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Assessment, differential diagnosis, and initial clinical evaluation of the pediatric patient with obesity: An Obesity Medical Association (OMA) Clinical Practice Statement 2022

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ABSTRACT

Background: The Obesity Medical Association (OMA) Clinical Practice Statement (CPS) on the assessment, differential diagnosis, and initial clinical evaluation of pediatric patients with obesity is intended to provide clinicians with an overview of clinical practices applicable to children and adolescents with body mass indexes greater than or equal to the 95th percentile for their ages, particularly those with adverse consequences resulting from increased body mass. The information in this CPS is based on scientific evidence, supported by the medical literature, and derived from the clinical experiences of members of the OMA.
Methods: The scientific information and clinical guidance in this CPS is based upon referenced evidence and derived from the clinical perspectives of the authors.
Results: This OMA Clinical Practice Statement on assessment, differential diagnosis, and initial clinical evaluation of pediatric patients with obesity provides clinical information regarding classification of children and adolescents with overweight or obesity, differential diagnoses to consider, and a roadmap for the initial clinical evaluation.
Conclusions: This OMA Clinical Practice Statement on assessment, differential diagnosis, and initial clinical evaluation of pediatric patients with obesity is an overview of current recommendations. Assessment of pediatric patients with obesity is the first step in determining treatments leading to the improvement of the health of children and adolescents with obesity, especially those with metabolic, physiological, and psychological complications.

1. Introduction

The purpose of the CPS on assessment, differential diagnosis, and initial clinical evaluation of pediatric patients with obesity is to provide clinicians with a tool to assess children with obesity. The OMA is an organization of providers in the field of obesity medicine dedicated to the comprehensive care of patients with obesity. OMA members are physicians, nurse practitioners, physician assistants, and other healthcare providers who take a comprehensive, evidence-based approach to treating obesity. This approach is comprised of the four pillars of nutrition, physical activity, behavior, and medication. While it is hoped many clinicians may find the recommendations in this CPS helpful, the final decision regarding the optimal care of the patient with overweight or obesity is dependent upon the individual clinical presentation and the judgment of the clinician who is tasked with directing a treatment plan that is in the best interest of the patient.

2. Assessment of pediatric patients

Weight assessment of children is age dependent. For children less than two years of age, weight percentiles and weight for length charts are used. Clinicians can choose between growth charts provided by the Centers for Disease Control and Prevention (CDC) or the World Health Organization (WHO). The CDC charts are based on a cohort of mainly white American children who were mostly non-breastfed. The WHO charts are based on children from diverse racial and ethnic backgrounds, mostly breastfed [1–3].

For children and adolescents ages 2–20, body mass index (BMI) percentile is used. For children and adolescents ages 2–20 with BMIs greater than the 95th percentile, use percent of the 95th percentile [4].

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Categories of weight status are as follows: less than 5th percentile is underweight, 5–84th percentile is healthy weight, 85–94th percentile is overweight, 95th percentile to 119% of the 95th percentile is Class 1 obesity, 120–139% of the 95th percentile or a BMI greater than 35 kg/m^2 and less than 39 kg/m^2 is Class 2 obesity, and greater than 140% of the 95th percentile or BMI greater than 40 is Class 3 obesity [5,6]. Any BMI greater than or equal to 120% of the 95th percentile is severe obesity. Different charts are used to track BMI for up to the 95th percentile and above the 95th percentile [4,6–8]. Body mass index categories and percentiles are shown in Fig. 1.

BMI z-scores, or BMI standard deviation scores, can be used to adjust for child age and sex [9] but should not be used to assess BMI changes among children and adolescents with BMIs greater than or equal to 120% of the 95th percentile. High BMI z-scores are compressed into a narrow range, which can result in a clinically significant reduction in BMI being represented by a small reduction in BMI z-score. Change in the percent of the 95th percentile is the preferred metric to use in the management of children and adolescents with severe obesity [7,8]. BMI charts for children and adolescents with severe obesity are shown in Fig. 2.

A BMI of 35 kg/m^2 is a higher threshold than a BMI greater than or equal to 120% of the 95th percentile among most children, but it is a somewhat lower threshold (and therefore expands the population that is categorized as severely obese) among boys approximately 18 years of age and older and girls approximately 16 years of age and older [7,10]. Overall, assessments should consider age as well as BMI status. The top 10 takeaway messages for assessing children and adolescents with obesity are shown in Table 1.

