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Compulsive Aspects of Impulse-Control Disorders

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Case vignette

Anna, a 32-year-old married woman, described herself as being “compulsive.” She reported a history, beginning in late adolescence, of uncontrollable shoplifting. She reports that over the course of a few months she became “obsessed” with stealing, thinking about it “all day.” She reports that her shoplifting started when she stole candy with friends and, over a period of a few months, developed into an almost daily ritual, which she did by herself. Anna reports that she currently shoplifts one to two times each week. She reports a “high” or a “rush” each time she steals. She primarily steals hygiene products, such as shampoo and soap. She usually steals multiple versions of the same item. Anna reports that she has boxes of the same shampoo and soap hidden in her closet. She steals shampoo and soap that she does not use and buys her preferred shampoo and soap at another store. When asked why she does not discard the shampoo, Anna reports that having these products “comforts” her. Anna’s shoplifting may consume 2 to 3 hours at a time. Anna also describes daily thoughts and urges to shoplift that preoccupy her for 3 to 4 hours each day. She may even leave work early, with projects unfinished, so that she can get to a store and steal something. In addition, she lies to her husband, telling him that she buys the items she steals. Anna reports feeling “compelled” to shoplift items.

Does Anna suffer from obsessive-compulsive disorder (OCD) or kleptomania? Is her behavior compulsive, impulsive, or both? How might conceptualization of her behavior influence the treatment of Anna’s behavior? Might Anna benefit most from a high dose of a selective serotonin reuptake inhibitor, or would a mood stabilizer or naltrexone be more effective options?

Impulsivity has been defined as a predisposition toward rapid, unplanned reactions to either internal or external stimuli without regard for negative consequences [1]. Although certain disorders are formally classified as impulse-control disorders (ICDs), impulsivity is a key element of many psychiatric disorders (eg, substance-use disorders, bipolar disorder, personality disorders, attention-deficit hyperactivity disorder).

The American Psychiatric Association defines compulsivity as the performance of repetitive behaviors with the goal of reducing or preventing anxiety or distress, not to provide pleasure or gratification [2]. Although OCD may be the most apparent disorder with compulsive features, compulsivity is often a prominent symptom in a number of psychiatric disorders (eg, substance-use disorders, personality disorders, schizophrenia) [3].

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Some have considered the domains of impulsivity and compulsivity to be diametrically opposed, but the relationship seems to be more intricate. Compulsivity and impulsivity may co-occur simultaneously in the same disorders or at different times within the same disorders, thereby complicating both the understanding and treatment of certain behaviors. ICDs, disorders classically characterized by impulsivity, have more recently been found to have features of compulsivity. A central aim of this article is to explore how compulsivity pertains to ICDs. In this process, the article also explores the relationship between OCD and ICDs.

Historically, one conceptualization of ICDs has been as part of an obsessive-compulsive spectrum [4]. This initial understanding of ICDs was based on available data on the clinical characteristics of these disorders, patterns of familial transmission, and responses to pharmacologic and psychosocial treatments. In the Diagnostic and Statistical Manual of Mental Disorders, edition four, text revised (DSM-IV-TR), the category of ICDs not elsewhere classified currently includes intermittent explosive disorder, kleptomania, pyromania, pathological gambling (PG), and trichotillomania. Other disorders have been proposed for inclusion based on perceived phenomenologic, clinical, and possibly biologic similarities: psychogenic excoriation (skin picking), compulsive buying, compulsive Internet use, and non-paraphilic compulsive sexual behavior. The extent to which these ICDs share clinical, genetic, phenomenologic, and biologic features is incompletely understood. Although ICDs are still relatively understudied, research in these disorders has increased recently. Data from these studies suggest a complex relationship between ICDs and OCD, heterogeneity within the ICDs, and a complicated overlap between impulsivity and compulsivity. Because rigorous research on most ICDs is limited, this article focuses largely on PG and trichotillomania, the two ICDs that have received the most research attention. It also reviews kleptomania, which, although less studied than other psychiatric disorders, is receiving increasing attention from clinicians and researchers. The article reviews the relationships between these ICDs and OCD, the compulsive aspects of the ICDs, and the clinical implications for assessing compulsivity in ICDs.

