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# Incretin Mimetics For **Obesity** Management In **Menopausal Women:** Insights From a Delphi Consensus

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*Indian Menopause Society & other Experts Opinion | 2026*



**Dr. Sudhaa Sharma**

President, IMS 2026-27

### **President's Message**

It gives me immense pleasure to present this important publication on “Obesity Management in Menopausal Women,” a topic of growing importance in women’s health.

Menopause is not merely the end of reproductive life; it is a significant metabolic transition. Declining estrogen levels, changes in body composition, reduced muscle mass, altered fat distribution, sleep disturbances and lifestyle changes can together increase the risk of weight gain, central obesity and related non-communicable diseases. Obesity, in turn, can amplify several menopausal concerns and adversely affect quality of life.

Management therefore needs to move beyond simply advising a woman to “lose weight.” It should encompass healthy nutrition, regular physical activity, preservation of muscle and bone, adequate sleep, psychological well-being and appropriate medical intervention when indicated. A personalized, evidence-based and compassionate approach is essential.

The Indian Menopause Society remains committed to promoting a holistic approach to midlife health and empowering women to navigate menopause with knowledge, dignity and vitality.

With warm regards,

**Dr. Sudhaa Sharma**  
President, IMS 2026-27

# Indian Menopause Society & Other Experts



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# INCRETIN MIMETICS FOR OBESITY MANAGEMENT IN MENOPAUSAL WOMEN: INSIGHTS FROM A DELPHI CONSENSUS

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## Background

Obesity is a chronic, relapsing, disease resulting from complex interactions among genetics, neurobiology, eating behaviour, environmental influences, access to healthy food, and wider commercial and social determinants. Its global burden has risen sharply over recent decades. The World Health Organization (WHO) estimates that 2.5 billion adults are overweight and 890 million obese worldwide, representing 43% of all adults, with women (44%) disproportionately affected. [1] In India, the burden is both enormous and epidemiologically distinctive. The ICMR-INDIAB-17 study estimated 254 million Indians have generalised obesity and 351 million have abdominal obesity, with abdominal obesity affecting 40% of Indian women overall, rising to 56.7% in the 40–49 year age group.[2,3] A substantial proportion of these women carry significant visceral adiposity at a normal or near-normal BMI, reflecting the Asian metabolic phenotype in which cardiometabolic complications develop at lower body weight thresholds than in Western populations. [4]

The menopausal transition (MT) represents a critical metabolic turning point that further amplifies obesity risk in women. Declining ovarian oestrogen leads to a preferential shift of fat from subcutaneous to visceral depots, a reduction in resting energy expenditure, impaired insulin sensitivity, and altered appetite regulation. These changes are not fully captured by BMI and therefore remain clinically under-recognized unless central adiposity and metabolic risk are assessed directly.[5,6] Women spend more

than 30 years in the postmenopausal state, making the cumulative cardiometabolic burden of menopausal visceral adiposity one of the most important and most under-addressed public health challenges in Indian women's health.[1]

Against this background, the Indian Menopause Society (IMS) developed the first evidence-based expert opinion on the role of Incretin Mimetic Drugs (IMDs), including semaglutide in the management of obesity during MT, with attention to not only body weight but also to cardiometabolic risk, body composition, quality of life, and safety in the Indian menopausal population.

## Methodology

A structured Delphi methodology was used to develop expert consensus statements on Glucagon Like Peptide (GLP-1) receptor agonists, focussing on semaglutide for obesity management in peri- and postmenopausal women. Delphi methods are widely used to generate structured expert consensus in clinical medicine, especially where randomized trial evidence in a broader population is comprehensive and robust or where evidence must be interpreted for a specific population.

### The process had two parts:

1. An evidence review phase, in which an up-to-date, evidence-based presentation covering the relevant clinical domains was prepared and presented to the expert panel.
2. An anonymous voting phase, in which panellists rated pre-specified statements on a 5-point Likert scale.

This approach ensured that voting was informed by current literature while also reflecting the panellists’ clinical experience with Indian menopausal women.

**Evidence Review Phase**

A systematic literature review was conducted with evidence sourced from published randomised controlled trials, systematic reviews and meta-analyses, large real-world cohort studies, and current national and international clinical guidelines. The review incorporated data from the STEP clinical programme, the SELECT cardiovascular outcomes trial, the SWAN Heart Study, the SEMALEAN body composition study, and the FOGSI Good Clinical Practice Recommendations on Obesity in Women (2026), among other sources. The evidence was compiled into a structured presentation.

**Anonymous Voting Phase**

The expert panel comprised 10 clinicians with expertise in menopausal medicine, gynaecology, endocrinology, and obesity management. Pre-specified clinical statements were developed based on the evidence review and grouped into five domains: (1) Menopause Transition (MT) and Burden of Obesity (2) Current Approaches in “Obesity and Menopause” and Unmet Needs (3) GLP-1 RAs and semaglutide in obesity and menopause; (4) benefits beyond weight loss with Semaglutide; and (5) safety and tolerability of GLP 1 RAs. Each statement was presented with supporting evidence, followed by anonymous voting on a 5-point Likert scale:

- 1) Strongly Disagree
- 2) Disagree
- 3) Neutral
- 4) Agree
- 5) Strongly Agree

**Grading of Statements**

After voting, statements were graded according to the proportion of panellists voting Agree or Strongly Agree (Table 1).

**Table 1. Grading system for expert consensus statements**

| Grade               | Level of Agreement | Description                                           |
|---------------------|--------------------|-------------------------------------------------------|
| Unanimous Consensus | 100%               | All 10 panellists voted Agree or Strongly Agree       |
| Strong Consensus    | ≥80%               | 8–10 panellists voted Agree or Strongly Agree         |
| Consensus           | ≥60%               | 6–7 panellists voted Agree or Strongly Agree          |
| No Consensus        | <60%               | Fewer than 6 panellists voted Agree or Strongly Agree |

## Section 1: Menopause Transition & Burden of Obesity

### Statement 1

**Menopause increases the risk of obesity by several folds.**

**Expert Agreement: 100% | Unanimous Consensus** (Strongly Agree 80% + Agree 20%; zero neutral, zero dissenting votes)

### Discussion

What makes menopause a uniquely potent driver of obesity risk is a specific biological programme triggered by the cessation of ovarian oestrogen production, one that operates independently of diet, physical activity, or prior weight history. Oestrogen simultaneously regulates adipose tissue distribution, skeletal muscle insulin sensitivity, and hypothalamic appetite control. When oestrogen declines, lipoprotein lipase activity shifts from subcutaneous to visceral fat depots promoting preferential visceral adipose tissue (VAT) accumulation; [7] myocellular Insulin Receptor Substrate (IRS)-1 insulin signalling is impaired, driving systemic insulin resistance); [8] and hypothalamic neuropeptide Y/ Agouti-related peptide (AgRP) appetite regulation is disrupted, increasing caloric intake and reducing energy expenditure.[9] These three converging mechanisms create a metabolic environment that actively promotes visceral fat gain even without any lifestyle change, explaining why many women gain weight around menopause despite unchanged eating and exercise habits.

Longitudinal DEXA and CT analyses demonstrate that fat mass increases markedly from the perimenopause onward, with the steepest rate of change in the two years surrounding the Final Menstrual Period (FMP), while lean mass simultaneously declines.[10] (Figure 1)

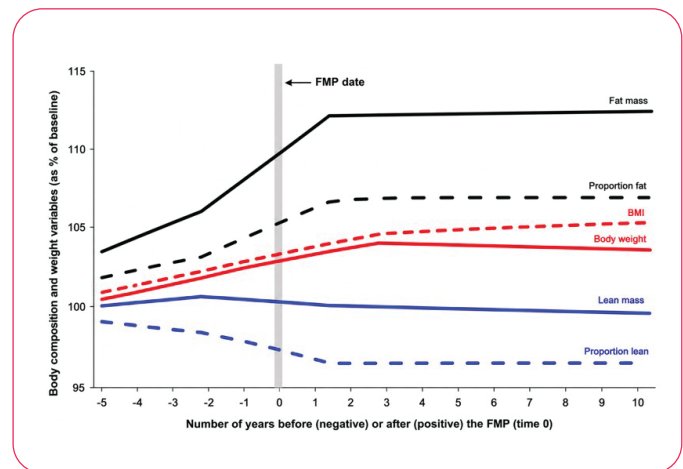


**Figure 1:** Model-predicted trajectories of body composition and body weight outcomes relative to the time prior to or after the FMP (First Menopausal Period) [10] Critically, BMI often remains stable because lean mass loss partially offsets fat mass gain in total body weight, masking the metabolic deterioration from standard anthropometric assessment.[11] This dissociation between BMI and fat mass trajectory is the mechanistic basis for the "normal weight obesity" phenotype prevalent in Indian menopausal women, and it establishes why the MT, not BMI, must be the trigger for metabolic evaluation.