3. Obesity as a disease

Obesity has been recognized as a disease by the American Medical Association since 2013 [11]. Response to the disease of obesity manifests differently in children and adolescents than in adults. A breakdown of responses into categories of endocrine or immune response, physical response, and psychological response is a useful way to work through the clinical presentation [12].

Endocrine and immune responses are due to adiposopathy or "sick fat." Common phenomena include impaired fasting glucose, metabolic syndrome, hypertension, menstrual dysfunction, early onset or a delay in onset of puberty, nonalcoholic fatty liver disease, dyslipidemia, insulin resistance, type 2 diabetes, increased uric acid, microalbuminuria, gynecomastia, cholecystitis, and accelerated growth [13,14].

Physical response is attributed to fat mass disease. Asthma, immobility, lipomastia, tissue compression (obstructive sleep apnea, gastroesophageal reflux, hypertension), tissue friction (intertrigo), stress on weight bearing joints, slipped capital femoral epiphysis, Blount's disease, scoliosis, and orthopedic disorders are all possible findings [15].

Psychological responses can impact quality of life and include isolation from peers, decreased ability to participate in normal childhood activities, victimization (bullying, emotional/physical abuse, or neglect), lack of social age-appropriate relationships, anxiety, depression, binge eating disorder, night eating disorder, and bulimia [15,16].

4. Differential diagnosis of children and adolescents with obesity

The diagnosis of exogenous obesity in children and adolescents due to nutritional origin is a diagnosis of exclusion. Clinicians must consider endocrinopathies and syndromal obesity as well as both monogenic and polygenic etiologies.

For approaching the differential diagnosis of childhood obesity, it is important to assess linear growth. Prepubertal and pubertal children with exogenous obesity (i.e., related to unhealthful nutrition and physical inactivity) usually have accelerated or consistent linear growth, in contrast to those with an endocrinopathy, who frequently present with a decrease in linear growth [17]. Precocious puberty can occur in both those with exogenous obesity and those with an endocrinopathy. The diagnosis of precocious puberty is made in females younger than 8 years of age with Tanner II breast development and in boys younger than 9 years of age with testicular enlargement greater than 4 cc [18]. In these children, a bone age assessment may show an advancement of bone age as compared to chronological age of 1–2 years or more. If a child with obesity presents with a decrease in linear growth, the clinician should consider possible hypothalamic/pituitary dysfunction (with growth hormone deficiency), hypercortisolism (consider a dexamethasone suppression test or 24-h urinary free cortisol), hypothyroidism [check thyroid-stimulating hormone (TSH), conduct a free thyroxine (T4) test], or genetic syndromes [19,20].

Syndromal obesity is obesity accompanied by other behavior, functional, or anatomic abnormalities, such as hyperphagia, cognitive delay, dysmorphic features, and organ-specific disorders. The clinician should consider syndromal obesity in children with obesity who present with a developmental delay. Developmental delay can be associated with decreased linear growth, and the evaluation of these children is dependent on presentation and family history. A child presenting with severe obesity (>120% of the 95th percentile) before 5 years of age may indicate a genetic etiology. If hyperphagia and/or a family history of extreme obesity is present, genetic testing is recommended [21]. The clinical history may include food-seeking behavior such as searching for or stealing food, waking at night to eat, and eating food left behind by others. Neurological causes should be excluded. These dysfunctional behaviors result from disruption of the hypothalamic pathways involved in the regulation of energy balance. Genetic testing for multi-gene panels and referral to a geneticist should be considered [19,22–24]. A summary of differential diagnoses regarding childhood obesity is shown in Fig. 3.

5. Focused Review of Systems

Children with obesity are commonly seen in primary care for a problem other than obesity. Therefore, specific symptoms secondary to obesity must be carefully assessed. Children may or may not complain of symptoms, as many have been living with symptoms for years and are not aware of what life is like without symptoms. Fig. 4 identifies symptoms with accompanying related co-morbidities.

