	• Poor response to induction of ovulation, requiring higher doses of gonadotropins and longer treatment and ovulatory cycles for follicular development. • Lower oocyte yield and a higher rate of cycle failure. • Ovarian stimulation producing fewer follicles, hence fewer harvested oocytes. • Low embryo quality and poor fertilization rates.
Miscarriage	The risk of miscarriage in obese women vs. normal weight patients

Women with obesity undergoing IVF have oocytes that are generally smaller than those of normal-weight women. While fertilization rates show inconsistent associations with BMI, blastulation and embryo metabolism appear adversely affected.[75] Evidence from 239,127 IVF cycles in the Society for Assisted Reproductive Technology (SART) registry shows that

the number of oocytes retrieved, embryo quality, implantation, clinical pregnancy, and live birth rates all decline as BMI exceeds 40.[78] The negative impact of obesity on reproduction is summarized in Table 5.

Management of obese PCOS women

RECOMMENDATIONS

- In women with PCOS and obesity, GLP-1 receptor agonists may be considered in the preconception period to improve insulin sensitivity, reduce metabolic inflammation, normalize reproductive hormones, and enhance ovulation and implantation potential, particularly when lifestyle modification alone is insufficient. (Level A/ Class I)
- In women with PCOS and obesity undergoing ovarian stimulation, oocyte retrieval, or IVF, GLP-1 receptor agonist appear safe and do not increase anesthesia or procedural complications; however, long-acting GLP-1 receptor agonists can be discontinued 7 days before any procedure requiring sedation or general anesthesia to reduce aspiration risk. (Level A/ Class I)
- For women with PCOS and obesity receiving fertility treatment, GLP-1 receptor agonist therapy can be considered to be integrated thoughtfully within fertility pathways, ensuring appropriate washout timing when transitioning to conception-focused treatment. (Level B/ Class I)
- For women with obesity and PCOS, a 5–15% weight loss may provide substantial benefit; semaglutide may be considered in eligible patients to achieve this target. (Level A/Class 1)

Discussion

In obese women with PCOS, a weight reduction of 5–10% has been shown to improve insulin resistance and reduce androgen excess, leading to improvements in menstrual cyclicity, ovulatory function, and fertility outcomes.[79] The use of GLP-1 receptor agonists in the preconception period offers benefits such as improved insulin sensitivity and better control of BMI-related metabolic factors, including blood pressure and inflammatory markers.[64] GLP-1 receptor agonists are a promising therapeutic option for obese women with PCOS. Beyond weight loss, they target mechanisms underlying insulin resistance. GLP-1 receptor agonists may enhance fertility through two pathways: (1) reducing hypothalamic–pituitary suppression driven by obesity-related estrogen excess, restoring luteinizing hormone (LH) levels; and (2) decreasing elevated LH levels associated with hyperinsulinemia and obesity.[80]

In the American Association of Clinical Endocrinology (AACE) 2025 consensus, PCOS is listed among obesity-associated complications for which clinically meaningful benefit is typically observed with approximately 5–15% weight loss. The same consensus notes that second-generation anti-obesity medications such as Semaglutide achieve, on average, ≥15% weight loss in phase III trials.[40]

The 2023 International Evidence-Based Guideline published by American Society of Reproductive Medicine (ASRM) for the Assessment and Management of Polycystic Ovary Syndrome also emphasize lifestyle intervention and weight management as core components of care, within a broader framework of long-term management addressing metabolic, cardiovascular, reproductive, and psychological risks. Semaglutide can be considered in addition to lifestyle intervention for the management of higher weight in adults with PCOS.[81]

In one prospective study, pretreatment with a short-acting GLP-1 receptor agonists before IVF was associated with improved embryo implantation rates. The authors proposed that the drug's anti-inflammatory effects may enhance endometrial receptivity and support implantation.[82]

A study assessed Semaglutide 0.5 mg subcutaneously once weekly in obese PCOS patients who did not respond to lifestyle modifications. After 3 months, nearly 80% achieved at least a 5% weight loss, and among responders, 80% normalized menstrual cycles. Low-dose Semaglutide was therefore effective in reducing body weight and restoring menstrual regularity in most obese PCOS patients, with minimal side effects.[83]

In overweight or obese women with PCOS, combination therapy with Semaglutide and metformin produced greater reductions in body weight, BMI, and waist-to-hip ratio compared with metformin alone (all $p<0.01$). Menstrual cycle recovery was more frequent, and natural pregnancy rates from weeks 16 to 40 were higher (35% vs. 15%, $p<0.05$) in the combination group.[80]

Incorporating obesity management into a reproductive endocrinology practice is practical in both academic and private settings. Reproductive endocrinologists commonly manage PCOS and are familiar with medications that have comparable adverse effect profiles, such as metformin. Therefore, when a patient with obesity and infertility presents to a fertility clinic, clinicians should either facilitate timely referrals to multidisciplinary services or integrate obesity management into their comprehensive reproductive treatment plan.[70]

