Journal of
Medical Case Reports

CASE REPORT Open Access

Semaglutide and laparoscopic sleeve gastrectomy in an adolescent with congenital adrenal hyperplasia due to 21-hydroxylase: a case report

Alaina P. Vidmar[1*], Linus Kaiser[1], Matthew J. Martin[2,3], Stuart Abel[2,3], Aimee G. Kim[2], Madeleine Weitzner[1], Cynthia E. Muñoz[1,4], Lynda K. Fisher[1], Mimi S. Kim[1] and Kamran Samakar[2,3]

Abstract

Background Classic congenital adrenal hyperplasia, primarily due to 21-hydroxylase deficiency, leads to impaired cortisol and aldosterone production and excess adrenal androgens. Lifelong glucocorticoid therapy is required, often necessitating supraphysiological doses in youth to manage androgen excess and growth acceleration. These patients experience higher obesity rates, hypertension, and glucose metabolism issues, complicating long-term health management. Despite this, there is limited literature on effective obesity management strategies in congenital adrenal hyperplasia patients, emphasizing the need for comprehensive care approaches.

Case presentation We present the case of an 18-year-old Hispanic male with classic congenital adrenal hyperplasia and class III obesity, who underwent a multimodal obesity treatment plan. Diagnosed shortly after birth, he experienced rapid weight gain starting at the age of 2 years, with his body mass index escalating to 52.5 kg/m^2 by age 15. Initial interventions included lifestyle modifications and pharmacotherapy with metformin and topiramate, which were ineffective alone. Subsequently, he was treated with semaglutide, achieving an 11% body mass index reduction. Owing to ongoing metabolic dysregulation, he underwent laparoscopic sleeve gastrectomy at the age of 17 years. The surgery was well tolerated, with careful intraoperative glucocorticoid management. Post-surgery, he experienced significant improvements in body mass index, hunger, and satiety, along with a reduction in emotional overeating.

Conclusions This case highlights the potential of an integrative, multidisciplinary approach to address severe obesity and its associated comorbidities in patients with classic congenital adrenal hyperplasia. The successful outcomes from both pharmacotherapy and bariatric surgery suggest that tailored obesity management strategies can optimize health in this unique population, reinforcing the need for further research into comprehensive obesity care in individuals with congenital adrenal hyperplasia.

Keywords Congenital adrenal hyperplasia, Pediatric obesity, Laparoscopic sleeve gastrectomy

*Correspondence:
Alaina P. Vidmar
avidmar@chla.usc.edu
Full list of author information is available at the end of the article

Introduction

Classic congenital adrenal hyperplasia (CAH), primarily due to 21-hydroxylase deficiency, is characterized by impaired cortisol and aldosterone synthesis, and excessive adrenal androgen production [1]. These patients require lifelong glucocorticoid therapy for cortisol replacement, with supraphysiological doses (12–15 mg/m^2/day) often required in youth with classic CAH to achieve androgen suppression and thereby avoid growth acceleration and bone age advancement [1]. While essential for the management of CAH, glucocorticoid treatment can present unique challenges related to metabolic health, including difficulty mimicking physiologic diurnal cortisol secretion with current glucocorticoid formulations and potential exacerbation of obesity-related complications [1–4]. Youth with CAH experience higher rates of obesity, hypertension, and impaired glucose metabolism compared with their peers, along with an early adiposity rebound, compounding the challenges in their long-term management [1–3, 5, 6].

Despite the established link between CAH and obesity, a significant gap exists in the literature regarding effective strategies for managing obesity in this population. The American Academy of Pediatrics and the American Society of Metabolic and Bariatric Surgery advocate for a comprehensive approach to managing severe obesity in all pediatric patients, regardless of concomitant chronic conditions [7, 8]. This multifaceted strategy integrates lifestyle modifications, pharmacotherapy, and, when appropriate, metabolic and bariatric surgery [9]. Emerging literature indicates that pharmacotherapy for obesity can offer benefits for patients with CAH, although comprehensive data remains limited [10]. Similarly, while bariatric surgery is the most effective and durable treatment option for severe obesity in the general pediatric population, its application in individuals with CAH has not been extensively explored, despite the potential for substantial improvements in weight and metabolic health.