6. Diagnostic workup

The diagnostic workup is determined by the age at presentation of the

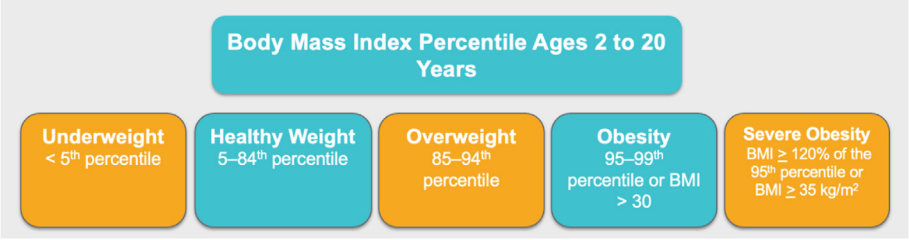

Fig. 1. Body Mass Index Categories in Children and Adolescents Ages 2–20. BMI categories are shown for children ages 2–20 who are underweight, at a healthy weight, overweight, obese, and severely obese [6]. Note that not all patients with BMI in the 85th percentile or above have excess adiposity, and many children and adolescents with BMIs below the 5th percentile are healthy and do not need treatment. The CDC recommends using the WHO growth charts to monitor growth for infants and children ages 0–2 years of age in the U.S. and using the CDC growth charts for children ages 2 years and older [2].

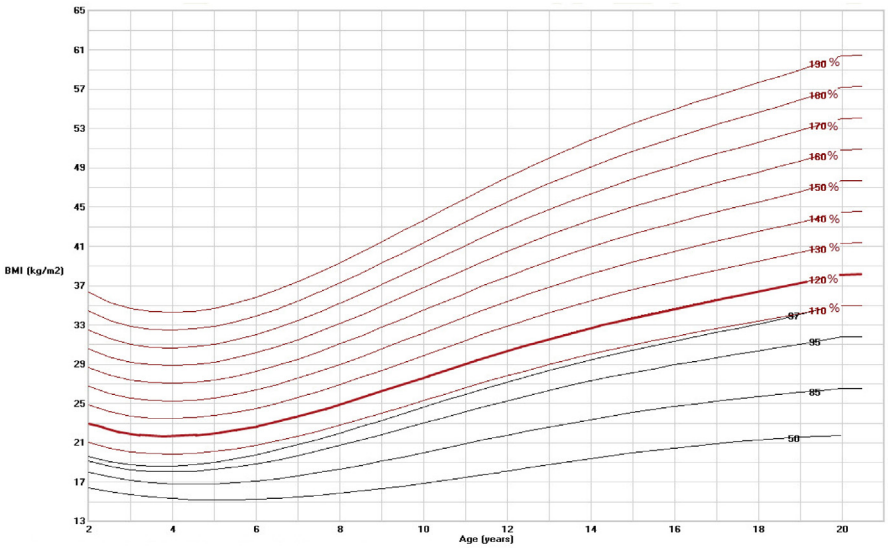

Fig. 2. Body Mass Index Charts for Children and Adolescents Ages 2–20 Years with Severe Obesity. BMI vs. age is shown for children ages 2–20 years.

Table 1
Top 10 Takeaway Messages: Assessment of the Child with Obesity. Shown are the top 10 messages from the OMA regarding weight assessment of children [1–8].

1.	For children less than two years of age, weight for length percentile is used to assess weight status.
2.	For children 2–20 years of age, BMI is used to assess weight status.
3.	Overweight is defined as a BMI percentile between the 85th percentile and the 94th percentile.
4.	Obese is defined as a BMI percentile between the 95th percentile and 119% of the 95th percentile.
5.	Severely obese defined as is a BMI percentile greater than or equal to 120% of the 95th percentile.
6.	Class 1 obesity is defined as a BMI percentile between the 95th percentile and 119% of the 95th percentile.
7.	Class 2 obesity is defined as a BMI percentile that is 120–139% of the 95th percentile.
8.	Class 3 obesity is defined as a BMI percentile greater than or equal to 140% of the 95th percentile.
9.	BMI z-scores should not be used to assess BMI change among children and adolescents with BMI greater than 120% of the 95th percentile.
10.	A BMI of 35 kg/m^2 is used for adolescent boys at approximately 18 years of age and adolescent girls at approximately 16 years of age as the threshold for severely obese and is somewhat lower than 120% of the 95th percentile.

child with obesity and the classification of obesity presented by the child.

Children with obesity are at risk for glycemic dysregulation [25]. A fasting blood glucose or a hemoglobin A1c (HbA1c) test will determine whether glycemic dysregulation is present. Dyslipidemia is the most common laboratory abnormality found in children with obesity [26]. Either a fasting lipid profile or a non-fasting lipid profile, if fasting is not feasible, can assess dyslipidemia. Liver function tests, specifically alanine aminotransferase (ALT) and aspartate aminotransferase (AST), can screen for non-alcoholic fatty liver disease. Many children are at risk for vitamin D deficiency, which is detected with a 25-hydroxy (25 OH) vitamin D test [27]. Blood pressure is checked if the child is older than three years of age. Other studies are indicated based on history: a sleep study for any history of snoring, daytime sleepiness, disrupted sleep cycle or even hyperactivity may be indicated. Liver imaging can be done if the liver function tests (ALT and AST) are high. Uric acid can be obtained in those children with diabetes or prediabetes. Uric acid can be elevated with high intake of high fructose corn syrup and even table sugar. Fig. 5 shows recommended diagnostics based on age ranges, BMIs, and risk factors [19,22–24].