Fat redistribution during the MT extends beyond the abdomen to include cardiac ectopic depots. The SWAN Cardiovascular Fat Ancillary Study (n=456, mean age 50.75 years) demonstrated that late peri- and postmenopausal women had significantly greater epicardial (EAT), paracardial (PAT), and aortic perivascular (PVAT) adipose tissue volumes compared with pre- and early perimenopausal women, independent of age and overall adiposity. [12] These metabolically active depots secrete pro-inflammatory adipokines and cytokines that exert local paracrine effects on the myocardium and coronary arteries, accelerating subclinical cardiovascular disease independently of systemic

adiposity.[5,12]

At the systemic level, visceral adipose tissue secretes TNF- $\alpha$ , IL-6, and monocyte chemoattractant protein-1 (MCP-1), driving low-grade inflammation, hepatic insulin resistance, endothelial dysfunction, and atherogenesis, while adiponectin paradoxically falls.[13] The result is a clinically observable acceleration of dyslipidaemia, C-reactive protein (CRP), blood pressure, and fasting insulin in the perimenopausal years, often before significant weight gain is apparent on examination.[5,13]

How does the evidence translate into the "several folds" increase in obesity risk stated in this consensus? Epidemiologic data indicate that central adiposity is approximately five times more likely after menopause, independent of BMI.[14] This menopausal shift toward central fat deposition is recognised by major public health bodies, including the World Obesity Federation and WHO, as an important driver of increased cardiometabolic risk in mid life women.[15]

SWAN longitudinal data show VAT increasing at 8.2% per year in late perimenopause and 5.8% per year in early postmenopause, rates that compound to a 40–50% increase in visceral fat area within the first five postmenopausal years alone.[16] (Figure 2)

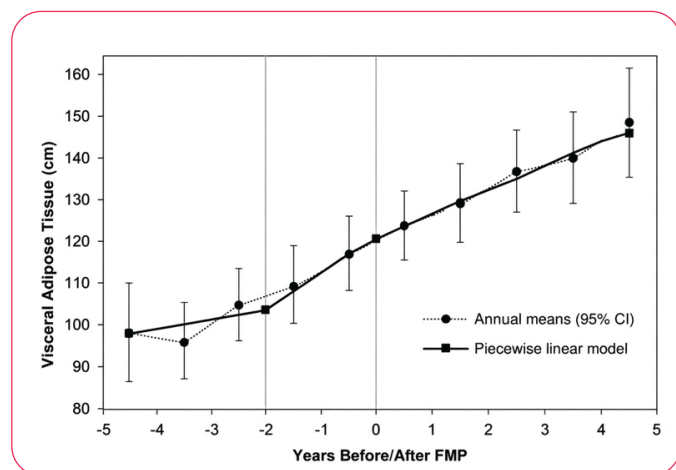


**Figure 2:** Means of Abdominal Visceral Adipose Tissue (VAT) in years around the final menstrual period (FMP) [16]

In Indian women, this risk is further amplified by the Asian metabolic phenotype. The same absolute visceral fat quantity confers greater insulin resistance, dyslipidaemia, and cardiovascular risk in South Asian compared with European women, meaning moderate VAT accumulation can translate into clinically significant metabolic disease at body weights that would not prompt concern in Western populations.[4]

*In this Delphi consensus, all ten expert panellists unanimously agreed, with 80% voting Strongly Agree that menopause increases the risk of obesity by several folds. The panel noted that this statement resonates directly with their daily clinical experience: peri- and postmenopausal women presenting with new-onset central adiposity, metabolic derangements, and weight gain despite unchanged lifestyle are a consistent and increasing feature of Indian gynaecological and endocrinology practice. The biology is the explanation and recognising it changes how these women are assessed and managed.*

## Statement 2

**Indian peri- and postmenopausal women should undergo proactive screening for central obesity and metabolic syndrome even when BMI appears near normal.**

**Expert Agreement: 100% | Unanimous Consensus** (Strongly Agree 70% + Agree 30%; zero neutral, zero dissenting)

## Discussion

As established in Statement 1, menopausal fat redistribution from subcutaneous to visceral depots occurs largely independent of changes in total body weight. The clinical consequence in India is particularly acute: South Asian women develop insulin resistance, dyslipidaemia, type 2 diabetes,

and cardiovascular disease at substantially lower BMI thresholds than Western populations, the well-documented Asian BMI paradox.[4] Revised Asian-Indian definitions classify overweight at BMI  $\geq 23$  kg/m<sup>2</sup> and obesity at  $\geq 25$  kg/m<sup>2</sup>, and abdominal obesity at waist circumference of  $\geq 80$  cm in women. [4] Standard WHO BMI thresholds, which most Indian clinicians still use in practice, will systematically misclassify a large proportion of metabolically high-risk menopausal women as normal.

The scale of this misclassification is captured by national data. The NFHS-5 (2019–2021) analysis found abdominal obesity (by waist circumference criteria) in 40% of Indian women overall, rising to 49.3% in the 30–39 year age group and 56.7% in the 40–49 year age group, the perimenopausal decade. [17] The more recent NFHS-6 (2023–24) confirms this trend is worsening: overall overweight/obesity among Indian women aged 15–49 years rose to 30.7%, up from 24.0% in NFHS-5, though an age-stratified abdominal obesity breakdown from NFHS-6 is not yet published. [18] The ICMR-INDIAB-17 study estimated 351 million Indian adults have abdominal obesity, many of whom would be classified as metabolically normal by BMI alone.[2] A substantial proportion of these women have normal or near-normal BMI, the "invisible" high-risk phenotype that proactive screening is designed to detect.

The urgency of early screening is further supported by evidence that metabolic deterioration begins in late perimenopause, before BMI changes are apparent. Subclinical atherosclerosis markers worsen in parallel with visceral fat accumulation regardless of total body weight, making the MT itself the appropriate screening trigger, not a threshold BMI.[5]

A practical screening protocol requires no specialist infrastructure. Waist circumference, waist-to-hip ratio, fasting glucose, HbA1c, and fasting lipid profile should be standard components, not optional add-ons, of the routine menopausal

health consultation for every Indian peri- and postmenopausal woman.[4,19]

*In this Delphi consensus, all ten expert panellists unanimously agreed that proactive metabolic screening, including for central obesity and metabolic syndrome should be conducted in all Indian peri- and postmenopausal women regardless of BMI. The panel noted that in their clinical experience, a substantial proportion of menopausal women presenting with apparently normal body weight are found to have significant abdominal adiposity, insulin resistance, and dyslipidaemia on targeted assessment, a pattern consistent with the Indian population data.*

### Statement 3

**Lifestyle modification should remain the first-line strategy for obesity management during the menopausal transition.**

**Expert Agreement: 100% | Unanimous Consensus** (Strongly Agree 100%; zero neutral, zero dissenting)

### Discussion

Structured lifestyle intervention is endorsed as the cornerstone of obesity management by all major guidelines, including American Association of Clinical Endocrinology (AACE) 2025, Endocrine Society of India (ESI) 2025 update, and FOGSI GCPR 2026.[20,21] The components are well defined: caloric restriction targeting approximately 1,200–1,500 kcal/day, individualized to body weight and activity, aerobic exercise >150 minutes/week, resistance training > twice weekly to preserve lean mass, and structured behavioural counselling addressing eating behaviours, and self-monitoring.[22] However, physiology actively defends against weight loss, particularly in

