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Intermittent Explosive Disorder

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Abstract:
Intermittent explosive disorder is characterized by recurrent episodes of inability to resist aggressive impulses, resulting in damage to other people or other people's property. In the history of DSM, it first appeared as 'isolated explosive disorder' in DSM-III. However, with some changes in the diagnostic criteria, it is included under the title of 'destructive, impulse control and conduct disorders' in DSM-5. Although previous studies have shown it is a rare disorder, recent epidemiological studies suggest it may be more common than thought. For this reason, it is essential to master the clinical features and learn the treatment methods in order to recognize people with this disorder and apply the appropriate treatment.

Keywords: Intermittent Explosive Disorder, Impulse Control, Conduct Disorders

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Introduction

Intermittent explosive disorder (IED) is an impulse control disorder characterized by recurrent episodes of inability to resist impulses of aggression, resulting in severe aggression or destruction of other people's property.[1] Diagnoses related to impulsive aggression have been included in the Diagnostic and Statistical Manual of Mental Disorders (DSM) of the American Psychiatric Association since its first edition.[2] In DSM 5, IED is classified under "Disruptive, Impulse Control, and Conduct Disorders" together with pyromania and kleptomania.[3]

The DSM has argued that in order for patients with anger outbursts or impulsive aggression to be diagnosed with IED, other psychiatric disorders that can be shown to cause these behaviors should be excluded. Anger, violence, and aggression are also seen in the presence of many other psychiatric conditions, such as personality disorders, psychotic disorders, substance use disorders, and conduct disorders. IED is included in this section precisely to emphasize that impulsivity is the main component and that uncontrollable aggressive and impulsive episodes are separate from those observed in these disorders.[4] The degree of aggression observed in patients is disproportionate to the stressor that elicited the presenting picture.

Considering that IED can lead to serious public health problems, that most people with this diagnosis do not seek treatment, and that this disorder is a common behavioral disorder that can be treated with specific pharmacological or psychological interventions, the importance of not overlooking this diagnosis in outpatients and inpatients stands out.[2,4]

History

IED was first named by French psychiatrist Jean-Étienne Dominique Esquirol in 1838 under the title "impulsive monomania," a "partial insanity" related to senseless impulsive attempts.[5] When we look at the history of IED, the only adult psychiatric diagnosis in which pathological aggression is primary, it is seen that it was called "passive-aggressive personality-aggressive type" in DSM I and "explosive personality" in DSM II.[3,6] In the International System of Classification of Diseases 9th Edition Clinical Modification (ICD-9-CM), and for the first time in the history of DSM, it was officially included in DSM-III together with the diagnosis of "Isolated Explosive Disorder" within "Axis I Disorders". Moreover, in this classification, the presence of an organic cause was not considered an exclusion criterion for the diagnosis of IED. In DSM-III-R, on the other hand, "isolated explosive disorder" was removed from the classification because it was artificial, and IED retained its place in the classification.[1,4]

In DSM-IV, the diagnostic criteria for APD were generally preserved.[7,8] However, "Criterion C" (absence of impulsivity or aggression between episodes) was removed as a diagnostic criterion because it makes it difficult to diagnose people who exhibit violent and aggressive attitudes between episodes.[8] Furthermore, an exclusion criterion has been added, stating that the symptoms of a person losing their control cannot be better explained by other psychiatric disorders such as schizophrenia, antisocial personality disorder, borderline personality disorder, attention deficit hyperactivity disorder, conduct disorder, also that it should not be directly attributed to the physiological effects of a substance or a general medical condition, simultaneously, if the aggression experienced by the individual aims at gaining material benefit, self-defense, obtaining social control, emphasizing political preference, or involves a gang-related attempt, then a diagnosis of IED should not be made.[8,9] However, it is noteworthy that there is a lack of definition of how often and in what period such aggressive behaviors occur.[4]

The diagnostic criteria have not changed in DSM-IV-TR.[9] This diagnosis, which was included in the category of 'impulse control disorders not elsewhere classified' in DSM-IV and DSM-IV-TR, was included in 'disruptive, impulse control and conduct disorders' with the removal of the category of 'impulse control disorders not elsewhere

classified' in DSM-5.[8,10] In DSM-IV-TR, it is stated that the patient generally does not have symptoms of generalized impulsivity or aggression except during periods of active illness. In contrast, in DSM-5, it is stated that milder episodes may be detected between severe episodes.[9,11] DSM-IV criteria focused on physical aggression, but DSM-5 allows the diagnosis of IED in the presence of frequent verbal aggression with or without physical aggression.[6] According to DSM-5, IED is defined as a failure to resist aggressive impulses resulting in repeated acts of verbal or physical aggression. The inclusion of verbal aggression represents a significant change from previous definitions of IED in the DSM.[6]