In this context, we present a case report of an 18-year-old Hispanic male with classic CAH and class III obesity, who underwent a multimodal obesity treatment plan incorporating both obesity pharmacotherapy and laparoscopic sleeve gastrectomy. This case underscores the potential for a tailored, integrative approach to effectively address severe obesity and its comorbidities in patients with classic CAH. In addition, we have performed a structured review of the literature on CAH and obesity management. Our findings highlight sustained weight reduction, resolution of weight-related complications, and improved management of androgen excess following the surgery. This report contributes to the growing body of evidence supporting comprehensive obesity care in this unique population, emphasizing the need for multidisciplinary strategies to optimize health outcomes.

Case presentation

History and early life

The patient, an 18-year-old Hispanic male diagnosed with classic CAH due to 21-hydroxylase deficiency, was identified shortly after birth, with a 17-hydroxyprogesterone (17-OHP) level of 60,601 ng/dL (918.2 nmol/L; conversion whole blood units × 66 to serum concentration [11]) detected from routine newborn screening at 29 hours of life. He was born full-term via normal spontaneous vaginal delivery and appeared well at birth. His mother and father identify as Hispanic. Maternal history included hypertension and gestational diabetes, while the family history was negative for CAH. During early childhood, he was managed with oral hydrocortisone and fludrocortisone to maintain adrenal function. Growth patterns during infancy and early childhood showed that he tracked along the 17th percentile for height and the 69th percentile for weight until 24 months of age, with a weight-for-length percentile of 87. However, from the age of 2 years, he began to experience a significant increase in weight, with a rapid weight gain velocity of approximately 6 kg per year from 6 to 13 years old. By the age of 11 years, the patient's body mass index (BMI) was recorded at 38.7 kg/m^2 (165% of the 95th percentile), and his 17-OHP levels were poorly controlled at 10,519 ng/dL, despite taking hydrocortisone 35 mg/m^2/day divided into three daily doses owing to adherence challenges. Notably, his bone age was advanced at 14.5 years, relative to his chronological age of 11 years, raising concerns about potential long-term implications for his growth and development, which may be attributed to a combination of obesity, hyperandrogenism, and poor hormonal control.

Comprehensive obesity care—lifestyle modification and obesity pharmacotherapy

He began intensive health and behavior lifestyle modification for obesity treatment at the age of 11 years. At his first presentation to the multidisciplinary comprehensive obesity care clinic, the patient's BMI was measured at 40 kg/m^2 (190% percent of the 95th percentile, Fig. 1). Leading up to this consultation, he reported extreme food cravings and difficulties with portion control, contributing to further weight gain. In response to these challenges, he was started on metformin 1000 mg twice daily and topiramate 100 mg nightly (started at 25 mg nightly and titrated to goal), which he tolerated without any side effects, and he noted that it helped suppress his appetite. In addition, lifestyle modifications were implemented, including an increase in physical activity, and changes in nutrition.

