

Obesity: Its Epidemiology, Comorbidities, and Management

Jana Jarolimova, BS; James Tagoni, DMD, MD; and Theodore A. Stern, MD

LESSONS LEARNED AT THE INTERFACE OF MEDICINE AND PSYCHIATRY

The Psychiatric Consultation Service at Massachusetts General Hospital sees medical and surgical inpatients with comorbid psychiatric symptoms and conditions. Such consultations require the integration of medical and psychiatric knowledge. During their twice-weekly rounds, Dr Stern and other members of the Consultation Service discuss the diagnosis and management of conditions confronted. These discussions have given rise to rounds reports that will prove useful for clinicians practicing at the interface of medicine and psychiatry.

Ms Jarolimova is a senior medical student at Harvard Medical School, Boston, Massachusetts.

Dr Tagoni is a resident in the Department of Oral and Maxillofacial Surgery, Massachusetts General Hospital, Boston. **Dr Stern** is chief of the Psychiatric Consultation Service at Massachusetts General Hospital, Boston, and a professor of psychiatry at Harvard Medical School, Boston, Massachusetts.

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Corresponding author: Jana Jarolimova, Massachusetts General Hospital, 55 Fruit St, Warren Building, Room 605, Boston, MA 02114 (Jana_Jarolimova@hms.harvard.edu).

Have you ever wondered why people allow themselves to become obese? Have you ever been frustrated by not knowing how to help your obese patients control their appetite and weight? Have you assumed that psychiatric conditions can drive weight gain and its downstream complications? If you have, then the following clinical vignette and discussion should prove to be a useful stimulus for learning more about obesity, its assessment, and its treatment.

CASE VIGNETTE

Ms A, a 49-year-old woman with a history of severe obesity, bipolar disorder, anxiety, and multiple medical problems, was admitted to the hospital with severe anasarca and a chronic pleural effusion.

At age 40 years, she underwent gastric bypass surgery. In the 2 years that followed, Ms A lost almost 200 lb. However, over the next 6 years, she regained most of that weight, and she reached her maximum weight of 375 lb at age 48. At that time, Ms A developed progressive, bilateral, lower-extremity edema that gradually became diffuse and most prominent in the dependent portions of her body. A workup revealed severe hypoalbuminemia as well as vitamin and nutrient deficiencies. Soon afterward, Ms A was admitted to a long-term rehabilitation facility for management of severe deconditioning, muscle wasting, chronic hypoalbuminemia, widespread anasarca, a nonhealing right leg wound, and a persistent right pleural effusion. Over the following months, staff at the rehabilitation facility became concerned about Ms A's capacity to make decisions about her eating. Despite strict nutritional recommendations, Ms A developed an intense aversion to food, citing lack of appetite and unhappiness about her weight and body habitus, while often blaming other facility residents for being too "disgusting" for her to eat around them. Ms A's clinical condition continued to deteriorate, and she required hospital admission for more active management.

WHAT IS OBESITY AND HOW IS IT CATEGORIZED?

The most basic definition of obesity refers to having excess fat. Measuring body fat can be cumbersome (ie, measuring the percentage of body fat). Two common methods to assess the degree of obesity are measuring waist circumference, a surrogate for abdominal fat, and calculating the body mass index (BMI). Large waist circumference (>40 inches for men and >35 inches for women) has been shown to be an independent predictor of risk factors for obesity and increased morbidity.¹ Waist circumference has been shown to be the anthropometric index that best predicts the distribution of adipose tissue in fat compartments such as the abdominal region.² Certain body shapes among obese individuals have a strong relationship with an increased incidence of type 2 diabetes mellitus and metabolic syndrome. Apple-shaped individuals have increased visceral fat and a higher incidence of these diseases.³ Pear-shaped individuals have increased subcutaneous fat and a lower risk of these diseases.³ The BMI quantifies the relationship between height and weight⁴ and is calculated as weight (kg)/height squared (m²). Obesity is defined as a BMI >30. This definition is further subdivided into obesity classes 1 (30–34.9), 2 (35–39.9), and 3 (>40). These classifications allow for identification of individuals at increased risk of obesity-associated complications and for

- Patients with psychiatric disorders are especially prone to the development of obesity and metabolic derangements.
- Physicians caring for patients with psychiatric disorders should conduct careful assessments of obesity risk factors and metabolic derangements.
- Numerous treatment modalities have shown success in the treatment of obesity in patients with psychiatric disorders as well as those in the general population.
- Patients with preexisting mental illness or disordered eating are especially vulnerable to complications following bariatric surgery.

specific disorders.⁴ Overweight in children is defined as a BMI >the 85th percentile,⁵ while obesity in children is defined as a BMI >the 95th percentile.²

HOW COMMON IS OBESITY?

