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Weight Restoration in Atypical Anorexia Nervosa: a Clinical Conundrum

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Abstract

The determination of the treatment goal weight for adolescents with a restrictive eating disorder (ED) and a prior history of obesity, such as in atypical anorexia nervosa (AAN), represents an ongoing challenge for clinicians. This determination is particularly complex given competing interests of physical recovery from significant weight loss, mental recovery from ED psychopathology, and the medical comorbidities associated with obesity. We report the case of a 15-year old female with a history of obesity and irregular periods who presented with significant weight loss and absence of menses for three months. She was hospitalized for severe malnutrition and bradycardia, initiated family based treatment, and regained periods. However, her vital sign instability and eating disorder cognitions did not resolve until she reached a weight at which point her periods again ceased due to polycystic ovary syndrome. This case highlights the need for regular monitoring during weight recovery and the clinical challenge of determining the treatment goal weight in adolescents with EDs and prior obesity.

Keywords

Obesity; overweight; eating disorders; atypical anorexia nervosa; polycystic ovary syndrome; amenorrhea

Introduction

Atypical anorexia nervosa (AAN) is a new diagnosis in the Diagnostic and Statistical Manual, 5th Edition (DSM-5), to describe patients who meet all criteria for anorexia nervosa (AN), except that their weight is within or above the normal range despite “significant weight loss” (American Psychiatric Association, 2013). This burgeoning patient population reflects the high prevalence of youth with obesity and disordered eating; nearly one third of adolescents considered overweight or obese are estimated to engage in disordered eating behaviors (Neumark-Sztainer et al., 2007). Indeed, it is estimated that 70% of AAN patients

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were previously overweight or obese (Sawyer, Whitelaw, Le Grange, Yeo, & Hughes, 2016) and that nearly half of adolescents admitted to a large inpatient eating disorder (ED) unit are not underweight, representing a fivefold increase over a six-year period (Whitelaw, Gilbertson, Lee, & Sawyer, 2014).

A clinical dilemma in the treatment of AAN involves the determination of the treatment goal weight. If an adolescent was previously obese prior to the development of an eating disorder, should their treatment goal be to "restore" their weight back to their previous weight classification of obesity? On the one hand, the malnutrition and bradycardia in AAN can be as severe as it is in anorexia nervosa (Sawyer et al., 2016), and weight gain is one approach to restore medical stability (Hughes, Le Grange, Court, & Sawyer, 2017; Peebles et al., 2017). On the other hand, obesity is associated with significant medical comorbidities including polycystic ovary syndrome (PCOS) (Ybarra et al., 2018) and cardiometabolic risk such as dyslipidemia, hypertension, and diabetes (Bibbins-Domingo, Coxson, Pletcher, Lightwood, & Goldman, 2007). Here we present the case of a 15-year-old female with AAN who developed PCOS as she was reaching weight restoration.

Case Report

The patient was a 15 year old with a history of obesity who presented to a specialized ED unit in the context of a 27.0 kg weight loss in one year (body mass index [BMI] from 97th to 25th percentile for age and sex (Centers for Disease Control, C D C, 2000), weight from 71.4 kg to 44.4 kg). She reported being teased by peers about her "heavy" weight so she decided to lose weight. Notable eating disorder behaviors included eating smaller portions and refusing sugary foods. She also reported over-exercising, going to gym five days a week in addition to her regular lacrosse team practice.

She was admitted to the hospital for severe malnutrition, given that she had lost 37% of her body mass in one year (Golden et al., 2015). She was bradycardic with a resting heart rate of 54 beats per minute and overnight nadir of 40 beats per minute (Golden et al., 2015). She had menarche at age 12, and although her periods were not regular prior to the weight loss, she previously had periods every one to two months. Immediately prior to presentation, she had not had a menstrual period for three consecutive months. Workup at presentation included normal total testosterone (20 ng/dL, normal <41 ng/dL), free testosterone (1.7 pg/mL, normal 0.5–3.9 pg/mL), thyroid stimulating hormone, free T4, dehydroepiandrosterone, 17-hydroxyprogesterone, prolactin, follicle stimulating hormone (6.86 mIU/mL, normal 0.64–10.98 mIU/mL), luteinizing hormone (11 mIU/mL, normal 0.4–11.7 mIU/mL), and estradiol (29 pg/mL, normal ≤283 pg/mL), with a negative pregnancy test. A 2000 calorie per day (kcal/d) diet increasing by 200 kcal/d was prescribed, eventually reaching a target of 2800 kcal/d based on estimated energy requirement equations (Institute of Medicine, 2006). After six days in hospital, she had gained 1.6 kg and reached the 32nd BMI percentile. She was discharged to outpatient family based treatment and weekly medical follow up. Within one month, she had resumption of menses at the 42nd BMI percentile. However, she continued to experience significant ED thoughts and cognitions and mild medical complications of malnutrition (Golden et al., 2015; Katzman, 2005), including bradycardia (heart rates of 55 beats per minute) and dizziness with

orthostatic heart rate changes (heart rate increase of 27 beats per minute from lying to standing). While her ED thoughts and cognitions persisted, she denied behaviors such as restricting food or fluid intake (urine specific gravity ranged from 1.010–1.020), skipping meals, fasting, binging, purging, or avoiding fats at each visit following her hospitalization. With the supervision of her mother, her meal plan was liberalized to three meals and three snacks regularly each day without the need to calculate exact caloric intake. She also abstained from exercise.

