



# Challenges in Managing Treatment-Refractory Obsessive-Compulsive Disorder and Tourette's Syndrome

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## CASE HISTORY

Mr. R was a 19-year-old, single, unemployed, homeless Hispanic male with a history of Tourette's syndrome (TS), obsessive-compulsive disorder (OCD), attention-deficit/hyperactivity disorder (ADHD), and posttraumatic stress disorder. In addition, he suffered from suicidal ideation, self-injurious behavior (superficial cutting and head banging), anxiety, depression, and anger outbursts. He was referred to our cognitive-behavioral therapy (CBT) partial hospitalization program (PHP) and associated homeless shelter for the targeted treatment of his OCD, TS, and anger outbursts after being discharged from a psychiatric hospital.

At the time of presentation, Mr. R had recently aged out of a long-term adolescent state hospitalization program. Since that time, he has had a short stay in a crisis stabilization unit and a lengthier admission to a psychiatric hospital for poor impulse control and anger outbursts. At the crisis stabilization unit, he became "stuck" after the lights unexpectedly turned off in the bathroom, which led to uncontrollable tics, exacerbation of OCD symptoms, screaming that lasted three hours, and, finally, an angry outburst and acute hospitalization. After discharge, his outbursts were so severe that his mother did not feel safe with him at home. He was admitted

to our PHP and, because of his outbursts, forced to live in the associated shelter. On admission, he endorsed his mood as "sad." He admitted to having tics (vocalizations, moving foot, head, scratching) and OCD symptoms (looking at things a certain number of times, obsession with symmetry, and staring at a corner) that lasted the whole day. He endorsed anxiety that consisted of general worry and ruminations about the past. He denied any manic or psychotic symptoms, as well as suicidal or homicidal ideation.

Developmentally, the patient was born at 40 weeks gestation, birth weight 8 lbs, 2 oz, and delivered by normal spontaneous vaginal delivery, with no prenatal or perinatal complications. He returned home from the hospital with his parents, and his developmental milestones were normal. He first developed tics at age 10, which consisted of quick shoulder jerks that progressed to coordinated shoulder movements, head rolling, and eye rolling as a combined set of movements. He also had throat clearing and simple vocalizations. The vocal tics did not involve syllables, words, phrases, or echolalia. His tics had not been responsive to clonidine or guanfacine. At age 14, he was started on haloperidol, which suppressed his tics but resulted in significant weight gain and tachyphylaxis. Due to escalated aggression, risperidone was tried, which offered fair behavioral control for about 2–3 years. He was briefly on ziprasidone and aripiprazole, with little effect. Topiramate reportedly helped to reduce his tics.

At age 16, his tics significantly increased, as he began to have intrusive thoughts that his food was poisoned—to the point that he stopped eating and lost over 100 pounds. This behavior resulted in an adolescent psychiatric unit admission. During the hospitalization his anger outbursts progressed, and he threatened and assaulted staff. He was felt not to be safe for discharge and was transferred to a state hospital. While at the state hospital, Mr. R's neuropsychiatric symptoms were difficult to control, particularly those with a prominent compulsive aspect. His OCD symptoms caused him to get "stuck" in the middle of activities of daily living, such as showering or

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dressing. Symptoms included checking the time on his phone, repeating words, and rituals related to washing in the shower, until he got them “just right.” These symptoms increased his frustration and anxiety, and when staff tried to redirect him, he became angry, lost control, punched walls, and threw nearby items at staff.

As for Mr. R's social history, he had an urban upbringing and was raised by his mother. His father was incarcerated when the patient was six years old. He had grown up as an only child until two years ago, when his mother had her second child. Mr. R witnessed domestic violence in the home, which, he reported, contributed to his anxiety and anger. He had not participated in any early-intervention program. He reached the 12th grade at a therapeutic school but did not graduate. He had an individualized education plan for physical, occupational, and speech therapy, as well as behavioral management. He had never worked. He enjoyed playing sports, specifically basketball, and liked to swim. He denied any history of alcohol, tobacco, or illicit drug use. His family history was notable for OCD in his mother, who was doing well off of medications, and two maternal cousins with ADHD. His paternal family history was unknown.

