# OBESITY

## National Standard Treatment Guideline

Ministry of Health
Republic of Maldives

ADB

JFPR
Japan Fund for Prosperous and Resilient Asia and the Pacific

From the People of Japan

World Health Organization
Maldives

# National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
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- Diabetes Mellitus Type 1
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- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
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- Hyperkalemia
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- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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# GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

## 1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

## 2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

## 3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

## 4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

## 5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

## 6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

## 7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

## 8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

# 9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.

- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.

- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.

- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

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The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

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It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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# OBESITY

# QUICK REFERENCE GUIDE

## Definitions and staging

Adults (BMI):
WHO: Underweight <18.5; Normal 18.5–24.9; Overweight 25.0–29.9; Obesity ≥30
South Asian programs often use lower risk thresholds: Overweight ≥23; Obesity ≥25
Central adiposity (South Asian cut points): Men ≥90 cm waist; Women ≥80 cm
Children (WHO):
<5 years: Overweight >+2 SD weight-for-height; Obesity >+3 SD
5–19 years (BMI-for-age): Overweight >+1 SD; Obesity >+2 SD

## Clinical risk staging Edmonton Obesity Staging System (EOSS) to guide therapy.

| Stage | Adults (EOSS) | Pediatrics (EOSS-P) | Therapeutic focus | Weight target | Treatment approach | When to add meds / surgery | Follow-up |
|---|---|---|---|---|---|---|---|
| 0 | No metabolic risk, no symptoms, normal function/QoL | No cardiometabolic risk, no psychosocial distress, good function | Prevent weight gain, reinforce healthy lifestyle | Children: aim for **weight maintenance** as height increases; Adults: 0–5% if desired | Family-based lifestyle, diet quality, active play, sleep hygiene | Not indicated | Routine monitoring (6-12 months) |
| 1 | Subclinical risk (e.g., impaired fasting glucose, borderline BP), mild symptoms or distress, mild functional limits | Borderline BP, dyslipidemia, early insulin resistance, mild psychosocial distress or teasing, mild limitations in activity | Early intervention, risk reduction | Children: weight stabilization or slow BMI reduction; Adults: ~5–10% | Structured nutrition/ activity programs, behavioural counseling, school + family involvement | Meds: not usually; Surgery: not indicated | Closer (3-6 months) |
| 2 | Established obesity-related disease needing treatment (T2DM, HTN, OSA, MASLD, OA), moderate functional limits | Established disease (e.g., T2DM, OSA, fatty liver, HTN, orthopedic problems, anxiety/ depression), moderate impairment in daily function or self-esteem | Disease control + weight reduction | Children: modest BMI reduction, aim 5–10% or ≥0.25–0.5 BMI z-score decrease; Adults: 10–15% | Multidisciplinary team (pediatrician, dietitian, psychologist, physiotherapist) | Adults: meds if BMI criteria met; Children: consider **anti-obesity meds in adolescents** if ≥12 years, BMI ≥95th percentile with comorbidities, after failed intensive lifestyle | Every (1-3 months) |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 3 | Significant end-organ damage or marked functional impairment (CAD, HF, advanced NASH fibrosis, disabling OA) | Clear end-organ damage or serious impairment (e.g., advanced fatty liver, severe OSA, diabetes complications, major psychosocial dysfunction, inability to participate in school/social life) | Aggressive risk reduction, complication management | Children: individualized goals, higher BMI reduction if feasible; Adults: 15–20% | Intensive multidisciplinary care, higher-level centres, combine behaviour + meds | Adults: strong indication for meds/ surgery; Pediatrics: **Metabolic/ bariatric surgery** considered in adolescents ≥13–14 yrs, BMI ≥120% of 95th percentile or ≥35 with comorbidity | Intensive, often monthly |
| 4 | End-stage disease, severe disability, frailty, very high risk | End-stage organ failure, extreme disability, or very high treatment risk | Focus on QoL, function, symptom relief | Individualized, not always weight-centred | Supportive / rehabilitative / palliative goals | Adults: rarely appropriate for surgery; Pediatrics: surgery generally not considered | Individualized; focus on Qol, safety |

- History and risks: weight history, medications that promote weight gain, sleep and OSA symptoms, mental health, eating patterns, alcohol, smoking, physical activity, pregnancy intentions.
- Comorbidity screen: type 2 diabetes, hypertension, dyslipidemia, MASLD/ MASH, OSA, osteoarthritis, GERD, PCOS, depression/anxiety.
- Baseline measures: BMI, waist, blood pressure, functional status, quality of life.
- Laboratory tests: A1c or fasting glucose, lipid profile, ALT/AST; TSH if clinically suspected; pregnancy test if relevant; consider FIB-4 and OSA screen.
- Set targets: agree on an initial 5–10% weight loss over 3–6 months. In children, prefer weight maintenance as height increases unless otherwise indicated.

**Care algorithm (stepwise)[1]**

**Step 1. Lifestyle foundation for all**
Nutrition: energy deficit with high diet quality; regular meals; adequate protein; reduce energy-dense, low-nutrient foods.
Activity: 150–300 min/week moderate aerobic plus 2 days/week resistance; start lower if deconditioned and progress.
Sleep and stress: 7–9 hours sleep; stress reduction; limit alcohol; stop tobacco.
Behavioural skills: self-monitoring, problem-solving, stimulus control, goal setting.

**Step 2. Multidisciplinary support**
Team may include clinician, dietitian, psychologist/ psychiatrist, and exercise professional. Address weight stigma and barriers.

**Step 3. Escalate if response inadequate**
Add pharmacotherapy when BMI ≥30 or ≥27 with comorbidity. Use earlier if EOSS stage is higher. Consider endoscopic or surgical options when criteria are met and non-surgical care is optimized.

**Step 4. Long-term maintenance**
Relapse prevention plan, continued self-monitoring, timely intensification if weight regain begins.

[1]Adopted from American Association of Clinical Endocrinology (AACE) and the Endocrine Society.

## Treatment goals

- Achieve and maintain at least 5–10% weight loss to improve glycemia, blood pressure, lipids, sleep, joint pain, and liver health.
- For type 2 diabetes, >10% weight loss improves chances of remission and reduces medication burden.
- Improve function and quality of life. Prevent progression of complications such as MASH and cardiovascular disease.

# PHARMACOTHERAPY

Eligibility: BMI ≥30, or ≥27 with comorbidity. Local policies may set different thresholds for coverage.

| Medicine | Expected mean weight loss at ~1 year | Indication | Key cautions |
|---|---|---|---|
| **Semaglutide (weekly)** | ~12–15% | T2D with obesity, high cardiometabolic risk, appetite suppression | Avoid in pregnancy; contraindicated with personal/family history of MTC/MEN2; monitor for GI effects, gallbladder issues, rare pancreatitis |
| **Tirzepatide (weekly)** | ~15–22% | Marked weight loss need, diabetes with high A1c | Same thyroid warnings as GLP-1 class; GI effects; gallbladder risk; watch for hypoglycemia if on insulin/ secretagogues |
| **Liraglutide (daily)** | ~6–8% | When weekly injectables unsuitable or unavailable | Same class cautions as above; daily injection may affect compliance |
| **Dulaglutide (weekly)** | ~3–5% | Diabetes first, modest weight loss | Same GLP-1 class cautions; less weight loss compared to semaglutide or tirzepatide |
| **Orlistat** | ~3–5% | Non-injectable option; meal-based dosing | GI side effects; supplement fat-soluble vitamins; avoid in chronic malabsorption |

## Diabetes overlap

- Prefer GLP-1 RA or SGLT2 inhibitor for weight reduction and cardio-renal benefit. If insulin is needed, co-prescribe metformin and consider a GLP-1 RA or pramlintide to limit weight gain.
- Start low and up-titrate. Stop or switch if weight loss <5% at 3 months on a therapeutic dose, unless other clinical gains justify continuation.