Pathological gambling

PG, characterized by persistent and recurrent maladaptive patterns of gambling behavior, is associated with impaired functioning, reduced quality of life, and high rates of bankruptcy, divorce, and incarceration [5]. PG usually begins in early adulthood, with males tending to start at an earlier age [6]. If left untreated, PG seems to be a chronic, recurring condition.

Compulsivity refers to repetitive behaviors performed according to certain rules or in a stereotyped fashion, and PG is associated with many features of compulsivity. PG is characterized by the repetitive behavior of gambling and impaired inhibition of the behavior. People who have PG often describe gambling as difficult to resist or control, and in this respect PG seems similar to the frequently excessive, unnecessary, and unwanted rituals of OCD. Additionally, individuals who have PG often have specific rituals associated with their gambling (eg, wearing certain clothes when gambling or gambling on particular slot machines). Another putative link between PG and OCD is the propensity of individuals who have PG to engage in excessive, possibly harmful behavior that leads to significant impairment in social or occupational functioning and causes personal distress [7]. As in OCD, the compulsive behavior of PG-gambling is often triggered by aversive or stressful stimuli [8]. Individuals who have PG often report that their urges to gamble are triggered by feelings of anxiety, sadness, or loneliness [9,10].

Studies consistently find that individuals who have PG have high rates of lifetime mood (60%–76%), anxiety (16%–40%), and other (23%) ICDs [5,11,12]. Rates of co-occurrence between PG and OCD have been largely inconsistent, however. For example, in samples of subjects

who have PG, rates of co-occurring OCD have ranged from 1% to 20% [5], with some, but not all, studies finding higher rates of OCD (approximately 2%) than found in the general population. The St. Louis Epidemiologic Catchment Area study, however, found no significant relationship between problem gambling and OCD (an odds ratio of 0.6 for OCD in problem gamblers as compared with non-gamblers) [13]. Although this study collected data in the 1980s, it is the only study published to date in which a community sample was assessed for DSM-based diagnoses for both OCD and PG.

Studies of PG among individuals who have OCD have reported little, if any, relationship between PG and OCD. Although studies of small OCD samples have reported PG rates ranging from 2.2% to 2.6% [14,15], a recently completed study of a large sample of subjects who had primary OCD (n = 293) found rates of current (0.3%) and lifetime (1.0%) PG [16] that were no greater than those in the general population (0.7–1.6%) [13]. These recent findings are consistent with those from a sample of more than 2000 individuals who had OCD in which both current and past rates of PG were lower than 1% [17]. Similarly, a family study of OCD probands did not find evidence of a significant relationship between OCD and PG or OCD and ICDs in general (with the exception of grooming and eating disorders) [18].

Family-history studies of subjects who have PG are limited. Black and colleagues [19] examined 17 subjects who had PG and 75 of their first-degree relatives. The study found that 1% of the first-degree relatives had OCD (similar to rates in the community), compared with none in the control group. Although the sample was small, the study used a control group as well as structured interviews for the subjects and first-degree relatives. As in the study of OCD probands, the family study of subjects who had PG and their relatives failed to find a link between PG and OCD.

Although on the surface PG shares many phenomenologic features with OCD, the majority of data suggests that the co-occurrence between these disorders is not elevated. Thus it seems that PG has multiple compulsive features but is not associated with high rates of OCD. One reason for this observation may involve limitations of categorical diagnoses. An alternate, not mutually exclusive explanation is that although compulsive features are observed in each disorder, the underlying biologies of the disorders differ. Another consideration is that aspects of compulsivity may differ between the disorders.