After the diagnostic criteria were updated in DSM-5, the difference was not as high as thought in the studies evaluating the difference between the frequency of IED based on DSM-IV and DSM-5 diagnostic criteria. This situation may be associated with the DSM-5 diagnostic criteria being expanded to include verbal aggression yet being more restrictive in duration/frequency compared to the DSM-IV diagnostic criteria.[4]

Epidemiology

Although IED is stated to be a rare disorder in DSM-IV-TR, some recent epidemiological studies suggest that this diagnosis may be seen more frequently than thought.[9,12,13] The lack of a comprehensive study on this subject has prevented a definite figure from being put forward. The frequently changing diagnostic criteria for IED is the most significant factor preventing the comparison of studies. In one of the first studies conducted after IED was included as a separate diagnosis in DSM-III, 20 of 830 patients (2.4%) admitted to a university clinic over two years were diagnosed with IED.[14] IED was the primary diagnosis in only 14 (%1.7) of these patients. While evaluating the results of the study, the researchers suggested that this diagnosis, which was known to be quite rare until then, was more common and suggested that the diagnostic criteria should be evaluated individually for this purpose. Felthous et al. conducted a study on 443 male volunteers with complaints of violence and aggression and found that only 15 (3.4%) met the diagnosis of IED according to DSM-III.[15] It has been reported that 80% of the patients could not be diagnosed because of the criterion in DSM-III, which prevents patients from being diagnosed with IED if impulsive and aggressive behaviors are present between attacks. Various studies have also been conducted according to the DSM-IV diagnostic criteria, which are expected to cover a more comprehensive patient group than other versions. In a field study conducted on 253 individuals in Baltimore, the lifetime prevalence of IED, according to DSM-IV, was found to be 3.95%. In the same sample group, when IED was diagnosed according to the research criteria developed by Coccaro, in which personality disorders were not considered as an exclusion criterion, the lifetime prevalence increased by 30% to 5.14%.[16]

In a community-based survey of 9282 individuals (National Comorbidity Survey-Replication (NCS-R)), the lifetime prevalence for IED was found to be 5.4% and 7.3%, respectively, depending on the limited and comprehensive diagnostic criteria used. When the 12-month prevalence was analyzed, it was determined as 2.7% and 3.9%.[12] In a study conducted in Iraq with 4332 individuals, the lifetime prevalence of IED was 1.7%, and the 12-month prevalence was 1.5% according to DSM-IV diagnostic criteria.[17]

When other studies conducted in Turkey are examined, in an epidemiological study conducted in Sivas, IED was determined at a rate of 0.059% in a study group of 1184 cases.[18] In another clinic-based study conducted in Adana, lifelong IED was found as an additional diagnosis in 15 (14.6%) of 103 adult patients who were hospitalized in the psychiatry service of a university hospital within six months.[19] In another study conducted in Adana in 2015 with 406 patients who applied to outpatient clinic services for the first time in 6 months, the lifetime and 12-month prevalence of IED according to DSM-5 were 16.7% and 11.3%, respectively.[4]

In summary, it was stated that these differences found between the studies could be attributed to the patient groups in which the studies were conducted,

the fact that the disease could not be interrogated as much and systematically as it should be, or cultural factors; moreover, these differences could also be explained by the fact that there is no diagnostic tool that can be accepted as the 'gold standard' for APD and that the preferred research methods were not designed in a way to recognize social diversity.[2,17,20]

Etiopathogenesis

Hypotheses about the etiology of impulsive aggression and IED have been a subject of psychiatry since its inception. Since the second half of the 19th century, explanations about cases showing episodic impulsive aggression have been under two main headings. The first of these states that impulsive aggression occurs due to childhood traumas or adverse early childhood experiences that may affect the development of traits such as willpower (self-control), planning, delaying pleasure, and the power to withstand inhibition, which is extremely important in the suppression of impulsive aggression (self-prevention). The second view is that impulsive aggression is associated with imbalances or variations in the brain's functioning, mediating behavioral arousal and behavioral inhibition. This view is gaining strength, with many results supporting it in research conducted over the past two decades.[21]