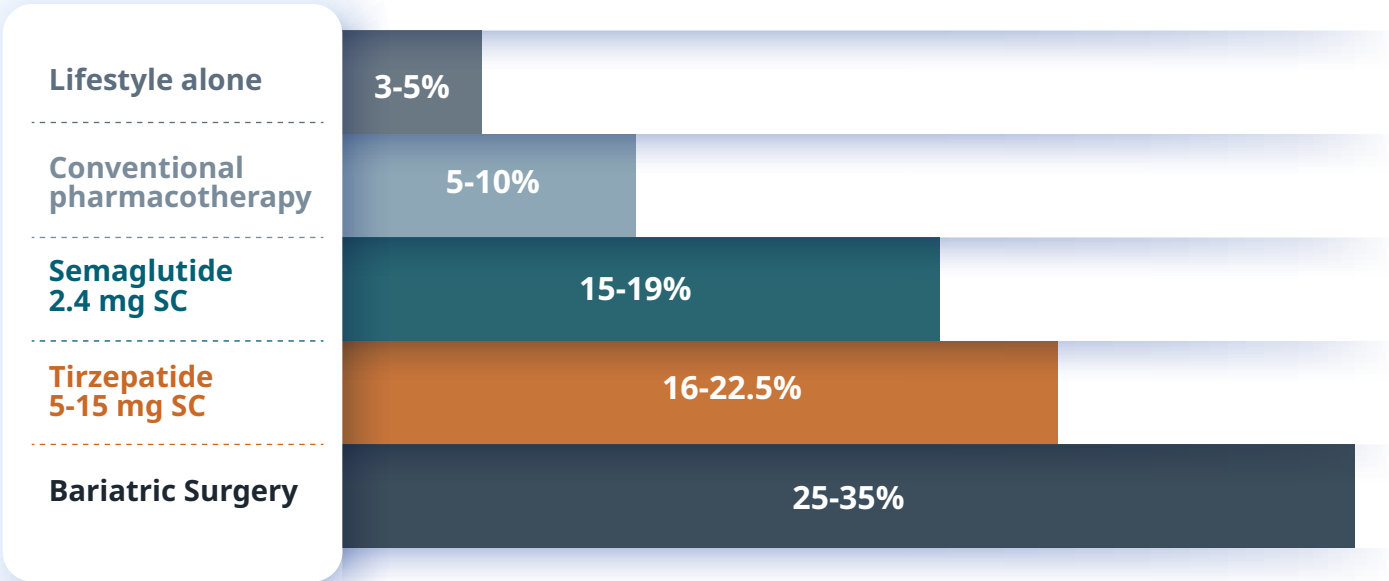
menopausal women. As caloric restriction is maintained, ghrelin rises promoting hunger; leptin falls reducing satiety; and resting metabolic rate decreases as an adaptive response, making weight maintenance progressively harder over time.[23] This is not failure of patient effort but physiological adaptation, and clinicians must explicitly counsel and empathize with patients on these biological barriers to avoid attributing limited weight loss to behavioural lapses. The limitations of available pharmacotherapy in India further contextualise the unmet need. Agents approved by CDSCO, Orlistat (2004), Sibutramine (withdrawn post-2010 for

cardiovascular risk), and Naltrexone-Bupropion, achieve only 5–10% weight loss and carry significant side-effect profiles and have demonstrated no cardiovascular outcome benefit.[24] Incretin Mimetic Drugs like semaglutide bridge this therapeutic gap, delivering 15–19% weight loss (four to five times the efficacy of lifestyle alone) with a proven cardiovascular outcome benefit, establishing it as the evidence-based adjunct when lifestyle is insufficient. [24,25] The treatment gap in obesity management, from lifestyle modification to pharmacotherapy and bariatric surgery, is summarized in Table 2 and illustrated visually in Figure 3.

**Table 2. Treatment gap in obesity management, from lifestyle modification to pharmacotherapy and bariatric surgery**

| Treatment Strategy                                            | Typical Mean Weight Loss | Cardiovascular Outcome Benefit                           | Remarks                                                                                                                                                                  |
|---------------------------------------------------------------|--------------------------|----------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Lifestyle modification alone                                  | 3–5%                     | None demonstrated                                        | Foundation of all management; insufficient alone for most patients                                                                                                       |
| Conventional pharmacotherapy (Orlistat, Naltrexone-Bupropion) | 5–10%                    | None demonstrated                                        | Limited efficacy; significant side-effect profiles                                                                                                                       |
| Semaglutide 2.4 mg SC (Once-weekly)                           | 15–19%                   | 20% MACE reduction (SELECT trial, NEJM 2023)             | First anti-obesity medication with proven CV outcome benefit; CDSCO-approved in India                                                                                    |
| Tirzepatide 5-15 mg SC (Once-weekly)                          | 16–22.5%                 | Not yet established -SURMOUNT- MMO ongoing (NCT05556512) | Greater mean weight loss than semaglutide (SURMOUNT-5: 20.2% vs 13.7%); dedicated CV outcomes trial still in progress; CDSCO- approved for T2DM and obesity (March 2025) |
| Bariatric surgery                                             | 25–35%                   | CV benefit demonstrated                                  | Appropriate for selected patients; not widely accessible in India                                                                                                        |

**Figure 3: Weight loss comparison across treatment strategies for obesity management (visual summary of Table 2).**



In Indian practice, the challenge is amplified by cultural dietary norms, socioeconomic constraints on physical activity, and the psychological vulnerabilities of the MT. Counselling must therefore be culturally sensitive and realistic, reframing the clinical conversation from behavioural failure to hormonal-metabolic disease requiring multimodal management.

*In this Delphi consensus, all ten expert panellists, unanimously voted Strongly Agree that lifestyle modification must remain the first-line strategy. The panel emphasised that this position does not diminish the role of pharmacotherapy; rather, it establishes lifestyle intervention as the non-negotiable foundation upon which all other treatments are built.*

## Section 2: GLP-1 Receptor Agonists in Obesity and Menopause

### Statement 4

**GLP-1 RAs should be considered an important pharmacologic option for managing obesity and cardiometabolic risk in peri- and postmenopausal women.**

**Expert Agreement: 90% | Strong Consensus** (Strongly Agree 20% + Agree 70%; Disagree 10%)

### Discussion

Glucagon-like peptide-1 (GLP-1) is a gut-derived incretin hormone secreted by intestinal L-cells in response to nutrient ingestion. It stimulates glucose-dependent insulin secretion, suppresses glucagon, slows gastric emptying, and, critically for obesity management, reduces appetite and food intake by acting on GLP-1 receptors in both the hypothalamus (homeostatic appetite regulation) and hindbrain regions including the area postrema and nucleus tractus solitarius (hedonic appetite and reward-related eating).[26] By targeting both physiological hunger and food craving, semaglutide addresses the full spectrum of mechanisms driving excess caloric intake, a breadth of action that older anti-obesity medications lack.

The relevance to menopausal women is supported by converging evidence specific to this population. The STEP programme enrolled predominantly female participants: 74.1% of STEP 1, 81.0% of STEP 3, and 77.6% of STEP 5, with mean participant ages of 46–55 years, directly reflecting the perimenopausal and early postmenopausal demographic.[27] Furthermore, semaglutide produces a statistically greater weight loss in females than males (MD 1.04 kg; 95% CI 0.45–1.63), and a real-world data in 7,847 people with T2DM confirmed significantly more women achieved  $\geq 5\%$  and  $\geq 10\%$  weight loss targets compared to men.[28,29] Women with menopausal

obesity therefore appear to be preferential responders.

The FOGSI GCPR 2026 further endorses GLP-1 RAs as first-line pharmacotherapy in postmenopausal women with BMI  $\geq 27$  kg/m<sup>2</sup> with comorbidities or  $\geq 30$  kg/m<sup>2</sup>. [30] New data from the European Congress 2026 show  $>41\%$  of premenopausal women on semaglutide (7.2 mg) achieved  $\geq 25\%$  weight loss and SELECT post-hoc analysis confirms cardiovascular risk reduction across all menopausal stages.[31]

The single dissenting vote (10%) in this consensus reflected a valid scientific request: the need for RCTs specifically designed with menopausal status as a stratification variable. This is an important research priority the panel endorsed, while noting that the existing female-predominant STEP data, sex-specific efficacy advantage, and SELECT cardiovascular outcome findings already provide compelling support for clinical use in this population.