## Starter dosing & titration

**Semaglutide (weekly, obesity)**: 0.25 mg SC weekly x4 weeks → 0.5 mg x4 → 1.0 mg x4 → 1.7 mg x4 → maintain 2.4 mg weekly (or 1.7 mg if not tolerated). If using Semaglutide for diabetes: 0.25 → 0.5 → 1.0 → up to 2.0 mg weekly as needed for glycemic control. **OR**

**Tirzepatide (weekly)**: 2.5 mg SC weekly x4 weeks, then increase by

2.5 mg every 4 weeks (5 → 7.5 → 10 → 12.5 → 15 mg) to the highest tolerated/ effective dose. **OR**

**Liraglutide (daily, obesity)**: 0.6 mg SC daily (week 1) → 1.2 mg (week 2) → 1.8 mg (week 3) → 2.4 mg (week 4) → 3.0 mg daily maintenance. If using Liraglutide for diabetes: 0.6 → 1.2 → 1.8 mg daily. **OR**

**Dulaglutide (weekly)**: Start 0.75 mg SC weekly; increase to 1.5 mg after ≥4 weeks; higher doses 3.0/4.5 mg weekly may be used for additional glycemic effect/weight loss. **OR**

**Orlistat**: 120 mg with each main meal (up to TID). Skip dose if skipping meal or if meal has very little fat. Take multivitamin at bedtime (separate ≥2 hours).

***Optional for diabetes where available***

**Oral Semaglutide**: 3 mg daily x30 days, then 7 mg; may increase to 14 mg. Take on empty stomach with ≤120 mL water; wait ≥30 min before food/ other medicines.

**Note** (GLP-1 coverage): current Maldives MOH interim policy uses BMI ≥37.5 without comorbidity or ≥32.5 with comorbidity for eligibility. Apply clinical judgment for non-covered cases.[2]

## Indications for metabolic/ bariatric surgery[3]

- Adults, Asia/South-Asian thresholds used by many programs: BMI ≥37.5 regardless of comorbidity or BMI ≥32.5 with at least one major comorbidity such as T2D, OSA, MASLD with fibrosis, or severe osteoarthritis.
- T2D not controlled despite optimal therapy: consider from BMI ≥30; some centers individualize at 27.5–32.4 in high-risk patients.
- Requirements: documented attempt at structured lifestyle with or without pharmacotherapy, psychosocial readiness, ability for long-term follow-up, acceptable peri-operative risk.

[2] health.gov.mv/storage/uploads/Eond5AYM/rws5pyqk.pdf
[3] Adopted from International Federation for the Surgery of Obesity (IFSO) and the American Society for Metabolic and Bariatric Surgery (ASMBS).

## Monitoring and follow-up

| Focus | What to do | Timing | Threshold and decision |
|---|---|---|---|
| **Clinical measures** | Track weight, waist, blood pressure, A1c/FBG, lipid profile, ALT/AST, patient-reported outcomes | Every 4–12 weeks initially | Use percent weight loss as the primary metric |
| **Success check** | Assess response to therapy | Around 12 weeks | ≥5% loss supports continuation |
| **Inadequate response** | If <5% loss or comorbidities persist | At 12 weeks | Intensify lifestyle, reassess dosing, add or switch therapy |
| **Maintenance** | Continue effective therapy and relapse prevention | Minimum 1 year; often longer | Plan for early action on any regain |
| **Safety** | Review side effects and adherence | Each visit | Modify or stop if intolerance or safety concerns arise |

## Pediatrics (EOSS-P pointers)

- Emphasize family-based lifestyle change, sleep, and activity. Weight maintenance with height gain may improve BMI percentile.
- Consider pharmacotherapy only in older adolescents with severe obesity and comorbidities after intensive lifestyle therapy.
- Surgery may be considered in selected adolescents meeting BMI percent-of-95th percentile criteria and with serious comorbidities in experienced centers.

## Counseling: Do's and Don'ts

- Do set a 5–10% target, plan meals, move daily, sleep 7–9 hours, log food and weekly weight, take medicines as prescribed, and bring a support person to key visits.
- Do not crash diet, double missed doses, stop medicines without advice, use unregulated supplements, chase detoxes or extreme workouts after long gaps, or ignore persistent side effects.
- Red flags that need urgent care: chest pain, severe shortness of breath, fainting, severe headache, marked polyuria with vomiting or confusion, severe or persistent abdominal pain, one-sided leg swelling with breathlessness, jaundice

## Documentation checklist

- Baseline anthropometry and comorbidity profile
- Informed discussion of options and agreed goals
- Chosen plan including lifestyle, medicines, and any referrals
- Monitoring schedule and safety plan
- Maintenance and relapse strategy

# INTRODUCTION

Obesity is a chronic, progressive disease caused by excess adiposity that impairs health and increases the risk of diabetes, cardiovascular disease, certain cancers, and musculoskeletal disorders. It requires the same medical attention and continuity of care as other chronic conditions. Globally, over 1 billion adults are obese, and projections suggest more than half the world's adult population could be obese by 2035. There is a rising prevalence of overweight and obesity in both men and women, with a concerning burden of central obesity linked to early-onset metabolic disease, even at normal BMI levels.  A substantial proportion of the South Asian population presents with normal weight obesity—characterized by normal BMI but elevated cardiometabolic risk, comparable to individuals with overt obesity. Prevalence varies across regions due to disparities in income, diet, and access to safe physical activity spaces, with urban women particularly affected by inactivity.

Childhood and adolescent obesity are rising rapidly, fueled by processed diets and sedentary lifestyles. Early weight gain often tracks into adulthood, with 30% of obesity originating in childhood and up to 80% of obese children remaining obese as adults. The older the child when overweight persists, the higher the likelihood of adult obesity, underscoring the urgency of early identification and intervention. In geographically dispersed settings, limited access to specialized care and trained professionals leads to fragmented, reactive obesity management. Recognizing obesity as a chronic condition—termed Adiposity-Based Chronic Disease (ABCD) by the American Association of Clinical Endocrinology—reinforces the need for sustained, systemic approaches to prevention and care.

In line with the Maldives National Multi-sectoral Action Plan for NCDs (2023–2031), there is an urgent need for structured, evidence-based guidelines tailored to the local context. These should address abdominal obesity, integrate early-life interventions, provide culturally appropriate recommendations, and offer simplified, tiered protocols for different levels of care. Strengthening capacity at the island and atoll level, including task-sharing with community health workers, will be central to improving access. By aligning with broader NCD programs, these guidelines aim to bridge gaps in care, promote prevention, and provide a standardized framework that empowers providers and supports sustainable weight control, ultimately reducing the long-term cardiometabolic burden in the Maldives.

# SCOPE OF THE GUIDELINES

These guidelines address the prevention, early detection, and management of obesity in both adults and children. They cover medical, nutritional, behavioral, and lifestyle-based interventions, with attention to co-morbidities and complications associated with obesity. Surgical interventions are acknowledged as part of the treatment spectrum, but detailed

procedural descriptions are beyond the scope of this document.

The focus is on evidence-based, practical recommendations that can be implemented at all levels of healthcare, with tiered applicability:

- **Primary Care:** Early recognition, growth and BMI monitoring, counseling on diet, physical activity, and lifestyle changes; screening for comorbidities.
- **Secondary Care:** Medical management of obesity-related complications (diabetes, hypertension, dyslipidemia), structured weight management programs, use of pharmacotherapy where indicated.
- **Tertiary Care:** Advanced management, multidisciplinary obesity clinics, evaluation for surgical interventions when necessary.

## Intended Users

- Primary care physicians, pediatricians, family practitioners, and general practitioners.
- Specialists managing obesity-related comorbidities (endocrinologists, cardiologists, psychiatrists, dietitians, physiotherapists, and public health practitioners).
- Nurses, mid-level health providers, and community health workers delivering frontline health services.
- Policymakers and program managers involved in designing and implementing obesity prevention and control strategies.

## Applicability and Bridging Gaps

These guidelines are tailored for use in settings where specialized obesity services may be limited. They standardize essential practices to help frontline providers deliver early, consistent, and practical interventions, with lifestyle and behavioral management as the first line of care. Clear referral pathways ensure appropriate escalation to higher levels when needed, while reducing reliance on costly, inaccessible options. In this way, the guidelines bridge gaps until tertiary care is available, ensuring safe and evidence-based management within existing infrastructure.

# DEFINITION

Obesity is a chronic, progressive disease characterized by abnormal or excessive accumulation of body fat that presents a risk to health. It is commonly assessed using body mass index (BMI), which is calculated as weight in kilograms divided by height in

meters squared (kg/m²). While the World Health Organization (WHO)defines overweight as a BMI ≥25 kg/m² and obesity as a BMI ≥30 kg/m² for the general population, South Asians have higher body fat percentage and greater metabolic risk at lower BMI levels compared to Caucasian populations.