Assessing compulsivity in OCD and in PG and other ICDs might clarify the role of compulsivity in each disorder. Although many studies have assessed impulsivity and related constructs (eg, sensation seeking) in PG [5,20], relatively few have explored the construct of compulsivity in PG. In one study (the Padua Inventory), pathologic gamblers scored higher than normal controls on a measure of compulsivity [21]. A recent study attempting to understand the compulsive and impulsive dimensions of PG used the Padua Inventory to examine 38 subjects before and after 12 weeks of treatment with paroxetine [22]. The Padua Inventory measures obsessions and compulsions and contains four factors [23]:

1. Impaired control over mental activities, which assesses ruminations and exaggerated doubts
2. Fear of contamination
3. Checking
4. Impaired control over motor activities which measures urges and worries related to motor behavior, such as violent impulses

At baseline, the severity of PG symptoms was associated with features of both impulsivity and compulsivity (specifically, factors 1 and 4 of the Padua Inventory). During treatment, overall scores on measures of impulsivity and compulsivity diminished, with significant decreases

seen in factor 1 of the Padua Inventory and the impulsiveness subscales of the Eysenck Impulsivity Questionnaire [22]. This study suggests that compulsivity and impulsivity in PG interact in a complex fashion, and that measures of impulsivity and compulsivity have relevance with respect to treatment outcome. A corollary of this finding is that compulsivity or impulsivity (or specific aspects of each) might represent treatment targets for PG.

Although pathogenesis is arguably the most valid indicator of whether disorders are related, only a sparse amount of research has investigated possible neurobiologic correlates of PG, and the evidence suggests a different pathology from that seen in OCD. A functional MRI study of gambling urges in male pathologic gamblers suggests that PG has neural features (relatively decreased activation within cortical, basal ganglionic, and thalamic brain regions in subjects who have PG as compared with controls) distinct from the brain activation pattern observed in cue-provocation studies of OCD (relatively increased cortico-basal-ganglionic-thalamic activity) [24,25]. Whereas research on the neurobiology of PG is increasing, the neurobiologic relationship of PG to OCD remains to be qualified. More systematic studies of PG and OCD (eg, those that directly compare and contrast subjects using the same paradigm) are needed.

Treatment of pathological gambling

Originally it was suggested that PG, like OCD, may demonstrate a preferential response to serotonin reuptake inhibitors (SRIs). Data from double-blinded, randomized pharmacotherapy trials of SRIs in the treatment of PG, have been inconclusive, however [7], with medication showing a significant advantage over placebo in some but not in other trials of SRIs [26–29]. In addition, PG has demonstrated responses to opioid antagonists [30,31], drugs that have not been shown to be effective in treating OCD. The response of PG to pharmacologic treatment has been studied insufficiently to determine clearly the choice of treatment. The extent to which measures of compulsivity may be used to match specific treatments with specific individuals who have PG or used to assess or predict treatment outcome remains to be examined.

Cognitive and behavioral treatments that address the compulsive aspect of PG have shown early benefit [32]. Cognitive-behavioral therapy for PG, however, differs from the exposure and response prevention treatment used for OCD [33]. Cognitive therapy focuses on changing the patient's beliefs regarding perceived control over randomly determined events. Cognitive therapy helps the patient understand that the laws of probability, not ritualistic behavior, control the outcome of gambling. In one study, individual cognitive therapy resulted in reduced gambling frequency and increased perceived self-control over gambling when compared with wait-list controls [34]. A second study that included relapse prevention also produced improvement in gambling symptoms compared with wait-list controls [35].

Cognitive-behavioral therapy has also been used to treat PG. The behavioral element addresses substituting alternative behaviors for gambling. One randomized trial compared four types of treatment: (1) individual stimulus control and in vivo exposure with response prevention, (2) group cognitive restructuring, (3) a combination of methods 1 and 2, and (4) a wait-list control. At 12 months, rates of abstinence or minimal gambling were higher in the individual treatment (69%) group than in the group cognitive restructuring (38%) and the combined treatment (38%) groups [36]. An independent, controlled trial, based upon cognitive-behavior therapies used in the treatment of substance-use disorders and including relapse-prevention strategies, is currently under way; initial results suggest the efficacy of manually driven cognitive-behavioral therapy [37].