7. Special populations

Turner syndrome (i.e., absence or dysfunction of an X chromosome), achondroplasia (i.e., mutation of fibroblast growth factor receptor 3

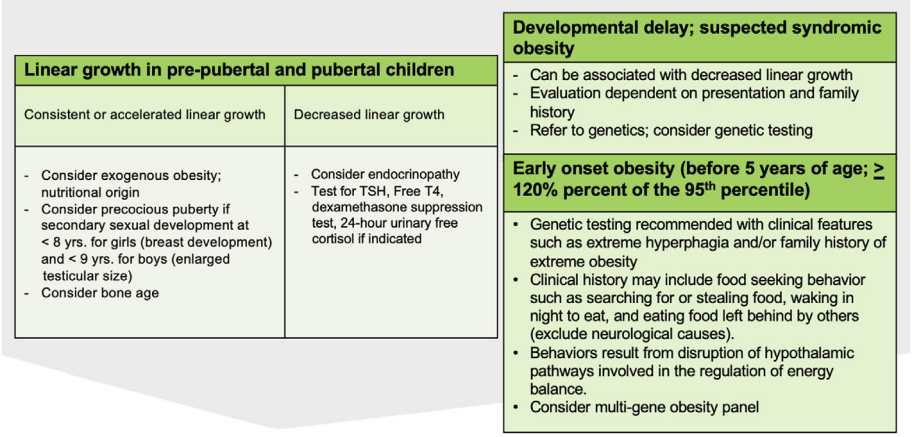

Fig. 3. Childhood Obesity: Differential Diagnosis. Considerations and associations are shown regarding linear growth, developmental delays, and early onset obesity in children. **Abbreviations:** TSH: thyroid-stimulating hormone; T4: thyroxine.

Symptoms	Related Co-Morbidities
Nervousness, school avoidance, social inhibitions	Depression, anxiety, bullying
Fatigue, Muscle aches	Vitamin D deficiency
Polyuria, polydipsia, fatigue, nocturia	Type 2 Diabetes (T2DM)
Headaches, facial numbness	Idiopathic Intracranial Hypertension (Pseudotumor cerebri)
Skin pigmenting, skin tags	Insulin resistance (IR)
Daytime somnolence, loud snoring, witnessed apnea, attention deficit	Obstructive sleep apnea (OSA)
Abdominal pain, indigestion	Gastroesophageal reflux disease (GERD), gall bladder disease, constipation
Hip or knee pain	Slipped capital femoral epiphysis (SCFE), early osteoarthritis
In-toeing, leg bowing, mild knee pain	Blount's disease
Hirsutism, acne, irregular menses	Polycystic Ovarian Syndrome (PCOS)

Fig. 4. Focused Review of Systems. Symptoms and associated co-morbidities for children with obesity are shown. Children may or may not complain of symptoms, so a careful assessment of symptoms associated with obesity is necessary.

Infancy (0-24 months)	Toddler (Age 2-4 years)	Early Childhood (Age 5-9 years)	Puberty (Age 10-14 years)	Adolescent (Age 15-18 years)
Weight >Length	BMI ≥ 95th percentile Or ≥ 85th percentile with 2 or more risk factors (24-48 months)	BMI ≥ 95th percentile Or ≥ 85th percentile with 2 or more risk factors	BMI ≥ 95th percentile Or ≥ 85th percentile with 2 or more risk factors	BMI ≥ 95th percentile Or ≥ 85th percentile with 2 or more risk factors
	- Fasting Blood Glucose and/or HbA1c - Fasting Lipid Panel/Non fasting if fasting not feasible - ALT, AST, consider GGT - Consider 25 OH Vitamin D - BP annually if ≥ 3 years			
		- Consider Sleep Study - Consider Liver Imaging - Consider Uric Acid - Consider fasting serum insulin		
			- Consider Urine Microalbumin/Creatinine ratio - Consider C-peptide, hs-CRP	

Fig. 5. Diagnostic Workup: Labs and Studies. Shown are recommended diagnostics based on age ranges, BMIs, and risk factors for pediatric patients [28,29]. **Abbreviations:** BMI: body mass index; HbA1c: hemoglobin A1c; ALT: alanine aminotransferase; AST: aspartate aminotransferase; GGT: gamma-glutamyl transferase; 25 OH: 25-hydroxy; hs-CRP: high-sensitivity C-reactive protein.

gene), and Down syndrome (i.e., trisomy 21 or three copies of chromosome 21) are commonly seen populations of children at risk for obesity. These populations require additional considerations regarding health impacts and screening tools for obesity.