Mr. R's medical history was significant for elevated liver function tests. A liver biopsy was performed and showed moderate steatosis, mild chronic portal inflammation, and portal fibrosis. Several syncopal episodes raised suspicion for a seizure disorder, which was ruled out after an electroencephalogram did not show epileptiform activity and after a brain MRI was negative for any intracranial pathology. He had been recently diagnosed with benign tachycardia that was thought to be due to anxiety. He had no known drug allergies.

Mr. R's laboratory values on his most recent psychiatric admission revealed a normal complete blood count, basic metabolic panel, and liver function tests, except for elevated fasting blood glucose. A toxicology screen was negative except for benzodiazepines, which he was prescribed and taking regularly. His valproic acid and clomipramine levels were 90 mcg/mL and 94 mcg/L, respectively. The patient's electrocardiogram had a normal QTc and QRS intervals.

On psychiatric evaluation at our CBT-based PHP, he was noted to have eye rolling and facial and shoulder tics. The latter tic was performed until it felt “just right,” which would take minutes to hours to complete, making him feel “stuck.” If interrupted, he became upset, necessitating the reinitiation of the ritual. Another tic involved the urge to look at an object around the room, look away, and look back again, until he “got it just right.” On mental status examination, Mr. R appeared calm, cooperative, and childlike. His speech was characterized by normal volume, tone, and prosody, with occasional periods of stuttering. His affect was anxious, euthymic, and occasionally irritable. He denied experiencing hallucinations, thought insertion or broadcasting, or ideas of reference. He did not exhibit any loosening of associations, and his thought process was largely linear and goal directed. He denied any thoughts of harm to self or others. The cognitive examination

was characterized by intact orientation, attention, calculations, and memory tasks.

The patient's clinical history was complex. The differential diagnosis included severe TS and OCD-related compulsive tics with difficulty inhibiting impulses that made him prone to sudden anger outbursts. In his treatment, we targeted his anger, which was thought to be multifactorial. Our work with him included identifying his triggers and reducing the frequency or severity of episodes. We also utilized pharmacotherapy and behavioral approaches to boost his capacity to inhibit compulsions.

During Mr. R's participation in the PHP, he experienced intermittent difficulty with his symptoms. On some days he was able to engage in CBT, whereas on other days he was unable to participate, because of his rituals. His rituals would also often land him in the infirmary, making it difficult for him to engage in groups and in individual treatment. Staff had to wait for Mr. R to finish his rituals before anyone could effectively work with him. In an attempt to avert his outbursts, a behavioral plan was instituted that allowed him to engage in rituals as long as he could maintain self-control. He also had a difficult time managing his symptoms during visits to his mother's home, which frequently resulted in emergency department visits. In an effort to reduce hospital visits and possible psychiatric hospitalizations, limits were set that kept Mr. R from visiting his mother's home and that encouraged him to stay at the PHP-affiliated shelter.

When Mr. R was able to participate in individual treatment, he performed some basic exposure response prevention work. Together with Mr. R, we created a hierarchy of his stressors. His least stressful trigger was having people walk closely by him; his most difficult stressor was loud noises. Other stressors included ritual interruption and being touched. On several occasions, he recorded his ritualistic episodes, including the trigger and duration of rituals. Additionally, on more productive treatment days, Mr. R identified coping skills and engaged in coping strategies when he was feeling overwhelmed. He identified listening to music and playing basketball to be some of his more effective coping skills.