*In this Delphi consensus, 90% of expert panellists agreed that GLP-1 receptor agonists should be considered an important pharmacologic option for managing obesity and cardiometabolic risk in peri- and postmenopausal Indian women. The panel noted that, in their respective clinical practices and within the current Indian regulatory framework, many members support the use of semaglutide in this population where clinically indicated, given the convergence of compelling mechanistic rationale, female-predominant trial data. In line with the Government of India circular (March 2026), panellists highlighted that semaglutide use should be initiated and managed by Endocrinologists and Internal Medicine Specialists, and for specified indications by Cardiologists only.*

## Statement 5

**Semaglutide provides clinically meaningful reductions in body weight and visceral adiposity and long-term CV benefits.**

**Expert Agreement: 70% | Consensus**  
(Strongly Agree 20% + Agree 50%; Neutral 30%)

### Discussion

The STEP phase 3 programme provides the most comprehensive efficacy dataset of any anti-obesity medication to date. In STEP 1 (n=1,961, 74.1% women, mean age 46 years), semaglutide 2.4 mg produced 14.9% mean weight loss versus 2.4% with placebo at 68 weeks; 50.5% achieved  $\geq 15\%$  weight loss and more than one in three achieved  $\geq 20\%$ .<sup>[32]</sup> STEP 5 extended follow-up to 104 weeks (n=304), confirmed 15.2% sustained weight loss vs 2.6% with placebo, with maintained cardiometabolic risk marker improvements.<sup>[33]</sup> STEP-UP demonstrated 18.7% weight loss with semaglutide 7.2 mg versus 3.9% with placebo at 72 weeks, with one in three participants (33.2%) achieving  $\geq 25\%$  weight loss with the 7.2 mg dose, compared with one in five (16.7%) on 2.4 mg and none on placebo, extending the dose-response relationship and offering a stepped-up option for women desiring greater weight loss based on baseline body weight.<sup>[34]</sup> The semaglutide 7.2 mg dose is not currently approved by CDSCO in India; all CDSCO-approved semaglutide formulations in India are presently licensed up to the 2.4 mg maintenance dose only.

Beyond weight, semaglutide produced comprehensive cardiometabolic risk reduction across STEP trials: waist circumference decreased by up to 16.3 cm; systolic blood pressure by up to 5.3 mmHg; triglycerides by up to 22%; CRP by up to 63.4%; and HbA1c by up to 1.9%.<sup>[35,36]</sup> These effects are directly relevant to the menopausal woman, in whom all of these

parameters typically worsen as a consequence of oestrogen deficiency, making semaglutide a pharmacotherapy that simultaneously targets multiple pathophysiological drivers of menopausal cardiometabolic risk.

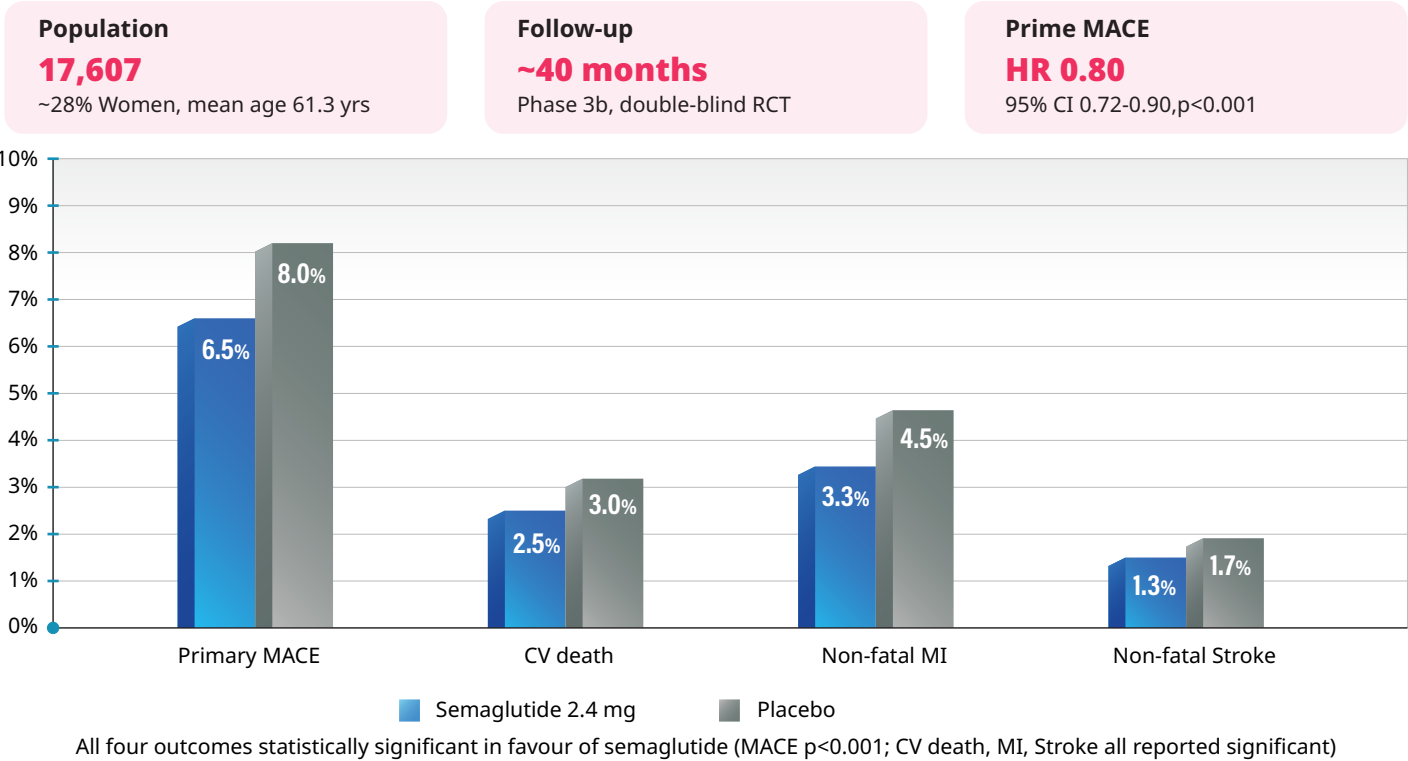
The SELECT trial (n=17,604 adults without diabetes, approximately 4,900–5,000 women) delivered landmark cardiovascular outcome evidence. Semaglutide 2.4 mg reduced major adverse cardiovascular events (MACE), cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke, by 20% versus placebo (HR 0.80; 95% CI 0.72–0.90;  $p < 0.001$ ) over a mean follow-up of approximately 40 months.<sup>[25]</sup>

**This makes semaglutide the first and only anti-obesity medication with a proven cardiovascular outcome benefit,** an unprecedented achievement of particular importance for menopausal women, in whom cardiovascular disease is the leading cause of mortality. (Table 3 and Figure 4)

Table 3. SELECT trial cardiovascular outcomes: semaglutide 2.4 mg in adults with overweight/obesity and established cardiovascular disease

| Parameter                | Semaglutide 2.4 mg                                       | Placebo | Result                                                 |
|--------------------------|----------------------------------------------------------|---------|--------------------------------------------------------|
| Trial design             | Randomised, double-blind, placebo-controlled             | -       | Phase 3b cardiovascular outcomes trial                 |
| Population               | 17,604 adults with overweight /obesity, CVD, no diabetes | -       | ~28% women; mean age 61.3 years                        |
| Primary endpoint (MACE)* | 6.5%                                                     | 8.0%    | HR 0.80 (95% CI 0.72–0.90); p<0.001                    |
| CV death                 | 2.5%                                                     | 3.0%    | HR 0.85; statistically significant                     |
| Non-fatal MI             | 3.3%                                                     | 4.5%    | HR 0.72; statistically significant                     |
| Non-fatal stroke         | 1.3%                                                     | 1.7%    | HR 0.73; statistically significant                     |
| Mean weight loss         | –9.4%                                                    | +0.2%   | Concurrent with CV benefit                             |
| Mean follow-up           | ~40 months                                               | -       | First anti-obesity drug with proven CV outcome benefit |

Figure 4: SELECT trial — cardiovascular outcomes with semaglutide 2.4 mg



The 30% neutral votes reflected the expert panel's scientifically measured position. The SELECT trial did not specifically enrol or stratify by menopausal status, and a dedicated menopausal cardiovascular outcome trial or pre - specified menopausal - stage subgroup analysis would further strengthen this statement. This is a legitimate research priority, not a doubt about semaglutide's fundamental efficacy or the validity of these data.