Children with Turner syndrome have restricted height growth (i.e., short stature) and increased adiposity, with an increase in total and visceral fat mass and a decrease in lean body mass. In addition, 70% of girls with Turner syndrome have abnormal glucose metabolism leading to an increased prevalence of both Type 1 and Type 2 diabetes [30,31]. Children with Down syndrome typically have short stature and have shorter limbs and poor coordination, leading to less activity and a propensity to gain weight. Growth charts for height and weight are used to follow children with Down Syndrome, but the American Academy of Pediatrics (AAP) recommends that clinicians use BMI guidelines from the CDC for normally developing children to classify BMI status [32–34]. Children with achondroplasia, the most common form of inherited disproportionate short stature, have no pubertal growth spurt and a relatively flat growth curve for height from infancy on. They do not have adiposity rebound and are at high risk for early cardiovascular disease, obstructive sleep apnea, and difficulty with mobility [35,36]. Fig. 6 shows a summary of considerations for children with Turner syndrome, Down syndrome, and achondroplasia. Table 2 shows the top eight takeaway messages from the OMA regarding differential diagnoses and diagnostic workups for special populations.

8. Conclusions

This Clinical Practice Statement on the assessment, differential diagnosis, and initial clinical evaluation of pediatric patients with obesity provides clinicians with assessment tools and recommendations regarding their pediatric patients. Assessment of pediatric patients with obesity is the first step in determining treatments that may lead to improvements in the health and wellbeing of children and adolescents with obesity, especially those with metabolic, physiological, and psychological complications.

8.1. Writing Process and ethics statement [38]

8.1.1. Transparency

This manuscript was largely derived and edited from the 2020–2022

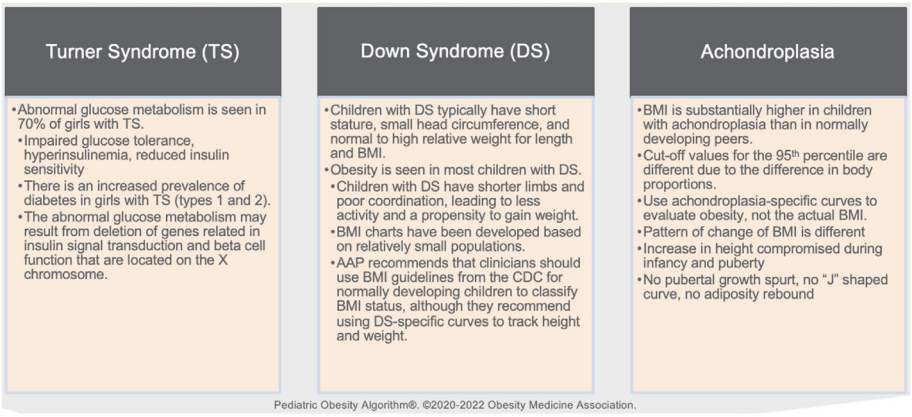

Fig. 6. Special Populations: Turner Syndrome, Down Syndrome, and Achondroplasia. Children with Turner syndrome, Down syndrome, and achondroplasia require special considerations regarding health risks and obesity. Takeaways for each condition are shown [30–37].

Table 2
Top Eight Takeaway Messages: Differential Diagnoses and Diagnostic Workups for Special Populations. The top eight messages from the OMA regarding differential diagnosis, diagnostic workups, and special populations are shown [30–37].