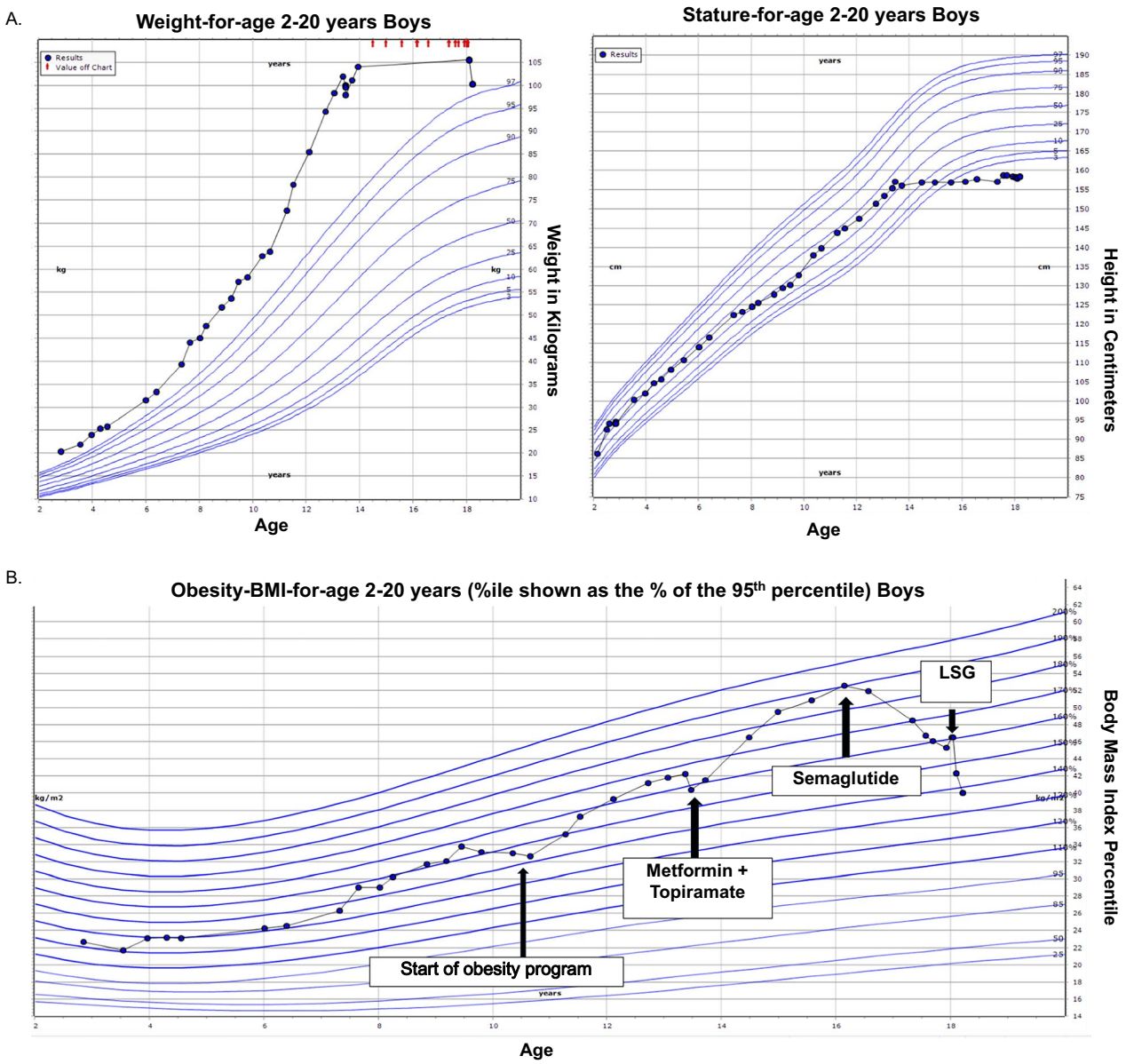


Fig. 1: An 18-year-old Hispanic male with congenital adrenal hyperplasia due to 21-hydroxylase deficiency and severe obesity: weight, height (**A**), and body mass index (**B**) trajectories over time, from age 2 to 18 years, in response to comprehensive obesity treatment

He remained on this regimen for 2 years, yet his BMI continued to rise, reaching 49.5 kg/m^2 despite optimizing topiramate to 200 mg nightly. By the age of 15 years, his BMI had escalated to 52.5 kg/m^2, and he was showing metabolic dysregulation with elevation in liver enzymes, blood lipid levels, and hemoglobin A1c (5.8%). Thus, he began treatment with semaglutide, initially starting at a dose of 0.25 mg and titrating up to 1 mg weekly, after 12 months of which he achieved an 11% BMI reduction to 46.7 kg/m^2 despite intermittent pauses in treatment due to supply access challenges. He reported no gastrointestinal side effects during his semaglutide titration and overall denied any side effects from the medication. Upon reaching the 1 mg weekly dose he reported a significant reduction in his appetite, improved satiety, increased energy, and improved overall mood. His maintenance daily hydrocortisone dose was decreased by 9% (34 mg/m^2/day) as his androgen control improved during the period of time that he was taking semaglutide, his weight was trending down, and his hydrocortisone adherence remained high.