*In this Delphi consensus, 70% of expert panellists agreed that semaglutide provides clinically meaningful reductions in body weight, visceral adiposity, and long-term cardiovascular benefit. The panel uniformly recognised the SELECT trial as a pivotal advance, and those who voted neutral expressed anticipation for menopause - stratified analysis of cardiovascular outcome data rather than doubt about semaglutide's clinical value.*

## Statement 6

**Combination of hormone therapy with semaglutide may enhance body weight loss outcomes in appropriately selected menopausal women**

**Expert Agreement: 60% | Consensus**  
(Strongly Agree 10% + Agree 50%; Neutral 40%)

## Discussion

Semaglutide addresses appetite dysregulation and promotes weight loss via GLP-1 receptor-mediated mechanisms, but does not restore oestrogen, the upstream hormonal deficiency driving menopausal fat redistribution. Menopause Hormone Therapy (MHT) or HT, by restoring physiological oestrogen levels, may attenuate the primary pathophysiological driver that semaglutide cannot, creating a mechanistically rational complementary combination. [6]

The key clinical evidence comes from a retrospective cohort of 106 postmenopausal women treated with semaglutide. Those concurrently receiving HT achieved significantly greater total body weight loss at every measured time points:  $7\pm3\%$  vs  $5\pm4\%$  at 3 months ( $p=0.01$ ),  $13\pm6\%$  vs  $9\pm5\%$  at 6 months ( $p=0.01$ ),  $15\pm6\%$  vs  $10\pm6\%$  at 9 months ( $p=0.02$ ), and  $16\pm6\%$  vs  $12\pm8\%$  at 12 months ( $p=0.04$ ).[37] A significantly higher proportion of HT users achieved  $\geq 5\%$  and  $\geq 10\%$  weight loss thresholds. These differences persisted after adjusting for age, BMI, race, and comorbidities, strengthening the inference of a true additive effect.

Regarding drug safety, the British Menopause Society (2025) reviewed available pharmacokinetic & pharmacodynamic data & concluded that transdermal oestrogen-based HRT is unaffected by concurrent semaglutide use and may be the preferred route in this setting. However, BMS noted a theoretical pharmacokinetic concern with oral HRT: delayed gastric emptying from semaglutide may reduce absorption of oral progestogens, potentially compromising endometrial protection, though no semaglutide-specific outcome data confirming this exist. [38] The AMOGS Delphi consensus 2026 included a specific Grade U (Unanimous) statement endorsing semaglutide in perimenopausal, menopausal, and postmenopausal women with obesity. This consensus has not yet been published and is currently available only as an unpublished society document.

The 40% neutral votes reflect the scientifically appropriate recognition that the current evidence base, though biologically plausible and clinically encouraging, rests primarily on a single retrospective cohort study. A prospective RCT, specifically examining the combination of HT and semaglutide in menopausal women, with body composition and cardiometabolic outcomes as pre-specified endpoints, is needed before this can be elevated to a strong consensus position.

*In this Delphi consensus, 60% of expert panellists agreed that HT combined with semaglutide may enhance weight loss in appropriately selected menopausal women. The panel strongly emphasised the word "appropriately selected", HT remains an individualised clinical decision requiring careful risk-benefit assessment for each patient, and its combination with semaglutide should always be guided by established HT indications and contraindications.*

### Statement 7

**Clinically meaningful weight loss with semaglutide can be achieved across all menopausal stages, including postmenopause.**

**Expert Agreement: 60% | Consensus**  
(Strongly Agree 30% + Agree 30%; Neutral 40%)

### Discussion

The STEP programme's predominantly female participants (77.6–81.0% women, mean ages 46–55 years across STEP 3–5), span the perimenopausal and early postmenopausal range, providing directly relevant large-scale evidence.[27] Real-world data extend this to broader menopausal populations. In a retrospective cohort of 1,882 women with overweight or obesity, mean body weight decreased by 10.73% with semaglutide, 82.4% achieved  $\geq 5\%$ , 52.1% achieved  $\geq 10\%$ , and 23.2% achieved  $\geq 15\%$  weight loss.[39]

Postmenopausal efficacy is specifically confirmed by the cohort study reported in Statement 6 (n=106 postmenopausal women), which showed a mean 12% weight loss at 12 months even in the non-HT subgroup.[37] A direct comparison of semaglutide 1 mg in postmenopausal versus premenopausal women found comparable percentage weight reductions at 4 months

(5.8% vs 5.1%) and similar fat mass reductions, despite postmenopausal women having significantly higher baseline body weight and fat mass, suggesting that the more adverse metabolic environment of postmenopause does not attenuate semaglutide's efficacy. [40]

European Congress 2026 SELECT post-hoc data confirm cardiovascular risk reduction across all menopausal stages, numerically 42% in perimenopausal and 13% in postmenopausal women versus placebo, confirming that the cardiovascular benefits of semaglutide are not restricted to younger menopausal women.[41]

The 40% neutral votes reflect an appropriate scientific caveat. Prospective RCTs specifically designed and powered to evaluate semaglutide in pre-specified menopausal subgroups, peri-, early postmenopause ( $\leq 5$  years of FMP), and late postmenopause ( $> 5$  years of FMP) are not yet available.

*In this Delphi consensus, 60% of expert panellists agreed that clinically meaningful weight loss with semaglutide can be achieved across all menopausal stages, including postmenopause. The panel noted that, consistent with available real-world and cohort data, postmenopausal women including those of advanced age who have previously been unable to achieve clinically significant weight loss with lifestyle intervention alone, have demonstrated meaningful reductions in body weight when semaglutide is used as an adjunct to, rather than a replacement for, comprehensive lifestyle measures.*

## Statement 8

**QoL improvement to be considered an important therapeutic outcome when evaluating semaglutide treatment in women with obesity during menopause.**

**Expert Agreement: 90% | Strong Consensus** (Strongly Agree 50% + Agree 40%; Neutral 10%)

### Discussion

Obesity during menopause impairs quality of life (QoL) across multiple overlapping domains that standard weight metrics do not capture. Beyond physical consequences, menopausal obesity worsens vasomotor symptoms (hot flushes are more frequent and more severe in women with higher BMI),[42] disrupts sleep architecture and promotes obstructive sleep apnoea (OSA),[43,44] contributes to depressive symptoms and anxiety through hormonal fluctuation and adipokine-mediated neuroinflammation,[45,46] and reduces physical mobility, sexual health and self-esteem. The QoL burden of menopausal obesity is therefore multidimensional and systematically underestimated in clinical practice.

Sleep disturbance deserves particular emphasis. Up to 60% of perimenopausal women report sleep difficulties.[43,44] Obesity drives this through both mechanical pathways (increased pharyngeal collapsibility promoting OSA) and metabolic pathways (adipokine dysregulation, systemic inflammation impairing sleep architecture). Emerging evidence suggests that semaglutide improves sleep quality and reduces OSA severity, primarily through weight reduction and metabolic improvement, offering a clinically important QoL benefit beyond the scale.[47,48]

Postmenopausal women frequently use stress eating as a coping strategy for depressive mood, a maladaptive cycle that worsens both obesity and psychological

distress.[49–51] Semaglutide's hedonic appetite effects, reducing food craving and improving eating control, may interrupt this cycle, providing psychological benefit beyond caloric restriction. Real-world data from ObesityWeek® 2025 (Atlanta) confirmed that approximately 50–70% of patients on semaglutide achieved meaningful improvement across multiple QoL domains, including physical functioning, mood, and weight-related self-assessment.

The expert panel strongly endorsed (90% agreement) the incorporation of QoL as a primary or co-primary outcome in all future clinical evaluations of semaglutide in menopausal women with obesity. This is not merely a recommendation for research design; it is a call for practising clinicians to routinely assess and document QoL as part of the treatment monitoring framework, rather than focusing exclusively on weight metrics.

*In this Delphi consensus, 90% of expert panellists, (the second-highest agreement level in the exercise), affirmed that QoL improvement is an important therapeutic outcome when evaluating semaglutide in menopausal women with obesity. The panel reflected that their patients consistently report improvements in energy, sleep, mood, and daily functioning with semaglutide treatment that are at least as meaningful as the number on the scale, a patient-centred perspective that should inform how we define and measure treatment success.*

## Section 3: Benefits Beyond Weight Loss

### Statement 9

**Semaglutide-associated weight loss may contribute to improvement in obesity-related urinary incontinence in menopausal women.**

**Expert Agreement: 50% | Consensus**  
(Strongly Agree 20% + Agree 30%; Neutral 50%)

#### Discussion

Urinary incontinence (UI) affects an estimated 30–50% of menopausal women and is one of the most prevalent yet underreported conditions in this age group.[52] Obesity drives UI through elevated intra-abdominal pressure: excess visceral and abdominal fat chronically loads the bladder and pelvic floor, overwhelming the urethral sphincter (stress UI) and driving bladder overactivity (urge UI).[53] The MT compounds this through oestrogen-mediated reduction in urethral mucosal coaptation and pelvic floor muscle tone, creating a convergence of mechanical and hormonal UI drivers pronounced in the obese menopausal woman.

The clinical evidence that weight loss improves UI is strongly supported by evidence. The PRIDE and ICI trials demonstrated that a 5–10% weight reduction produces clinically meaningful reductions in UI frequency and severity, with a dose-response relationship; greater weight loss correlates with greater UI improvement.[54] Given that semaglutide achieves 15–19% weight loss, well above the proven therapeutic threshold, its potential impact on pelvic floor outcomes is both clinically plausible and mechanistically well-grounded.