Due to this increased susceptibility to insulin resistance, type 2 diabetes, dyslipidemia, and cardiovascular disease at lower BMI values, revised cut-offs for South Asian populations are recommended:

| Category | BMI (kg/m²) |
|---|---|
| **Underweight** | <18.5 |
| **Normal range** | 18.5 – 22.9 |
| **Overweight (at risk)** | 23.0 – 24.9 |
| **Obesity (Class I)** | 25.0 – 29.9 |
| **Obesity (Class II)** | ≥30.0 |

In addition to BMI, waist circumference is an important measure of abdominal (central) obesity, which is strongly linked to metabolic risk. For South Asians:

- **Men:** Waist circumference ≥90 cm indicates central obesity
- **Women:** Waist circumference ≥80 cm indicates central obesity

WHO defines obesity in children using Body Mass Index (BMI)-for-age percentiles or Z-scores.

| Category | Children <5 years (WHO, weight-for-height) | Children 5–19 years (WHO, BMI-for-age) | Adults (WHO BMI, kg/m²) | Adults (South Asian BMI, kg/m²) |
|---|---|---|---|---|
| **Underweight / Wasting** | < −2 SD | < −2 SD(< 5th percentile) | < 18.5 | < 18.5 |
| **Normal weight** | ≥ −2 SD to ≤ +2 SD | ≥ −2 SD to ≤ +1 SD(5th – 85th percentile) | 18.5 – 24.9 | 18.5 – 22.9 |
| **Overweight** | > +2 SD to ≤ +3 SD | > +1 SD to ≤ +2 SD(85th – 95th percentile) | 25.0 – 29.9 | 23.0 – 24.9 |
| **Obesity** | > +3 SD | > +2 SD(≥ 95th percentile) | ≥ 30.0 | ≥ 25.0 |
| **Obesity Grade 1** | | | | 25.0 – 29.9 |
| **Obesity Grade 2** | | | | 30.0 – 34.9 |
| **Obesity Grade 3** | | | | ≥ 35 |

# CAUSES, RISK FACTORS & TRIGGERS

Obesity is a multifactorial, chronic disease resulting from a sustained positive energy balance, where energy intake consistently exceeds energy expenditure. Its development reflects a complex interplay of biological, behavioural, environmental, and social determinants.

| Category | Details |
|---|---|
| **Genetic predisposition** | Variations in genes regulating appetite (leptin, melanocortin receptors), satiety, energy expenditure, and fat storage increase susceptibility to weight gain and metabolic inefficiency. |
| **Ethnic-specific factors** | South Asians and similar groups have higher body fat percentage and central adiposity at lower BMI, raising metabolic risk even without overt obesity. |
| **Dietary factors** | High-calorie diets with refined carbs, added sugars, and saturated fats. Overconsumption of ultra-processed foods (low satiety, high palatability). Portion size expansion, frequent snacking, and sweetened beverages. |
| **Physical inactivity** | Sedentary lifestyle in urban settings. Reduced active transport (walking, cycling). Limited recreational spaces, screen-based entertainment, and mechanized occupations lower energy expenditure. |
| **Sleep and circadian disruption** | Sleep deprivation → higher ghrelin, lower leptin → increased hunger. Irregular cycles (e.g., shift work) disrupt circadian rhythm and metabolism, raising obesity risk. |
| **Medications** | Psychotropics: atypical antipsychotics (olanzapine, clozapine), some antidepressants - Glucocorticoids: long-term steroid use - Anticonvulsants: valproate, carbamazepine - Antidiabetics: insulin, sulfonylureas (in some cases) |
| **Psychosocial & behavioral factors** | Chronic stress → HPA axis activation → high cortisol → visceral fat. Emotional eating, food as coping, irregular meals. Cultural norms favoring calorie-dense foods or limiting activity (especially in women). |
| **Early-life & developmental factors** | Maternal obesity, gestational diabetes, or intrauterine exposures alter offspring metabolism. Low birth weight with rapid catch-up growth increases adult obesity risk. Childhood obesity strongly predicts adult obesity. |
| **Environmental & societal factors** | Obesogenic environment: easy access to unhealthy food, aggressive marketing, poor urban planning, unsafe neighborhoods. Nutrition transition toward processed foods, reduced occupational activity due to mechanization. |
| **Medical & hormonal conditions** | Obesogenic environment: easy access to unhealthy food, aggressive marketing, poor urban planning, unsafe neighborhoods. Nutrition transition toward processed foods, reduced occupational activity due to mechanization. |

# EVALUATION FOR DIAGNOSIS

Patients may present asymptomatically or with weight gain, reduced exercise tolerance, acanthosis nigricans, central adiposity, and features of insulin resistance. The diagnosis of obesity requires a structured assessment that includes anthropometric measurements, clinical evaluation, and risk stratification for obesity-related comorbidities. The aim is not only to confirm excess adiposity but also to evaluate its health impact and identify underlying causes or contributing factors.

## Medical History

| Aspect | Details |
|---|---|
| **Weight history** | Onset, progression, and any periods of rapid weight gain |
| **Lifestyle factors** | Dietary habits, frequency of eating out, physical activity, sedentary behaviour, sleep patterns |
| **Medication history** | Drugs linked to weight gain: atypical antipsychotics, steroids, valproate, carbamazepine, insulin, sulfonylureas |
| **Past medical history** | Type 2 diabetes, hypertension, dyslipidaemia, cardiovascular disease, fatty liver disease, osteoarthritis, sleep apnoea |
| **Family history** | Obesity, diabetes, premature cardiovascular disease |
| **Reproductive history (women)** | Menstrual irregularities, PCOS, gestational diabetes |
| **Psychosocial history** | Depression, anxiety, stress, emotional eating, social habits, substance use |
| **Endocrine disorder history** | Hypothyroidism, Cushing's syndrome, growth hormone deficiency, PCOS, hypogonadism. |
| **Genetic syndromes** | Prader–Willi syndrome, Bardet–Biedl syndrome (rare). |
| **Hypothalamic damage history** | Tumours, trauma, surgery, or inflammation affecting appetite regulation. |

## Physical Examination

| Parameter | Details / Cut-offs |
|---|---|
| **Body Mass Index (BMI)** | Weight (kg)/Height ($m^2$); South Asian cut-offs: Overweight ≥23.0, Obesity ≥25.0 |
| **Waist circumference (WC)** | Men ≥90 cm; Women ≥80 cm → central obesity |
| **Waist–hip ratio (WHR)** | Men >0.90; Women >0.85 → increased cardiometabolic risk |
| **Body fat percentage** | Measured by bioelectrical impedance or DEXA scan |
| **General assessment** | Blood pressure, heart rate, insulin resistance signs (acanthosis nigricans, skin tags), endocrine features (goitre, Cushingoid features) |
| **Musculoskeletal assessment** | Joint pain, mobility or back issues |
| **Respiratory assessment** | Risk of Obstructive sleep apnoea (OSA): Epworth Sleepiness Scale, STOP-Bang score (for details see guidelines on obstructive sleep apnoea). |

## Laboratory Investigations

To assess metabolic risk, comorbidities, and exclude secondary causes:

| Test | Purpose |
|---|---|
| **Fasting plasma glucose, HbA1c, OGTT (if indicated)** | Screening for diabetes and prediabetes |
| **Lipid profile** | Total cholesterol, LDL-C, HDL-C, triglycerides |
| **Liver function tests** | Assessment for MASLD |
| **Thyroid function tests** | Exclude hypothyroidism |
| **Serum cortisol / Overnight dexamethasone suppression test** | Rule out Cushing's syndrome if suspected |
| **Renal function tests** | Baseline comorbidity assessment |
| **Sleep study (polysomnography)** | Confirm OSA if suspected |

