Obesity

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Clinical Trials and Investigations

Understanding the Increasing Prevalence of Obesity in Patients With Type 1 Diabetes: Strategies for Improving Clinical Care

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ABSTRACT

The presence of excess weight is no longer a distinguishing feature between patients with type 1 diabetes (T1D) and those with type 2 diabetes (T2D). Obesity treatment in patients with T2D improves glycemic control and reduces or even eliminates medication burden. Robust evidence and clear guidelines exist to support and direct effective weight management in patients with T2DM. Now, however, rates of obesity in patients with T1D rival those found in the general population, yet little is known about the efficacy, safety, and unique considerations of obesity treatment (lifestyle modifications, pharmacology, and surgery) in this population. This review tackles these topics and the gaps in evidence and clinical care.

1 | Introduction

Type 1 diabetes (T1D) is an autoimmune disease targeting pancreatic beta cells that results in lifelong absolute insulin deficiency. In the United States, there are over 300,000 youth and 1.7 million adults with T1D, and the numbers are increasing [1, 2]. Evidence that diabetes-related complications can be prevented and better treated has resulted in longer life expectancies, especially in high-income countries [3]. Compared to 40 years ago, obesity is now an acknowledged coexisting comorbidity in patients with T1D, and the reasons for this are complex, while effective and safe obesity treatments have not had the years of study as those seen in patients with both obesity and T2D.

2 | Background

In the Diabetes Control and Complications Trial (DCCT) where recruitment of 1441 adult subjects with T1D were randomized to intensive (three or more injections per day or insulin pump therapy) vs. conventional therapy (one or two injections per day) between 1983 and 1989, mean BMI at study entry was 23.5 ± 2.6 and $23.9 \pm 2.8\,kg/m^2$, respectively [4]. Importantly, HA1c levels at the same time were $8.8\% \pm 1.5\%$ and $8.7\% \pm 1.6\%$ for the two groups. BMI increased much more in the intensive therapy (where HA1c decreased to approximately 7%) compared to the conventional therapy group (where HA1c stayed close to 9%), especially in the first 1.3 years (BMI increase of 1.1 ± 0.49 vs. $0.35 \pm 0.049\,kg/m^2$/year, $p < 0.001$) [4]. Those with family histories of T2D had even greater weight gain.

Modern day management has resulted in no difference in obesity rates for those with T1D compared to the general population. For adults with T1D in the United States, those with overweight or obesity are approximately one-third of each category for those above the age of 26 years. For those patients in the 18– to 25-year-old group, only about half have a normal weight or underweight status (Figures 1 and 2) [5, 6].

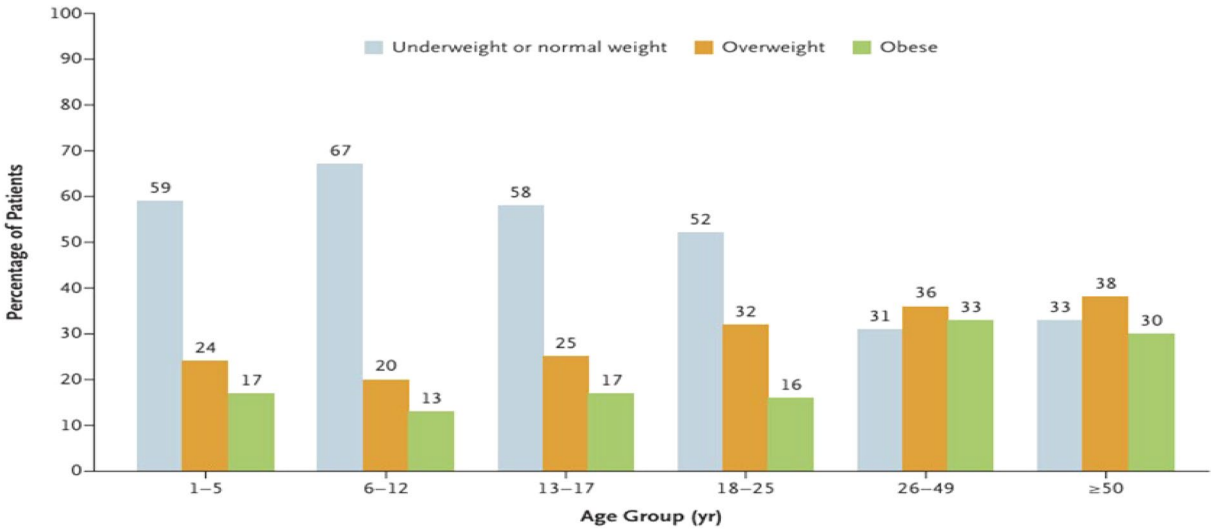

FIGURE 1 | Prevalence of overweight and obesity in patients with T1D.

T1D	T2D
Controlled: ✓ Reduce excess body fat ✓ Improve CVD risk factors ✓ Reduce insulin dose ✓ Minimize hypoglycemia	**Controlled:** ✓ Reduce excess body fat ✓ Improve CVD risk factors ✓ Eliminate or minimize insulin and/or sulfonylurea use ✓ Partial or complete remission of T2D
Uncontrolled: ✓ Reduce excess body fat ✓ Improve glycemic control ✓ Improve CVD disease risk ✓ Reduce insulin dose ✓ Minimize hypoglycemia	**Uncontrolled:** ✓ Reduce excess body fat ✓ Improve glycemic control ✓ Improve CVD disease risk ✓ Eliminate/reduce any insulin or oral hypoglycemic use ✓ Partial or complete remission of T2D

FIGURE 2 | Key differences in treatment goals. Controlled indicates HA1c < 7% and time in range > 70%. Uncontrolled indicates HA1c > 7% and time in range < 70%. CVD, cardiovascular disease.

This change in T1D phenotype has caused confusion for the appropriate classification of diabetes. First, for unclear reasons, T1D is presenting later in life, not the typical childhood-onset T1D seen almost exclusively over 30 years ago. It is now estimated that over half of T1D presents in adulthood, misclassification is common, and this trend is growing [7]. For example, in an analysis of US individuals from a commercially insured database, the total number of new cases in adults over a 14-year period was 19,174 compared to 13,303 in youth [8]. Clinically, these adult-onset patients do not present with severe insulin deficiency and ketoacidosis is rare; in fact, many of these patients don't require insulin at diagnosis, quite different than childhood-onset T1D [9, 10]. This specific population is called "latent autoimmune diabetes of adults" (LADA) which is generally considered a subtype of T1D, although the autoimmunity, genetics, and clinical course are different than childhood-onset disease. Some degree of endogenous insulin can be present for decades for these older-onset patients, while, in general, c-peptide secretion is generally unmeasurable in weeks to months in most children after their diagnosis.