Metabolic and bariatric surgery consideration and intraoperative care

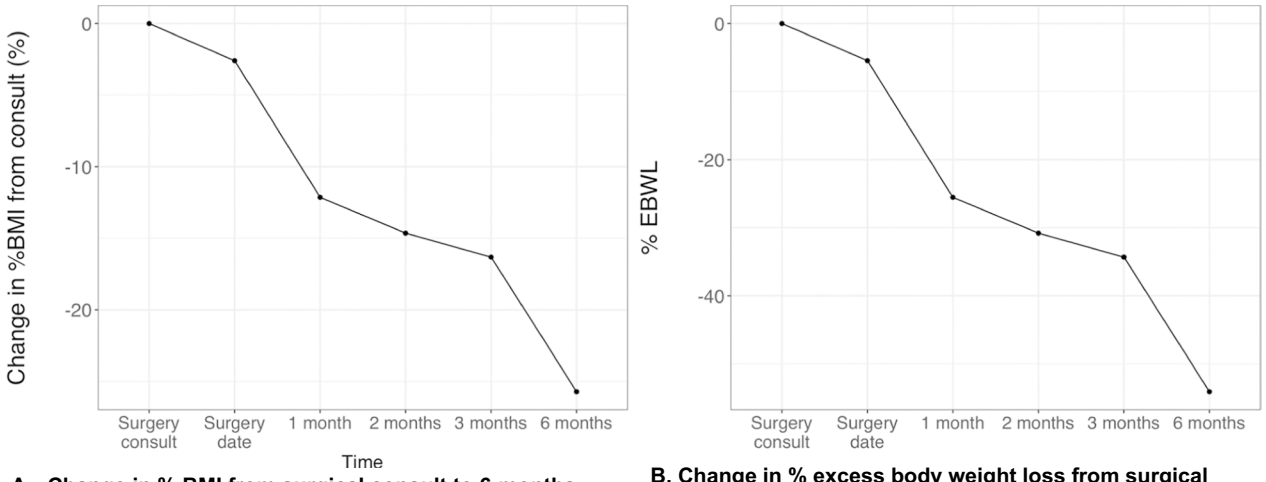
Despite significant reductions in his BMI, the patient continued to have an elevated hemoglobin A1c (5.9%), liver enzymes, and blood lipid levels (Table 1). As a result, he was referred to the metabolic and bariatric surgery program for consideration of metabolic and bariatric surgery. After a thorough evaluation by the interdisciplinary metabolic and bariatric surgery team, he was assessed as a good candidate for laparoscopic sleeve gastrectomy and underwent the procedure at the age of 17 years. Intraoperatively, he received standard stress-dose hydrocortisone at 50 mg/m^2/day, followed by a 48-hour moderate stress-dose regimen to support pain control and stress response. The surgery was well-tolerated, with no intraoperative or perioperative complications. He required no additional stress-dose coverage and was able to return to his maintenance hydrocortisone regimen (35 mg/m^2/day) 2 days postoperatively. At 2 and 4 weeks postoperatively, he completed a validated eating behavior questionnaire, reporting a threefold reduction in hunger and a twofold increase in satiety. However, as his emotional overeating and food responsiveness scores remained elevated compared with baseline, semaglutide was restarted to help manage his self-reported emotional overeating behaviors. A total of 6 months postoperatively, his BMI had decreased to 39 kg/m^2 (Fig. 2), and his emotional overeating and food responsiveness scores had improved, showing a twofold reduction. His maintenance daily hydrocortisone dose was decreased by an additional 9% (34 mg/m^2/day).

Glucocorticoid therapy and surgical risk

It is important to recognize that hydrocortisone, commonly used in CAH management, has a significantly lower glucocorticoid potency compared with other longer-acting steroids, such as prednisone. As well, the glucocorticoid dosing in CAH, as a primary adrenal insufficiency, is relatively near-physiological compared with chronic steroid use seen in patients with systemic disease. This distinction may reduce concerns about anastomotic leaks or gastric perforations in patients with CAH compared with those on higher-potency, high-dose steroids who are known to have increased risk of

Table 1 Biochemical analysis over time in response to comprehensive obesity treatment in an 18-year-old male with congenital adrenal hyperplasia due to 21-hydroxylase deficiency