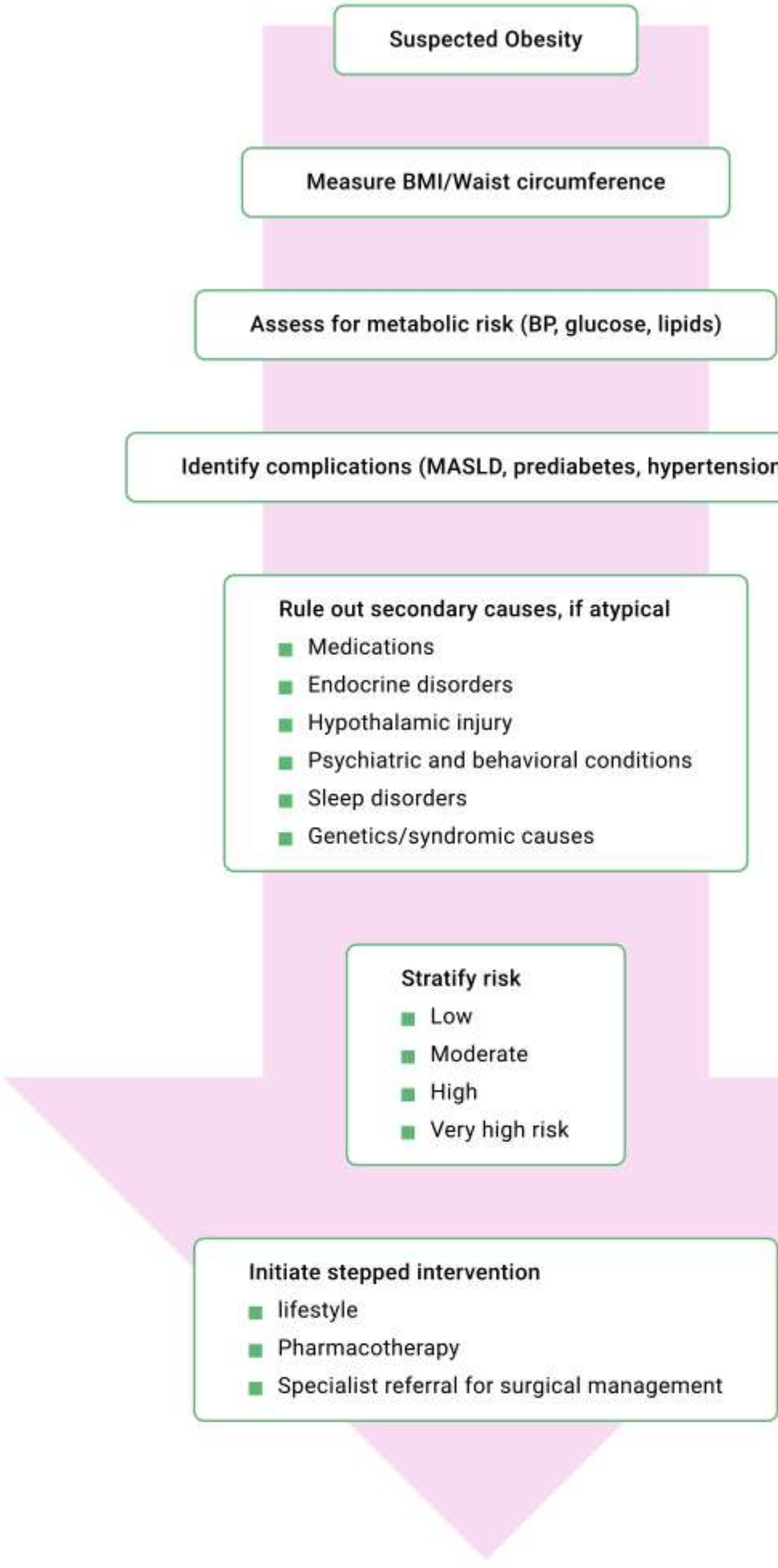
**Note**: Genetic testing in patients with obesity is indicated when specific clinical features suggest a monogenic or syndromic etiology. These include early-onset obesity before 10 years of age, morbid obesity, and a family history of consanguinity or young-onset obesity. Additional red flags include persistent hyperphagia, developmental delays, visual or hearing impairments, and macrocephaly. Identifying these markers can guide personalized management and uncover underlying genetic contributions that may not be evident through routine evaluation.

# CONFIRMATION OF DIAGNOSIS

Obesity diagnosis is primarily clinical, based on elevated BMI or central adiposity accompanied by metabolic abnormalities. Imaging modalities like ultrasound or transient elastography can help confirm hepatic steatosis and exclude other causes. When atypical features are present, secondary contributors to weight gain should be ruled out.

**Note:** obesity should not be diagnosed solely on body weight; a comprehensive assessment must identify the underlying drivers in each individual—such as reduced metabolic rate, low physical activity, or excessive caloric intake. BMI should be interpreted in the context of its metabolic impact, associated comorbidities, and potential secondary etiologies.

# Confirmation of diagnosis



# DIFFERENTIAL DIAGNOSIS

Differential diagnosis of obesity requires distinguishing true excess adiposity from conditions that cause weight gain or central fat redistribution and from mimics where apparent "obesity" is actually fluid accumulation or abnormal fat deposition.

| Condition | Features | Confirmatory Tests / Distinguishing Points |
|---|---|---|
| **Cushing's syndrome** | Rapid central weight gain, moon face, buffalo hump, purple striae | Dexamethasone suppression test |
| **Hypothyroidism** | Modest gain, fatigue, cold intolerance, bradycardia | TSH, free T4 |
| **PCOS** | Irregular menses, hirsutism, acne, abdominal weight gain | Androgens, metabolic profile |
| **Lipedema** | Painful, symmetric leg fat with sparing of feet; poor diet response | Clinical diagnosis |
| **Fluid retention (cardiac/renal/hepatic)** | Pitting edema, ascites, dyspnea | Organ-specific labs, imaging |
| **Medications** | Weight gain or fat redistribution with steroids, antipsychotics, antidepressants, insulin | Drug history |
| **Hypothalamic obesity** | Post-CNS insult, hyperphagia, rapid gain | Neuroimaging |
| **Genetic syndromes** | Early severe obesity + dysmorphic features | Genetic testing |
| **Psychiatric / behavioral** | Binge eating, depression, emotional triggers | History, screening tools |
| **Simple (exogenous) obesity** | Gradual weight gain linked to excess intake & inactivity; no endocrine stigmata | Normal labs (unless metabolic sequelae present) |

# MANAGEMENT

Treatment of obesity must be individualized, considering the patient's clinical profile, preferences, and what therapies are accessible. Lifestyle change is the cornerstone of overweight and obesity management and the platform for all further therapies. If lifestyle change alone is insufficient, add pharmacotherapy, endoscopic remodeling, or metabolic surgery in a stepped fashion to achieve and sustain meaningful weight loss.

Assess baseline risk, prioritize lifestyle modification, add pharmacotherapy for insufficient response in high-risk individuals, and refer for advanced care (e.g., metabolic surgery) when criteria are met. Address barriers, ensure psychosocial support, and target sustainable weight loss (5–10% initial). Combining these options in a coordinated, comprehensive plan yields the best long-term results.

Effective long-term care is a partnership between a motivated patient and a multidisciplinary team tailored to the individual's comorbidities. That team can include a physician, psychologist or psychiatrist, physical/exercise therapists, dietitians, and other specialists.

# MANAGEMENT GOALS

The goals of obesity management are to achieve and sustain clinically meaningful weight loss (an initial 5–10% reduction), reverse or improve metabolic comorbidities such as diabetes, hypertension, and dyslipidemia, and lower overall cardiovascular risk. Equally important is preventing

progression of liver disease, particularly metabolic dysfunction–associated steatotic liver disease (MASLD), while improving physical function and quality of life. These targets work together: modest sustained weight loss yields measurable benefits across metabolic, hepatic, and functional domains.