It needs to be emphasized that LADA is still an autoimmune condition, but given the age of diagnosis and the fact that many of these individuals are clinically indistinguishable from those with T2D, it is not surprising that this population is frequently misdiagnosed [11]. Patients with obesity, compared to normal weight individuals with LADA, also have lower GAD antibodies and greater insulin secretion and are more likely to have low-risk HLA-genotypes [9]. Adult-onset T1D needs to be considered a continuum, as not all meet the criteria of LADA. Those who present with severe insulin deficiency (especially with diabetic ketoacidosis [DKA]) may evolve into classical T1D with no significant endogenous insulin secretion within weeks or a few months, similar to children. Those

with LADA, on the other hand, will usually require insulin anywhere from 2 to 5 years after diagnosis. Many will start all GAD antibody positive patients on insulin, although data that this preserves beta-cell function are conflicting. Importantly, clinicians should be ready to start insulin on any patient with a positive GAD antibody if glycemia is not controlled. Those with obesity are more likely to have endogenous insulin secretion for a longer period of time.

Ahlqvist and colleagues have attempted to clarify the different phenotypes of diabetes by performing a cluster analysis of 8980 newly diagnosed patients with diabetes in Sweden [12]. Cluster 1, characterized by early-onset disease and poor metabolic control, GAD positivity, and insulin deficiency, is termed severe autoimmune diabetes (SAID). Cluster 2 was GAD antibody negative but otherwise similar to cluster 1. This cluster is termed severe insulin deficient diabetes (SIDD). The final three clusters are different phenotypes of T2D. While both SAID and SIDD appear to be different phenotypes of T1D (and may partly explain the increased incidence of T1D), it is clear our current classification of diabetes is outdated as there is much overlap between classic T1D and T2D. Hopefully, the specific diagnosis of diabetes will be improved as genetic testing, specifically the T1D genetic risk score, enters routine clinical medicine [13].

3 | Pathophysiology

Despite the change in age of T1D with increased obesity in adults, childhood-onset disease has also seen this increase in weight. The term "double diabetes" was first noted in 1991, and it was pointed out that given the high prevalence of T2D in the community, it is not surprising those with family histories of T2D could have both [14]. The genetic predisposition not only for dysglycemia but also for obesity (and the entire metabolic syndrome) is a simplistic rationale for the appearance of obesity in childhood-onset T1D over the past three decades. The reduction of glycosuria, however, is not the only etiology for the obesity seen in T1D today. Still, in uncontrolled T1D, renal glucose losses can account for 300–400 kcal/day in obligate energy losses [15].

Insulin resistance (IR) is another physiological contributor that is associated with excess adiposity in T1D [16], and certain markers of IR have been found to correlate with increased micro- and macrovascular risk in T1D [17, 18]. While multiple indirect measures of IR have been developed and validated in this population [19–21], there is no established standard clinical assessment for IR in T1D. Total daily insulin dose per kg body weight is commonly used to quantify insulin resistance in clinical practice; however, secondary analyses of data from the DCCT and from the Pittsburgh Epidemiology of Diabetes Complications Study showed total daily insulin dose was not correlated with clinical risk for micro- or macrovascular complications [17, 18, 22]. Quantifying IR using a model that correlates with cardiovascular risk may be the most clinically relevant; however, further data are needed to determine if targeting these markers of IR in treatment carries clinical benefit.

Behavioral snacking to avoid hypoglycemia is another likely contributor to the development of obesity, particularly in those patients treated in the era before insulin analogues when snacking was required to avoid hypoglycemia. Especially with the use of long-acting analogues replacing neutral protamine Hagedorn (NPH) insulin, that is not generally required today [23]. There is also a suggestion from canine models of T1D that energy expenditure is higher with portal vs. systemic insulin administration [24]. Other mechanisms have been proposed resulting in the weight gain seen in T1D as glycemic control has improved over the past few decades. For example, an increase in sympathetic nervous system activity [25], change in protein turnover and substrate transport across cell membranes [26], alterations in the gut microbiome [27], and an increase in the size of metabolically active organs [28] may all contribute to changes in alterations in energy expenditure with T1D.

Previous reviews have highlighted the lack of data regarding the treatment of obesity in patients with T1D, particularly with obesity pharmacotherapy and surgical obesity treatment [29, 30]. These reviews also did not include the newer incretin therapies now approved for obesity treatment. In this context, this narrative review aims to update the treatment options for patients with both obesity and T1D and the unique risks and benefits clinicians must consider. We conducted a literature review using PubMed and Google Scholar with key words focusing on behavior modifications, obesity pharmacotherapy, incretin therapy, and surgical treatment in patients with both T1D and obesity.

4 | Lifestyle Modification

4.1 | Dietary Changes

As the Endocrine Society advises, patients with obesity would benefit from a treatment strategy using a structured lifestyle intervention program that includes a reduced calorie diet and feedback from a dietitian or nutrition educator as part of a multidisciplinary team [31]. Patients with T1D and obesity who participated in a multidisciplinary weight management program had greater weight loss and reduction in daily insulin requirements after 12 months when retrospectively compared with patients receiving standard care [32]. However, there was no significant change in glycemic control between the two groups despite changes in weight and decreased insulin requirements. Similarly, patients from the T1D Registry who gained 20 lb or more only had a 0.2% difference in HA1c compared to those who had stable weight at 5 years [33]. This is in contrast to meta-analyses of weight loss in T2D, in which glycemic control improved significantly with a total body weight loss of 5% or more [34] and demonstrated a 0.1% reduction in HA1c for every kilogram of weight lost [35]. Changes (or lack of change, for T1D) in glycemic control as a reflection of weight gain between patients with T1D and T2D illustrate an important point of contrast in the treatment of obesity between these groups. While improvement in glycemic control is often a desired outcome of weight loss in T2D, the relationship between excess weight and glycemic control is of much less clinical significance in the setting of T1D. Rather than glycemic control, secondary goals of treating obesity in patients with T1D should primarily focus on prevention of hypoglycemia, with careful attention to changing insulin requirements during the active weight loss phase, and frequent review and adjustment of insulin dosing.