Test	Prior to antiobesity medication start	12-mo post topiramate and metformin	6-mo post semaglutide	6-mo post sleeve gastrectomy	Normal range
Sodium	142 mEq/L (mmol/L)	140 mEq/L (mmol/L)	139 mEq/L (mmol/L)	140 mEq/L (mmol/L)	135–140 mEq/L (mmol/L)
Potassium	3.9 mEq/L (mmol/L)	3.7 mEq/L (mmol/L)	3.5 mEq/L (mmol/L)	3.8 mEq/L (mmol/L)	3.6–5.0 mEq/L (mmol/L)
Aspartate aminotransferase	**57 U/L (IU/L)**	23 U/L (IU/L)	25 U/L (IU/L)	23 U/L (IU/L)	15–46 U/L (IU/L)
Alanine aminotransferase	**89 U/L (IU/L)**	21 U/L (IU/L)	20 U/L (IU/L)	21U/L (IU/L)	3–35 U/L (IU/L)
Hemoglobin A1c	**5.9%**	5.6%	5.1%	5.1%	< 5.7%
Triglyceride	**297 mg/dL (3.36 mmol/L)**	72 mg/dL (0.81 mmol/L)	88 mg/dL (0.99 mmol/L)	72 mg/dL (0.81 mmol/L)	40–160 mg/dL (0.45–1.81 mmol/L)
Cholesterol	**265 mg/dL (6.85 mmol/L)**	225 mg/dL (5.83 mmol/L)	192 mg/dL (4.97 mmol/L)	166 mg/dL (4.30 mmol/L)	65–175 mg/dL (1.69–4.54 mmol/L)
High-density lipoprotein	**26 mg/dL (0.67 mmol/L)**	36 mg/dL (0.93 mmol/L)	41 mg/dL (1.07 mmol/L)	52 mg/dL (1.35 mmol/L)	35–70 mg/dL (0.91–1.81 mmol/L)
Low-density lipoprotein	**174 mg/dL (4.51 mmol/L)**	136 mg/dL (3.52 mmol/L)	121 mg/dL (3.13 mmol/L)	94 mg/dL (2.43 mmol/L)	< 100 mg/dL (< 2.59 mmol/L)
17-hydroxyprogesterone	**1212 ng/dL (4.24 nmol/L)**	**3388 ng/dL (11.71 mmol/L)**	142 ng/dL (0.5 mmol/L)	73 ng/dL (0.25 mmol/L)	Target in CAH management 500 ng/dL
Androstenedione	61 ng/dL (0.21 nmol/L)	**191 ng/dL (0.66 nmol/L)**	21 ng/dL (0.07 nmol/L)	23 ng/dL (0.08 nmol/L)	21–154 ng/dL (0.07–0.54 nmol/L)
Adrenocorticotropic hormone	**104 pg/mL (23.4 pmol/L)**	37 pg/mL (8.2 pmol/L)	25 pg/mL (5.5 pmol/L)	< 5 pg/mL (< 1.1 pmol/L)	9–57 pg/mL (2–12.5 pmol/L)
Plasma renin activity	5.28 ng/mL/h (5.28 µg/L/h)	**11.98 ng/mL/h** (11.98 µg/L/h)	1.83 ng/mL/h (1.83 µg/L/h)	0.3 ng/mL/h (0.3 µg/L/h)	0.25–5.82 ng/mL/h (0.25–5.82 µg/L/h)
Total testosterone	354 ng/dL (1.23 nmol/L)	312 ng/dL (1.09 nmol/L)	379 ng/dL (1.32 nmol/L)	433 ng/dL (1.51 nmol/L)	300–950 ng/dL (1.04–3.3 nmol/L)



A. Change in % BMI from surgical consult to 6-months postoperatively

B. Change in % excess body weight loss from surgical consult to 6-months postoperatively

Fig. 2 Change in percent body mass index (**A**) and percent excess weight loss (**B**) from surgical consult to 6 months after a laparoscopic sleeve gastrectomy in an 18-year-old Hispanic male with congenital adrenal hyperplasia due to 21-hydroxylase deficiency

surgical complications, including impaired wound healing, ulcer disease, increased infection rates, and higher risk of gastrointestinal leaks [12, 13]. Bariatric surgery can still be a safe option in this population when managed appropriately, with careful preoperative planning and postoperative management. Optimizing intraoperative glucocorticoid dosing, ensuring meticulous perioperative care, and closely monitoring for complications helped mitigate the risks associated with surgery. A multidisciplinary approach was essential in determining the appropriate timing for surgery and minimizing potential complications, ultimately contributing to a successful outcome [14].