## Edmonton Obesity Staging System (EOSS) to guide therapy.

| Stage | Adults (EOSS) | Pediatrics (EOSS-P) | Therapeutic focus | Weight target | Treatment approach | When to add meds / surgery | Follow-up |
|---|---|---|---|---|---|---|---|
| 0 | No metabolic risk, no symptoms, normal function/QoL | No cardiometabolic risk, no psychosocial distress, good function | Prevent weight gain, reinforce healthy lifestyle | Children: aim for weight maintenance as height increases; Adults: 0–5% if desired | Family-based lifestyle, diet quality, active play, sleep hygiene | Not indicated | Routine monitoring (6-12 months) |
| 1 | Subclinical risk (e.g., impaired fasting glucose, borderline BP), mild symptoms or distress, mild functional limits | Borderline BP, dyslipidemia, early insulin resistance, mild psychosocial distress or teasing, mild limitations in activity | Early intervention, risk reduction | Children: weight stabilization or slow BMI reduction; Adults: ~5–10% | Structured nutrition/ activity programs, behavioural counseling, school + family involvement | Meds: not usually; Surgery: not indicated | Closer (3-6 months) |
| 2 | Established obesity-related disease needing treatment (T2DM, HTN, OSA, MASLD, OA), moderate functional limits | Established disease (e.g., T2DM, OSA, fatty liver, HTN, orthopedic problems, anxiety/ depression), moderate impairment in daily function or self-esteem | Disease control + weight reduction | Children: modest BMI reduction, aim 5–10% or ≥0.25–0.5 BMI z-score decrease; Adults: 10–15% | Multidisciplinary team (pediatrician, dietitian, psychologist, physiotherapist) | Adults: meds if BMI criteria met; Children: consider anti-obesity meds in adolescents if ≥12 years, BMI ≥95th percentile with comorbidities, after failed intensive lifestyle | Every (1-3 months) |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 3 | Significant end-organ damage or marked functional impairment (CAD, HF, advanced NASH fibrosis, disabling OA) | Clear end-organ damage or serious impairment (e.g., advanced fatty liver, severe OSA, diabetes complications, major psychosocial dysfunction, inability to participate in school/social life) | Aggressive risk reduction, complication management | Children: individualized goals, higher BMI reduction if feasible; Adults: 15–20% | Intensive multidisciplinary care, higher-level centres, combine behaviour + meds | Adults: strong indication for meds/ surgery; Pediatrics: **Metabolic/ bariatric surgery** considered in adolescents ≥13–14 yrs, BMI ≥120% of 95th percentile or ≥35 with comorbidity | Intensive, often monthly |
| 4 | End-stage disease, severe disability, frailty, very high risk | End-stage organ failure, extreme disability, or very high treatment risk | Focus on QoL, function, symptom relief | Individualized, not always weight-centred | Supportive / rehabilitative / palliative goals | Adults: rarely appropriate for surgery; Pediatrics: surgery generally not considered | Individualized; focus on QoL, safety |

## Note: How to use EOSS staging in decisions

- **Stage sets intensity:** higher stage → earlier escalation from lifestyle to meds to surgery. In children multidimensional approach including medical, mental health, social milieu, and functional domains. Bullying, self-esteem, depression, and school participation are heavily weighted in staging and decisions.
- **Targets scale with stage:** ~5–10% at Stage 1, ~10–15% at Stage 2, ~15–20% at Stage 3 (as tolerated). Weight goal differs by age in younger children, weight maintenance with height gain may improve BMI percentile. In adolescents, active weight loss may be appropriate.
- **Modalities:** combine behavioural therapy for all stages; add pharmacotherapy when BMI criteria are met and lifestyle alone isn't enough; consider surgery from Stage 2 upward if eligible and fit.
- **Track the stage over time** to see if care is de-risking the patient (stage down) or if you need to intensify care (stage up).

## BMI eligibility notes (adapt to local policy):

- **Medications:** South Asian settings - lower thresholds are used (e.g., ≥27.5, or ≥23 with comorbidities). Limited to older adolescents with severe obesity + comorbidities (WHO/Endocrine Society criteria).
- **Surgery:** commonly BMI ≥40, or ≥35 with comorbidities; in South Asian settings lower cut-offs are often applied (e.g., ≥37.5, or ≥32.5 with comorbidities). Reserved for

adolescents meeting strict BMI + comorbidity thresholds and after failed intensive lifestyle therapy.

### Management of Obesity

**Step 1. Initial Assessment**

- Record BMI, waist circumference, comorbidities, functional status, and psychosocial context
- Identify barriers and patient preferences
- Set realistic goals (initial 5–10% weight loss or weight stabilization in children)

**Step 2. Lifestyle Modification (Cornerstone)**

- Structured nutrition plan (reduce energy density, portion control, diet quality)
- Regular physical activity (aerobic + resistance, age- and ability-appropriate)
- Sleep hygiene and stress management
- Behavioural strategies (self-monitoring, goal setting, problem-solving)

**Step 3. Multidisciplinary Support**

- Team approach: physician, dietitian, psychologist/psychiatrist, physiotherapist/exercise specialist
- Provide psychosocial support, address stigma, improve motivation
- Regular monitoring of weight, metabolic profile, liver health, and function

**Step 4. Escalation of Therapy (if insufficient response)**

- Pharmacotherapy: add when lifestyle change alone is inadequate, especially in high-risk patients
- Endoscopic bariatric/metabolic interventions: consider in selected cases where available
- Metabolic/bariatric surgery: offer if criteria are met (BMI and comorbidity thresholds), after failed structured therapy, and with adequate peri-operative support

**Step 5. Long-Term Care & Goals**

- Sustain ≥5–10% weight loss to improve diabetes, hypertension, dyslipidemia, and lower CV risk
- Prevent or slow progression of MASLD and other obesity-related organ damage
- Enhance physical function, mobility, and quality of life
- Maintain ongoing follow-up (frequency tailored to stage and therapy used)

## Non-pharmacological interventions

- All patients with a BMI ≥25 kg/m² should receive diet, exercise, and behavioral modification:
- Creating a sustained caloric deficit of 500–750 kcal/day can yield 4–8% weight loss in one year. This should be paired with at least 150 minutes of moderate activity weekly for maintenance, increasing to around 300 minutes for active weight loss.
- Durable weight loss is challenging, but when sustained it drives meaningful health benefits, lower blood pressure, improved glycemic control, and reduced metabolic risk. It requires behavior modification (self-monitoring, goal setting, stimulus control, relapse prevention), and sleep hygiene.
- Incorporate culturally appropriate dietary advice and leverage community support. Address psychological factors with cognitive behavioral therapy when needed.

# PHARMACOLOGICAL THERAPY

Pharmacotherapy is added for BMI ≥27 kg/m² with at least one comorbidity ((hypertension, dyslipidemia, type 2 diabetes, obstructive sleep apnea), after failing adequate lifestyle intervention or BMI >30 kg/m² to help control comorbidities and reinforce behavior change.

**GIP/GLP-1 dual agonist:** Tirzepatide (weekly SC injection) shows superior weight loss and metabolic benefits. Start with 2.5 mg once weekly for 4 weeks (for gastrointestinal tolerability; not intended for glycemic control). Increase in 2.5 mg increments every 4 weeks based on tolerability and treatment goals. Usual maintenance doses: 5 mg, 10 mg, or 15 mg once weekly. Maximum dose: 15 mg once weekly. If a dose is missed, it can be given within 4 days (96 hours); otherwise, skip and resume on the next scheduled day.

**Or**

**GLP-1 receptor agonists:** Semaglutide (Start 0.25 mg weekly and titrate every 4 weeks through 0.5 mg, 1 mg, 1.7 mg to maintenance 2.4 mg weekly **OR** liraglutide (Start at 0.6 mg once daily, increasing by 0.6 mg each week; Titrate to a maintenance dose of 3.0 mg once daily. Administered independently of meals; SC injections in abdomen, thigh, or upper arm.

**Or**

**Orlistat:** 120 mg orally, three times daily with each main meal containing fat during the meal or up to 1 hour after eating. If a meal is skipped or contains no fat, the dose should be omitted. It reduces fat absorption; watch for gastrointestinal intolerance and fat-soluble vitamin deficiency.

These drugs produce substantial weight loss, with tirzepatide showing the largest effect (mean ~21% body weight reduction). Weight loss medications can support adherence to behavior change and improve physical function, easing the increase in activity for those initially unable to exercise. Sustained use is required to maintain weight loss; discontinuation typically leads to regain. Beyond weight reduction, they lower incident diabetes and decrease the need for antihypertensive and lipid-lowering therapies and confer cardiovascular risk reduction. Their effectiveness comes with the trade-off of long-term continuation and high cost compared with lifestyle-only approaches.

Combine pharmacotherapy with lifestyle support; discontinue if weight loss <5% at defined timepoints (typically 12–16 weeks) unless benefit is evident.