To help reduce the frequency and severity of Mr. R's behavioral outbursts, his clomipramine was increased to 250 mg daily, as his blood level was subtherapeutic. Risperidone was continued at 3 mg twice daily, as antipsychotics had previously been effective in managing his anger. The dose of divalproex was decreased, with a plan to discontinue this medication if symptoms improved. Clonazepam was discontinued, as it would interfere with therapy. A review of the literature suggested that memantine, a targeted glutamate modulator, could be an effective augmentation strategy for the presumed glutamatergic mechanism of OCD. Memantine was started at 5 mg daily and was slowly uptitrated to 10 mg twice daily, with no noticeable improvement in symptoms. This medication was ultimately discontinued. We considered evaluation for neurosurgical interventions, but the team was informed that the patient was a

poor candidate, given his degree of behavioral dysregulation and suicidal ideation with self-injurious behavior.

After a few months in the PHP, Mr. R had a decrease in behavioral outbursts and increased his participation in treatment; thus, he was felt to be stable enough to begin discharge planning. A visit to a step-down program, however, triggered another episode of getting “stuck.” Mr. R became physically violent upon interruption of his rituals and was brought to the nearest hospital.

After Mr. R was transported to the emergency department, he was placed at a private psychiatric facility. A few days later, the patient’s mother picked up the patient’s belongings from the shelter and closed his bed there. The PHP team was in touch with the hospital, his case manager, and his mother in an attempt to support them. His aftercare plan after his psychiatric admission is unknown.

In addition to Mr. R’s challenging presentation, several other external issues made this case difficult to manage. Systematic issues arose when Mr. R became an adult because he was no longer allowed to access resources in the child system that had provided him with various supports in the past. For example, after his most recent psychiatric admission, it was recommended that he go to one of the state’s structured shelters, but he was sent to a general shelter instead. While Mr. R was being treated within the child system, a general shelter would likely not have been a housing option.

An additional challenge arose from inconsistencies in working with Mr. R’s mother and legal guardian to establish a behavioral plan. His mother typically called daily for updates and to communicate her concerns about her son. But when Mr. R had an episode of getting “stuck” while visiting his mother at home that resulted in property destruction, his mother informed providers that she would no longer be involved in his care. Given this incident, the team established a plan with Mr. R and his mother to suspend all visits for two weeks or until he was in better behavioral control. In spite of this plan, his mother allowed Mr. R to visit the home prior to the completion of the two-week trial period. This one example illustrates the difficulty in creating behavioral plans with Mr. R while involving his family in treatment planning.\*

### QUESTIONS TO THE CONSULTANTS:

1. To Dr. Scharf: Please describe the pathophysiology, diagnosis, and clinical management of TS/OCD. How should clinicians approach treatment-refractory cases?
2. To Dr. Berman: At times, family may have a difficult time understanding how to best support a family member that suffers from a severe mental illness. In what ways can family be effectively educated in order to best help the patient?
3. To Drs. Widge, Dougherty, and Eskandar: What factors determine if deep brain stimulation is an appropriate

intervention for TS/OCD? Is this patient a good candidate for deep brain stimulation?

### Jeremiah Scharf, MD, PhD

To place the discussions below into context, I will provide an overview of the clinical features, diagnosis, presumed pathophysiology, and management of TS and OCD, followed by a targeted discussion of Mr. R’s symptoms and how they relate to our current understanding of the diagnostic overlap between TS and OCD, along with the particular challenges that can arise in treatment-refractory cases.