The prevalence of obesity in the United States continues to increase. In the United States, obesity has increased from 20.9%⁶ to 33.8%⁷ over the last decade. The prevalence of obesity among immigrants to the United States varies among subgroups, but it is more common among Latin/Caribbean immigrants who are living above the poverty level.⁸ Those who have lived in the United States longer also have higher rates of obesity. Interestingly, a higher prevalence of obesity among immigrants living in the western United States has been noted, contrary to trends seen in the native population.⁸ The prevalence of obesity in Hispanic women and men is higher than in other ethnic groups.⁴ Black women tend to have higher rates of obesity than white women, while white men tend to be obese more often than black men.⁴ Unlike the adult population, which has seen an increase of obesity in the last decade, the level of obesity among children has stabilized over the last decade. During 2007–2008, 16.9% of children (2–19 years old) were obese (ie, > the 95th percentile).⁹

The trend of increasing obesity is also of concern because of its economic impact, increased comorbidities, and associated decreased lifespan. Most Americans tend to have an increase in weight of 0.2–0.8 kg (0.44–1.76 lb) per year.^{10,11} This weight gain has been largely attributed to the holiday period between Thanksgiving and New Year's Day when American adults tend to gain 0.48 kg (1.06 lb).¹² When applied to the population as a whole, these trends become alarming, as some studies estimate that, by 2030, nearly 51.1% will be obese.¹³

WHAT BIOLOGICAL AND PSYCHOLOGICAL FACTORS CONTRIBUTE TO OBESITY?

The biology of obesity is multifactorial; it includes genetic and endocrine defects. To date, only a handful of well-characterized single-gene disorders have been associated with obesity. Contributing factors include the melanocortin peptides that play a role in energy homeostasis¹⁴; the “ob” (obese) gene, which appears to regulate the body fat reserve¹⁵; and gastric inhibitory peptide, which aids in the absorption

of ingested fat.¹⁶ However, these disorders account for only a small proportion of obese individuals. In addition, there appears to be a genetic influence and a degree of heritability of BMI and obesity.¹⁷ The current understanding of obesity does not completely align itself with the notion that weight gain versus weight loss is simply a balance between energy intake and energy expenditure. A “set-point” of body weight is believed to play a role in some individuals' difficulty in reducing their body weight. A study of 18 obese subjects and 23 nonobese subjects showed that a weight loss of 10% to 20% correlated with a decreased resting energy expenditure; the same was true of a 10% weight increase for increased resting energy expenditure.¹⁴

There is no clear psychological or psychiatric cause for obesity, but there are psychiatric conditions that contribute to the development of obesity. Some of the more clearly elucidated of these are seasonal affective disorder and atypical depression.¹⁸ Among obese patients undergoing bariatric surgery, nearly 50% have a history of mood or anxiety disorder.^{19,20} Thus, there is evidence that psychosocial factors are implicated in the development of obesity.

WHAT COMPRISES AN EVALUATION OF OBESITY?

It is important for primary care physicians to evaluate obesity, as it is associated with higher mortality and comorbid conditions (eg, diabetes mellitus, hypertension). Once a diagnosis of obesity has been made, an assessment of risk factors and a treatment plan can be formulated; however, evaluation is not as common (or as easy) as it may seem. A retrospective study from the Mayo Clinic, Rochester, Minnesota, identified 9,827 patients who underwent general medical examinations; of these, 2,543 were found to be obese, but only 19.9% of these obese individuals received a diagnosis of obesity and 22.6% had an obesity management plan formulated.²¹ A correlation between childhood/adolescent obesity has been found in most studies, although the strength of this correlation varies. Guo and Chumlea²² conducted a retrospective cohort study of 555 women and men and found that being overweight/obese at younger ages (which included 18, 13, and <13 years old) was predictive of one's BMI at 35 years of age. The strongest predictor in this study was the BMI at age 18 years (odds ratio of 14.9 and 27.7 for men and women, respectively).²¹ In contrast, a study from an Australian cohort by Magarey and associates²³ of 155 boys and girls showed that one-third of overweight and obese adults (20 years old) were overweight or obese in childhood at the ages of 2, 8, and 11 years old. They also noted that the relative risk increased with increasing age, due to the fact that the interval to early adulthood decreases with increasing age; however, the relative risk for being obese was greater than 1 at every age.²³

A large impact of obesity is derived from the psychosocial burden on patients. Chronic obesity that begins in childhood as opposed to onset in adolescence is associated with oppositional defiant disorder and depressive disorders.²⁴ A cohort study of obese women noted that they were more likely to have completed less schooling, were less likely to

be married, and were more likely to have lower household income and higher poverty rates compared to their nonobese counterparts.²⁵ Eating disorders are also common (nearly 20%–40% of obese patients in at least 1 study²⁶) among obese patients. Mood disorders constitute another class of comorbid conditions among obese individuals.²⁷ Obese patients additionally tend to have higher rates of depression,²⁸ a condition that may affect treatment adherence; hence, a thorough psychiatric evaluation may facilitate treatment.

WHAT COMPLICATIONS CAN ARISE AS A CONSEQUENCE OF OBESITY?