**Note 1:** Eligibility for government insurance coverage (obesity indication): BMI ≥ 37.5 with no comorbidities, or BMI ≥ 32.5 with any obesity-related comorbidity. Must be combined with lifestyle modifications. Internal medicine specialists, endocrinologists, pulmonologists, cardiologists, bariatric/metabolic surgeons, family medicine and specialized GPs can prescribe these medicines. Medical Officers (MOs) cannot renew. Renewals must be reviewed and approved by a listed specialist based on clinical progress (MoH MV).

**Note 2:** Tirzepatide shares a tolerability profile with GLP-1 receptor agonists, with gastrointestinal symptoms—nausea, vomiting, diarrhea, constipation, and abdominal discomfort—being the most common, typically mild to moderate and improving with continued use or dose titration. Less frequent effects include dyspepsia, fatigue, and injection site reactions. Rare but serious risks include pancreatitis, gallbladder disease, and worsening diabetic retinopathy with rapid glycemic control. Tirzepatide delays gastric emptying, potentially affecting absorption of oral medications; caution is warranted with drugs that have a narrow therapeutic index (e.g., warfarin, levothyroxine, certain antibiotics) and with insulin or sulfonylureas due to increased hypoglycemia risk. No dose adjustment is needed in renal or hepatic impairment, but gastrointestinal side effects may exacerbate renal dysfunction through dehydration, and limited data in severe liver disease calls for close monitoring.

Semaglutide is generally well tolerated, with mild and transient gastrointestinal side effects such as nausea, vomiting, diarrhea, and constipation being most common. Less frequent reactions include dyspepsia, abdominal pain, fatigue, and occasional injection site issues. Rare but serious risks include pancreatitis, gallbladder disease, and worsening diabetic retinopathy, especially with rapid glucose reduction. Semaglutide is contraindicated in individuals with a personal or family history of medullary thyroid carcinoma, MEN2, or hypersensitivity to the drug. Caution is advised in patients with a history of pancreatitis, severe gastrointestinal disorders like gastroparesis, or advanced diabetic retinopathy. It is not recommended during pregnancy or breastfeeding. Due to delayed gastric emptying, semaglutide may affect absorption of oral medications, requiring monitoring for drugs with narrow therapeutic indices such as warfarin and levothyroxine. When used with insulin or sulfonylureas, the risk of hypoglycemia increases, necessitating dose adjustments. Renal and hepatic impairment also warrant careful monitoring and dose modification.

**GLP-1 receptor agonist: Dulaglutide,** may aid weight reduction in individuals with obesity, though it's primarily approved for type 2 diabetes. Begin at 0.75 mg, with gradual titration up to 4.5 mg based on tolerance and response  once-weekly subcutaneous. Treatment duration is long-term, with reassessment at 12–16 weeks to ensure ≥5% weight loss.

**Caution:** in patients with a history of medullary thyroid carcinoma, MEN2, pancreatitis, or severe gastrointestinal disease. Common side effects include nausea and delayed gastric emptying, which may affect absorption of oral medications.

# OBESITY & COMORBIDITIES

## Obesity & Diabetes:

Adults with BMI ≥30 kg/m², or ≥27 kg/m² with at least one weight-related comorbidity (e.g., hypertension, dyslipidemia, T2DM). Target sustainable weight loss (5–10% initial to improve diabetes control and for diabetes remission >10%.

## Glucose-lowering choice in overweight/obese T2D

- Use metformin unless contraindicated.
- Prefer weight-reducing agents:
  - GLP-1 receptor agonist (e.g., liraglutide, semaglutide, dulaglutide, exenatide, lixisenatide) or dual GIP/GLP-1 (tirzepatide).
  - SGLT-2 inhibitor.
- Use as monotherapy if metformin is not tolerated, or in combination with other glucose-lowering drugs, especially in patients with obesity or cardiovascular disease.

## If insulin is required

- Co-prescribe at least one of: metformin, pramlintide, or a GLP-1 RA / dual GIP-GLP-1 to counter insulin-associated weight gain.
- Prefer basal insulin over insulin alone in escalating regimens; avoid pairing insulin with a sulfonylurea when possible.
- Consider adding an SGLT-2 inhibitor if not contraindicated.

## Follow-up

- Reassess weight, A1c, comorbidities, and tolerance at ~3 months; intensify therapy if targets aren't met.

## Obesity with Hypertension

In hypertensive patients with obesity and type 2 diabetes, consider ACE inhibitors, ARBs, or calcium channel blockers as first-line agents, avoid first-line beta-blockers unless specifically indicated.

## Obesity & Contraception

For women with BMI ≥27 kg/m² with comorbidities or BMI ≥30 kg/m² seeking contraception, prefer oral contraceptives over injectables, after counseling on risks and benefits and confirming no contraindications.

## Management of Secondary obesity

Management of secondary obesity begins with identifying and treating the underlying cause—correct hormonal imbalances (e.g., treat Cushing syndrome, optimize thyroid or gonadal function), address hypothalamic or neuroregulatory injury, and adjust or switch weight-promoting medications when possible.

Concurrently apply standard lifestyle measures and treat comorbid contributors such as sleep apnea with CPAP and address psychiatric drivers (binge eating, depression) with therapy. When appropriate, add pharmacotherapy for weight control per guidelines, and refer high-risk or refractory cases for metabolic/bariatric evaluation.

# ASSESSMENT OF RESPONSE

| Focus | What to do | Timing | Threshold / Decision rule | Next action |
|---|---|---|---|---|
| **Clinical monitoring** | Track weight, waist circumference, blood pressure, glycemic markers (A1c/FBG), lipid profile, patient-reported outcomes | Every **4–12 weeks** initially | Establish baseline and trend | Adjust plan based on changes |
| **Adherence & tolerability** | Review lifestyle adherence and medication side effects | **Each visit** | Barriers or intolerance identified | Address barriers, modify regimen, or provide targeted support |
| **Primary success metric** | Use **percent weight loss** as main metric | Check at **~3 months (~12 weeks)** | **≥5%** loss = adequate response | **Continue** current therapy |
| **Pharmacotherapy continuation & maintenance** | Continue anti-obesity medication if effective and well-tolerated; begin maintenance | If ≥5% at 3 months | Maintain therapy; **maintenance ≥1 year** (may be lifelong per clinical need) | Schedule relapse-prevention follow-ups |
| **Inadequate response** | If weight loss <5% or comorbidities persist/ worsen | Reassess at **~3 months** | **<5%** loss = inadequate | Intensify behavioral support, reassess dosing, **add/switch** pharmacotherapy |
| **Escalation / referral** | Re-evaluate eligibility for specialist care (e.g., metabolic surgery) | When BMI thresholds and burden persist despite optimized care | Meets local BMI/ comorbidity criteria | Refer to bariatric/ metabolic team; proactively manage any **weight regain** by revisiting core interventions |

# OBESITY IN PREGNANCY

The DHS 2016–2017 reports that about half of women aged 15–49 is overweight or obese (BMI ≥25). At booking: Measure weight and height with calibrated equipment, calculate BMI, and record in handheld notes and the electronic record.

| Classification | BMI (kg/m²) |
|---|---|
| **Underweight** | < 18.5 |
| **Normal range** | 18.5–24.9 |
| **Overweight** | 25.0–29.9 |
| **Obesity class I** | 30.0–34.9 |
| **Obesity class II** | 35.0–39.9 |
| **Obesity class III (morbid)** | 35.0–39.9 |
| **Super obesity (risk stratification)** | ≥ 50.0 |

**Note:** Calculate and classify BMI at the booking visit and document it.

## Recommended gestational weight gain

(aligned with widely used IOM ranges; apply clinical judgment for local protocols)

| Pre-pregnancy BMI | First trimester total gain (kg) | 2nd–3rd trimester rate (kg/week) | Recommended total gain (kg) |
|---|---|---|---|
| **Underweight (<18.5)** | ~1.0–2.0 | 0.44–0.58 | 12.5–18 |
| **Normal (18.5–24.9)** | 0.5–2.0 | 0.35–0.50 | 11.5–16 |
| **Overweight (25.0–29.9)** | ~0.5–2.0 | 0.23–0.33 | 7–11.5 |
| **Obesity (≥30.0)** | ~0.5–2.0 | 0.17–0.27 | 5–9 |

## Counsel before, between, and during pregnancies

- Preconception and inter-pregnancy: Measure weight and BMI, explain risks of obesity in pregnancy and childbirth, and support weight loss before conception. Weight reduction between pregnancies lowers risks of stillbirth, hypertensive disorders, and fetal macrosomia, and improves the chance of successful Vaginal Birth After Cesarean (VBAC).