Managing obesity in perimenopausal and menopausal women

RECOMMENDATIONS

- Postmenopausal women should be assessed for sarcopenia, osteoporosis, and metabolic disorders (insulin resistance, dyslipidemia, MASLD, and CVD risk) prior to initiating lifestyle, pharmacologic, or surgical weight-management interventions. (Level A/ Class I)
- Pharmacologic therapy may be considered for menopausal women with BMI ≥27 kg/m^2 with comorbidities or ≥30 kg/m^2, in combination with a hypocaloric diet, regular physical activity, and structured clinical support. (Level B/ Class I)
- GLP-1 receptor agonists, particularly once-weekly subcutaneous Semaglutide 2.4 mg, may be considered as first-line pharmacological option for sustained weight loss and improving cardiometabolic parameters in postmenopausal women, including those on hormone therapy. (Level A/ Class I)

Discussion

The onset of menopause is characterized by declining estrogen levels, leading to physiological changes such as reduced resting energy expenditure, increased appetite, and preferential central fat accumulation. These body composition shifts increase fat mass and reduced lean mass, increase the risk of insulin resistance, dyslipidemia, metabolic dysfunction-associated steatotic liver disease (MASLD-previously referred to as nonalcoholic fatty liver disease), and CVD.[84]

Higher BMI is linked to more severe vasomotor symptoms, joint pain, sleep disruption, and genitourinary issues. It also contributes to hormonal imbalance, increasing the likelihood of anovulatory cycles and abnormal uterine bleeding. Additionally, obesity affects overall quality of life, and the chronic low-grade inflammation and prolonged estrogen exposure due to HRT is associated with a higher risk of hormone-sensitive cancers, including breast and endometrial cancer.[84] Weight loss improves cardiometabolic conditions, thereby lowering CVD and mortality risk. CVD is the leading cause of death in women and menopause independently increases this risk, interventions to prevent weight gain and manage overweight and obesity are critical in women.[85]

Pharmacologic therapy is an important adjunct to lifestyle modification for managing obesity in menopausal women, particularly those with BMI ≥27 kg/m^2 with comorbidities or ≥30 kg/m^2. These treatments should be integrated with a hypocaloric diet, regular physical activity, and optimal clinical support. GLP-1 receptor agonists are reported to be effective and safe, with once-weekly Semaglutide 2.4 mg considered a first-line choice due to its potent weight-loss effects and cardiovascular benefits.[84]

The International Menopause Society acknowledges GLP-1 receptor agonists, such as semaglutide, as effective weight-loss therapies that enhance satiety and promote significant weight reduction.[85]

GLP-1 receptor agonists can be effective in reducing weight in post-menopausal women.

In a retrospective cohort study of 106 postmenopausal women, Semaglutide produced sustained weight loss in both groups. Compared with non-hormonally treated women, those treated with hormone therapy experienced total body weight loss percentage of 7±3% vs. 5±4% (p=0.01); 13±6% vs. 9±5% (p=0.01); 15±6% vs. 10±6% (p=0.02); and 16±6% vs. 12±8% (p=0.04) at 3, 6, 9, and 12 months. Despite this difference, overall, Semaglutide was effective in reducing weight and enhancing cardiometabolic health in postmenopausal women.[86]

A multidisciplinary approach engaging gynecologists, endocrinologists, bariatric specialists, and behavioral therapists and other relevant specialists such as cardiologists, hepatologists/gastroenterologists, nephrologists, or pulmonologists, is essential to deliver comprehensive care to this high-risk menopausal population.[84]