The 50% neutral vote in this consensus reflects that the panel members were unanimous in their view that the evidence is

promising and the mechanism is sound, but prospective controlled trials with UI as a primary endpoint in menopausal populations are needed before a strong consensus can be reached. This statement is an evidence-based hypothesis identifying a research question, not as an expression of doubt about semaglutide's pelvic floor potential.

*In Delphi consensus, 50% of expert panellists agreed that semaglutide-associated weight loss may contribute to improvement in obesity-related urinary incontinence in menopausal women. The panel called for prospective Indian trials evaluating pelvic floor outcomes, including stress and urge UI severity, pad use, and quality of life as pre-specified endpoints in future menopausal obesity pharmacotherapy studies.*

### Statement 10

**Semaglutide therapy may provide additional neurological and psychological benefits including lower risk of migraine and depression in menopausal women with obesity.**

**Expert Agreement: 40% | Consensus**  
(Strongly Agree 10% + Agree 30%; Neutral 60%)

#### Discussion

Migraine affects 18–25% of women versus 6% of men and undergoes a complex transition during perimenopause. Oestrogen fluctuations during perimenopause often worsen migraine frequency before gradual decline in postmenopause.[55,56] Obesity independently increases migraine risk through central sensitisation, hypothalamic

dysfunction, and adipokine-mediated neuroinflammation.[57] Depression is similarly prevalent during the MT, driven by oestrogen's effects on serotonin, noradrenaline, and dopamine signalling, compounded by sleep disruption, and psychosocial stressors, with obesity further increasing depressive risk through systemic inflammation.[58]

GLP-1 receptors are expressed widely in the central nervous system, including the hypothalamus, brainstem, limbic system, and prefrontal cortex, regions critical to pain modulation, mood regulation, reward processing, and stress responses. This neuroanatomical distribution provides a direct mechanistic basis for neuropsychological effects of semaglutide, independent of weight loss alone. Animal and early neuroimaging data suggest GLP-1 receptor agonism modulates hypothalamic activity, reduces neuroinflammation, and alters reward circuit sensitivity, all of which are plausible pathways to both migraine and depression benefit.[59]

A large real-world observational study involving more than 34,000 women across the menopausal transition, presented at the 33rd European Congress on Obesity (ECO 2026), found that semaglutide use was associated with a 42–45% lower risk of migraine beginning approximately six months after treatment initiation, compared with MHT alone, with the association persisting throughout one year of follow-up. Semaglutide use was also associated with a 25% lower risk of depression. As these findings are derived from observational data presented at a scientific meeting, prospective studies are needed to confirm these associations. [60]

The 60% neutral vote correctly reflects the strength of current evidence. These data, while impressive in magnitude, derive from a single observational study with inherent confounding, presented in abstract form ahead of full peer-reviewed publication. A prospective RCT with pre-specified neuropsychiatric endpoints is required

before this can be endorsed strongly into formal clinical guidance. The appropriate clinical position is that these findings are mechanistically plausible and statistically compelling and need more evidence.

*In this Delphi consensus, 40% of expert panellists agreed that semaglutide may provide neurological and psychological benefits in menopausal women with obesity. The panel unanimously identified this as one of the most exciting and clinically important emerging areas in semaglutide research, one that, if confirmed with more evidence in prospective trials, would significantly expand the clinical case for semaglutide in menopausal women beyond weight loss and cardiovascular outcomes.*

#### Statement 11

**Semaglutide preferentially reduces fat mass over lean mass, supporting its role in improving body composition in women with obesity.**

**Expert Agreement: 80% | Strong Consensus** (Strongly Agree 10% + Agree 70%; Neutral 20%)

#### Discussion

The question of lean mass preservation during pharmacological weight loss is of particular clinical relevance in menopausal women, who face age-related sarcopenia, progressive loss of skeletal muscle mass and function, which is accelerated by oestrogen deficiency and inadequate physical activity.[61] A weight loss intervention that effectively reduces fat mass but accelerates lean mass loss could worsen sarcopenia, impair functional capacity, increase fall risk, and reduce bone density in this already vulnerable population. The body composition effects of semaglutide therefore warrant careful, evidence-based evaluation.

The most comprehensive and directly relevant data come from the SEMALEAN study, a prospective cohort study that evaluated body composition longitudinally during semaglutide therapy. Key findings were: semaglutide produced 10% total weight loss at 7 months and 13% at 12 months. Fat mass decreased by 14% at 7 months and 18% at 12 months, while lean mass declined modestly at 7 months (–3 kg) but then stabilised, demonstrating preferential fat-over-lean reduction. Handgrip strength, a validated functional marker of muscle performance, increased by approximately 4.5 kg, suggesting preserved and possibly improved neuromuscular function despite the caloric deficit. Sarcopenic obesity prevalence fell from 49% to 33% at 12 months, a clinically meaningful reduction in one of the most metabolically dangerous phenotypes in menopausal women. [62]

Resting energy expenditure (normalised to lean mass) increased between 7 and 12 months in the SEMALEAN cohort, a finding that distinguishes semaglutide from caloric restriction alone, where adaptive metabolic suppression typically reduces lean-mass-normalised energy expenditure. This suggests that semaglutide may improve metabolic efficiency rather than suppressing metabolism, an important advantage for long-term weight maintenance.[62]

The panel strongly emphasised that these favourable body composition outcomes are contingent on two co-interventions that must be actively recommended alongside semaglutide in menopausal women: adequate protein intake (target  $\geq 1.2$  g/kg ideal body weight/day) to support lean mass retention, and structured resistance training at least twice weekly to preserve muscle mass and neuromuscular function.

*In this Delphi consensus, 80% of expert panellists agreed that semaglutide preferentially reduces fat mass over lean mass in women with obesity. The panel noted that the SEMALEAN data on handgrip*

*strength improvement and sarcopenic obesity reduction are of particular clinical significance for their postmenopausal patients, in whom body composition, rather than total body weight, is the primary determinant of functional health and cardiometabolic risk.*

## Section 4: Safety and Tolerability of GLP-1 Receptor Agonists

### Statement 12

**Semaglutide has a predictable and manageable safety profile for obesity treatment in peri- and postmenopausal women.**

**Expert Agreement: 50% | Consensus**  
(Strongly Agree 10% + Agree 40%; Neutral 50%)

#### Discussion

Evidence from multiple RCTs and extensive real-world data, characterises a predictable, dose-dependent, and predominantly gastrointestinal adverse effect profile for semaglutide.[27] Nausea, vomiting, diarrhoea, and constipation are the most common adverse events, typically mild-to-moderate in severity, transient, peak during dose escalation, and diminish substantially once the maintenance dose is established.[27,32] Treatment discontinuation due to GI events occurred in approximately 4–7% of semaglutide-treated patients across the STEP programme, far below the 18–20% discontinuation rates reported with older anti-obesity agents such as orlistat. (Dose escalation management is addressed separately in Statement 13.)

Data from the SUSTAIN 1–5 pooled elderly subgroup analysis (n=854, age ≥65 years), directly relevant to older postmenopausal women, confirmed that weight reductions were comparable to those in younger patients, with no increased risk of severe or confirmed hypoglycaemia and no new safety signals specific to the elderly population.[63] Absolute contraindications are personal or family history of medullary thyroid carcinoma (MTC) or Multiple Endocrine Neoplasia type 2 (MEN2). Clinical caution is warranted in patients with a history of pancreatitis, gallbladder disease, or gastroparesis, and baseline evaluation should include glycaemic status, renal

function, and retinopathy screening in women with diabetes.

The most discussed emerging safety signal is the potential association between semaglutide and non-arteritic anterior ischaemic optic neuropathy (NAION). A recent systematic analysis [64] and the North American Neuro-Ophthalmology Society (NANOS) Consensus Statement [65] concluded that current evidence does not establish a confirmed causal association between semaglutide and NAION, though monitoring in patients with established optic nerve disease or high IOP risk factors is recommended.

The 50% neutral vote in this consensus reflects the panel's appropriately cautious position on the NAION signal, and the limited long-term safety dataset specifically in women >60 years.