- Antenatal booking: Record BMI, use the correct BP cuff size, and set individualized gestational weight gain goals.
- Public Health Unit and RHC: Offer structured lifestyle advice and refer to dietitians when needed.

| Maternal complications | Babies Fetal/Neonatal complications |
|---|---|
| **Miscarriage** | Congenital anomalies (including neural tube defects) |
| **Gestational diabetes** | |
| **Preeclampsia** | |
| **Venous thromboembolism** | |
| **Induction of labour** | Neonatal death |
| **Dysfunctional or prolonged labour** | Higher risk of childhood obesity and metabolic disorders |
| **Caesarean section** | |
| **Anaesthetic complications** | |
| **Postpartum haemorrhage** | |
| **Shoulder dystocia** | |
| **Wound infection** | |
| **Puerperal urinary tract infections** | |
| **Mortality** | |
| **Difficulty initiating and maintaining breastfeeding** | |

## Management of obesity in pregnancy

### Pre- and inter-conception

- Comprehensive health assessment and discussion of risks and options
- Personalized weight and lifestyle plan; dietitian referral when available
- Aim to normalize weight before conception
- Higher-dose folic acid as per protocol
- After bariatric surgery: micronutrient supplementation and monitoring
- Identify and optimize comorbidities, including diabetes

## Antenatal

- Early booking and documentation of pre-pregnancy BMI
- Use correctly sized BP cuff
- After bariatric surgery: continue micronutrient supplements and monitoring
- Discuss healthy eating, physical activity, and expected gestational weight gain; consider a weight-gain chart
- Assess risk and consider:
  - **Low-dose aspirin** for preeclampsia prevention when indicated
  - **VTE risk** and need for thromboprophylaxis
- Refer for psychosocial and mental health support when needed

## Labour and birth

- Early assessment of IV access
- If prophylactic antibiotics are indicated, consider higher dose
- Vigilance for shoulder dystocia and postpartum haemorrhage
- Active management of the third stage of labour

## Postpartum

- Monitor airway risks immediately after delivery if relevant
- Encourage early mobilization
- Assess VTE risk and provide thromboprophylaxis when indicated
- Monitor for wound infection
- Provide additional support for breastfeeding
- Continue healthy lifestyle support and weight-management counseling

# SURGICAL MANAGEMENT

Use Edmonton Obesity Staging (EOSS/EOSS-P) to gauge urgency and intensity—higher stage strengthens the indication even at lower BMI.

# Indications for metabolic/bariatric surgery (MBS)

| Group | Indication (BMI & context) | Examples of qualifying comorbidities |
|---|---|---|
| **Adults – International (ASMBS/IFSO 2022)** | BMI ≥35 kg/m² → surgery recommended, regardless of comorbidities | N/A |
| | BMI 30–34.9 kg/m² with metabolic disease (e.g., T2DM not at goal despite optimal therapy) → surgery can be considered | T2DM, HTN, dyslipidemia, OSA, NAFLD/ MASLD, severe OA |
| | T2DM specifically: consider MBS from BMI ≥30 kg/m² when hyperglycemia persists despite guideline-directed therapy | T2DM ± insulin use, high ASCVD risk |
| **Adults – South Asian/Asia-Pacific & MoH MV (lower thresholds used by many programs)** | BMI ≥37.5 kg/m² → surgery recommended | - |
| | BMI ≥32.5 kg/m² with ≥1 major comorbidity → surgery recommended/ appropriate | T2DM, HTN, OSA, NAFLD with fibrosis, debilitating OA |
| | T2DM not controlled despite optimal meds: consider BMI 27.5–32.4 kg/m² on a case-by-case basis in experienced centers | T2DM with high CVD risk, insulin requirement |
| **Adolescents (AAP/ASMBS)** | Class II obesity: BMI ≥120% of the 95th percentile or ≥35 kg/m² with a serious comorbidity → consider | T2DM, moderate–severe OSA, HTN, NAFLD with fibrosis, slipped capital femoral epiphysis |
| | Class III obesity: BMI ≥140% of the 95th percentile or ≥40 kg/m² (with or without comorbidities) → consider | As above; comorbidity not required |

## Common prerequisites

- Documented failure of structured, multidisciplinary non-surgical therapy (diet, activity, behavioral ± pharmacotherapy).
- Psychosocial readiness, ability to adhere to long-term follow-up, contraception planning for patients of child-bearing potential.
- Risk stratification: cardiopulmonary evaluation, OSA assessment, liver disease screen (MASLD/MASH), micronutrient baseline.

## Relative/absolute contraindications

- Untreated severe psychiatric illness or active substance use disorder.
- Unstable cardiac/respiratory disease prohibiting anesthesia.

- Inability to provide informed consent or to participate in long-term care.
- Pregnancy (defer); active GI cancer; uncontrolled coagulopathy.

## Endoscopic Sleeve Gastroplasty (ESG)

ESG is a minimally invasive endoscopic procedure that reduces gastric volume by suturing the stomach internally. Lower thresholds may apply for high-risk cases. Typical excess weight loss at one year is 13–16%, with durability shown up to five years. Improvements in metabolic parameters are common: up to 92% of patients experience better glycemic control, and roughly half see reductions in blood pressure and cholesterol. Long-term data are still accumulating, but early outcomes support ESG as an effective bridging option between medical therapy and more invasive surgery.

## Metabolic Surgery (Sleeve Gastrectomy, Roux-en-Y Gastric Bypass)

Metabolic surgery—primarily sleeve gastrectomy and Roux-en-Y gastric bypass—achieves weight loss by reducing gastric capacity and, in the case of bypass, rerouting nutrient flow to alter metabolism. These procedures typically produce 25–30% total body weight loss at 12 months with durable long-term results. Metabolic benefits—resolution or improvement of diabetes, hypertension, dyslipidemia, and cardiovascular risk—exceed those seen with non-surgical therapies. They do carry operative risks and demand lifelong follow-up to monitor and manage nutritional deficiencies, micronutrient supplementation, and potential complications.

## Referral

- Primary care: initial diagnosis, lifestyle program, basic metabolic workup, and first-line pharmacotherapy.
- Refer to secondary-level weight management clinics for patients with inadequate response, complex comorbidities, or needing advanced anti-obesity medications.
- Tertiary centers: multidisciplinary obesity clinics, consideration of metabolic/bariatric surgery, management of complications (e.g., advanced Metabolic Dysfunction–Associated Steatotic Liver Disease (MASLD), and access to specialist behavioral health, endocrinology, and nutrition. Ensure clear handoff with documentation of prior interventions, weight trajectory, and risk profile.