Obesity has long been associated with increased morbidity and mortality. Among the well-known complications of severe obesity are increased risks of developing diabetes, hypertension, and hyperlipidemia. The development of type 2 diabetes mellitus has been associated with obesity in all ethnic groups and is positively correlated with BMI.²⁹ Similarly, excess body weight is thought to account for up to one-fourth of cases of hypertension in adults.³⁰ Obese individuals, especially those with central fat distribution, are at increased risk for several abnormalities in lipid metabolism, namely, high serum cholesterol, low-density lipoproteins, and very low-density lipoproteins and triglycerides, as well as a mild reduction in serum high-density lipoproteins.^{31,32} Heart disease and ischemic stroke are other significant and well-evidenced complications of morbid obesity.^{33,34} Obesity also increases mortality from a number of cancers, most significantly of the esophagus, colon, rectum, liver, gallbladder, pancreas, and kidney, as well as non-Hodgkin's lymphoma and multiple myeloma.³⁵ Severe obesity has been associated with an increased rate of death from all causes³⁶ and decreased life expectancy³⁷ regardless of age, smoking, educational achievement, geographic region, and physical activity levels.

WHEN (AND HOW) CAN PREVENTION OR TREATMENT OF OBESITY BEGIN?

Prevention of obesity should begin in childhood and adolescence, as research in women has shown that being overweight in adolescence is independently correlated with an increased risk of premature death in adulthood.³⁸ However, for those adults who present to their physicians with preexisting obesity, primary care providers play a key role in screening and treatment. The US Preventive Services Task Force recommends that clinicians screen all adult patients for obesity (calculating the BMI) and offer intensive counseling and behavioral interventions to promote sustained weight loss for obese adults.^{39,40} Guidelines from the American College of Physicians recommend that clinicians counsel all obese patients on lifestyle and behavioral modifications and work with each patient to determine individual goals for weight loss and improvements in other parameters (such as blood pressure and fasting blood glucose).⁴¹ Unfortunately, brief counseling interventions in the clinical setting have not reliably led to significant reductions in weight over the long-term. Instead, modest success has been seen with

more intensive interventions, such as nutrition education, individualized goal-setting, behavioral approaches, walking groups, and other social activities.⁴² Physicians should provide information to all obese patients on their increased health risks and should refer patients to intensive, multidisciplinary programs for weight loss and physical activity (if they have access).

WHAT MODALITIES EFFECTIVELY TREAT OBESITY?

Management of overweight and obesity starts with a combination of diet, exercise, and behavioral modification. Behavioral treatments for obesity have aimed at either decreasing energy intake or increasing energy expenditure; those that focus on decreasing energy intake generally have a greater potential for achievement of weight loss. A plethora of diets exists, and the selection of a diet should start with the patient's preferences. A realistic expectation for dietary therapy is a loss of at least 5% of body weight after 6 months; if this is maintained and accompanied by improvement in associated risk factors, then dietary therapy can be considered successful. Unfortunately, over half of all patients reach a plateau before losing 10% of their initial (prediet) body weight. Exercise, similarly, should be tailored to the patient's preferences; often, the incorporation of short periods of activity into one's daily schedule can be as effective as achieving weight loss with structured exercise (eg, aerobics classes).⁴³ Structured behavioral interventions in primary care settings have produced significant weight loss compared with no intervention at all.⁴²

The American College of Physicians recommends that pharmacologic therapy be offered to obese patients who have failed to achieve their weight loss goals through diet and exercise alone. Pharmacologic therapy can be considered for patients with a BMI > 30 or a BMI of 27–30 with comorbid conditions⁴⁴; the choice of agent depends on the specific side effect profile and the patient's tolerance of those side effects. Prior to July 2012, orlistat was the primary pharmacologic agent used for treatment of obesity; however, the US Food and Drug Administration has approved 2 new agents for the treatment of obese patients: a controlled-release preparation of phentermine and topiramate and lorcaserin. When considering pharmacologic treatment, it is critical to involve the patient in a discussion of drug side effects, the lack of long-term safety data, and the temporary nature of weight loss achieved with medications.⁴⁴

Finally, bariatric surgery is becoming an increasingly common therapy for obesity that has failed to respond to other treatments. Indications for bariatric surgery stipulate that the patient must be well-informed and motivated, have a BMI > 40 or between 35 and 40 with serious comorbidities (such as diabetes, sleep apnea, or degenerative joint disease), have an acceptable surgical risk, and have failed previous trials at nonsurgical weight loss.⁴¹ Bariatric surgery encompasses a range of procedures, from purely restrictive procedures (such as gastric lap banding) to combined restrictive and malabsorptive procedures (such as the Roux-en-Y gastric bypass). Although bariatric surgery carries inherent risks,

the safety of the procedures has improved significantly since the early days of these operations.⁴⁵ The majority of patients undergoing bariatric surgery for obesity experience improvement or complete resolution of diabetes, hypertension, hyperlipidemia, and obstructive sleep apnea⁴⁶ in addition to significant and sustained weight loss.

WHAT ARE THE LONG-TERM WEIGHT LOSS RESULTS FOLLOWING BARIATRIC SURGERY?