Discussion

This case report highlights the challenges of managing severe obesity in patients with congenital adrenal hyperplasia, emphasizing the need to consider a multimodal approach. The hormonal imbalances inherent to CAH can contribute to various metabolic complications, including obesity, insulin resistance, and dyslipidemia, making effective management of cases in young patients particularly challenging. Our patient exhibited rapid weight gain at an early age, reflecting the early adiposity rebound and trend toward severe obesity seen in youth with CAH. This case underscores the necessity of early intervention and comprehensive management strategies of obesity in this high-risk population.

A comprehensive literature review found two additional cases discussing the use of bariatric surgery in individuals with classic CAH. Zatsepina (2023) described a 45-year-old female patient with classic CAH who experienced a 72% reduction in excess weight following a sleeve gastrectomy, along with a 25% reduction in her daily prednisolone dose after 7 months [15]. Mallappa (2017) focused on a 19-year-old female with classic CAH who also had a sleeve gastrectomy, resulting in a 48% reduction in percent BMI and a 34% decrease in her daily hydrocortisone dose 15 months later after starting at a daily dose of 16 mg/m^2/day [16]. Collectively, these case reports highlight the potential for metabolic and bariatric surgery to facilitate significant weight loss and improve glucocorticoid management in patients with CAH and severe obesity. These case reports collectively illustrate a promising trend: bariatric surgery can facilitate significant weight loss and improve glucocorticoid replacement dosing in patients with CAH, with no increase in perioperative morbidity or mortality. Specifically, the studies highlight consistent outcomes in both weight reduction and the potential for decreased glucocorticoid requirements, suggesting a beneficial interplay between surgical intervention and hormonal balance.

This case study builds on these findings by presenting the first comprehensive report of an adolescent male with classic CAH undergoing a multimodal treatment approach that integrated both bariatric surgery and pharmacotherapy. By situating our findings within the broader medical context, we emphasize the need for further investigation into the efficacy of combined treatment modalities in pediatric populations with complex medical conditions like CAH. This growing body of evidence underscores the potential for metabolic and bariatric surgery to serve as a

critical component in the comprehensive management of severe obesity and related complications in this unique demographic.

The patient's unabating weight trajectory across various treatment modalities for obesity underscores the importance of a tailored approach to obesity management in youth. Initial lifestyle modifications, including increased physical activity and dietary changes, were ineffective in halting the progression of obesity. This aligns with findings in the literature that demonstrate lifestyle interventions alone often yield limited results for severe obesity, particularly in the context of endocrine disorders [7]. The introduction of pharmacotherapy, starting with topiramate and later semaglutide, marked critical advancements in the patient's treatment. Semaglutide, a GLP-1 receptor agonist, has garnered attention for its effectiveness in promoting weight loss and improving metabolic outcomes in pediatric populations [17, 18]. Studies have indicated that GLP-1 agonists can enhance satiety, reduce appetite, and lead to meaningful weight reduction, which was evident in this case as the patient experienced a significant decrease in BMI following the initiation of semaglutide [18–20].

Despite advancements with pharmacotherapy, the decision to pursue metabolic and bariatric surgery was made in accordance with the American Academy of Pediatrics' guidelines, recognizing that the patient met the criteria for surgical intervention [7, 8]. Laparoscopic sleeve gastrectomy has emerged as a valuable option for youth with severe obesity, particularly when other interventions have been insufficient [8]. Therefore, this case illustrates the potential benefits of metabolic and bariatric surgery in achieving substantial and sustained weight loss; it additionally holds potential for improvements in associated cardiometabolic comorbidities in patients with CAH.

Limitations

While this case report highlights a successful multimodal approach to treating severe obesity in a young adult with CAH, several limitations should be acknowledged. First, as a single case study, the findings may not be generalizable to all patients with CAH or obesity, as individual responses to treatment can vary widely based on genetic, environmental, and psychosocial factors. Second, the relatively short follow-up period post-surgery limits our ability to assess the long-term sustainability of weight loss and the potential for recurrence of obesity or metabolic complications. The reliance on self-reported data regarding dietary intake and physical activity may introduce bias, as patients and families may overestimate adherence with recommended lifestyle changes. Lastly, while pharmacotherapy and surgical intervention were effective in this case, the absence of a control group or comparative data limits our ability to draw definitive conclusions about the efficacy of each individual component of the treatment strategy.