In a consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes, there was no singular nutrition plan recommended for patients with T1D, and individualized nutrition planning was emphasized [36], with insufficient data on this subject to support more specific guidelines. Numerous diet variations have been examined in the setting of T2D, and a meta-analysis of these demonstrated a greater HA1c-lowering benefit from ketogenic diets (defined as carbohydrate intake making up 5%–10% of total caloric intake per day), as well as low-carbohydrate and low-fat diets, among others [37]. However, there is not yet sufficient evidence to recommend the safety of a ketogenic diet in T1D, with ongoing concerns for the risk of hypoglycemia, reduced glycogen stores blunting the response to glucagon [38], and the risk of relative insulin deficiency leading to DKA [39]. Specifically for patients treated with sodium-glucose cotransporter-2 (SGLT-2) inhibitors, the ADA recommends against following a ketogenic diet due to the increased risk for DKA [40]. Concerns for potential loss of bone mass in correlation with ketogenic diets have also been raised, with excess beta-hydroxybutyrate and the resulting acidic environment contributing to impaired bone growth through suppression of growth hormone and insulin-like growth factor-1 (IGF-1) activity, impairment of bone mineralization, and interference with the conversion of 25-hydroxyvitamin D to active 1,25-hydroxyvitamin D. Another mechanistic pathway suggests that high fat intake associated with the ketogenic diet causes replacement of osteoblasts in bone marrow with adipocytes as well as increased expression of inflammatory cytokines, resulting in decreased osteoblast differentiation and increased osteoclast activity [41]. With multiple possibly harmful metabolic effects, the benefits of ketogenic diets are unlikely to outweigh the risk of possible adverse effects in the T1D population.

While meta-analysis of low-carbohydrate diets (containing between 50 and 130 g of carbohydrate, or less than 26% of total energy intake from carbohydrate per day) in T2D resulted in 7.41 kg greater weight loss ($p < 0.001$) and greater reduction in HA1c at 6 months compared with controls [42], the data for low-carbohydrate diets in T1D are less conclusive. A review of eight studies examining low-carbohydrate diets meeting these criteria in T1D showed mixed results regarding effects on BMI, HA1c, and total daily insulin doses, and the sample size was not sufficient to complete a true meta-analysis [43]. Other data suggest that dietary macronutrient composition may not play a significant role in T1D, particularly on weight loss or HA1c. In a comparison of a low-calorie, low-carbohydrate diet to a low-calorie, low-fat diet and a Mediterranean diet without calorie restriction in T1D, all interventions resulted in similar proportions of modest weight loss and HA1c reduction (by −2.7 kg and −0.91% on average, respectively) regardless of macronutrient composition or caloric deficit [44]. Given the limitations in our current knowledge, the ADA gives no strict recommendation on the amount of daily carbohydrate to consume. General dietary principles, such as promoting intake of nonstarchy vegetables, minimizing consumption of red meat, sugar-sweetened beverages, and refined grains, and choosing whole foods over processed foods, are encouraged [40, 45].

When advising on protein intake in T1D, the renal effects of high protein intake should be considered. Increased dietary protein intake can result in renal hyperfiltration, which may manifest with an increased glomerular filtration rate (GFR) [46] and increased albuminuria [47]. Early nephropathy in T1D commonly presents with the development of albuminuria prior to a reduction in GFR, and albuminuria may be accompanied by a concurrent elevation in blood pressure [48]. For patients without existing diabetic nephropathy, careful attention should be paid to renal laboratory parameters and blood pressure if recommending increased protein intake from baseline. If a new elevation in urinary albumin or in blood pressure appears within 6 months of dietary protein increase, alternative dietary modifications should be recommended. Because diabetic nephropathy (albuminuria or reduced GFR) in T1D is associated with increased cardiovascular risk [49, 50] and higher overall mortality [51], the risk of morbidity and mortality for patients with preexisting kidney disease should be weighed against the potential weight loss benefit from following a high-protein diet.

Recent guidelines carry conflicting recommendations on protein intake. The 2022 Kidney Disease: Improving Global Outcomes (KDIGO) guideline for diabetes management in chronic kidney disease (CKD) recommends a daily protein intake equal to or less than 0.8 g/kg (of ideal body weight) for patients with T1D or T2D and any stage CKD not being treated with dialysis [52]. Based on a 2023 Cochrane review, the 2025 ADA Standards of Care in Diabetes suggests it is not necessary to limit protein intake below this 0.8 g/kg daily threshold [40]. Though this review found no effect on mortality or progression to kidney failure with protein-restricted diets (0.6–0.8 g/kg per day) compared to unrestricted protein intake (> 1 g/kg per day) in adults with diabetes and CKD, it included data from both T1D and T2D, and the authors reported these results as low-certainty evidence [53]. Additional analysis of data specific to T1D is needed to clarify these outcomes further. If recommending increased protein intake from baseline in patients with CKD, obtaining input from the patient's nephrologist to build a dietary plan based on individual factors would foster integration of care across the patient's medical team and may help reduce the risk of adverse renal outcomes.

Preparatory counseling about possible fluctuations in glycemic control is another important component of care when initiating a dietary intervention for weight loss in T1D. The postprandial period requires particular attention when modifying dietary macronutrient distribution. It is well known that meals higher in carbohydrates generally cause early postprandial hyperglycemia, but it has been demonstrated that meals combining carbohydrate with higher amounts of fat or protein can produce an initial blunting of glycemic response followed by sustained, late postprandial hyperglycemia appearing 1 to 2 h following a meal and lasting up to 6 h [54]. Combining fat with carbohydrate and a moderate amount of protein can have an additive effect, producing a greater glycemic excursion in the late postmeal period compared to meals mainly consisting of protein and carbohydrate [55]. Giving anticipatory guidance on these expected changes in both early and late postprandial glycemic response based on meal composition can help mitigate potential patient anxiety about glycemic variability during the transition into a new dietary plan and may help with adherence to a new diet plan. With dietary modification, clinicians should discuss potentially higher prandial insulin requirements or the need

for additional correction of late hyperglycemia. With interindividual differences in glycemic response and insulin sensitivity, insulin dosage adjustments should be made on a case-by-case review of glycemic trends, ideally using continuous glucose monitoring (CGM) data. Though patients using automated insulin delivery (AID) therapy are less likely to experience these challenges due to automated pump responses to glycemic changes, patients using open-loop insulin pumps may want to explore the use of extended or split mealtime bolus delivery to manage these changes.