One study of a brief intervention in the form of a workbook (which included cognitive-behavioral and motivational enhancement techniques) was compared with the use of the workbook plus one clinician interview [38]. Both groups reported significant reductions in gambling at a 6-month follow-up. Similarly, a separate study assigned gamblers to use of a

workbook, use of a workbook plus a telephone motivational enhancement intervention, or a wait list. Compared with those using the workbook alone, the gamblers assigned to the motivational intervention plus workbook reduced gambling throughout a 2-year follow-up period [39].

Two studies have also tested aversion therapy and imaginal desensitization in randomized designs. In the first study, both treatments resulted in improvement in a small sample of patients [40]. In the second study, 120 pathologic gamblers were assigned randomly to aversion therapy, imaginal desensitization, in vivo desensitization, or imaginal relaxation. Participants receiving imaginal desensitization reported better outcomes at 1 month and up to 9 years later [41].

Trichotillomania

Trichotillomania has been defined as repetitive, intentional hair pulling that causes noticeable hair loss and results in clinically significant distress or functional impairment [2]. As discussed elsewhere in this issue, trichotillomania seems to be relatively common, with an estimated prevalence between 1% and 3% [42]. The mean age at onset for trichotillomania is approximately 13 years [43].

The repetitive motor behavior of hair pulling with perceived diminished control bears a striking resemblance to OCD. In contrast to OCD, in which compulsions occur in a variety of situations, individuals who have trichotillomania tend to pull most often when engaged in sedentary activities [44]. Although the hair pulling in trichotillomania decreases anxiety, as do compulsions in OCD, it may also produce feelings of pleasure, whereas OCD compulsions typically do not.

Trichotillomania traditionally has been considered a disorder predominantly affecting females [45] and frequently is associated with depression (39%–65%), generalized anxiety disorder (27%–32%), and substance abuse (15%–20%). In particular, rates of co-occurring OCD are significantly higher (13%–27%) [43] than found in the community (1%–3%) [46], and this comorbidity raises the possibility of an underlying common neurobiologic pathway for the compulsivity seen in these two disorders. Trichotillomania is not associated with higher rates of obsessive-compulsive symptoms, with scores generally in the normal range [44].

Rates of trichotillomania among individuals who have OCD are inconsistent across studies. Three studies of small samples of OCD subjects have reported rates ranging from 4.6% to 7.1% [14,15,47]. One larger study of 293 subjects who had OCD reported lifetime and current rates of trichotillomania of 1.4% and 1.0%, respectively [16]. As with PG, the question remains whether examining the domain of compulsivity across these disorders would provide insight into possible pathophysiology.

A relationship between trichotillomania and OCD is supported partially by findings that OCD is common in relatives of subjects who have trichotillomania. Although family-history studies of trichotillomania are limited, one study has suggested a familial relationship with OCD. The study involved 22 subjects who had trichotillomania and 102 first-degree relatives. When compared with a control group ($n = 33$, with 182 first-degree relatives), significantly more relatives of the trichotillomania probands had OCD (2.9%) compared with the control group [48]. A family study of OCD probands found a higher proportion of case subjects than control subjects had trichotillomania (4% versus 1%), although the difference was not statistically significant given the sample size [18].