Bariatric surgery results in significant and relatively sustained weight loss. In one meta-analysis of 10,172 patients,⁴⁶ the mean excess weight loss for all surgery types was 61.2% (95% CI, 58.1%–64.6%). In most cases, weight loss outcomes did not differ significantly at 2 years or less or at more than 2 years after the procedure. Thus, the majority of the excess weight was lost during the first 2 years following surgery, and a significant percentage of this weight was not regained.

In a prospective Swedish study of post-bariatric surgery patients,⁴⁷ weight change was maximal 1 year after the procedure. After 2 years, total weight had decreased by a mean of 23.4% in the surgical group (compared to 0.1% in a conventionally treated control group). After 10 years, however, mean weight loss in the bariatric surgery group was 16.1% below inclusion weight, indicating some measure of weight was regained despite persistently significant weight loss.⁴⁷ However, at 10 years after the procedure, 8.8% of gastric bypass patients, 13.8% of vertical-banded gastroplasty patients, and 25.0% of gastric-banding patients had a loss of less than 5% of their inclusion weight, indicating either initial failure of weight loss or relapse. Bariatric surgery is thus an effective method of achieving clinically significant weight loss for the vast majority of patients.⁴⁷

WHAT COMPLICATIONS CAN DEVELOP FROM THE TREATMENT OF OBESITY?

While the health risks of obesity justify targeted treatment, available therapies are not without risks. Diet and exercise therapy are associated with the lowest risk, while pharmacotherapy and bariatric surgery carry significantly higher risks of complications and adverse events. Orlistat, until recently the most commonly used adjunctive drug therapy, has predominantly gastrointestinal side effects (including cramps, flatus, fecal incontinence, oily spotting, and flatus with discharge)^{48,49} that typically occur early following initiation of treatment and subside with enhanced adherence to the recommended dietary intake of no more than 30% fat.⁵⁰ Decreased absorption of fat-soluble vitamins A, D, E, and β -carotene has been reported, and benefit may result from supplementation with these vitamins during therapy.⁴⁸ Safety profiles of the newly approved pharmacologic agents are still incompletely understood, although some cardiovascular effects such as an increase in heart rate of the controlled-release preparation of phentermine and topiramate have already been noted.⁵¹

Bariatric surgery is associated with a number of perioperative and postoperative complications, some of

which, such as band erosions or marginal ulcers, are specific to bariatric surgery, while others, such as deep vein thrombosis or venous thromboembolism, are common to many types of surgery.⁴⁵ While these complications occur more frequently in extremely obese patients,⁴⁵ the overall rate for experienced surgeons is low.^{45,46} Metabolic derangements and electrolyte abnormalities occur most frequently with the malabsorptive or combination procedures and are due to inadequate intake of nutrients as well as to decreased absorption by the anatomically altered gastrointestinal tract.⁵²

Additionally, following bariatric surgery, many patients who have experienced massive weight loss continue to suffer from perceived deformities due to excess skin. This excess skin is unsightly, can be painful, and can increase susceptibility to intertriginous infections. Consequently, the rate of surgical “body contouring” procedures has grown dramatically in recent years, with over 50,000 such procedures performed in 2004 alone.⁵³ Furthermore, many patients experience a profound alteration in their psychosocial function following the dramatic weight loss that usually results from bariatric surgery, and these changes may lead to exacerbations of preexisting psychological conditions or difficulties with coping that may be best addressed by an experienced psychologist or psychiatrist through a long-term support group.

DO PHYSICIANS AND LAYPEOPLE FEEL NEGATIVELY ABOUT OBESE INDIVIDUALS AND TREAT THEM DIFFERENTLY?

Unfortunately, research has repeatedly shown that obese individuals are subject to obesity stigmatization both in the public sphere and in the health care setting. The overwhelmingly negative views of obesity prevalent in the public arena and in the media lead to a negative self-perception by obese individuals and a shared negative perspective by health care practitioners.⁵⁴ Some of the common negative stereotypes associated with obesity include laziness, lack of willpower, and emotional and moral instability.⁵⁴ These perceptions may lead obese individuals to avoid contact with the health care profession through primary care and thus fail to receive adequate care and follow-up for obesity-related health conditions (such as diabetes).⁵⁴ A number of studies, mostly of women, have shown that those with a higher BMI are more likely to delay seeking routine care, including screenings such as pap smears or mammography.^{55,56} Another study found a positive correlation between patients' negative perceptions of their weight and weight-related reasons for a delay in or avoidance of seeking care.⁵⁷ Smaller qualitative studies in obese men have also found that stigma against obesity plays a role in their ability to make positive lifestyle changes.⁵⁸ In addition, studies have identified lower levels of physician respect⁵⁹ for obese patients as evidenced, for example, in one study showing a negative correlation between patients' BMIs and their physicians' perceived levels of their medication adherence.⁶⁰ Since obesity has repeatedly been associated with increased rates of depression, decreased quality of life,^{61,62} and significant comorbidities and adverse

health outcomes, it is important to address the stigma of obesity as a factor that negatively affects the willingness of obese patients to seek care.

ARE THERE PSYCHIATRIC PRECURSORS TO AND PSYCHIATRIC IMPACTS OF OBESITY?