TS is a childhood-onset neuropsychiatric disorder characterized by the presence of multiple motor tics and at least one vocal tic that are present before age 18 and persist for at least one year.<sup>1</sup> Motor tics are repeated, usually rapid, non-rhythmic motor movements that most often involve the eyes, head, face, and shoulders, but can represent any fragment of otherwise normal motor movements or sequences of movements. Similarly, vocal tics range from repeated sounds such as sniffing, coughing, throat clearing, or squeaking to complex syllables, words, or phrases. Coprolalia, the utterance of socially inappropriate words or phrases, is not required for the diagnosis of TS and, in fact, is present only in 10%–20% of TS patients.<sup>2</sup> Tics are briefly suppressible and are usually preceded by a premonitory sensation or urge that builds up when tics are suppressed. This distinctive feature of tics is useful in confirming the diagnosis and serves as the basis for behavioral interventions for tics, as will be discussed below. TS is now considered to exist along a continuous spectrum of developmental tic disorders; chronic (persistent) motor (or vocal) tic disorder (CT) has the same clinical features, developmental time course, and treatment as TS but involves only motor (or only vocal) tics instead of both motor and vocal tics. Persistent tic disorders are much more common than previously appreciated, with population prevalence estimates of 0.3%–0.9% of school-age children for TS and 1%–2% for CT.<sup>3,4</sup> While the majority of children with TS experience significant reduction or remission of symptoms in late adolescence or early adulthood, approximately 20% have persistent impairing symptoms as adults, as in Mr. R’s case.<sup>5</sup> A diagnosis of TS or CT is made solely on the presence of a characteristic presentation and a normal neurological examination. If both of these are typical/normal, no laboratory tests or neuroimaging is required.<sup>6</sup>

OCD is defined by the presence of recurrent, intrusive, ego-dystonic (unwanted) thoughts, urges, or images (i.e., obsessions) that cause intense anxiety and distress, and that are neutralized by performance of ritualistic, repetitive behaviors (compulsions).<sup>1</sup> Obsessions vary widely and can include thoughts of contamination, harm to oneself or others, absence of symmetry or order, and taboo thoughts (aggressive, sexual, or religious). Compulsions can be any repeated behavior (or thought) performed to relieve or prevent anxiety generated by obsessions, but most often consist of cleaning, checking, repeating, counting, ordering/arranging, praying/

\*Paulo Lizano, MD, PhD, and Ami Popat-Jain, MA, prepared the case history.

confessing, or, in some cases, repeated motor sequences. While obsessive-compulsive (OC) symptoms in the absence of significant distress are common, an OCD diagnosis requires that symptoms are severe enough to be time-consuming (>1 hour per day) or to cause distress or impairment. The prevalence of OCD is approximately 1%–2% of adults.<sup>7</sup>

About 10%–20% of OCD patients have a co-occurring tic disorder, particularly those with childhood-onset OCD.<sup>7,8</sup> By contrast, 30%–50% of TS patients meet full diagnostic criteria for OCD, with many more manifesting non-interfering OC symptoms.<sup>9,10</sup> While patients with TS or chronic tics can have any of the obsessive or compulsive symptoms seen in OCD patients without tics, OCD in the context of tics will often involve themes of symmetry or “evening up.”<sup>11</sup> Interestingly, while functional imaging studies of OCD patients exposed to contamination triggers result in increased activity in the orbitofrontal cortex,<sup>12,13</sup> provocation with asymmetric stimuli in TS patients with symmetry OCD activates both the orbitofrontal and supplementary motor cortex, suggesting that these obsessions may indeed be more “tic-like.”<sup>14</sup> TS patients will also feel a need to complete tics a certain number of times or until they feel “just right,” which then relieves their uncomfortable, physical urge to tic.<sup>15</sup> In addition, a small subset of TS patients will develop frank anxiety when tics are not completed correctly, as seen in the case of Mr. R with his shoulder or eye tics. In this case, tics may be functioning as the compulsive arm of an OC loop, where correct performance of a tic neutralizes anxiety as opposed to providing physical relief of a somatic tension/urge that is more classically associated with tics.