There are several additional psychosocial components of care to consider when prescribing dietary modification for patients with T1D. Diabetes distress, or the emotional response to living with diabetes [56], should be taken into account. Between 20% and 30% of patients with T1D are projected to experience clinically elevated diabetes distress scores, which can negatively impact healthy diabetes self-management behaviors such as participation in physical activity and adherence to dietary modification [57]. Given that carbohydrate counting is a commonly required skill and often a daily task for patients managing T1D, recommending further self-monitoring in the form of calorie tracking or other macronutrient tracking through a new dietary program may be a trigger for increased diabetes distress. Having obesity and being inactive are also known to increase diabetes distress [57, 58], which inherently may put this population at higher risk and increase the importance of screening for diabetes distress in this population. The ADA also recommends that all patients with diabetes have a discussion regarding their history of prior diets and undergo screening for current disordered eating behaviors [40], which, although data are much more robust in the pediatric population, have been observed in 25%–31% of adults with T1D [59, 60] and can have an impact on diabetes management and weight management goals. This is particularly true in women with T1D, who are at higher risk for disordered eating behaviors than nondiabetic women [61]. Screening can include assessment for extreme exercise patterns, patterns of insulin use or restriction, HA1c trends, history of DKA, and assessment of patient concern regarding weight and body shape. Recognition of maladaptive eating behaviors can be helpful in identifying patients who are at higher risk for exacerbation of these behaviors while engaging in a weight loss program [40]. Finally, screening for food insecurity should be included in the initial assessment for people with T1D and obesity. Prevalence of food insecurity is greater than 25% in patients with T1D [62], almost double the rate (13.5%) within the United States as a whole [63], and it represents another risk factor not only for hypoglycemia due to insufficient food availability, but also for poor diabetes self-management and adherence to a dietary plan, particularly if insulin and food costs compete.

Taking measures to reduce diabetes distress can facilitate behavioral changes in a weight loss program. Interventions that have shown a reduction in diabetes distress include diabetes self-management and support (DSMES) that focused on improving knowledge about diabetes and the technical skills needed for self-management, such as goal setting and problem solving. These skills also translate well into a weight loss program. Behavioral health interventions that can reduce diabetes distress by improving coping strategies for diabetes management and modification of unhelpful cognitive processes include supportive counseling, cognitive behavioral therapy, and the use of self-determination approaches. Finally, the use of technology such as CGM and sensor-augmented insulin pump therapy can either improve or stabilize diabetes distress [57]. Remembering these distinct clinical challenges that people with T1D face will help facilitate behavioral changes and encourage successful lifestyle modification.

4.2 | Exercise

We face another gap in knowledge regarding how to recommend the optimal type, duration, and frequency of exercise for patients with T1D and obesity. The ADA 2025 Standards of Care in Diabetes recommends that patients with both T1D and T2D participate in a minimum of 150 min of moderate level physical activity per week, ideally distributed over 3 days per week and avoiding more than 2 days without physical activity. Specific goals include resistance training two to three times per week on nonconsecutive days, increasing active leisure activity (or non-exercise activity thermogenesis), and reducing overall sedentary time by interrupting prolonged sitting every 30 min [40]. The American Association of Clinical Endocrinologists (AACE) recommends similar physical activity goals for patients with obesity [64]. For all patients with overweight or obesity, the ADA recommends a goal of at least 3%–7% total body weight loss through a treatment plan based on medical nutrition therapy, physical activity, and behavioral therapy [40]. However, these recommendations are based largely on T2D and obesity trials and are mainly limited to expert opinion for those with T1D.

The health benefits of exercise for patients with T1D have yet to be fully clarified. One meta-analysis showed a very modest HA1c-lowering benefit (only 0.29% reduction) associated with exercise [65]. Longer duration of moderate to vigorous physical activity and higher daily step counts were associated with lower 10-year HA1c, waist circumference, and BMI, as well as higher insulin sensitivity (based on estimated glucose disposal rate) and lower total daily insulin doses in a cross-sectional study of patients with T1D. Other measures of body composition (percent fat mass and lean mass) were not significantly correlated with physical activity measures. Of note, higher sedentary time was correlated with lower insulin sensitivity, independent of time spent in moderate to vigorous physical activity [66], suggesting that guidelines recommending to limit sedentary time are likely of benefit in this population, but additional high-quality data with larger sample sizes are needed in this area.

Physiological responses to specific exercise modalities are also variable. A 6-week progressive high-intensity interval training (HIIT) program in sedentary adults with T1D resulted in higher exercise motivation and enjoyment, better sleep quality, and better health-related quality of life scores in domains of physical functioning, general health, pain, and physical role limitations [67]. Compared with resistance training, aerobic exercise was shown to produce a greater drop in blood glucose levels during activity with wider glucose variability, with an average decrease of 71.0 ± 48.1 mg/dL during aerobic exercise, compared to a glucose decrease of 24.0 ± 32.1 mg/dL during resistance exercise ($p = 0.007$) [68]. Another small study showed that glycemic variations differed between men and women, as men had lower

TABLE 1 | Key points regarding exercise management in T1D [23, 78–84].

Treatment modality	Recommendation
Basal insulin	Choose basal insulins that have less associated hypoglycemia: [78–80] • U-300 glargine • Insulin degludec
Prandial insulin	• Avoid using prandial insulins with higher risk of hypoglycemia [84] ◦ Regular insulin • Avoid initiating exercise at the peak of a prandial insulin dose (approximate time to peak effect below) [23, 81] ◦ Lispro: 60–150 min ◦ Aspart: 60–180 min ◦ Fiasp: 90–132 min ◦ Glulisine: 90 min ◦ Lispro-aabc: 120–174 min ◦ Regular insulin: 120–240 min ◦ Afrezza: 30–54 min
Diabetes technology	• Recommend CGM for all patients with T1D [82] • Consider AID pump for all patients with T1D [82, 83] ◦ Discuss AID pump modifications surrounding a bout of exercise ◦ Modifications should be based on timing of last meal, timing of last insulin dose, type of exercise, blood glucose and glucose trends prior to exercise
Supportive measures	• Engage diabetes self-management and support curriculum for promoting long-term healthy behaviors • Assess level of social support from family and friends

glucose after both continuous and interval aerobic activity, while women only had lower glucose after continuous aerobic activity [69]. Improvements in cardiovascular health observed in a meta-analysis were associated with aerobic exercise interventions with a higher frequency of exercise (3 or more days per week), as well as interventions with a more prolonged duration (> 12 weeks). Aerobic exercise alone had no significant effect on HA1c in this group, whereas a combination of aerobic and resistance exercise reduced HA1c. This HA1c-lowering effect was only associated with combination interventions with greater frequency and duration (more than 3 days weekly over 12 or more weeks) [70].

Many adults living with T1D do not achieve currently recommended levels of physical activity [71]. In a secondary analysis of the T1D Exchange Clinic Registry data, only 33% of adult participants reported exercising 150 min or more per week, and 12% of participants reported not participating in any exercise [72]. In other survey-based analyses, between 8% and 44% of participants reported being inactive [73, 74], although in one group, 78% of participants reporting light activity or inactivity desired to increase their activity level [74]. In some cases, overestimation of exercise volume may be a contributing factor. When interviewed about their exercise habits, many patients with T1D were found to initially overestimate the frequency, duration, or intensity of their exercise [75]. Similar results were reported when physical activity was measured in patients with T1D subjectively (by participant self-report) and objectively (using an accelerometer). While 97% of participants met recommended moderate-intensity physical activity goals based on self-report, only 32% of participants met criteria when physical activity was measured by accelerometer. Those who met physical activity criteria based on accelerometry data had significantly lower HA1c, BMI, waist circumference, and body fat mass, which correlates with positive cardiovascular benefits of physical activity in the T1D population. However, this group also had higher rates of hypoglycemia [76].