Early experiences of "good enough mothering," which encourage a stage-appropriate delay of gratification, and the potential to imitate and identify with the mother are essential for normal development. Too much or too little frustration and too much or too little satisfaction can impair the normal development of the ability to anticipate frustration and delay satisfaction, which is thought to lead to impulsive aggression.[22]

A second line of work, which has yielded numerous positive findings over the last 20 years, states that impulsive aggression may result from variations or imbalances in brain mechanisms mediating behavioral arousal and behavioral inhibition. A rapidly growing body of evidence has shown that impulsive aggression may be associated with defects in the brain's serotonergic system, which inhibits motor activity.[23,24] Animal studies suggest that serotonergic neurons are involved in behavioral inhibition, thus providing impetus to explore the role of serotonin in human impulsivity. Studies on neuropsychiatric patients with localized brain lesions have shown that some bilateral lesions in the PFC may be associated with a chronic pattern of particularly impulsive aggressive behavior. Neurological studies suggest that PFC regions associated with impulsive aggression syndromes are involved in the processing of emotional information and inhibition of motor responsiveness, both of which are impaired in impulsive aggressive patients.

Interictal episodes of aggression may also occur in some people with epilepsy. In a quantitative MRI study examining such episodes among people with temporal lobe epilepsy (TLE), three groups (24 TLE patients with aggressive behaviour, 24 TLE patients without such behaviour and 35 non-patient controls) were compared and aggressive behaviour was associated with reduced frontal neocortical grey matter.[25]

Clinical observation and family history data indicate that IED is a familial disorder. In a study conducted by McElroy et al. in 1998, it was reported that approximately one-third of the first-degree relatives of IED patients had the same diagnosis. In another family study, the risk of disease was found to be 26% in relatives of IED patients, whereas this rate was 8% in relatives of the control group.[26] Twin studies have confirmed the hypothesis that both impulsivity and aggression are under significant genetic influence, but twin studies of IED itself have not been conducted.[27,28] The genetic influence of these two traits varies between 28% and 47%, and unshared environmental effects account for most of the remaining variance.

Very few neuroimaging studies have examined impulsive aggression or IED. Impulsive aggressive behavior is defined as an imbalance between excessive, aggressive impulses originating from limbic brain structures such as the amygdala and inadequate control of these impulses by cortical structures such as the orbitofrontal cortex

and anterior cingulate cortex.[29] Coccaro, et al.'s functional magnetic resonance imaging study of 20 unmedicated patients (10 patients with IED and 10 control group patients) who were shown emotionally salient facial expressions, supports this hypothesis.[30] Compared to controls, patients with IED have been shown to have increased activation of the amygdala and decreased activation of the orbitofrontal cortex towards faces expressing anger. In another perspective, unlike the healthy control group, aggressive individuals responded to angry facial expressions but failed to establish an amygdala-orbitofrontal cortex connection. This study found dysfunction in the amygdala-orbitofrontal cortex connection in subjects with a history of impulsive aggressive behavior when faced with a signal that can be considered socially threatening (angry facial expressions). In addition, it revealed the link between aggression and the problem in the functioning of the corticolimbic network. In an earlier study using fluorodeoxyglucose positron emission tomography (FDG-PET), Siever et al. found blunted glucose utilization responses to serotonin stimulation in the orbitofrontal cortex (an area associated with impulsive aggression) of IED patients. A similar finding has been reported in impulsive-aggressive individuals' anterior cingulate and anteromedial orbit cortex.[31]

Clinical Features

IED is one of several impulse control disorders, manifested by an inability to control emotions and behavior, resulting in behavior that violates social norms and the rights of others.[2] Patients with IED are periodically unable to restrain their impulses, resulting in verbal or physical aggression.[32] The aggressive behavior is unplanned and out of proportion to the provocation. It causes distress or psychosocial impairment in patients. In addition, cognitive impairment and self-harm (e.g., suicide attempts) may occur.[20]

Psychotic experiences (delusions and hallucinations) are observed in many mental disorders, including IED.[33] In a cross-country study conducted in 18 countries, individuals with a lifetime diagnosis of intermittent explosive disorder were identified, and it was found that psychotic experiences occurred in 15% of them.[34] It has been observed that psychotic experiences typically occur after the onset of IED rather than before or at the same time as its onset.