Treatment of trichotillomania

Treatments evaluated for trichotillomania include pharmacologic and behavioral interventions. It is well established that the pharmacologic first-line treatment for OCD is an SRI (eg, clomipramine, fluvoxamine, or fluoxetine). The data regarding the efficacies of SRIs for trichotillomania are less convincing, however. One study compared clomipramine with desipramine in a 10-week double-blinded, cross-over design (5 weeks for each agent after 2 weeks of single-blind placebo lead-in) [49]. Twelve of 13 subjects had significant improvement when receiving clomipramine. Although SRIs are effective for OCD, these medications have demonstrated mixed results in three randomized trials of trichotillomania [50–52]. In addition, individuals who have trichotillomania and who are successfully treated with an SRI tend to have higher rates of symptom relapse than do SRI-treated people who have OCD [51].

Other pharmacologic agents that have shown benefit for trichotillomania have not been effective for OCD. This lack of efficacy raises questions about the overlap between these disorders. Christenson and colleagues [51] compared the opioid antagonist naltrexone with placebo in a 6-week randomized, double-blinded, parallel study. Significant improvement was noted for the naltrexone group on one measure of trichotillomania symptoms. In an open-label study of lithium, 8 of 10 subjects reported decreases in pulling frequency, amount of hair pulled, and extent of hair loss [53]. Lithium has often been beneficial in treating individuals who have disorders characterized by impaired impulse control [54]. The positive results from the open-label trial of lithium [53] raise the possibility that impulsive rather than compulsive features represent an important treatment target in some individuals who trichotillomania. Direct testing of this hypothesis is needed before this claim can be verified.

Both OCD and trichotillomania respond to behavioral interventions; however, the modes of behavioral treatment differ quite substantially. Azrin and colleagues [55] randomly assigned 34 subjects to habit-reversal therapy or negative practice (in which subjects were instructed to stand in front of a mirror and act out motions of hair-pulling without actually pulling). Habit reversal reduced hair pulling by more than 90% for 4 months, compared with a 52% to 68% reduction for negative practice at 3 months. No control group was included, and therefore time and therapist attention could not be assessed.

A recent study examined 25 subjects randomized to 12 weeks (10 sessions) of either acceptance and commitment therapy/habit reversal or wait list [56]. Subjects assigned to the therapy experienced significant reductions in hair-pulling severity and impairment compared to those assigned to the wait list, and improvement was maintained at 3-month follow-up.

Kleptomania

The core features of kleptomania include (1) a recurrent failure to resist an impulse to steal unneeded objects; (2) an increasing sense of tension before committing the theft; (3) an experience of pleasure, gratification, or release at the time of committing the theft; and (4) stealing not performed out of anger, vengeance, or because of psychosis [2].

Like OCD, kleptomania usually appears first during late adolescence or early adulthood [57]. The course is generally chronic with waxing and waning of symptoms. Unlike OCD, however, women are two times more likely than men to suffer from kleptomania [57]. In one study, all participants reported increased urges to steal when trying to stop stealing [57]. The diminished ability to stop often leads to feelings of shame and guilt, reported by most subjects (77.3%) [57].

Although people who have kleptomania often steal various items from multiple places, most steal from stores. In one study, 68.2% of patients reported that the value of stolen items had

increased over time [57]. Many (64%–87%) have been apprehended at some time because of their behavior [58], and 15% to 23% report having been jailed [57]. Although most of the patients who were apprehended reported that their urges to steal were diminished after the apprehension, the symptom remission generally lasted only for a few days or weeks [58]. Together, these findings demonstrate a continued engagement in the problematic behavior despite adverse consequences.

This repetitive behavior seen in kleptomania is suggestive of a compulsion, as in the case vignette that opened this article. In addition, most individuals who have kleptomania (63%) hoard particular items that they steal [57]. Personality examinations of individuals who have kleptomania suggest, however, that they are generally sensation seeking [59] and impulsive [60] and thereby differ from individuals who have OCD, who are generally harm avoidant with a compulsive risk-averse endpoint to their behaviors [4]. Unlike individuals who have OCD, people who have kleptomania may report an urge or craving before engaging in the stealing and a hedonic quality during the performance of the thefts [7].