*In this Delphi consensus, 50% of expert panellists agreed that semaglutide has a predictable and manageable safety profile in peri- and postmenopausal women. Those who voted neutral reflected scientific caution regarding the NAION signal and the limited postmenopausal-specific long-term safety dataset, not a belief that the safety profile is unacceptable. The panel recommended that a formal pre-treatment evaluation protocol, including a structured monitoring framework, be incorporated into every semaglutide prescription in older menopausal women.*

## Statement 13

**Careful dose escalation and patient counselling are important to reduce GI side effects, improve tolerability and long-term adherence to semaglutide therapy.**

**Expert Agreement: 100% | Unanimous Consensus** (Strongly Agree 56% + Agree 44%; zero neutral, zero dissenting)

### Discussion

Gastrointestinal tolerability is the single most important determinant of long-term adherence to semaglutide therapy, and adherence, in turn, is the primary determinant of sustained weight loss outcomes. The relationship between weight loss and treatment duration with semaglutide is near-linear up to the maintenance dose, meaning that patients who discontinue early due to GI intolerance forfeit the majority of their potential therapeutic benefit.[66] Understanding and actively managing GI adverse effects is therefore not merely a comfort issue; it is a clinical priority that directly affects outcomes.

The approved titration protocol for semaglutide subcutaneous injection 2.4 mg provides the pharmacological framework for GI management: initiation at 0.25 mg once weekly for 4 weeks, followed by stepwise escalation every 4 weeks, to 0.5 mg, then 1.0 mg, then 1.7 mg before reaching the 2.4 mg maintenance dose. This slow escalation allows the gastrointestinal tract to adapt progressively to the gastric motility effects of semaglutide, substantially reducing the severity of nausea and vomiting. Data from the STEP programme confirm that the incidence and severity of GI adverse events peak during dose escalation and diminish significantly once the maintenance dose is established.[67] Clinicians should not accelerate the escalation schedule to achieve faster weight loss, as this predictably

increases GI intolerance and risk of early discontinuation.

Patient counselling is equally critical and should address: dietary modifications (smaller portion sizes; low-fat, low-spice meals; avoiding high-fat or ultra-processed trigger foods; avoiding carbonated beverages); behavioural measures (eating slowly; sitting upright for at least 30 minutes after meals; maintaining regular meal timing); hydration (adequate fluid intake to prevent nausea and constipation); and expectation setting (clearly communicating that initial GI discomfort is expected, transient, and manageable, and does not indicate treatment failure or intolerance requiring discontinuation).[68]

Two additional counselling imperatives deserve specific attention in menopausal women. First, protein adequacy: reduced food intake during semaglutide therapy must not compromise protein ingestion, as protein is essential for lean mass preservation in a population already at risk of sarcopenia. A target of  $\geq 1.2$  g protein/kg ideal body weight/day is recommended. Second, resistance exercise: structured strength training must be recommended alongside semaglutide to preserve muscle mass and function; this is particularly important in postmenopausal women who face age-accelerated sarcopenia.[62]

*In this Delphi consensus, all ten expert panellists unanimously agreed, (the joint strongest result in the exercise) that careful dose escalation and patient counselling are critical to GI tolerability and long-term adherence. The panel collectively reflected that inadequate patient education about expected GI effects is the most common and most preventable cause of premature semaglutide discontinuation in their clinical practice, a problem that structured counselling protocols can largely eliminate.*

## Statement 14

**Current evidence supports semaglutide use in older women with obesity, including postmenopausal populations, with appropriate monitoring**

**Expert Agreement: 70% | Consensus**  
(Strongly Agree 20% + Agree 50%; Neutral 30%)

### Discussion

Postmenopausal women with obesity represent a high-need clinical population that has historically been underrepresented in pharmacotherapy trials and underserved in clinical guidelines focused predominantly on younger, reproductive-age patients. Yet it is precisely this group, in whom decades of postmenopausal visceral fat accumulation have driven the highest absolute cardiometabolic, musculoskeletal, and functional risk, that may derive the greatest absolute benefit from effective anti-obesity pharmacotherapy.

The direct evidence for semaglutide in older women comes from multiple sources. The SUSTAIN 1–5 pooled elderly subgroup analysis (n=854, age ≥65 years) demonstrated that semaglutide produced body-weight reductions comparable to those in younger patients, with no increased risk of severe hypoglycaemia and no new or unexpected safety signals.[63] The STEP programme's predominantly female, midlife-aged participants (mean 46–55 years) provide directly applicable data for peri- and early postmenopausal women.[27] The real-world cohort study of 106 postmenopausal women confirmed effective and sustained weight loss in postmenopausal women across a range of ages, including those on concurrent HRT.[37]

The AMOGS Delphi consensus on obesity in women (2026) reached Grade U (unanimous) agreement on a dedicated statement that semaglutide should be considered for weight management in perimenopausal,

menopausal, and postmenopausal women with obesity, given its demonstrated benefits on weight reduction and quality of life. (Unpublished data) The FOGSI GCPR on Obesity in Women (2026) similarly endorsed GLP-1 receptor agonists, particularly semaglutide 2.4 mg, as a first-line pharmacological option for sustained weight loss in postmenopausal women.[30]

Monitoring in older postmenopausal women should be structured and comprehensive: weight and BMI trajectory; glycaemic status (particularly relevant as age increases T2DM risk); gastrointestinal tolerability; renal function (which may reduce drug clearance in older patients); nutritional and protein status (to prevent sarcopenic weight loss); muscle mass and sarcopenia assessment using validated tools (e.g., SARC-F questionnaire, grip strength measurement); and bone health, with DEXA scanning where indicated. A multidisciplinary team approach, integrating gynaecology, endocrinology, cardiology, and dietetics, is recommended for complex older patients.

*In this Delphi consensus, 70% of expert panellists agreed that current evidence supports semaglutide use in older postmenopausal women with obesity, with appropriate monitoring. Those who voted neutral reflected a scientifically cautious position: need more evidence in women aged >65 years, and the need for careful attention to sarcopenia, bone health, and nutritional adequacy in late postmenopause. The panel recommends that future studies specifically enrol and stratify older postmenopausal women as a defined subgroup, with functional outcomes alongside weight and cardiometabolic measures as co-primary endpoints.*

## Stepwise Clinical Algorithm for Obesity Management in Menopausal Indian Women

**Table 4. Stepwise clinical algorithm for the management of obesity in peri- and postmenopausal Indian women**

| Step                                                 | Action                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                | Key Clinical Points                                                                                                                                                                                                                                                                                                                                                                                                      |
|------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Step 1</b><br>Initial Assessment                  | <ul style="list-style-type: none"> <li>Measure BMI (Asian cutoffs: Overweight <math>\geq 23</math> kg/m<sup>2</sup>; Obese <math>\geq 25</math> kg/m<sup>2</sup>)</li> <li>Record waist circumference, WHR, BP</li> <li>Determine menopausal status (peri-/postmenopause)</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                  | <ul style="list-style-type: none"> <li>Do not rely on BMI alone, assess central adiposity</li> <li>Waist circumference <math>\geq 80</math> cm indicates abdominal obesity in Indian women</li> <li>Menopause itself warrants metabolic screening regardless of BMI</li> </ul>                                                                                                                                           |
| <b>Step 2</b><br>Metabolic Evaluation                | <ul style="list-style-type: none"> <li>Order Fasting glucose, PPBS, HbA1c, lipid profile, LFTs, RFTs, TSH</li> <li>Screen for sarcopenia and osteoporosis (especially postmenopause)</li> <li>Consider additional tests as indicated: liver ultrasound (MASLD), anemia workup, sleep study, echocardiography</li> </ul>                                                                                                                                                                                                                                                                                                                                               | <ul style="list-style-type: none"> <li>Baseline cardiometabolic profiling is essential prior to pharmacotherapy</li> <li>Sarcopenia and bone health assessment are critical in late postmenopause</li> </ul>                                                                                                                                                                                                             |
| <b>Step 3</b><br>Lifestyle Intervention (First-Line) | <ul style="list-style-type: none"> <li>Caloric restriction: <math>\sim 1,200</math>–<math>1,500</math> kcal/day (individualised)</li> <li>Aerobic activity: <math>\geq 150</math> min/week (moderate) or <math>\geq 75</math> min/week (vigorous) <ul style="list-style-type: none"> <li>Example: brisk walking 30 min / day, 5 days/week (5–6 km/h)</li> <li>Alternatives: jogging, cycling, swimming, dancing, water aerobics</li> </ul> </li> <li>Resistance training: <math>\geq 2</math> sessions/week (all major muscle groups)</li> <li>Behavioural strategies: self-monitoring, goal-setting, stimulus control, relapse prevention, social support</li> </ul> | <ul style="list-style-type: none"> <li>Preferred starting exercise: brisk walking (safe, accessible, culturally acceptable); progress as tolerated</li> <li>Address biological barriers: metabolic adaptation, increased hunger, oestrogen deficiency – related visceral fat gain</li> <li>Expected weight loss with lifestyle alone: <math>\sim 3</math>–<math>5\%</math>; add pharmacotherapy if inadequate</li> </ul> |