Although her caloric goals were liberalized, she continued to gain approximately 1.33 kg/month for nine months. She was allowed to resume participation on the lacrosse team at the 59th BMI percentile given that her mild bradycardia and orthostasis were not clinically significant concerns, but did not exercise outside of her team's practice schedule. She continued regular monitoring with her medical and mental health providers during her transition to exercise. Her bradycardia persisted until she reached the 73rd BMI percentile, but at this weight she still had orthostatic heart rate changes with dizziness. Her autonomic instability first resolved when she reached the 77th BMI percentile. However, at that point, she had developed notable hirsutism including severe acne and her periods again ceased. Repeat labs revealed an elevated total testosterone (52 ng/dL) and free testosterone (5.8 pg/mL). Thyroid stimulating hormone, free T4, follicle stimulating hormone, luteinizing hormone, and estradiol remained normal, with a repeat negative pregnancy test. Based on these findings, a diagnosis of polycystic ovary syndrome was made. She was started on oral contraceptive pills (norgestimate-ethinyl estradiol 0.25–35 mg-mcg tablet). Since her vital signs stabilized and ED cognitions resolved she was considered "weight restored."

Discussion

This case highlights a 15-year-old female with a history of obesity who presented with significant weight loss and amenorrhea in the context of AAN. Within a month of treatment, her menses resumed; however, nine months into recovery, she developed PCOS at a BMI within the normal range (77% for age and sex). This case underscores (1) the clinical conundrum of identifying a treatment goal weight in AAN, (2) the importance of monitoring for medical complications including menstrual irregularities throughout the treatment course, and (3) the need for further research to inform guidelines for the treatment of AAN.

Although we prescribed initial weight gain in the context of medical instability in AAN, this approach lacks definitive evidence from studies such as randomized controlled trials. In addition, as the patient's vital signs normalized, her caloric intake was liberalized to general guidance of three meals and three snacks per day, but her weight continued to increase. An alternate approach could have been weight maintenance to see if vital sign stabilization would ensue over time. However, given that her natural course on a liberalized diet was to gain weight, it is possible that maintaining her weight may have felt restrictive for the patient. The lack of a clear evidence or guidelines for weight management in this case highlights our call for more research and guidelines in this topic.

The patients' vital sign instability persisted until she reached the 77th percentile of BMI. While her orthostatic heart rate changes were thought to be attributed to her malnutrition and

suppressed weight, other potential factors in the differential diagnosis could include dehydration from surreptitious behaviors (although her urine specific gravity ranged from 1.010–1.020 in clinic), longer time needed for normalization of vital signs, or postural orthostatic tachycardia syndrome (POTS). Further research is needed to determine the time course of normalization of vital signs and ED thoughts and cognitions in AAN.

Clinicians caring for adolescents with AAN face a treatment dilemma, balancing ED psychopathology and potential medical instability with medical comorbidities that may accompany weight gain. This is a particular challenge given the lack of a standard evidence-based method to calculate treatment goal weight even in AN (Lebow, Sim, & Accurso, 2017). The degree of malnutrition and bradycardia in AAN can be just as severe as in AN (Sawyer et al., 2016), which indicates the importance of refeeding to restore weight. On the other hand, this case may suggest that there is an upper limit at which point the medical complications may outweigh the benefits of weight restoration. The question of whether there is a rate of weight gain that prevents complications also warrants examination. Other important comorbidities of adolescent obesity include risk factors for future cardiovascular disease, such as dyslipidemia, hypertension, and diabetes (Bibbins-Domingo et al., 2007).

Resumption of menses has been identified as an important treatment goal for restrictive EDs as a marker of improved bone mineral density (Golden et al., 2015). One study in previously overweight participants (who would meet DSM-5 criteria for AAN) found that resumption of menses occurred on average just above the 50th percentile of BMI; however there was a large standard deviation (Seetharaman et al., 2017). These findings indicate that patients with prior obesity do indeed need to gain weight for recovery and, further, that the degree of variation in recovery weight among individuals is large. The development of PCOS in this case occurred at the 77th BMI percentile, which is within the normal range (Centers for Disease Control, C D C, 2000). It is not known whether the patient had premorbid PCOS with her prior obesity and oligomenorrhea as she had not been previously screened. However, her total testosterone was within normal limits upon presentation to ED care (at the 25th BMI percentile) whereas it became elevated at a higher BMI percentile.