High rates of other psychiatric disorders have been found in patients who have kleptomania. Rates of lifetime comorbid affective disorders range from 59% [61] to 100% [58]. Studies have also found high lifetime rates of comorbid anxiety disorders (60% to 80%) [58,62] and substance-use disorders (23% to 50%) [58,61].

The extent to which OCD and kleptomania co-occur is not well understood. Rates of co-occurring OCD in samples of individuals who have kleptomania have ranged from 6.5% [61] to 60% [63]. Conversely, rates of kleptomania in OCD samples suggest a higher rate of co-occurrence than found in the community (2.2%–5.9%) [14,15]. A recent study of 293 subjects who had OCD reported current and lifetime rates of kleptomania (0.3% and 1.0%) [16] that were lower than rates found in a population of general psychiatric inpatients (7.8% and 9.3%, respectively) [64]. Large psychiatric epidemiologic studies have typically excluded measures of kleptomania, thus limiting the available knowledge regarding its prevalence and patterns of co-occurrence with other psychiatric disorders.

A family history study compared 31 individuals who had kleptomania and 152 of their first-degree relatives with 35 control subjects and 118 of their first-degree relatives [61]. The study found that 0.7% of the relatives of the kleptomania proband suffered from OCD compared with 0% in families of the controls.

Treatment of kleptomania

Only case reports, two small case series, and one open-label study of pharmacotherapy have been performed for kleptomania. Various medications have been studied in case reports or case series, and several have been found effective: fluoxetine, nortriptyline, trazodone, clonazepam, valproate, lithium, fluvoxamine, paroxetine, and topiramate [65]. Unlike the treatment of OCD, there does not seem to be a preferential response of kleptomania to serotonergic medications. The only formal trial of medication for kleptomania involved 10 subjects in a 12-week, open-label study of naltrexone. At a mean dose of 150 mg/d, medication resulted in a significant decline in the intensity of urges to steal, thoughts about stealing, and stealing behavior [66].

Although multiple types of psychotherapies have been described in the treatment of kleptomania, no controlled trials exist in the literature. Forms of psychotherapy described in case reports as demonstrating success include psychoanalytic, insight-oriented, and behavioral techniques [58,67]. Because no controlled trials of therapy for kleptomania have been published, the efficacies of these interventions are difficult to evaluate, but the range of psychosocial interventions, as with medications, suggests that kleptomania is heterogeneous.

Summary

As seen in the introductory case vignette, ICDs are characterized by repetitive behaviors and impaired inhibition of these behaviors. The difficult-to-control behaviors characteristic of ICDs suggest a similarity to the frequently excessive, unnecessary, and unwanted rituals of OCD. There are, however, differences between ICDs and OCD (eg, the urge or craving state seen in ICDs, the hedonic quality during the performance of the ICD behavior, and the sensation-seeking personality type often seen in individuals who have ICD) [7]. Despite the differences between ICDs and OCD, features of compulsivity have been observed in association with ICDs, and preliminary data suggest that features of compulsivity, as well as impulsivity, might represent important treatment targets in some ICDs.

Future directions

Because research is limited, and findings are varied, it seems premature to identify ICDs too closely with OCD. The extent to which there exist specific ICDs or subtypes of ICD that are more closely associated with OCD remains to be investigated more systematically. In addition, the construct of compulsivity as related to ICDs and OCD warrants additional investigation to identify the similarities and differences and to examine the implications for prevention and treatment strategies. For example, given that the treatment of ICDs with SRIs has demonstrated mixed results, future investigations are needed to determine whether specific subgroups (eg, individuals who have PG with specific features of compulsivity or impulsivity) respond better or worse to specific treatments (eg, SRIs). Similarly, specific aspects of compulsivity might represent targets for behavioral interventions for ICDs. Future biologic studies of ICDs (eg, genetic, neuroimaging) should also include measures of compulsivity to understand better its relevance to the OC spectrum disorders.

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