Fear of hypoglycemia (FOH) is one of the most commonly reported barriers to engaging in physical activity for patients with T1D [73, 76, 77]. There are many methods to help patients avoid hypoglycemia during exercise. Basic strategies include choosing basal insulins with lower risk of hypoglycemia, such as U-300 glargine and degludec [78–80]. Additionally, physical activity can be avoided during the peak effect of a prandial insulin dose. Time to peak action for commonly used prandial insulins is listed in Table 1, along with a summary of recommendations for exercise management [23, 78–84]. During exercise, patients using a multiple daily injection (MDI) insulin regimen can target a slightly more liberal glucose range of 126–180 mg/dL and consume 10–35 g of carbohydrate if glucose levels fall below this range, depending on the trajectory of glucose trends [85]. Consumption of additional calories associated with a bout of exercise may be perceived as counterproductive, especially for individuals following a reduced calorie diet plan. However, a snack before or during exercise can be viewed as a positive behavior that promotes safety during exercise and may even make patients more likely to exercise. Patients with T1D who routinely consumed carbohydrates prior to exercise were found to be more likely to complete the weekly recommended 150 min of exercise [72], a fact that health care providers can discuss with patients to encourage this behavior as a reasonable safety measure. As previously noted in our discussion of dietary modification, macronutrient composition of meals can have a prolonged effect on

glycemic patterns; thus, pre-exercise meal composition can be utilized strategically to support the type of exercise a patient desires to perform.

There are multiple other modalities for glycemic management implementing insulin dosing surrounding a bout of exercise (including adjustment of pre-exercise prandial insulin dose, changing basal rate for those using insulin pumps, and adjusting target glucose level in AID systems), but these must be personalized based on individual responses to exercise, timing and content of the last meal, type of exercise to be performed, and type of insulin delivery. Because they are numerous and highly individualized, we will not detail each option here, but we provide references for resources to utilize for this purpose [85–89]. Glycemic management during exercise should be facilitated by the use of diabetes technology such as CGM, which is standard of care in all patients with T1D [82], or AID pumps, which should also be considered for use in all patients with T1D [82, 83]. Patients with T1D who used real-time CGM or AID systems had lower FOH survey scores [71], and use of technology in maintaining euglycemia during exercise is a positive facilitator of physical activity [90]. CGM used in conjunction with activity trackers has also been studied in the development of a prandial insulin dosing algorithm before and after exercise to reduce hypoglycemia and hyperglycemia [91]. All commercially available AID systems have options to change insulin delivery surrounding a bout of exercise (e.g., use of increased glucose targets, temporary basal or bolus reductions), but these generally must be changed manually, and some changes must be manually stopped after exercise as well [83]. Use of CGM has been evaluated during exercise in patients with T1D, and while CGM data have historically been shown to lag somewhat behind changes in blood glucose, more recent sensors have been less impacted by exercise [92–94]. Future developments including CGM with higher accuracy during exercise and automated insulin delivery systems that do not require a manual adjustment surrounding a bout of exercise would likely be beneficial for facilitating exercise habits in this group. As diabetes technology continues to develop, we may see a shifting mind-set regarding safety and ease of glycemic management during exercise in T1D, but more data will be needed to assess patient perspectives on exercise as technology evolves.

Choosing specific types of exercise may also modify risk for hypoglycemia. A 6-week HIIT program resulted in only mild hypoglycemia (average 67 mg/dL ±2.6 mg/dL) in 1.5% of observed HIIT training sessions, which totaled 3 hypoglycemic episodes out of 198 total training sessions [67]. Another HIIT-based protocol showed no increase in hypoglycemia frequency when compared to no exercise, and those who participated in HIIT workouts had lower rates of nocturnal hypoglycemia [95]. HIIT sessions being generally shorter in duration than typical moderate-intensity aerobic exercise may also help mitigate the barrier of lack of time to exercise. This factor combined with low rates of associated hypoglycemia may make HIIT an attractive option for patients with T1D as it overcomes two separate barriers to participating in exercise in this population.

Other barriers to exercise based on patient surveys include perceived low level of fitness, loss of control over diabetes, lack of time, cost, lack of knowledge, and restrictive work schedules [71, 73–77]. Social factors that can help facilitate engagement in regular physical activity include increased support from family, friends, and health care providers [71]. Ensuring that people living with T1D have supportive social structures in place that encourage healthy behaviors is a practical measure to consider in constructing a lifestyle intervention for obesity management. Use of DSMES as a continuing curriculum is another resource for facilitating healthy behaviors. Topics of healthy eating and physical activity are encompassed in the national standards of the DSMES curriculum, and this resource can be reintroduced at any time within a treatment plan; but its use is encouraged annually, if patients are not meeting treatment goals, if major life transitions occur, or if complicating factors arise [40].

5 | Pharmacotherapy

5.1 | First-Generation Obesity Medications

Current guidelines recommend obesity medications for patients with BMI > 27 kg/m^2 and weight-related comorbidities or BMI > 30 kg/m^2 [96]. The use of BMI-centric criteria for treatment warrants some discussion. While easy to determine quickly in clinic, the BMI remains very rudimentary and problematic when diagnosing overweight and obesity and determining treatment. By only capturing one's height and weight, the BMI ignores body composition, adipose distribution, gender, age, or race, all necessary factors for determining risk and disease severity. Until a superior clinical marker is determined, clinicians should weigh all other key aspects and, most importantly, gauge the change in BMI to better develop treatment plans and goals of care. Particularly for patients with T1D and obesity, an elevated waist circumference (greater than 35 in for females or 40 in for males) and/or evidence of metabolic disease including hypertension, dyslipidemia, and MASLD (metabolic associated steatotic liver disease) should prompt greater concern and intensity of obesity treatment.

There are six medications approved for chronic weight management, three approved for short-term use (phentermine, phendimetrazine, diethylpropion), and one approved for specific syndromal and monogenic causes of obesity (setmelanotide) [96]. Generally, orlistat, extended-release phentermine/topiramate, bupropion/naltrexone, and phentermine are regarded as first-generation obesity medications, with all the others considered to be second-generation obesity medications. Ongoing research and development in this field contribute to a rapidly changing landscape, with a greater number of medications, more effective medications, and a strong pipeline of future medications. Despite this advancement with obesity medications, a critical question remains. Are these safe and effective treatment options for patients with both obesity and T1D? While not contraindicated for patients with T1D, not one of the first-generation obesity medications has been studied in patients with T1D and obesity. Little data exist to elicit the utilization of obesity medications in patients with T1D and obesity. Overall, rates of prescribing obesity medications remain substantially low, particularly compared to rates of prescribing medications for other chronic diseases such as T2D or hypertension [97]. An initial study investigating the use of weight loss medications in the T1D Exchange Clinic Registry revealed only 0.9% of patients

TABLE 2 | Obesity and diabetes medications and associated weight changes in patients with and without diabetes [100–129].