Considerations for clinicians prescribing anti-obesity medications

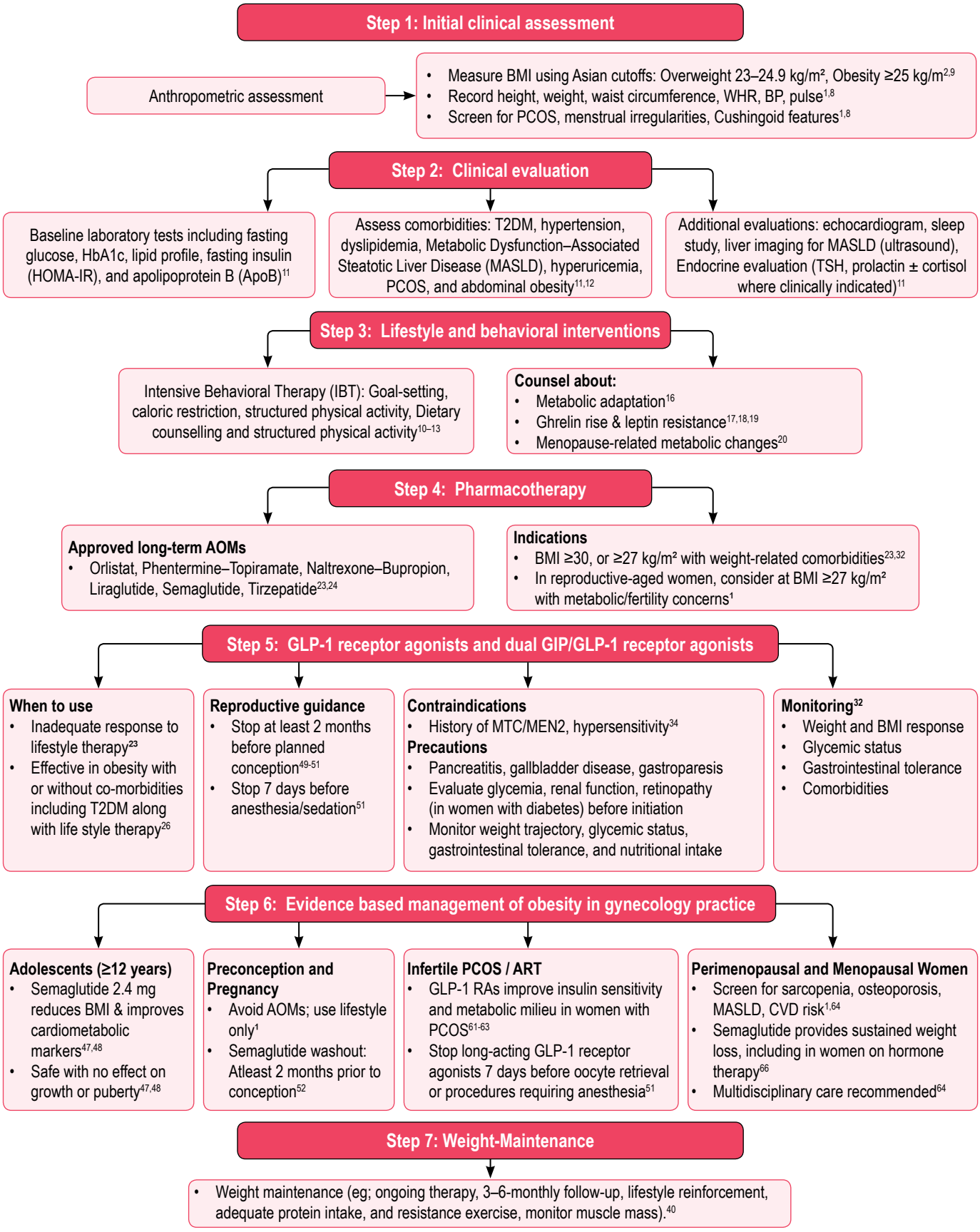
- Anti-obesity medications approved by the FDA are evidence-based adjuncts to lifestyle therapy and should be considered a standard component of obesity management.
- Because obesity can be chronic, patients should be counselled that long-term use of medications is often necessary to sustain weight loss, as discontinuation commonly leads to weight regain.
- Treatment decisions should be individualized through shared clinician and patient discussions that prioritize patient needs and associated comorbidities.
- A practical approach is to initiate therapy at a low dose and gradually titrate upward based on weight-loss response and medication tolerability.[27]
- Semaglutide and Tirzepatide are administered via once-weekly subcutaneous injections.
- Treatment should begin at a low dose and escalated according to the titration schedule.
- For Semaglutide, therapy should start at 0.25 mg weekly for 4 weeks, and then increase to 0.5 mg. Based on clinical response and tolerability, the dose may be further increased to 1.0, 1.7, or 2.4 mg once weekly with escalation every 4 weeks as per product label and then continued at the effective maintenance level.[70]
- Tirzepatide may be initiated at 2.5 mg once weekly, with dose escalation in 2.5 mg increments at 4-week intervals to maintenance doses of 5 mg, 10 mg, or 15 mg once weekly, based on clinical response and tolerability.[87]

Action and awareness-based approach for obesity management in India

Management of obesity through action and patient awareness has been recognized as an important approach for the prevention and long-term management of obesity, with emphasis on early identification, patient education, and sustained lifestyle-based interventions.[88]

This approach, referred to as Managing Obesity Through Action and Patient Awareness, is abbreviated as MOTAPA. This reflects the broader shift toward comprehensive, patient-centered and lifestyle-based obesity management strategies. The term MOTAPA highlights the importance of proactive action and patient awareness as a central element of obesity management. In the Indian context, the word "MOTAPA" is commonly used to denote obesity, and the term also reflects the commonly used terminology in an action-oriented framework.

STEPWISE CLINICAL ALGORITHM FOR THE ASSESSMENT AND MANAGEMENT OF OBESITY IN WOMEN



BMI: Body mass index; PCOS: Polycystic ovarian syndrome: HbA1c: Glycated hemoglobin; LFTs: Liver function tests; TSH: Thyroid stimulating hormone; MASLD: Metabolic Dysfunction-Associated Steatotic Liver Disease; HOMA-IR: Homeostasis Model Assessment for Insulin Resistance; T2DM: Type 2 diabetes mellitus; GLP-1: Glucagon-Like Peptide-1; CVD: Cardiovascular disease; AOMs: Antiobesity medications; ART: Assisted reproductive technology; MTC: Medullary thyroid carcinoma:; MEN2: Multiple endocrine neoplasia type 2

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