Many studies have assessed the association between psychiatric illness and obesity and have evaluated various models of obesity treatment and behavioral change in patients with mental illness. A positive association between psychiatric disorders and the development of obesity has been demonstrated for multiple mental disorders, including mood disorders,^{63–65} personality disorders,⁶⁶ and schizophrenia.⁶⁷ These correlations vary from 1.2–1.5 for major depression and bipolar disorder to 2.8–3.5 for schizophrenia. Similarly, obesity has been identified as one of the common comorbid conditions associated with bipolar disorder.⁶⁸ The associations between obesity and psychiatric disorders are complex and most likely involve a combination of poor self-care/lifestyle, illness-related symptoms, lower income, metabolic side effects of various psychiatric medications, and possibly the subsequent development of mood disorders related to effects of the stigma associated with obesity. While the exact causal and temporal relationships between these 2 variables remain to be fully elucidated, the need for adequate obesity-related care in the primary care setting for patients with psychiatric disorders is clear.

While obviously not responsible for the full association between mental illness and obesity, psychotropic medications are a well-known contributing factor to obesity rates among psychiatric patients. Weight gain is a side effect of antipsychotic medications that affects 15% to 72% of patients being treated for schizophrenia, and antipsychotic medication has been associated with weight gain in patients with bipolar disorder as well.⁶⁷ Weight gain is known to be highest with clozapine and olanzapine compared with other antipsychotic agents. However, all antipsychotics have been associated with weight gain in treatment-naive patients; antidepressants and mood stabilizers have also been associated with weight gain.⁶⁷

WHICH INTERVENTIONS CAN TREAT OR PREVENT OBESITY IN PATIENTS WITH PSYCHIATRIC ILLNESS?

The majority of outpatient interventions aimed at reducing obesity among patients with psychiatric illness have been studied in patients with severe mental disorders, most commonly schizophrenia and psychotic disorders.^{69–71} Current rates of screening for metabolic risk factors by either psychiatrists or primary care physicians are low,⁷² and many experts have called for greater monitoring by mental health providers of weight-related parameters (waist circumference, BMI, metabolic parameters, change in BMI) for all psychiatric patients as well as screening for metabolic risk factors in patients taking antipsychotic medications.^{67–71} This strategy should also be offered by primary care physicians who care for such patients. Additionally, patients should be encouraged to monitor and chart their own weight.⁶⁸

As increasing numbers of mental health patients are treated in the community rather than in the inpatient setting, attention must be paid to the physical health of these patients in regular clinical encounters. Various interventions for preexisting obesity and psychiatric medication-induced weight gain in psychiatric patients have been studied. One meta-analysis focusing on patients with a variety of psychotic disorders showed that various nonpharmacologic lifestyle interventions (including diet, physical activity, counseling, and information sessions) were generally able to reduce weight by an average of -0.98 BMI points compared to control patients who received treatment as usual.⁷² While this decrease in the BMI does not meet the threshold of 5% body weight reduction required to classify these as “successful” interventions, such a change in body weight can still have a significant positive impact on the metabolic derangements more commonly found in this patient population.⁷¹ Similarly, experts advocate for the integration of lifestyle modifications into education and treatment programs for patients with mental illness.⁶⁸

Cognitive-behavioral therapy, group counseling, and motivational interviewing are all behavioral approaches to the treatment of obesity that have shown a significant degree of success in achieving and maintaining weight loss,^{71,73,74} although maintenance of weight loss has consistently proved challenging with these techniques. Improvement of long-term weight maintenance with these treatment modalities has been linked to extended periods of treatment and follow-up, combinations of various dietary and physical activity strategies, and increased patient-provider contact.⁷³ Some of these methods have been studied among patients with mental illness, revealing similar results.^{71,75} Pharmacotherapy has also shown promise in patients with mental illness; however, success has been limited by significant medication side effects and long-term weight regain.⁷⁵ With regard to pharmacologic contributors to obesity in the population of patients with mental illness, experts have advised that providers not be reluctant to consider switching psychiatric medications in patients who have gained $>5\%$ of initial body weight on treatment or who have signs of metabolic side effects, such as hyperglycemia or hyperlipidemia.⁶⁸

CASE DISCUSSION

Ms A was hospitalized for more than 1 month, during which time she underwent a thorough workup for any potential causes of malabsorption, including protein-losing enteropathy (stool α_1 -antitrypsin), a malignancy due to a persistently elevated mean corpuscular volume, a 24-hour stool fat study, and a 14-day trial of metronidazole for suspected bacterial overgrowth secondary to long-term effects of her bariatric procedure. The workup was negative, and the antibiotics did not significantly improve her nutritional status.