Medication	Average long term (1 year) weight loss in patients without T2D	Average long term (1 year) weight loss in patients with T2D not using insulin therapy	Average long term (1 year) weight loss in patients with T2D on insulin therapy	Average long term (1 year) weight loss in patients with T1D
Phentermine (8 mg-37.5 mg)	3%–8% (12–24 weeks)	Unknown	Unknown	Unknown
Phendimetrazine	Unknown	Unknown	Unknown	Unknown
Diethylproprion	Unknown	Unknown	Unknown	Unknown
Orlistat	8.5%	4.7%	3.76%	Unknown
Extended-release phentermine-topiramate	10.5%	9.0%	Unknown	Unknown
Bupropion/naltrexone	6.4%	5.0%	Unknown	Unknown
Liraglutide	8.0%	6.0%	3%–4% (at 6 months)	4.9 kg
Semaglutide	14.9%	9.6%	7.6% (at 6 months)	15.9 lb
Tirzepatide	22.5%	14.7%	8.8% (at 40 weeks)	18.%
Metformin[a]	2.0–5.0 kg	2%–3%	N/A (studies shower lower weight gain)	3.8 kg
Pramlintide[a]	N/A	N/A	N/A	0.4–1.3 kg
Dapagliflozin[a]	N/A	1.8–3.5 kg	2.39–3.5 kg	3.90 kg
Empagliflozin[a]	N/A	4-5 kg	−2.39−3.5 kg	3.60 kg

[a]These medications are not FDA-approved obesity medications but are associated with weight loss.

utilized first-generation weight loss medications, and 2.6% utilized a second-generation weight loss medication [98]. A more recent study investigating specifically glucagon-like peptide-1 (GLP-1)/GLP-1 receptor agonist (RA) use in the T1D Exchange found the rate has increased to 5.8%, with the majority using semaglutide (67%) followed by tirzepatide (16%) [99]. This lack of data regarding obesity pharmacotherapy safety and efficacy in patients with T1D and obesity must be addressed to improve the care delivered to this increasing patient population.

All of the obesity medications approved for chronic treatment of polygenic obesity have data regarding weight loss response in patients without diabetes and patients with T2D. Patients with T2D consistently lose less weight on average compared to patients without diabetes. Table 2 includes this comparison [100–118]. Data suggest that patients with T1D and obesity develop insulin resistance and other pathophysiology that overlaps with T2D, occasionally called “double diabetes” [130, 131]. Therefore, it may be reasonable to assume that the effectiveness of these medications for patients with T1D and obesity parallels the effectiveness of that seen in patients with T2D and obesity. Unfortunately, studies of weight loss medications in patients with obesity and T2D often exclude people who are on insulin treatment, a critical factor in treatment response as insulin tends to promote weight gain [132, 133]. The existing data as shown in Table 2 confirm that patients requiring insulin for treatment of T2D experience less weight loss. Patients with T1D and obesity typically require much higher doses of insulin for a longer duration, which risks mitigating the weight loss response. Similarly, patients with T1D and obesity face greater risk of hypoglycemia, which promotes additional energy intake often in the form of simple carbohydrates and processed or ultraprocessed foods. This may trigger cravings and further disordered eating behaviors, all of which lessen the energy deficit and subsequent weight loss. In fact, those patients utilizing obesity medications in the T1D Exchange Clinic Registry did not have statistically significant weight changes after starting or stopping obesity medications [98].

Until more data emerge regarding the safety and efficacy of AOMs in patients with T1D and obesity, patients may benefit from clinicians using a more unique approach to medication management for obesity treatment. Minimizing the use of medications with high weight gain potential and maximizing the use of medications associated with weight loss for patients with T1DM and overweight/obesity may at least stabilize weight or enhance the response to behavioral modifications for weight management. Important ones to consider include insulin, incretins, antidepressants, antipsychotics, and neuropathic pain medications [134] (for example, utilizing topiramate or zonisamide for migraine prevention). Also, patients treated with basal insulin Glar-300 tend to have less weight gain and mild weight

TABLE 3 | Weight-gaining medications [136–138].

Medication class	Examples of weight-gaining medications	Examples of weight-favorable or weight-neutral alternative
Insulin	NPH, deludec, Glar-100	Detemir, Glar-300
Antidepressants or mood stabilizers	TCAs (nortriptyline, amitriptyline), SSRIs (paroxetine, escitalopram, citalopram, duloxetine), mirtazapine, lurasidone, lithium	Bupropion Fluoxetine Sertraline Fluvoxamine Lamotrigine
Antipsychotics	Quetiapine, risperidone, olanzapine, valproic acid	Ziprasidone, aripiprazole
Antieleptics	Gabapentin, pregabalin	Topiramate Zonisamide
Beta-blockers	Metoprolol, propranolol, atenolol	Carvedilol Angiotensin receptor blockers (valsartan, olmesartan) Angiotensin-converting enzymes (lisinopril, enalapril)
Contraceptives	Depo-medroxyprogesterone	Intrauterine device Oral contraceptives
Steroids	Prednisone, cortisone, prednisolone	Alternate day dosing Limit exposure

loss compared to NPH insulin, degludec, or Glar-100 [80, 135]. Table 3 [136–138] lists common weight-gaining medications and weight-neutral or weight-favorable alternatives. Furthermore, patients with T1D treated to more aggressive glycemic targets tend to experience more weight gain [133, 139]. For example, the DCCT/EDIC study found that over the 30-year period, females in the conventional treatment arm had a lower mean weight than females in the intensive group, yet the small difference (2.7 kg) may be of little clinical significance as the mean BMI in the intensive group increased from 23.3 to 27.2 kg/m^2 without worsening metabolic parameters [4].

The amylin analogue pramlintide deserves special mention. Amylin is a 37-amino acid peptide hormone which is secreted by the pancreatic beta cells and, like insulin, in response to nutrient stimuli [140]. Therefore, not surprisingly, those with T1D with complete fibrosis of the beta cells are amylin deficient [141]. Pramlintide was approved by the US FDA in 2005 for bolus premeal administration adjunct to insulin therapy for individuals with T1D or T2D [142]. While the drug is approved for diabetes and not obesity, it has a similar action as GLP-1 RAs in that it diminishes satiety, retards gastric emptying, and inhibits glucagon secretion [143].