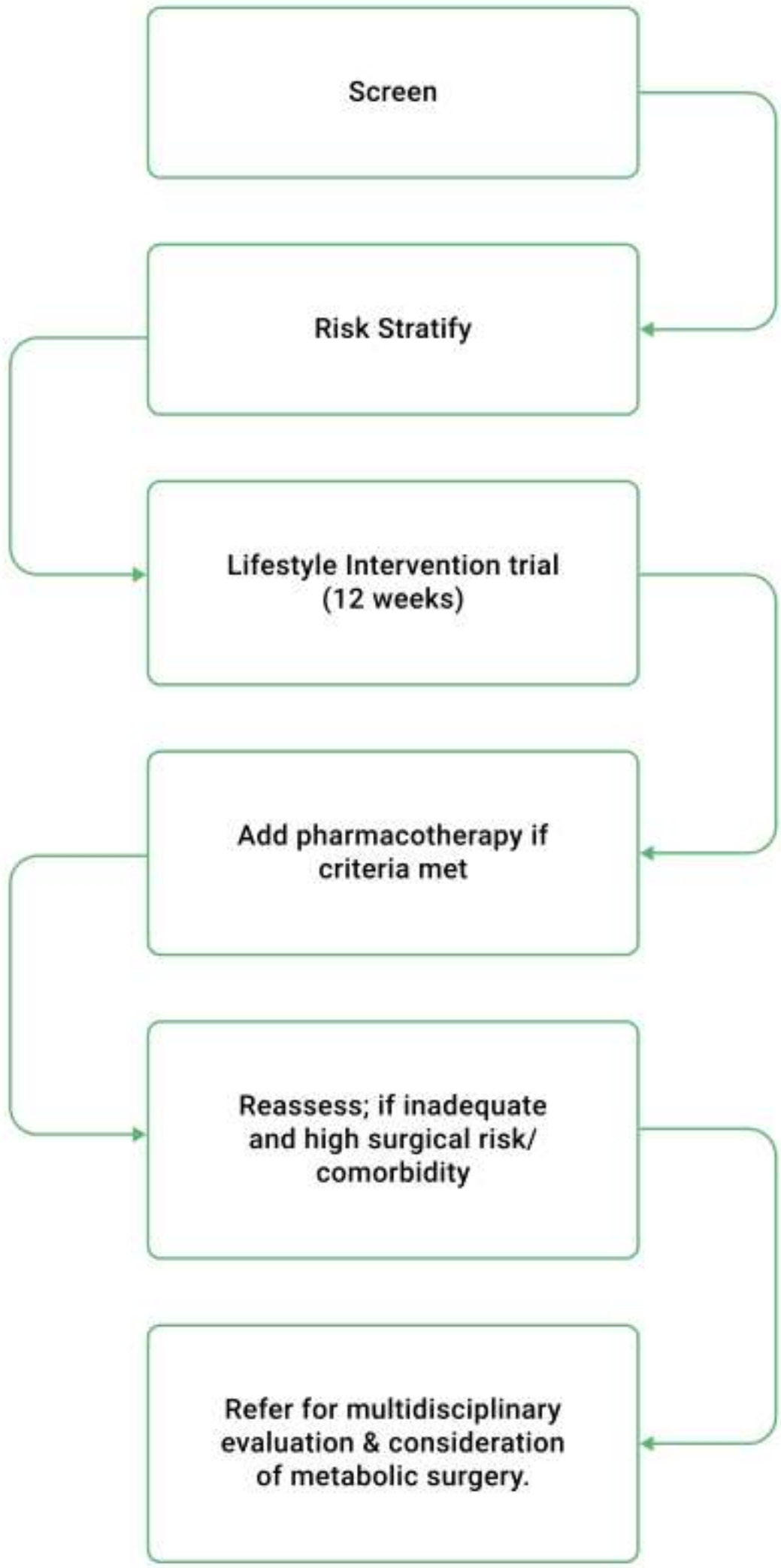
# COMPLICATIONS

Obesity is a complex chronic disease with far-reaching effects on nearly every organ system. Its complications arise from the combined impact of excess adiposity, metabolic dysfunction, and systemic inflammation. These consequences can be life-threatening, progressive, and multifactorial, affecting both physical and mental health.

| Complication / Domain | Why obesity increases risk | Key consequences / notes |
|---|---|---|
| **Cardiovascular disease** (MI, stroke, heart failure, atrial fibrillation) | Atherogenic dyslipidemia, hypertension, insulin resistance, systemic inflammation, structural cardiac remodeling | Higher rates of MI and stroke, HFpEF (Heart Failure with Preserved Ejection Fraction) /HFrEF (Heart Failure with Reduced Ejection Fraction) AF from atrial enlargement; risk rises with BMI and waist circumference (For details see heart failure guidelines) |
| **Type 2 diabetes mellitus** | Visceral adiposity → insulin resistance, β-cell stress, impaired glucose tolerance | Strongest modifiable driver of T2DM; risk escalates with BMI/waist; South Asians develop diabetes at lower BMI |
| **MASLD / MASH** | Hepatic fat accumulation with metabolic dysfunction and inflammation | Progression to fibrosis, cirrhosis, hepatocellular carcinoma; major cause of liver morbidity |
| **Obstructive sleep apnea (OSA)** | Peripharyngeal fat narrows airway and increases collapsibility during sleep | Repeated apneas, hypoxia, daytime sleepiness, higher CV risk; weight loss improves severity |
| **Osteoarthritis** | Increased mechanical load on knees/ hips plus inflammatory mediators | Chronic pain, reduced mobility, disability; weight reduction lowers joint load and symptoms |
| **Cancers** | Hyperinsulinemia, excess estrogens, chronic inflammation, adipokine imbalance | Higher risk of endometrial, postmenopausal breast, colorectal, esophageal adenocarcinoma, pancreatic, kidney, gallbladder cancers |
| **Mental health disorders** | Stigma, body image concerns, biological stress pathways; some psych meds cause weight gain | Higher prevalence of depression and anxiety; bidirectional relationship with obesity |
| **Reduced life expectancy** | Increased mortality from CVD, cancers, diabetes complications, respiratory disease | Severity and earlier onset of obesity shorten lifespan; risk rises progressively with BMI |
| **Impaired mobility and quality of life** | Pain, fatigue, deconditioning, functional limits | Loss of independence, work limitations, social impact, lower overall well-being |

# MANAGEMENT ALGORITHMS

## Obesity Management Algorithm



# PREVENTION & PROMOTION OF HEALTHY LIFESTYLE

Diet-related ill health is a pressing public health challenge. Key targets for halting the rise of obesity (and diabetes) as per Multi-sectoral Action Plan for the Prevention and Control of Noncommunicable Diseases in Maldives (2023-2031) By 2031, keep obesity prevalence at no more than 26% and diabetes at no more than 4.7%; i.e., "halt the rise" rather than reverse it. Keep obesity ≤26% and diabetes ≤4.7% by 2031 (halt the rise).
Core strategies

1. Use multi-level public health approaches—policy, environmental, community, organizational, clinical, and individual— to promote healthy diet, physical activity, and tobacco cessation across schools, workplaces, communities, and health services.
2. Improve food environments: reduce availability and marketing of high salt/sugar/fat foods, enhance access to nutritious options, and address food/nutrition insecurity.
3. Scale physical activity by making built environments safer and more accessible and embedding promotion in schools, workplaces, and communities.
4. Advance equity by tackling social determinants (economic, access, built environment, healthcare gaps) and closing clinic-to-community implementation gaps.
5. Align public health and healthcare efforts for coordinated prevention and management, reducing disparities in access and treatment.
6. Mainstream obesity prevention across sectors and create default healthy settings (workplaces, events) through policy, systems, and environmental changes.
7. Reframe communication to reduce weight stigma—focus on positive behavior change rather than labels.
8. Establish and sustain multisectoral governance and coordination to support these integrated actions.

# PATIENT EDUCATION

Explain Obesity is chronic, complex, and relapsing. Even a 5–10% weight loss improves sugar control, blood pressure, lipids, sleep, and joint pain. Change takes time; staying engaged matters more than short bursts. Teach:

- **Self-monitoring:** food logs, activity minutes/steps, weekly weight (and waist monthly).

- **Warning signs:** polyuria/polydipsia, unusual fatigue, chest pain, severe breathlessness, vision changes, persistent abdominal or right-upper-quadrant pain.
- **Medication safety:** how to take it, common side effects, when to call.
- **Mindset:** tackle stigma, set realistic, stepwise goals, celebrate non-scale wins (fitness, sleep, energy).

## Instructions to patient/caregiver

- Follow individualized calorie and activity plan. Do plan meals and keep healthy options visible. Don't crash diet or skip meals to "make up" for overeating. Don't let a single lapse derail the week—reset at the next meal.
- Record daily intake and weekly weight.
- Do move daily (aerobic + strength, as advised).
- Take prescribed medications as directed; report side effects (nausea, gallbladder pain). Don't double a dose if you miss one—follow the plan your clinician gave for missed doses.
- Attend scheduled follow-ups for labs and adjustment.
- Avoid quick fixes (Don't use unregulated weight-loss supplements; Don't chase detoxes/cleanses or extreme workouts after long gaps); sustain behavior change.
- Seek early care for symptoms of diabetes, chest pain, rapid leg swelling or one-sided calf pain with breathlessness, yellowing of eyes/skin, dark urine, or severe fatigue with right-upper-abdominal pain or worsening liver health.

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