However, after further questioning, concern grew over the possibility that Ms A had anorexia and significant anxiety surrounding food, with long periods of eating very little over the preceding 2 years. It was unclear whether Ms A had been

taking her prescribed vitamin and nutrient supplements, and she had missed countless recent appointments with primary care and mental health providers. She described intense anxiety about eating, with explanations ranging from early satiety and loss of appetite to fear of gaining more weight, shame over her current appearance, and loss of desire to eat anywhere near other residents in her rehabilitation facility due to disgust for them. She had met with nutritionists in the past who had given her advice on what to eat, but she intermittently refused to follow their recommendations. She had been unable to adhere to her nutrition regimen at home and was similarly nonadherent in her rehabilitation facility. Notably, she had already been hospitalized several months earlier for psychiatric concerns at another hospital and had her psychiatric medications adjusted there. During this hospitalization, she was seen by the psychiatry team and was prescribed lorazepam before meals (to reduce anxiety) and mirtazapine (to stimulate appetite). However, she was unable to maintain sufficient oral intake and was started on total parenteral nutrition during her admission. Ms A had a semipermanent catheter placed for drainage of her persistent pleural effusion. Once her albumin levels improved, her pleural drainage slowed, and her right leg wound began to heal, she was discharged to a rehabilitation facility with the plan to eventually transition to home with visiting nurse services.

Unfortunately, records were unavailable to assess how thoroughly Ms A had been psychologically screened before gastric bypass surgery, and she was unable to provide a coherent history regarding the events that had led to her regaining weight and developing anorexia.

CONCLUSION

Health care providers commonly care for patients with obesity, a problem that has become an increasingly serious threat to the health of the general population. The causes and presentations of obesity are multifaceted and complex and lead to numerous comorbidities and medical complications. Advances in health care over the past several decades have provided us with a wider and more effective set of tools to treat obesity; however, many of these treatments come with their own risks that can lead to significant health complications. It is therefore critical that the benefits and risks be weighed for each individual patient when recommendations are made for obesity treatment. Importantly, psychosocial factors play a significant role in the development, perpetuation, and management of obesity; therefore, it is important that patients are evaluated from a psychiatric and social standpoint when being counseled on obesity prevention and treatment options.

Drug names: lorazepam (Ativan and others), lorcaserin (Belviq), metronidazole (Flagyl and others), mirtazapine (Remeron and others), orlistat (Xenical).

REFERENCES

1. Simpson JA, MacInnis RJ, Peeters A, et al. A comparison of adiposity measures as predictors of all-cause mortality: the Melbourne Collaborative Cohort Study. *Obesity (Silver Spring)*. 2007;15(4):994–1003.
2. Chan DC, Watts GF, Barrett PH, et al. Waist circumference, waist-to-hip ratio and body mass index as predictors of adipose tissue compartments in men. *QJM*. 2003;96(6):441–447.
3. Kissebah AH, Krakower GR. Regional adiposity and morbidity. *Physiol Rev*. 1994;74(4):761–811.
4. National Institutes of Health. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: the Evidence Report. *Obes Res*. 1998;6(suppl 2):51S–209S.
5. Baker S, Barlow S, Cochran W, et al. Overweight children and adolescents: a clinical report of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition. *J Pediatr Gastroenterol Nutr*. 2005;40(5):533–543.
6. Mokdad AH, Ford ES, Bowman BA, et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA*. 2003;289(1):76–79.
7. Flegal KM, Carroll MD, Ogden CL, et al. Prevalence and trends in obesity among US adults, 1999–2008. *JAMA*. 2010;303(3):235–241.
8. Choi JY. Prevalence of overweight and obesity among US immigrants: results of the 2003 New Immigrant Survey. *J Immigr Minor Health*. 2012;14(6):1112–1118.
9. Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of high body mass index in US children and adolescents, 2007–2008. *JAMA*. 2010;303(3):242–249.
10. Jeffery RW, French SA. Preventing weight gain in adults: design, methods and one year results from the Pound of Prevention Study. *Int J Obes Relat Metab Disord*. 1997;21(6):457–464.
11. Williamson DF. Descriptive epidemiology of body weight and weight change in US adults. *Ann Intern Med*. 1993;119(7, pt 2):646–649.
12. Yanovski JA, Yanovski SZ, Sovik KN, et al. A prospective study of holiday weight gain. *N Engl J Med*. 2000;342(12):861–867.
13. Wang Y, Beydoun MA, Liang L, et al. Will all Americans become overweight or obese? estimating the progression and cost of the US obesity epidemic. *Obesity (Silver Spring)*. 2008;16(10):2323–2330.
14. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. *N Engl J Med*. 1995;332(10):621–628.
15. Zhang Y, Proenca R, Maffei M, et al. Positional cloning of the mouse obese gene and its human homologue. *Nature*. 1994;372(6505):425–432.
16. Miyawaki K, Yamada Y, Ban N, et al. Inhibition of gastric inhibitory polypeptide signaling prevents obesity. *Nat Med*. 2002;8(7):738–742.
17. Wardle J, Carnell S, Haworth CM, et al. Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *Am J Clin Nutr*. 2008;87(2):398–404.
18. Tam EM, Lam RW, Robertson HA, et al. Atypical depressive symptoms in seasonal and nonseasonal mood disorders. *J Affect Disord*. 1997;44(1):39–44.
19. Sarwer DB, Wadden TA, Fabricatore AN. Psychosocial and behavioral aspects of bariatric surgery. *Obes Res*. 2005;13(4):639–648.
20. Wadden TA, Sarwer DB, Womble LG, et al. Psychosocial aspects of obesity and obesity surgery. *Surg Clin North Am*. 2001;81(5):1001–1024.
21. Bardia A, Holtan SG, Slezak JM, et al. Diagnosis of obesity by primary care physicians and impact on obesity management. *Mayo Clin Proc*. 2007;82(8):927–932.
22. Guo SS, Chumlea WC. Tracking of body mass index in children in relation to overweight in adulthood. *Am J Clin Nutr*. 1999;70(1):145S–148S.
23. Magarey AM, Daniels LA, Boulton TJ, et al. Predicting obesity in early adulthood from childhood and parental obesity. *J Int Assoc Study Obesity*. 2003;27(4):505–513.
24. Mustillo S, Worthman C, Erkanli A, et al. Obesity and psychiatric disorder: developmental trajectories. *Pediatrics*. 2003;111(4, pt 1):851–859.
25. Saunders MR, Watson KT, Tak HJ. Social factors in childhood and adulthood associated with adult obesity in African American and white women. *ISRN Public Health*. 2012.
26. Pagoto S, Bodenlos JS, Kantor L, et al. Association of major depression and binge eating disorder with weight loss in a clinical setting. *Obesity (Silver Spring)*. 2007;15(11):2557–2559.
27. Amianto F, Lavagnino L, Leombruni P, et al. Hypomania across the binge eating spectrum: a study on hypomanic symptoms in full criteria and subthreshold binge-eating subjects. *J Affect Disord*. 2011;133(3):580–583.
28. Stunkard AJ, Faith MS, Allison KC. Depression and obesity. *Biol Psychiatry*. 2003;54(3):330–337.
29. Nguyen NT, Magno CP, Lane KT, et al. Association of hypertension, diabetes, dyslipidemia, and metabolic syndrome with obesity: findings from the National Health and Nutrition Examination Survey, 1999 to 2004. *J Am Coll Surg*. 2008;207(6):928–934.
30. Wilson PW, D'Agostino RB, Sullivan L, et al. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. *Arch Intern Med*. 2002;162(16):1867–1872.
31. Hubert HB, Feinleib M, McNamara PM, et al. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation*. 1983;67(5):968–977.