Both tics and OCD are thought to arise from aberrant development or maintenance of regulatory brain circuits connecting specific areas of the cerebral cortex to corresponding regions in the basal ganglia and thalamus (cortico-striato-thalamo-cortical [CSTC] loops).<sup>16,17</sup> These circuits are involved in top-down control and selection of intended actions, thoughts, and behaviors—which, in a simplified model, correlate with dysregulated motor and premotor CSTC loops in TS and with orbitofrontal CSTC loops in OCD.<sup>18,19</sup> As is the case for Mr. R, approximately 30%–50% of TS patients also have ADHD, with presumed dysregulation of CSTC loops between the dorsolateral prefrontal cortex and corresponding areas of the anterior caudate, pallidum, and associative thalamic nuclei.<sup>9,10,17</sup> As such, the frequent co-occurrence of TS, OCD, and ADHD can be viewed as arising from abnormal development of a shared set of CSTC circuit components that manifest clinically as distinct, though sometimes overlapping, symptoms.

In this case, Mr. R has symptoms of complex tics, OCD, and ADHD, as well as severe anger outbursts (“rage attacks”), the latter of which are the major source of Mr. R’s impairment and have resulted in institutionalization. Severe anger outbursts in TS patients are similar to those seen in ADHD patients without tics, and are thought to arise from impaired

frontally mediated inhibitory control of impulsive anger.<sup>20–22</sup> These outbursts can arise, however, from a wide range of triggers that are usually related to frustration or anxiety arising from incomplete treatment of other symptoms such as severe tics, difficulty initiating or completing homework due to ADHD, or interruption of OC rituals. In Mr. R’s case, his slowness in performing activities of daily living and responding to directions from staff because of his need to complete tics and compulsions was not tolerated by his institutional environment, which led to increased anxiety and subsequent explosive anger outbursts.

Treatment of refractory TS, such as Mr. R’s, first requires a functional analysis to identify both the antecedents of anger outbursts and the degree to which other co-occurring neuropsychiatric disorders are contributing to his combustible mix of behaviors. In Mr. R’s case, his severe impulsivity and disinhibition place him at high risk for anger outbursts at baseline. On top of that, plus his baseline obsessive and non-obsessive anxiety, interruption of his treatment-refractory compulsive tics appears to be the major trigger for his outbursts. Sleep disruption, due either to anxiety or to another sleep disorder (which is present in 30%–65% of TS patients), will further add fuel to the fire.<sup>23</sup> Finally, the environmental context of his placement in a correctional facility, where staff may take a confrontational approach to disruptive behavior, can further escalate the situation.

For treatment-refractory cases of OCD and TS, multimodal treatment approaches are needed—including medications addressing anxiety, OCD, tics, and anger, as well as behavioral therapy. Intensive CBT was initially effective for Mr. R, though as noted by Dr. Berman below, training of parents and other care providers is also crucial. In multicenter, randomized, controlled trials, behavioral therapy for tics (comprehensive behavioral intervention for tics; CBIT) has been demonstrated to be effective in reducing tics in both children and adults;<sup>24,25</sup> it could be employed to target specific impairing tics. From a medication standpoint, tics that lie at the border zone between TS and OCD require treatment for both the tic and OC component. While the alpha-2 agonists clonidine and guanfacine, supplemented with topiramate in some cases, are the mainstay first-line agents for tics,<sup>26</sup> severe compulsive tics will often require use of an atypical neuroleptic combined with a selective serotonin reuptake inhibitor (SSRI) or clomipramine.<sup>27</sup> This combination is consistent with results from controlled clinical trials indicating that OCD with tics is more refractory to SSRI therapy than OCD without tics, and often requires neuroleptic augmentation, with the best data currently indicating risperidone.<sup>27</sup> For OCD alone, while fluvoxamine is often cited as the SSRI of choice, meta-analyses have demonstrated that all SSRIs are equally effective in treating OCD.<sup>28,29</sup> For severe OCD, clomipramine has been proven to be the most effective medication treatment,<sup>28</sup> and CBT plus clomipramine (or an SSRI) has been shown to be more effective in combination than with either treatment alone.<sup>30,31</sup>

Most recently, emerging data support augmentation of SSRIs with a glutamatergic modulator such as memantine,