Conclusion

This case report underscores the importance of a comprehensive, multimodal approach to managing severe obesity in a young adult with classic CAH. The integration of pharmacotherapy, specifically GLP-1 receptor agonists, and laparoscopic sleeve gastrectomy holds the potential for significant and sustained weight loss, along with improvements in metabolic health in patients with CAH and severe obesity. As pediatric obesity rates continue to rise, particularly in individuals with endocrine disorders, tailored treatment strategies that combine lifestyle modifications, obesity medication, and surgical options will be essential. Future research is needed to explore long-term outcomes of integrated approaches to severe obesity and their applicability across diverse pediatric populations, to contribute to more effective management strategies for severe obesity in youth with endocrine disorders such as CAH.

Abbreviations

MBS	Metabolic and bariatric surgery
LSG	Laparoscopic sleeve gastrectomy
BMI	Body mass index
zBMI	Body mass index Z-score
$\%BMI_{p95}$	Percent of the 95th percentile
CAH	Congenital adrenal hyperplasia
AOM	Antiobesity medication

Acknowledgements

The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Author contributions

Conceptualization: APV, LK, MJM, SA, AGK, MW, CEM, LKF, MSK, and KS; methodology: APV, LK, MJM, SA, AGK, MW, CEM, LKF, and KS; formal analysis: APV; data curation: LK; writing—original draft preparation: LK and AVP; writing—review and editing: APV, LK, MJM, SA, AGK, MW, CEM, LKF, MSK, and KS; and supervision: APV and KS. All authors have read and agreed to the published version of the manuscript.

Funding

This work was supported by grants: (1) K23DK134801 NIH NIDDK, (2) Sacchi Foundation Research Scientist, (3) Supported by American Diabetes Association grant #11-22-ICTSN-32, and (4) The Southern California Center for Latino Health Pilot Award 2022. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Availability of data and materials

The datasets from this study will be available from the corresponding author on written request.

Declarations

Ethics approval and consent to participate

Written informed consent was obtained from all participants and a parent or guardian involved in the study. The study was conducted according to the

guidelines of the Declaration of Helsinki and approved by the Institutional Review Board (or Ethics Committee) of Children's Hospital Los Angeles (CHLA-000243, date of approval—12/20/2023).

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors have no financial relationships or conflict of interest relevant to this article to disclose.

Author details
[1]Department of Pediatrics, Center for Endocrinology, Diabetes and Metabolism, Children's Hospital Los Angeles and Keck School of Medicine of USC, 4650 Sunset Boulevard, Mailstop #61, Los Angeles, CA 90027, USA. [2]Department of Surgery, Center for Endocrinology, Diabetes and Metabolism, Children's Hospital Los Angeles and Keck School of Medicine of USC, Los Angeles, CA, USA. [3]Division of Upper Gastrointestinal and General Surgery, Department of Surgery, Keck Medical Center of University of Southern California, Los Angeles, CA, USA. [4]Department of Psychology, Children's Hospital Los Angeles, Los Angeles, CA, USA.

Received: 6 November 2024 Accepted: 25 December 2024
Published online: 25 January 2025

References:

1. Fraga NR, Minaeian N, Kim MS. Congenital adrenal hyperplasia. Pediatr Rev. 2024;45(2):74–84. https://doi.org/10.1542/PIR.2022-005617.
2. Balagamage C, Arshad A, Elhassan YS, *et al*. Management aspects of congenital adrenal hyperplasia during adolescence and transition to adult care. Clin Endocrinol (Oxf). 2024. https://doi.org/10.1111/CEN.14992.
3. Arlt W. CAHtalyzing change in congenital adrenal hyperplasia. N Engl J Med. 2024;391(6):559–61. https://doi.org/10.1056/NEJME2407740.
4. Kim MS, Fraga NR, Minaeian N, Geffner ME. Components of metabolic syndrome in youth with classical congenital adrenal hyperplasia. Front Endocrinol (Lausanne). 2022. https://doi.org/10.3389/FENDO.2022.848274.
5. Bhullar G, Tanawattanacharoen VK, Yeh MY, *et al*. Early adiposity rebound predicts obesity and adiposity in youth with congenital adrenal hyperplasia. Hormone Res Paediatrics. 2021. https://doi.org/10.1159/000514130.
6. Speiser PW, Arlt W, Auchus RJ, *et al*. Congenital adrenal hyperplasia due to steroid 21-hydroxylase deficiency: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2018;103(11):4043–88. https://doi.org/10.1210/JC.2018-01865.
7. Hampl SE, Hassink SG, Skinner AC, *et al*. Clinical practice guideline for the evaluation and treatment of children and adolescents with obesity. Pediatrics. 2023. https://doi.org/10.1542/PEDS.2022-060640.
8. Pratt JSA, Browne A, Browne NT, *et al*. ASMBS pediatric metabolic and bariatric surgery guidelines, 2018. Surg Obes Relat Dis. 2018;14(7):882–901. https://doi.org/10.1016/J.SOARD.2018.03.019.
9. Kelly AS, Armstrong SC, Michalsky MP, Fox CK. Obesity in adolescents: a review. JAMA. 2024. https://doi.org/10.1001/JAMA.2024.11809.
10. Seagroves A, Ross HM, Vidmar AP, *et al*. Weight loss during topiramate treatment in a severely obese adolescent with congenital adrenal hyperplasia and migraine. J Clin Res Pediatr Endocrinol. 2023;15(1):81–5. https://doi.org/10.4274/JCRPE.GALENOS.2021.2020.0310.
11. CDC, Nceh, DLS, NSMBB. 2022 ANNUAL SUMMARY REPORT Newborn Screening Quality Assurance Program 2 Newborn Screening Quality Assurance Program 2022 Annual Summary Report, Volume 40. Published online 2022.
12. Kaplan JA, Schecter SC, Rogers SJ, Lin MYC, Posselt AM, Carter JT. Expanded indications for bariatric surgery: should patients on chronic steroids be offered bariatric procedures? Surg Obes Relat Dis. 2017;13(1):35–40. https://doi.org/10.1016/J.SOARD.2015.10.086.
13. Mazzei M, Zhao H, Edwards MA. Perioperative outcomes of bariatric surgery in the setting of chronic steroid use: an MBSAQIP database analysis. Surg Obes Relat Dis. 2019;15(6):926–34. https://doi.org/10.1016/J.SOARD.2019.02.007.
14. Bornstein SR, Allolio B, Arlt W, *et al*. Diagnosis and treatment of primary adrenal insufficiency: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2016;101(2):364–89. https://doi.org/10.1210/JC.2015-1710.
15. Zatsepina EA, Samoilov VS, Volynkina AP, Stepanenko AV, Novichikhina EE. Experience of successful laparoscopic sleeve resection of the stomach and treatment of morbid obesity in a patient with a classic form of congenital adrenal dysfunction. Probl Endokrinol (Mosk). 2023;69(3):83–9. https://doi.org/10.14341/PROBL13206.
16. Mallappa A, Nella AA, Kumar P, *et al*. Alterations in hydrocortisone pharmacokinetics in a patient with congenital adrenal hyperplasia following bariatric surgery. J Endocr Soc. 2017;1(7):994. https://doi.org/10.1210/JS.2017-00215.
17. Ryan DH. Next generation antiobesity medications: setmelanotide, semaglutide, tirzepatide and bimagrumab: what do they mean for clinical practice? J Obes Metab Syndr. 2021;30(3):196–208. https://doi.org/10.7570/JOMES21033.
18. Weghuber D, Barrett T, Barrientos-Pérez M, *et al*. Once-weekly semaglutide in adolescents with obesity. N Engl J Med. 2022. https://doi.org/10.1056/NEJMOA2208601.
19. Bonnet JB, Tournayre S, Anitcheou J, *et al*. Semaglutide 2.4 mg/wk for weight loss in patients with severe obesity and with or without a history of bariatric surgery. Obesity (Silver Spring). 2024;32(1):50–8. https://doi.org/10.1002/OBY.23922.
20. Jamal M, Alhashemi M, Dsouza C, *et al*. Semaglutide and tirzepatide for the management of weight recurrence after sleeve gastrectomy: a retrospective cohort study. Obes Surg. 2024;34(4):1324–32. https://doi.org/10.1007/s11695-024-07137-0.

Publisher's Note