| Step                                   | Action                                                                                                                                                                                                                                                                                                                                  | Key Clinical Points                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             |
|----------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Step 4</b><br>Pharma co-therapy     | <ul style="list-style-type: none"> <li>Initiate if BMI <math>\geq 30</math> kg/m<sup>2</sup> or <math>\geq 27</math> kg/m<sup>2</sup> with comorbidities</li> <li>Start semaglutide 2.4 mg SC once weekly (preferred agent)</li> <li>Initiation by physician / gynaecologist; refer to endocrinologist or MDT when indicated</li> </ul> | <ul style="list-style-type: none"> <li>Pharmacotherapy is adjunct to lifestyle, not a replacement</li> <li>Screen for diabetic retinopathy in patients with diabetes before initiation</li> <li>Contraindications: hypersensitivity, history/risk of pancreatitis or cholecystitis, diabetic retinopathy (caution), MTC/MEN2</li> <li>Refer for bariatric evaluation if BMI <math>\geq 40</math> or inadequate response (&lt;5% weight loss)</li> </ul>                                                                                                                                                                                                         |
| <b>Step 5</b><br>Semaglutide Titration | <ul style="list-style-type: none"> <li>Dose escalation: 0.25 → 0.5 → 1.0 → 1.7 → 2.4 mg (every 4 weeks)</li> </ul>                                                                                                                                                                                                                      | <ul style="list-style-type: none"> <li>Do not accelerate titration</li> <li>Monitor GI tolerability at each step</li> <li>Reinforce protein intake (<math>\geq 1.2</math> g/kg IBW/day) and resistance training</li> </ul>                                                                                                                                                                                                                                                                                                                                                                                                                                      |
| <b>Step 6</b><br>MHT Consideration     | <ul style="list-style-type: none"> <li>Evaluate eligibility based on individual risk-benefit profile</li> <li>Indications: VMS, GUSM, bone loss prevention</li> <li>Prefer transdermal oestrogen when combined with semaglutide</li> </ul>                                                                                              | <ul style="list-style-type: none"> <li>Oral progestogen absorption may be reduced due to delayed gastric emptying</li> <li>Do not delay semaglutide initiation pending MHT decision</li> <li>MHT use should be individualised</li> <li>Clinical note <ul style="list-style-type: none"> <li>Distinct mechanisms: MHT (oestrogen / progesterone receptors) vs semaglutide (GLP-1 gut-brain axis)</li> <li>No clinically significant pharmacokinetic interaction, especially with transdermal MHT</li> <li>Combination is safe and potentially synergistic, with greater weight loss and cardiometabolic benefit vs semaglutide alone.[37]</li> </ul> </li> </ul> |

| Step                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     | Action                                                                                                                                                                                                                                                                                                                                                        | Key Clinical Points                                                                                                                                                                                                                                                          |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Step 7</b><br>Monitoring                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              | <ul style="list-style-type: none"> <li>Follow-up every 3–6 months: weight, BMI, waist circumference, glycaemia, GI tolerance, comorbidities</li> <li>Assess muscle mass, bone health, and nutritional/protein status</li> <li>Evaluate symptom relief and monitor side effects in women on MHT</li> <li>Involve multidisciplinary team when needed</li> </ul> | <ul style="list-style-type: none"> <li>Long-term therapy is usually required; discontinuation leads to weight regain (STEP 4 evidence)</li> <li>Obesity management should not be isolated from menopause care; an integrated approach optimises clinical outcomes</li> </ul> |
| <b>BMI:</b> Body Mass Index   <b>WHR:</b> Waist-to-Hip Ratio   <b>BP:</b> Blood Pressure   <b>PPBS:</b> Postprandial Blood Sugar   <b>HbA1c:</b> Glycated Hemoglobin<br><b>LFTs:</b> Liver Function Tests   <b>RFTs:</b> Renal Function Tests   <b>TSH:</b> Thyroid-Stimulating Hormone   <b>MASLD:</b> Metabolic Dysfunction-Associated Steatotic Liver Disease   <b>SC:</b> Subcutaneous   <b>MTC:</b> Medullary Thyroid Carcinoma   <b>MEN2:</b> Multiple Endocrine Neoplasia Type 2   <b>IBW:</b> Ideal Body Weight   <b>MHT:</b> Menopausal Hormone Therapy   <b>VMS:</b> Vasomotor Symptoms   <b>GUSM:</b> Genitourinary Syndrome of Menopause   <b>GI:</b> Gastrointestinal   <b>MDT:</b> Multidisciplinary Team. |                                                                                                                                                                                                                                                                                                                                                               |                                                                                                                                                                                                                                                                              |

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## Strengths and Limitations

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This consensus has several strengths that support its clinical relevance and scientific credibility. It is the first Indian expert opinion to address semaglutide and GLP-1 receptor agonists specifically in the context of menopause, filling an important gap at the intersection of two highly prevalent conditions in women. The recommendations were built on a structured evidence review presented to the panel before voting, ensuring that the process was evidence-informed rather than based on unaided opinion. All 14 statements achieved at least consensus, with no dissenting votes recorded, reflecting strong convergence among the experts. The scope is also broad, covering screening, lifestyle management, pharmacotherapy, quality of life, urinary incontinence, neuropsychological outcomes, body composition, and safety of semaglutide in peri- and postmenopausal women. In addition, the consensus is clearly framed for India by incorporating the Asian BMI paradox, ICMR-INDIAB, and NFHS-5/NFHS-6 data, while its transparent methodology and published voting patterns allow readers to judge the strength of each recommendation. It is also broadly aligned with current Indian guidance, including the FOGSI GCPR on Obesity in Women (2026) and the AMOGS Delphi Consensus on Obesity in Women (2026) (Unpublished).

Several limitations should also be considered. The panel was relatively small, with 10 clinicians drawn mainly from gynaecology and endocrinology, while representation from bariatric surgery, dietetics, and geriatric medicine was limited. Most supporting evidence comes from studies in predominantly female, middle-aged populations rather than trials designed specifically around menopausal status. India has contributed substantially to the global semaglutide clinical development programme: 13 of 129 STEP 1 trial sites were located in India. (<https://clinicaltrials.gov/study/NCT03548935>) Despite this participation, none of the publicly available trial reports have published a menopause-stratified sub-group analysis of Indian participants. Direct evidence specific to Indian menopausal women therefore remains absent from the public domain. Some of the more emerging statements, particularly those on hormone therapy combination, urinary incontinence, and neuropsychological outcomes, rely mainly on retrospective cohorts, real-world data, or conference abstracts and therefore require confirmation in prospective randomised trials. Finally, the evidence base is rapidly evolving, so several statements may need future refinement as new data on higher-dose semaglutide, menopause-stratified analyses, neuropsychiatric outcomes, and Indian real-world experience become available.

## Indian Menopause Society Takeaway: Role of Incretin Mimetics/GLP-1 RAs in Obesity and Menopause

- Menopause significantly increases the risk of obesity and cardiometabolic complications, necessitating early and proactive screening, particularly for central obesity even in women with near-normal BMI.
- Lifestyle modification remains the cornerstone and first-line strategy for obesity management during the menopausal transition.
- GLP-1 receptor agonists, including semaglutide, are recognised as important pharmacological options for managing obesity and cardiometabolic risk in peri- and postmenopausal women.
- Semaglutide demonstrates clinically meaningful reductions in body weight, visceral adiposity, and favourable cardiovascular outcomes, with additional benefits on body composition through preferential fat mass reduction.
- Quality of life should be considered a key therapeutic endpoint alongside weight loss when evaluating treatment outcomes in menopausal women.
- Combination approaches, including semaglutide with hormone therapy, show potential but require further evidence for routine clinical adoption.
- Evidence for benefits beyond weight loss (e.g., urinary incontinence, neurological and psychological outcomes) remains mainly from real-world /observational data and needs further prospective investigation.
- Semaglutide has a generally acceptable safety profile; however, careful dose escalation and patient counselling are essential to optimise tolerability and long-term adherence.
- Use of semaglutide in older and postmenopausal women is supported with appropriate clinical monitoring.
- Overall, expert consensus is strong, with highest agreement on foundational care principles and emerging but evolving evidence in adjunct and extended pharmacotherapeutic benefits.

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