N-acetylcysteine, or riluzole in treatment-refractory OCD.<sup>32</sup> Whether these agents are also effective for treating OCD in the context of tics—and, in particular, OCD where tic completion is the anxiety-provoking symptom—has yet to be determined. Treatment-refractory tics, particularly those that reemerge in adults after years of quiescence, are often driven by undertreated non-obsessive anxiety, OCD, or sleep disruption. In my experience, these areas of functioning should be targeted as aggressively as direct tic suppression, and may result in the need for lower doses of neuroleptics. Finally, let me mention that Drs. Widge, Dougherty, and Eskandar provide a detailed discussion below on the use of deep brain stimulation (DBS) in treatment-refractory TS patients. A recent position paper from the Tourette Association DBS Registry and Study Group suggests that additional randomized, controlled trials are needed to identify the optimal location and settings for DBS in TS, as well as to monitor and report the effects of DBS on TS-related comorbidities (OCD, ADHD, disinhibition, anxiety, anger), which often cause more impairment than the tics themselves.<sup>33</sup>

#### Noah Berman, PhD

The family's role in the development, maintenance, and treatment of severe mental illness has been extensively studied for decades. I will focus on how the family influences one of Mr. R's most impairing disorders: OCD. In particular, I will discuss how Mr. R's mother could be effectively educated to improve his treatment and functional impairment. Indeed, much research has demonstrated the effect of family accommodation (i.e., the process of family members assisting or participating in the patient's compulsions) and maladaptive communication styles (i.e., hostility and criticism) on OCD symptom severity and treatment outcome. Therefore, I will first describe the conceptual model of family accommodation and how it maintains, and often exacerbates, OCD symptoms. Next, I will describe how Mr. R's significant rage relates to family accommodation and will suggest a self-report measure that can both guide treatment and educate the family. Finally, I will describe how to incorporate the family into treatment by targeting maladaptive behaviors through CBT—specifically, exposure with response prevention.

**FAMILY ACCOMMODATION: FUNCTION AND CORRELATES** Research has demonstrated that family accommodation is associated with (1) poorer treatment outcome in adults<sup>34</sup> and youth,<sup>35</sup> (2) greater depression and anxiety in family members,<sup>36</sup> and (3) poorer family functioning.<sup>37</sup> Broadly speaking, family members participate in a patient's rituals in an effort to reduce or neutralize the patient's distress. This process does not occur overnight; instead, the dynamic tends to evolve over months or years. For instance, Mr. R's family likely accommodated his poison-related intrusions in his early teenage years by helping him avoid feared stimuli or by identifying safety foods. In that scenario, Mr. R's mother likely accommodated his OCD in an effort to reduce his distress and the time spent on lengthy rituals.<sup>38</sup> Although accommodation

would have been effective in the short term (i.e., reducing anxiety), these behaviors ultimately prevented Mr. R from adaptively confronting his obsessional thoughts and coping with the subsequent anxiety. Not surprisingly, accommodating behaviors maintain the severity of OCD symptoms and mitigate the motivation to engage in adaptive behavioral change.<sup>39</sup>

Although accommodation is a common familial response to a loved one with OCD, evidence suggests that Mr. R's mother is even more likely to engage in this set of maladaptive behaviors, given her own OCD diagnosis.<sup>40</sup> Although no research has concluded why parental anxiety is associated with accommodating behaviors, one hypothesis is that parents have difficulty tolerating negative affect in themselves and others, and therefore engage in a short-term solution—accommodation—to minimize discomfort. This same negative reinforcement dynamic applies to parents' responses to children's rage. Recent research indicates that rage attacks within the context of OCD are often associated with the disruption of rituals (as seen in Mr. R) and are directly related to the degree of family accommodation.<sup>41</sup> Paralleling the motivation to accommodate OCD symptoms, family members facilitate avoidance or abandon limit setting when the patient is “raging,” in order to shorten the duration of the episode. Paradoxically, these accommodating and avoidant behaviors reinforce the frequency and intensity of the rage attacks. In fact, mediation analyses indicate that the severity of the patient's rage leads to an increase in family accommodation prior to reducing functional impairment.<sup>41</sup> Applying these findings to Mr. R: when he becomes “rageful,” proximal individuals (i.e., mother, nurses) ultimately engage in accommodating behaviors in an effort to reduce the intensity of his anger. As a result, these short-term solutions negatively reinforce the patient's maladaptive behaviors and lead to greater functional impairment. Fortunately, these behavioral responses can be directly targeted in treatment.