Over time, weight loss is modest with pramlintide. In the registration study for T1D, weight loss over 12 months was 0.4 kg on the highest dose taken three times daily (60 mcg). The placebo control group had a 0.8 kg weight gain [141]. Similarly, in a meta-analysis assessing different doses and different amounts of drug exposure, weight loss was minimal with small reductions in HA1c and insulin dosing [144]. The greatest impact of pramlintide appears to be the first few weeks after starting the drug. Reported side effects and concerns with tachyphylaxis after about a year of treatment limit its popularity [145, 146]. Interest in amylin analogues' ability to influence weight loss has been resurrected, particularly with the promise of combination semaglutide and cagrilinitide (GLP-1 RA and amylin analogue). This duo demonstrated better weight loss and glycemic control than either agent alone in phase 2 trials [147]. Unfortunately, participant inclusion criteria prevent applicability to patients with obesity and T1D or patients with T2D and insulin use.

Finally, metformin and sodium-glucose cotransporter inhibitors, when used as adjunct agents to insulin for glycemic control, deserve mention. Metformin remains an optimal first-line treatment for T2D as it increases insulin sensitivity, reduces hepatic glucose production, and results in modest weight loss [119]. Studies of metformin with insulin in patients with T1D have shown some weight reduction (−1.17 kg to −3.8 kg) [148]. Both dapagliflozin and empagliflozin have shown weight reductions in patients with T1D (−2.95% to −4.54% and −1.5 kg to −3.6 kg) when compared to placebo, albeit at the risks of higher rates of DKA (2.6%–3.4% and 3.3%–4.3%, respectively) compared to placebo (1.2%–1.9%) [120, 121, 149].

The present day intense obesity medication research and development accelerate an already rapidly changing landscape in pharmacotherapy for obesity treatment. Many of these promising agents for obesity treatment are different doses or combinations of therapies with proven effectiveness for treating T2D. While meager, some data exist on the use of the second-generation weight loss medication in patients with T1D, as discussed later in this review. Clinicians do not have a "standard of care" for obesity and weight management in patients with T1D as they do for patients with T2D or patients without diabetes. The field of obesity medicine desperately needs increased attention to and investigation of best practices to treat patients with T1D and obesity.

5.2 | Newer Agents: GLP-1 and GLP/GIP Receptor Agonists

Including the two GLP-1 RAs that are combined with basal insulin (and therefore would not be able to easily be used with T1D as there would be no flexibility in the insulin dosing), there are eight preparations available in the United States. They are all approved for T2D while the same molecules with different trade names are approved for obesity (liraglutide for Saxenda, semaglutide for Wegovy, and tirzepatide for Zepbound). It needs to be recalled that tirzepatide is a combination GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) RA. While the package inserts for the diabetes-approved GLP-1 RAs (Victoza, Ozempic, and Mounjaro) specifically note they are not approved for T1D, there is no mention of T1D for the labeling for their obesity-approved twins. For obesity, therefore, these three GLP-1 RAs are not contraindicated.

Retrospective observational trials have shown these agents to be effective with T1D. In one report with a matched control group, after 12 months weight was reduced 15.9 lb compared to an increase of 2.1 lb in the control group [122]. There was also a nonsignificant improvement of HA1c of 0.3% with less hypoglycemia measured by CGM ($p = 0.04$) [122]. Another group noted in T1D patients receiving tirzepatide, at 8 months, a 10.1% and significant 0.45% reduction of body weight and HA1c, respectively [150]. Still, its use in this population requires a deep knowledge of insulin management, both for those receiving MDI and continuous subcutaneous insulin infusion (CSII), both open-loop and AID. The details of current day insulin therapy [151] and insulin adjustments with GLP-1 RA therapy in T1D are beyond the scope of this article but are reviewed elsewhere [152, 153]. It is important to appreciate the use of CSII is increasing dramatically [154], especially now that AID is so widely available.

Hypoglycemia is always a concern in T1D due to a well-understood reduction of glucagon counter-regulation [155]. The situation is potentially more concerning with the use of GLP-1 RAs, as mealtime insulin may be too much for a suppressed appetite. The need to reduce insulin with weight loss also needs to be considered, but perhaps most concerning is the mismatching of insulin with food given the delay of gastric emptying. A major point is the need to not administer mealtime insulin 15–20 min prior to eating as normally recommended. Rather, anecdotally, we've learned to give insulin at the beginning of the meal and to avoid the ultra-rapid acting analogues (fast-aspart and lispro-aabc) or Technosphere insulin (pulmonary inhaled insulin). In ADJUNCT 2 (primary outcome was HA1c reduction), the 1.2 mg dose of daily liraglutide resulted in more symptomatic hypoglycemia than placebo ($p = 0.03$) [156]. However, anecdotally, severe hypoglycemia (requiring the assistance of another person) has been described with the weekly medications [157].

6 | Bariatric Surgery in Patients With T1D

Patients with BMI > 35 kg/m^2 and weight-related complications or BMI > 40 kg/m^2 meet criteria for bariatric surgery [158]. Diabetes guidelines also now recommend bariatric surgery for patients with T2D and BMI > 30 kg/m^2 [159]. To date, surgery offers the highest long-term effective treatment for severe obesity, granted with the greatest risk. Fortunately, the rapid progress in surgical treatment has markedly lowered the risk for complications and mortality. Data have consistently demonstrated the ability of surgical obesity treatment to improve and often normalize glycemic control, alleviate medication burden for T2D, and improve quality of life. Of note, patients with longer duration of T2D and insulin use tend to have residual disease or more disease recurrence but still experience great benefit [160]. Table 4 [160–167] lists the most common bariatric surgical techniques and the average weight loss for patients with and without T2D.