In summary, we found that menstrual irregularities occurred at both ends of a narrow weight spectrum within a short recovery period in a 15-year old with AAN. This case underscores the limitations of relying on weight alone as a marker of AAN recovery. Nonetheless, weight is a cardinal metric that should be assessed along with ED cognitions and behaviors, exercise, and menstrual status. The identification of markers of recovery in patients with AAN is clinically pressing as these patients represent a rapidly growing proportion of adolescents with EDs (Sawyer et al., 2016; Whitelaw et al., 2014).

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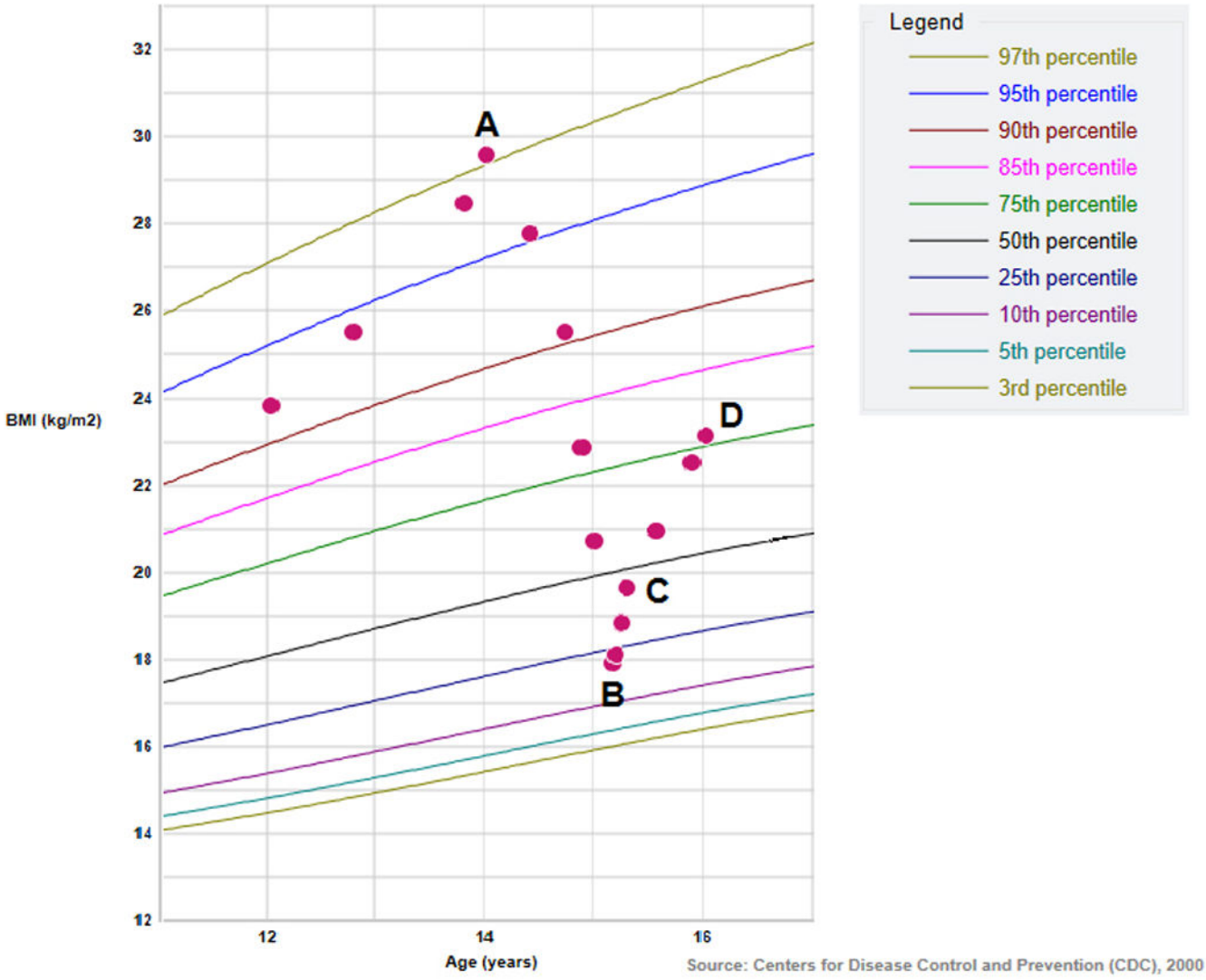

Figure 1.

An illustration of the patient's body mass index (BMI) trajectory. The patient had a BMI at the 97th percentile of BMI for age and sex at age 14 (A), at which point she lost 27.0 kg over a year, falling to the 25th BMI percentile and developing amenorrhea (B). After a hospitalization for severe malnutrition and starting family based treatment, the patient had resumption of menses at the 42nd BMI percentile (C) and developed PCOS at the 77th BMI percentile (D).