**EDUCATING AND INCORPORATING THE FAMILY INTO TREATMENT** Given the significant impact of family accommodation on symptom severity and treatment outcome, researchers have developed brief assessment tools (e.g., Family Accommodation Scale–Parent Report)<sup>42</sup> to guide treatment (i.e., identify the domains of accommodation for the therapist) and to educate the patient and family on how accommodating behaviors correspond to the patient's OCD symptoms and severity. Given Mr. R's age, the therapist should begin treatment by introducing him to the cognitive-behavioral model of OCD. If Mr. R is interested in involving his mother, she could attend the next session, and Mr. R could teach the model directly to her. In the process, the therapist would be in a position to determine whether Mr. R fully understands the relationship among his thoughts, emotions, and behaviors. Importantly, this approach offers an ideal segue into a discussion of how family accommodation functions similarly to a ritual in the maintenance of symptoms.

Following the educational intervention, Mr. R's mother should possess a preliminary understanding of how to engage in more adaptive behaviors that are in service of reducing her son's symptom severity. Assuming that the mother wishes to be involved in the remainder of treatment, I would suggest the following familial treatment targets: (1) normalize and validate her frustration and distress, (2) reduce accommodation of symptoms through collaborative problem solving, (3) learn to support Mr. R by acting as a co-therapist or coach, and if necessary, (4) reduce her criticism and hostility in response to Mr. R's rituals.<sup>43,44</sup> Although Mr. R's mother may "buy into" the theoretical model of reducing accommodating behaviors, she may be fearful to watch her child be in debilitating distress. To identify obstacles to implementing behavioral change, the therapist could ask the parent-child dyad to role-play a high-intensity scenario, or if that is too difficult, the therapist could act as either the patient or the parent in the role playing. Observing the dyad communicate in the context of the role playing has the potential to elucidate ineffective (and damaging) language. For instance, in response to the child's rituals, Mr. R's mother might employ hostile or critical language (e.g., "It's so stupid that you have to keep staring at different parts of the room—what's wrong with you?"). Indeed, this communication style is associated with greater OCD symptom severity and should therefore be addressed and targeted. To do so, the therapist could provide the family with psychoeducation and offer adaptive strategies to both communicate emotions and disengage from conflict.<sup>45</sup>

Family members often can be overwhelmed by the amount of changes (e.g., daily routine, communication, limit setting) that the therapist suggests during the educational phase of treatment. Therefore, it is crucial that both the patient and family member understand that CBT utilizes a hierarchy to gradually challenge behavioral patterns each week. Thus, both the parent and child should expect homework assignments that align with the week's exposure goals and with the broader aim of reducing OCD symptom severity. To increase motivation for both parties, the therapist is encouraged to identify rewards associated with completing homework, even if their feeling skillful or their arguing less frequently is already providing strong reinforcement.

