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.

Kleptomania treated with naltrexone in a patient with intellectual disability

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Kleptomania occurs in 0.3%–0.6% of the population and is defined as "the recurrent failure to resist impulses to steal items even though the items are not needed for personal use or for their monetary value."[1] It is currently classified in the disruptive, impulse control, and conduct disorder section of the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition* (DSM-5). However, many authors point to the similarities between kleptomania and broadly defined addictive behaviour, suggesting common pathophysiological pathways and shared sensitivity to antiaddictive drugs.[2]

We report the case of a 32-year-old woman referred to our department from a residential institution, where she had been living for 15 years, for severe aggressive behaviour and repeated assaults toward staff members and fellow residents.

The patient had a history of maltreatment, abuse and emotional deprivation in early childhood. At the age of 6 years, she received a diagnosis of intellectual disability (ID) and was referred to a specialized school and to community child psychiatric follow-up.

Ten years later, she developed oppositional and defiant behaviours, which rapidly evolved into regular and indiscriminate violent assaults. This overwhelming condition led to her first psychiatric hospitalization. Her IQ at that time, assessed using the Wechsler Intelligence Scale (WAIS-R), was 53. Biological and genetic workup did not indicate a known metabolic disease, genetic disorder, or chromosome abnormality. Institutional therapy and educative measures led to a progressive improvement of her global behaviour, allowing her placement in a specialized residential institution.

At the index hospitalization, the patient had poor speech. She denied staff-reported assaults and justified her aggressive behaviour toward fellow residents as a legitimate defence. We noted no mood disturbances or guilt. The patient did not report hallucinations, nor did she express delusional ideas. In the ensuing days, The nursing staff reported up to 5 agitation or aggression episodes daily toward patients and staff members, necessitating recourse to seclusion and restraint measures. Antipsychotic treatment was initiated, and doses were increased up to 600 mg of chlorpromazine and 60 mg of haloperidol. Lithium was added and titrated up to 800 mg/d.

Disruptive episodes persisted and appeared to happen following attempted appropriation of other patients' objects. The appropriation occurred according to the patterns of kleptomania described in DSM-5. The patient reported an increased sense of tension immediately before committing the theft and relief after the theft was committed. She scored 32/40 on the Yale–Brown Obsessive–Compulsive Scale Modified for Kleptomania (K-YBOCS).

Naltrexone was then introduced and titrated up to 100 mg/d (50 mg twice daily). No clinical or biological adverse effects were reported. The clinical evolution was favourable, with a substantial reduction of stealing and aggression episodes. Two months later, the patient's K-YBOCS score was 5/40. Her haloperidol dose was decreased to 10 mg/d; chlorpromazine was gradually reduced and stopped. Lithium was maintained at 800 mg/d. The patient accessed social-educational care before returning to her residential institution.

A careful characterization of psychiatric comorbidities in patients with ID is of utmost importance. Estimates suggest a high prevalence (up to 40%) of mental health issues in individuals with ID.[3] Overshadowing of psychiatric disorders by the cognitive condition leads to inappropriate therapeutic strategies and worsening of the mental, physical and social conditions of many patients.[4] In our patient's case, attributing the behavioural disturbances to the cognitive condition led to antipsychotic drug escalation as well as restrictive measures of seclusion and restraints. Furthermore, severe and persistent aggressive behaviour could have compromised the patient's social integration and worsened the global prognosis.

Naltrexone is a competitive antagonist of μ-opioid receptors in the central nervous system approved for the treatment of opioid and alcohol-use disorders. A double-blind placebo-controlled study of 25 patients[5] showed that naltrexone (50–150 mg/d) led to a significant reduction in behavioural disorders related to kleptomania. A recent meta-analysis of 6 randomized placebo-controlled studies assessing the effectiveness of naltrexone on broadly defined behavioural addictions, including gambling disorder, trichotillomania and kleptomania, also supports the beneficial effect of naltrexone.[6] Family history data suggest that individuals with kleptomania are more responsive to naltrexone if they have first-degree relatives with alcohol-use disorder.[7] Our patient's mother was alcohol-dependent. The remarkable effectiveness of naltrexone, in this case, lends support to a shared substrate between alcohol-use disorder and kleptomania, potentially targeted by naltrexone.

There is evidence that the endogenous opioid system may be critical to the maintenance of aggressive behaviour, particularly self-directed

aggression.[8] Aggression or self-directed aggression is associated with opioid release, which in turn increases the pain threshold and dopamine release, reinforcing self-destructive behaviour.[9] By antagonizing opioid receptors, naltrexone hinders opioid activity and breaks the reinforcing cycle.[9] In our patient's case, the reduction of aggressive behaviour could be attributed either to the kleptomania remission or to a more direct effect. So far, limited and controversial clinical evidence supports prescribing naltrexone for the treatment of aggression in patients with ID.

In the present case, the remarkable evolution with naltrexone therapy highlights the importance of careful screening for comorbid impulsive–compulsive behaviours in individuals with ID and points to the opioid system as potential therapeutic leverage.

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