1.	Although most children with obesity have exogenous obesity of nutritional origin, endocrinopathies and syndromal obesity must be considered.
2.	Not all children with syndromal obesity have developmental delays and/or a decrease in linear growth, but, if either is present, consider syndromal obesity.
3.	Children with severe early-onset obesity and hyperphagia are at increased risk for genetic etiologies for obesity.
4.	The review of systems and initial work up is determined by the age at presentation and degree of obesity of the child. Glycemic dysregulation, dyslipidemia, and hepatic function are generally assessed.
5.	Other testing is directed by presenting symptoms/physical findings.
6.	Turner syndrome is a special population at high risk for abnormal glucose metabolism.
7.	Children with Down syndrome are at high risk for obesity. BMI is assessed using the CDC charts for normally developing children; height and weight are followed using specific charts for Down syndrome.
8.	Achondroplasia is a form of inherited disproportionate short stature in which there is no pubertal growth spurt, no typical "J" shaped BMI curve, and high rates of obesity. Specific height, weight, and BMI curves should be used.

Obesity Medicine Association (OMA) Pediatric Obesity Algorithm. Beginning in 2016, the OMA created and maintained an online Pediatric "Obesity Algorithm" (i.e., educational slides and eBook) that underwent updates approximately every two years by OMA authors and was reviewed and approved annually by the OMA Board of Trustees. Authors of prior years' versions are included in Supplement #1. This manuscript is the first published version of the applicable chapter/s of the 2020–2022 OMA Pediatric Obesity Algorithm.

8.1.2. Group composition

Over the years, the authors of the OMA Pediatric Obesity Algorithm have represented a diverse range of clinicians, allied health professionals, clinical researchers, and academicians. (Supplement #1) The authors reflect a multidisciplinary and balanced group of experts in obesity science, patient evaluation, and clinical treatment.

8.1.3. Author contributions

SEC transcribed the first draft from the 2020–2022 OMA Pediatric Obesity Algorithm. MC and SEC then reviewed, edited, and approved the document for pre-peer review submission and post-peer review publication.

8.1.4. Disclosures (declaration of potential competing interest)

Potential dualities or conflicts of interest of the authors are listed in the Individual Disclosure section. Assistance of a medical writer paid by the Obesity Medicine Association is noted in the Acknowledgements section. Neither the prior OMA Pediatric Algorithms nor the publishing of this Clinical Practice Statement received outside funding. The authors of prior OMA Pediatric Obesity Algorithms never received payment for their writing, editing, and publishing work. Authors of this Clinical Practice Statement likewise received no payment for their writing, editing, and publishing work. While listed journal Editors received payment for their roles as Editors, they did not receive payment for their participation as authors.

8.1.5. Individual Disclosures

SEC declares a relationship with Novo Nordisk as a member of an Advisory Board and a relationship with Rhythm Pharmaceuticals as a member of their Gold Panel.

MC reports no disclosures pertaining to this project.

8.1.6. Evidence

The content of the OMA Pediatric Obesity Algorithm and this manuscript is supported by citations, which are listed in the References section.

8.1.7. Ethics review

After approval by the authors, a draft manuscript was peer-reviewed and approved by the OMA Board of Trustees prior to publication. This submission did not involve human test subjects or volunteers.

8.1.7.1. Conclusions and recommendations. This Clinical Practice Statement is intended to be an educational tool that incorporates the current medical science and the clinical experiences of obesity specialists. The intent is to better facilitate and improve the clinical care and management of patients with pre-obesity and obesity. This Clinical Practice Statement should not be interpreted as "rules" and/or directives regarding the medical care of an individual patient. The decision regarding the optimal care of the patient with overweight and obesity is best reliant upon a patient-centered approach, managed by the clinician tasked with directing an individual treatment plan that is in the best interest of the individual patient.

8.1.7.2. Updating. It is anticipated that sections of this Clinical Practice Statement may require future updates. The timing of such an update will depend on decisions made by Obesity Pillars Editorial team, with input from the OMA members and OMA Board of Trustees.

8.1.7.3. Disclaimer and limitations. Both the OMA Obesity Algorithms and this Clinical Practice Statement were developed to assist health care professionals in providing care for patients with pre-obesity and obesity

based upon the best available evidence. In areas regarding inconclusive or insufficient scientific evidence, the authors used their professional judgment. This Clinical Practice Statement is intended to represent the state of obesity medicine at the time of publication. Thus, this Clinical Practice Statement is not a substitute for maintaining awareness of emerging new science. Finally, decisions by practitioners to apply the principles in this Clinical Practice Statement are best made by considering local resources, individual patient circumstances, patient agreement, and knowledge of federal, state, and local laws and guidance.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Suzanne Elizabeth Cuda reports financial support and travel were provided by Novo Nordisk Inc. Suzanne Elizabeth Cuda reports a relationship with Novo Nordisk Inc that includes: consulting or advisory and travel reimbursement.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.obpill.2022.100010.

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