Taken together, family members can be integrated into the cognitive-behavioral treatment of complex OCD. To educate the family member effectively, the therapist should ensure that the parent understands the cognitive-behavioral model of OCD, how family accommodation functions similarly to a ritual in terms of negative reinforcement, and the effect of parental hostility and criticism on OCD symptoms and treatment outcome. Undoubtedly, there are many permutations regarding the family's involvement. Parents can attend the end of every session (as is often done with children and early adolescents) or attend only one or two sessions of psychoeducation and collaborative problem solving/role playing. Although no research has determined the optimal degree of family involvement, family-based treatments, including

limited adjunctive interventions (e.g., parent attends only two sessions), have shown a large effect on patients' overall global functioning and are associated with a significant reduction in (1) OCD severity, (2) depression symptoms, and (3) parental hostility and criticism.<sup>39,43,46</sup>

#### **Alik Widge, MD, PhD, Darin Dougherty, MD, MSc, and Emad Eskandar MD, MBA**

As happened in the case of Mr. R, the potential use of brain stimulation for any psychiatric condition should involve multispecialty consultation among psychiatry, neurology, and neurosurgery. DBS remains experimental for Tourette's syndrome but is FDA approved for OCD under a Humanitarian Device Exemption. Guidelines exist for DBS in TS and are very similar to those used for OCD.<sup>47</sup> The core criteria are the following:

- severe and life-impairing disease (Yale-Brown Obsessive Compulsive Scale > 30 for OCD; Yale Global Tic Severity Scale > 35 for tics)
- evidence of adequate trials of both multiple gold-standard pharmacologic agents and exposure/response-prevention therapy, for both the tic and obsessive-compulsive components of the symptoms
- for TS, age > 25, on the ground that the probability of spontaneous improvement with age is limited
- medical suitability to undergo neurosurgery
- absence of bipolar-spectrum disorder, as DBS at the most common OCD target can provoke hypomania to mania
- absence of active suicidality
- capacity to understand and consent to the extended and lifelong restrictions of living with and managing an implanted medical device
- adequate psychosocial support, particularly to manage patients' expectations regarding clinical outcomes and the possibility of non-response

There are various lesser inclusion criteria and relative contraindications; for a more complete discussion, see Mink and colleagues (2006)<sup>47</sup> and Widge and Dougherty (2015).<sup>48</sup>

The other complicating factor is targeting. For OCD, most centers target (with variation) a ventral striatal site that is referred to as anterior limb of the internal capsule, nucleus accumbens, or ventral capsule/ventral striatum. Some case-series evidence suggests that this same target can relieve tics in TS.<sup>49–51</sup> Other groups have reported good outcomes in the centromedian thalamus<sup>52,53</sup> and globus pallidus.<sup>54</sup> No head-to-head trials have been conducted.

As described in the case history, Mr. R likely would not have been a good candidate for DBS at any target. The presence of persistent suicidality is concerning, as the device itself creates a variety of means for self-injury. Further, patients with this level of behavioral dysregulation tend to have difficulty attending necessary appointments. This is doubly true when head banging is present as a common self-soothing

behavior. DBS devices have extension cables that tunnel subcutaneously in the neck and cross onto the anterior cranium. The patient's head-banging behavior could easily damage a connector and render the device nonfunctional, potentially even creating an increased risk of infection or inadvertent electrical discharge. The risks may be further exacerbated by the tics, depending on the severity. There are also case reports of injury to deep neck structures from violent tics,<sup>55</sup> suggesting that they could also damage an implanted neurostimulator.

A stereotactic lesion procedure may have been appropriate in this patient. Although the literature is less extensive than for DBS, case reports of capsulotomy, cingulotomy, and thalamotomy have demonstrated some success in TS.<sup>56</sup> These procedures, in TS, can often have complications, and there may be a divide between surgeries that relieve OCD and those that relieve tics.<sup>57–59</sup> In one case report, a combined capsulotomy and cingulotomy (limbic leucotomy) relieved self-injurious behavior, but with unknown effects on OCD or other TS symptoms.<sup>60</sup> A lesion does have the advantage of not requiring the extensive follow-up for DBS. For this specific patient, very detailed discussion would have been needed to ensure that he had the decisional capacity to request and undergo the procedure, with a full understanding of the limited probability for benefit. Active suicidality would still have been a contraindication during the time period covered by the case history.

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