The information in this column is not intended as a definitive treatment strategy but as a suggested approach for clinicians treating patients with similar histories. Individual cases may vary and should be evaluated carefully before treatment is provided. The patient described in this column is a composite with characteristics of several real patients.

Binge eating disorder hidden behind a wall of anxiety disorders

Amy Pipe, MB; Beth Patterson, MSc; Michael Van Ameringen, MD

A 23-year-old university student was initially seen for symptoms of long-standing social anxiety disorder (SAD). Her primary symptoms included excessive shyness and avoidance of peer-related activities, large family gatherings, speaking with authority figures, public speaking and class participation. She was particularly concerned about being negatively evaluated by others for her body weight, despite not being obviously obese (her body mass index was 28 kg/m^2). She reported that 2 years prior, she had had a treatment trial of mirtazapine 30 mg/d; however, this resulted in a 25 lbs weight gain. Her current treatment at initial assessment was venlafaxine extended-release (ER) 225 mg/d, which she felt had little benefit for her anxiety. We elected to change her treatment to escitalopram, titrated to 20 mg/d, which substantially improved her social anxiety. She was able to participate in class, stopped avoiding social situations and performed well at school.

Her anxiety symptoms remained stable over the subsequent 5 years; however, her concerns about her weight persisted. She had tried many different diets without success and had attended 2 medically supervised weight loss programs. She attributed her lack of success to stress eating and disclosed that she engaged in significant eating binges at least 4 times per week — sometimes 2 or 3 binges in 1 day. She would consume substantial quantities of cookies and ice cream in the span of an hour despite not feeling hungry. She reported a lot of self-deprecating thoughts, guilt and distress following her binges. Although this behaviour had been ongoing since mid-adolescence, she did not disclose this during her 5 years of psychiatric care as she was concerned with being negatively judged. Additionally, screening for binge eating was not part of our usual clinical assessment of anxiety disorders at that time.

Binge-eating disorder (BED) is characterized by recurrent episodes of rapid, uncontrolled eating accompanied by a sense of loss of control and psychological distress.[1] Known as the most common eating disorder, the average lifetime prevalence of BED is 1.4%.[2] About 47% of people with BED have at least 1 psychiatric comorbidity, including anxiety disorders, which occur in 12%–70% of patients with BED.[3–6] BED and SAD comorbidity is also common; it is reported by 10%–59% in community and BED samples.[7–12] Unfortunately, BED often goes unnoticed by health care professionals, as patients typically seek medical or psychiatric care for the treatment of conditions caused by their BED rather than the BED itself.[13]

Current evidence suggests that individuals with BED have decreased reward sensitivities, greater cognitive attentional biases toward food and altered brain activation in regions associated with impulsivity and compulsivity than individuals who do not have BED.[14,15] Although multiple neurotransmitter systems are likely involved (including μ-opioid receptors and norepinephrine), it is thought that dysregulated dopamine systems, which mediate eating and reward-seeking behaviours, lie at the core of BED.[16] This is evidenced by studies in animals[15] and humans.[17]

It has been theorized that BED results from an altered balance of signalling between the direct striatonigral output pathway (D1 receptors, linked to reward) and indirect striatonigral pathways (D2 receptors linked to behavioural flexibility).[15,18,19] Psychostimulants, typically used to treat attention-deficit/hyperactivity disorder (ADHD), target both norepinephrine and dopamine pathways, which are involved in the regulation of eating behaviour and reward.[17,20] Preclinical studies have shown that stimulants block the reuptake of dopamine and norepinephrine into the presynaptic neuron, thereby increasing the availability of both neurotransmitters in the synaptic cleft.[21] These agents may also alter the perception of food-related reward, thereby increasing patients' control over their eating behaviour.[22]

The stimulants lisdexamfetamine dimesylate (LDX) and methylphenidate ER as well as the antiseizure agent topiramate have demonstrated efficacy in randomized controlled trials for the treatment of BED, although LDX is the only agent with an indication for this condition.[23] Hydrolysis of LDX releases pharmacologically active d-amphetamine, which, unlike methylphenidate, has a direct effect on dopamine release.[20] Compared with placebo, LDX has demonstrated short-term (12-week) efficacy in reducing the number of weekly binge episodes, as well as significantly decreasing functional disability in a randomized, double-blind, large multi-site study (n = 514).[24] It has also demonstrated a significantly decreased risk of relapse at 6 months compared with placebo in a similarly large, randomized, multi-site withdrawal study (n = 418).[25] Reduced brain activation has been found in the globus pallidus, ventrolateral prefrontal cortex, and striatum (associated with responses to palatable foods) following 12 weeks of LDX treatment for BED.[26] Although LDX is generally well-tolerated, the most common adverse events include dry mouth, decreased appetite and insomnia.[24]

Many clinicians are reticent to use stimulants in patients with comorbid anxiety disorder, for fear of worsening anxiety symptoms. However, the ADHD literature does not support this. The bulk of studies examining ADHD with comorbid anxiety disorders have

demonstrated either improvement or no change in the anxiety symptoms with stimulant treatment.[27–30] Cognitive behavioural therapy (CBT), a first-line treatment for anxiety disorders,[31] has also demonstrated efficacy in reducing bingeing in BED.[24] CBT has been examined in combination with several pharmacological agents in BED (equivocal results), but not in combination with LDX.[24,32]

Our patient was subsequently treated with LDX titrated to 50 mg/d. Complete cessation of bingeing occurred within 4 weeks. Over the course of 5 months she lost 30 lbs without changing her diet. Around the 6-month mark, she reported experiencing significant stress from external life circumstances, but did not resume binge eating.

BED is easily overlooked by clinicians while treating common comorbid conditions. Unlike the other eating disorders, BED is highly responsive to treatment with stimulants and often results in early, sustained remission. Our patient's experience highlights the need to screen for BED in patients who struggle with weight management and who present with anxiety and mood disorder symptoms.

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