Like data on use of obesity medications in patients with T1D and obesity, data on bariatric surgery in patients with T1D and obesity are deficient and necessitate more studies. It is thought that because patients with T1D lack the beta-cell reserve patients with T2D have, the decreased insulin resistance and improved metabolic disease may be much less with bariatric surgery [171, 172]. A small study of 10 patients with severe obesity (mean baseline BMI 41.6 kg/m^2) and poorly controlled T1D (mean baseline HA1c 10.0%) showed favorable outcomes after bariatric surgery with weight reduction (mean BMI 27 kg/m^2 at 36 months), improved glycemic control (mean HA1c 8.9%), and improved blood pressure control [173]. Of note, 70% of the patients had Roux-en-Y gastric bypass. Another study reporting outcomes of bariatric surgery in adults in the T1D Exchange Clinic Registry found limited statistically significant difference in metabolic outcomes [98]. Over 5 years, weight decreased more modestly from BMI 38 to 33 kg/m^2 and likewise mean HA1c levels dropped to only 8.1% from 8.8%. Although lipid profile improved, blood pressure changed minimally before and after surgery. Similarly, the Roux-en-Y gastric bypass was the most common surgery performed. When compared to matched case controls, patients had a lower BMI after surgery, yet higher HA1c. A more recent systemic review and meta-analysis that included over 600 patients revealed substantial weight loss (from mean BMI 42.6 to 29 kg/m^2), reduced insulin requirements, and improved HA1c levels [174]. Again, the Roux-en-Y gastric bypass was the most common surgery performed, and mean follow-up was just shy of 3 years. Clinicians must recognize this key difference when caring for patients with T1D undergoing bariatric surgery versus those with T2D. Clinicians should focus on ensuring adequate weight response, nutritional quality, physical activity, and reduction in other cardiovascular risk factors (blood pressure, cholesterol) and minimizing these patients' unique risk for hypoglycemia or DKA and not expect tremendous improvement in glycemic control.

Important questions need to be answered to help clinicians determine which patients with T1D and severe obesity would benefit the most from surgical obesity treatment and which treatment to recommend. Current data suggest patients with LADA likely gain the most benefit when treated early so that improved insulin sensitivity stops beta-cell apoptosis, preserving beta-cell mass and avoiding complete insulin deficiency [175, 176]. Yet intervening too early could be problematic as a patient with classic T1D may still be making c-peptide, preventing a confirmed diagnosis of LADA or classic T1D. Furthermore, the risk of complications with each type of bariatric surgery must be carefully weighed. Hypoglycemia dominates as the most frequent complication after bariatric surgery in patients with T1D, with an

TABLE 4 | Common procedures for surgical obesity treatment [160–170].

Type of surgery (and number performed per year in 2022)	Average % total weight loss in patients without T2D	Average % total weight loss in patients with T2D	Rate of remission of T2D	Average reduction in BMI in patients with T1D	Common complications
Adjustable gastric banding (2500)	17%	10%–20%	56.7%	N/A	Band erosion Band slippage Nausea Esophageal dysmotility
Sleeve gastrectomy (160,609)	20%–25%	19%	47%	10.5	GERD Nutritional deficiencies (vitamin B12, iron, vitamin D) Esophageal stricture
Roux-en-Y gastric bypass (62,097)	25%–30%	23%	93%	8.7–16.5	Dumping syndrome and post-bariatric surgery hypoglycemia (PBH) Marginal ulcer Nutritional deficiencies (protein, vitamin D, iron, calcium, vitamin B12, thiamine) Internal hernia
Biliopancreatic duodenal switch (6096)	37%–41%	31%	95.1%	N/A	Steatorrhea Nutritional deficiencies (fat soluble vitamins, selenium, zinc, copper, vitamin B12, protein, iron, calcium)
Single anastomosis duodenal switch (1567)	38%	Not well established	81%	23	Diarrhea Nutritional deficiencies (protein, fat soluble vitamins, vitamin B12, iron, selenium, zinc, copper)

incidence of well over 50% [174, 177]. A better understanding of the factors contributing to this, such as carbohydrate absorption, dietary intake, physical activity, and insulin titration, could tremendously reduce this risk and improve patient outcomes. As such, patients would be best served using an AID system following bariatric surgery. Similarly, studies need to dedicate more attention to how best to mitigate DKA, a life-threatening complication of T1D, which has been reported in up to 25% of patients following bariatric surgery [178]. Furthermore, patients with T1D planning to have bariatric surgery may require unique assessments, such as baseline screening for osteoporosis and autoimmune thyroid disease. The initial insult to bone mass often occurs at a young age, and patients with T1DM longer than 5 years have lower bone mass; thus, premenopausal women and men should be screened prior to bariatric surgery rather than the recommended 2 years after surgery [179, 180]. Finally, questions around improvements in other aspects of health such as sleep, nutritional quality, quality of life, mobility, and depression and health care costs in patients with T1D and obesity who undergo bariatric surgery need to be resolved. Unfortunately, all existing studies remain highly limited by the size of evidence and study design to draw the best conclusions. Despite these limitations, bariatric surgery has proven benefits and should be a treatment option for patients with T1D and severe obesity.

7 | Screenings and Health Maintenance in T1D During Obesity Treatment

There are multiple areas in the management of T1D that should remain under close monitoring in general care; this component of care is especially pertinent during treatment for obesity, as changes in weight and glycemic control can result in changes to other areas including surveillance for retinopathy, foot health, blood pressure, lipid profiles, and bone health. Other autoimmune diseases (such as hypothyroidism and celiac disease) that can affect weight loss and nutritional status should also be considered and screened for in this population at higher risk for other autoimmune processes. Finally, the effects on fertility and contraception needs in females of childbearing age should also be given some thought during the treatment of obesity. In Table 5 [181, 182], we have compiled our recommendations regarding screenings and other health metrics in T1D to consider while treating obesity.

8 | Conclusion

More patients than ever before have both T1D and obesity. The advancements in obesity treatment have been fast and furious

TABLE 5 | Recommended Screenings in T1D During Obesity Treatment [181, 182].

Health measure	Recommendation
Eye health	Recommend prompt eye exam if patients experience rapid reduction in A1c due to risk of worsening retinopathy [181]
Foot health	Recommend more frequent personal foot exams when increasing levels of physical activity, especially for individuals with loss of protective sensation
Autoimmune comorbid diseases	Screen for autoimmune thyroid disease and celiac disease based on clinical signs and symptoms
Fertility	Recommend A1c < 6.5% prior to pursuing fertility [182]; recommend contraception in all females of childbearing age who are not planning fertility
Osteoporosis	Recommend screening for risk factors for osteoporosis in perimenopausal women with T1D starting weight loss treatment and continued screening for osteoporosis risk during weight loss

over the past two decades. Multiple different guidelines and recommendations for obesity treatment exist to direct clinical decisions and facilitate quality patient care. While these resources address the general population and certain specific populations (patients with T2D, pediatric and adolescent patients, etc.), they consistently exclude patients with T1D. Unfortunately, the answer is not to simply extrapolate data and apply it to patients with both T1D and obesity. Well-performed studies focusing on current obesity treatments in patients with T1D need to be a priority. When treating patients with both T1D and obesity, clinicians need to be well informed of the unique needs, responses, and complications these patients face and apply an interdisciplinary approach with experts in both T1D and obesity.

Conflicts of Interest

Dr. Hirsch reports research with Tandem, Dexcom, and Mannkind and a consulting role with Abbott, Roche, and Hagar. The other authors declare no conflicts of interest.

Data Availability Statement

Data sharing are not applicable to this article as no new data were created or analyzed in this study.

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