32. Grundy SM, Barnett JP. Metabolic and health complications of obesity. *Dis Mon.* 1990;36(12):641–731.
33. Klein S, Burke LE, Bray GA, et al; American Heart Association Council on Nutrition, Physical Activity, and Metabolism. Clinical implications of obesity with specific focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. *Circulation.* 2004;110(18):2952–2967.
34. Rexrode KM, Hennekens CH, Willett WC, et al. A prospective study of body mass index, weight change, and risk of stroke in women. *JAMA.* 1997;277(19):1539–1545.
35. Calle EE, Rodriguez C, Walker-Thurmond K, et al. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of US adults. *N Engl J Med.* 2003;348(17):1625–1638.
36. McTigue K, Larson JC, Valoski A, et al. Mortality and cardiac and vascular outcomes in extremely obese women. *JAMA.* 2006;296(1):79–86.
37. Peeters A, Barendregt JJ, Willekens F, et al; NEDCOM, the Netherlands Epidemiology and Demography Compression of Morbidity Research Group. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med.* 2003;138(1):24–32.
38. van Dam RM, Willett WC, Manson JE, et al. The relationship between overweight in adolescence and premature death in women. *Ann Intern Med.* 2006;145(2):91–97.
39. US Preventive Services Task Force. Screening for obesity in adults: recommendations and rationale. *Ann Intern Med.* 2003;139(11):930–932.
40. US Preventive Services Task Force. Behavioral counseling in primary care to promote physical activity: recommendation and rationale. *Ann Intern Med.* 2002;137(3):205–207.
41. Snow V, Barry P, Fitterman N, et al; Clinical Efficacy Assessment Subcommittee of the American College of Physicians. Pharmacologic and surgical management of obesity in primary care: a clinical practice guideline from the American College of Physicians. *Ann Intern Med.* 2005;142(7):525–531.
42. Leblanc ES, O'Connor E, Whitlock EP, et al. Effectiveness of primary care-relevant treatments for obesity in adults: a systematic evidence review for the US Preventive Services Task Force. *Ann Intern Med.* 2011;155(7):434–447.
43. Andersen RE, Wadden TA, Bartlett SJ, et al. Effects of lifestyle activity vs structured aerobic exercise in obese women: a randomized trial. *JAMA.* 1999;281(4):335–340.
44. Bray GA, Greenway FL. Pharmacological treatment of the overweight patient. *Pharmacol Rev.* 2007;59(2):151–184.
45. Flum DR, Belle SH, King WC, et al; Longitudinal Assessment of Bariatric Surgery (LABS) Consortium. Perioperative safety in the longitudinal assessment of bariatric surgery. *N Engl J Med.* 2009;361(5):445–454.
46. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA.* 2004;292(14):1724–1737.
47. Sjöström L, Lindroos AK, Peltonen M, et al; Swedish Obese Subjects Study Scientific Group. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med.* 2004;351(26):2683–2693.
48. Hollander PA, Elbein SC, Hirsch IB, et al. Role of orlistat in the treatment of obese patients with type 2 diabetes: a 1-year randomized double-blind study. *Diabetes Care.* 1998;21(8):1288–1294.
49. Padwal R, Li SK, Lau DC. Long-term pharmacotherapy for obesity and overweight. *Cochrane Database Syst Rev.* 2004;(3):CD004094.
50. FDA approves weight-management drug Qsymia. FDA News Release. July 17, 2012. <http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm312468.htm>.
51. Bloomberg RD, Fleishman A, Nalle JE, et al. Nutritional deficiencies following bariatric surgery: what have we learned? *Obes Surg.* 2005;15(2):145–154.
52. Spector JA, Levine SM, Karp NS. Surgical solutions to the problem of massive weight loss. *World J Gastroenterol.* 2006;12(41):6602–6607.
53. Teixeira ME, Budd GM. Obesity stigma: a newly recognized barrier to comprehensive and effective type 2 diabetes management. *J Am Acad Nurse Pract.* 2010;22(10):527–533.
54. Wee CC, McCarthy EP, Davis RB, et al. Obesity and breast cancer screening. *J Gen Intern Med.* 2004;19(4):324–331.
55. Wee CC, McCarthy EP, Davis RB, et al. Screening for cervical and breast cancer: is obesity an unrecognized barrier to preventive care? *Ann Intern Med.* 2000;132(9):697–704.
56. Drury CA, Louis M. Exploring the association between body weight, stigma of obesity, and health care avoidance. *J Am Acad Nurse Pract.* 2002;14(12):554–561.
57. Lewis S, Thomas SL, Hyde J, et al. A qualitative investigation of obese men's experiences with their weight. *Am J Health Behav.* 2011;35(4):458–469.
58. Huizinga MM, Cooper LA, Bleich SN, et al. Physician respect for patients with obesity. *J Gen Intern Med.* 2009;24(11):1236–1239.
59. Huizinga MM, Bleich SN, Beach MC, et al. Disparity in physician perception of patients' adherence to medications by obesity status. *Obesity (Silver Spring).* 2010;18(10):1932–1937.
60. Fine JT, Colditz GA, Coakley EH, et al. A prospective study of weight change and health-related quality of life in women. *JAMA.* 1999;282(22):2136–2142.
61. Dixon JB, Dixon ME, O'Brien PE. Depression in association with severe obesity: changes with weight loss. *Arch Intern Med.* 2003;163(17):2058–2065.
62. Simon GE, Von Korff M, Saunders K, et al. Association between obesity and psychiatric disorders in the US adult population. *Arch Gen Psychiatry.* 2006;63(7):824–830.
63. Carpenter KM, Hasin DS, Allison DB, et al. Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: results from a general population study. *Am J Public Health.* 2000;90(2):251–257.
64. Weber NS, Fisher JA, Cowan DN, et al. Psychiatric and general medical conditions comorbid with bipolar disorder in the National Hospital Discharge Survey. *Psychiatr Serv.* 2011;62(10):1152–1158.
65. Stanley SH, Laugharne JD, Addis S, et al. Assessing overweight and obesity across mental disorders: personality disorders at high risk. *Soc Psychiatry Psychiatr Epidemiol.* 2013;48(3):487–492.
66. DE Hert M, Correll CU, Bobes J, et al. Physical illness in patients with severe mental disorders, 1: prevalence, impact of medications and disparities in health care. *World Psychiatry.* 2011;10(1):52–77.
67. De Hert M, Cohen D, Bobes J, et al. Physical illness in patients with severe mental disorders, 2: barriers to care, monitoring and treatment guidelines, plus recommendations at the system and individual level. *World Psychiatry.* 2011;10(2):138–151.
68. Stanley SH, Laugharne JD. Obesity, cardiovascular disease and type 2 diabetes in people with a mental illness: a need for primary health care. *Aust J Prim Health.* 2012;18(3):258–264.
69. Marder SR, Essock SM, Miller AL, et al. Physical health monitoring of patients with schizophrenia. *Am J Psychiatry.* 2004;161(8):1334–1349.
70. Bonfilioli E, Berti L, Goss C, et al. Health promotion lifestyle interventions for weight management in psychosis: a systematic review and meta-analysis of randomised controlled trials. *BMC Psychiatry.* 2012;12(1):78.
71. Hasnain M, Fredrickson SK, Vieweg WV, et al. Metabolic syndrome associated with schizophrenia and atypical antipsychotics. *Curr Diab Rep.* 2010;10(3):209–216.
72. Van Dorsten B, Lindley EM. Cognitive and behavioral approaches in the treatment of obesity. *Med Clin North Am.* 2011;95(5):971–988.
73. Laddu D, Dow C, Hingle M, et al. A review of evidence-based strategies to treat obesity in adults. *Nutr Clin Pract.* 2011;26(5):512–525.
74. Taylor VH, Stonehocker B, Steele M, et al. An overview of treatments for obesity in a population with mental illness. *Can J Psychiatry.* 2012;57(1):13–20.