1. Aggressive behavior: The violent, impulsive outbursts that characterize IED are usually preceded by a brief prodrome period. The first impulse to attack typically occurs in response to a minor provocation but can also occur spontaneously.[15] The urge is often accompanied by a rapidly increasing tension or arousal.[35] Somatic symptoms such as paresthesia, tremors, palpitations, and chest tightness may be accompanied by irritability, anger, increased energy, racing thoughts, difficulty in communicating, and impaired cognitive functioning.[36] Little or no thought is given to the consequences of this sudden violent behavior.

Impulsive, aggressive behavior consists of one or more of the following:

- Physical hitting of other people or animals - Ranging from shoving or slapping to fist fighting, using a weapon against someone, injuring someone badly enough to require medical attention, or even murder.
- Verbal outbursts, arguments, and threats to physically attack others - It often occurs during tantrums or heated arguments, characterized by shouting and loss of control.
- Physical aggression towards objects - Ranges from throwing things, slamming doors, kicking objects to breaking objects, or injuring an animal. It often occurs during tantrums or heated arguments, characterized by shouting and loss of control.

The outburst of anger typically lasts less than 30 minutes and is often followed immediately by a feeling of relief.[2] The feelings that follow often include fatigue, dysphoria, regret, and shame.

In IED, the intensity and frequency of aggressive behavior are often inversely proportional:[2,37]

- Low-intensity (non-destructive and harmless) aggression is relatively common.
- High-intensity (destructive or injurious) aggression rarely occurs.

A retrospective study of individuals with IED (n = 380) found that approximately 70% of individuals showed both forms of aggression (low intensity/high frequency and high intensity/low frequency).[38] Approximately 20% showed only low-intensity/high-frequency impulsive aggression, and 10% showed only high-intensity/low-frequency aggression.

In patients with intermittent explosive behavior in the form of physically attacking and injuring other individuals or animals or damaging property, high-intensity aggressive behavior may occur three times a year. In contrast, verbal aggression and threats or physical aggression towards property, animals, or other individuals that does not result in physical damage or injury (i.e., low-intensity aggression) may occur on average twice a week for at least three months.[2,12,32]

A nationally representative survey in the United States found that among 463 people with a lifetime history of IED, the average number of physical (high and low intensity) and verbal outbursts was 28 per year.[12] Symptoms of general aggression may occur between explosive episodes.[35] Patients with IED may experience chronic anger on an ongoing basis as well as subthreshold events during these periods when they can resist aggressive impulses.

2. Cognitive impairment: Cognitive impairments may occur in IED. One study found that impulsive, aggressive male prisoners scored significantly lower on tests of attention, concentration, memory, and intelligence compared to a non-aggressive control group matched for age, race, and education.[39]

IED is also associated with abnormal processing of social and emotional information. Those with IED were more likely to misinterpret others' behavior, make hostile attributions about their intentions, and respond to this with negative emotions, having a more positive view of aggressive behavior compared to each control group. In addition, strategic emotional intelligence (the ability to understand what the other person is feeling and to use this information) is lower in individuals with IEDs compared to healthy controls; this deficit is likely related to abnormalities in social-emotional information processing.[40]

Clinical Prognosis

Retrospective studies show that IED is persistent and chronic. One study found that the average duration of the disease ranged from about 12 years to almost the entire life span.[2]

Treatments

A significant portion of the treatment approaches preferred in the treatment of IED was determined by considering the approaches preferred in the treatment of impulsivity or aggression observed in other psychiatric disorders. The main goal in treating IED is the complete resolution of symptoms or the creation of a picture in which only a few symptoms are mild. In cases where regression of symptoms cannot be achieved, it is aimed to ensure the safety of the person and the immediate environment and to minimize the number, frequency, and severity of attacks as much as possible. A 50% decrease in "Modified Open Aggression Scale" scores after treatment is considered as response to treatment.[20]

In the last decade, double-blind, placebo-controlled clinical trials have been conducted in patients with impulsive aggression or IED (research criteria). Early studies reported a decrease in impulsive aggressive behavior in IED patients with comorbid personality disorder, especially with fluoxetine treatment.[41]

Clinicians are advised to treat IED with CBT in addition to pharmacotherapy, based on randomized clinical trials showing the limited benefit of medication alone.[41,42]

1. Pharmacotherapy

a. Primary treatment approach

Selective serotonin reuptake inhibitors (SSRIs) are recommended as first-line pharmacotherapy for IED in terms of demonstrated efficacy, tolerability, and ease of use. Fluoxetine is frequently preferred because it is the most commonly studied agent. However, other SSRIs are also suitable alternatives. Most randomized trials recommend 6 to 12 weeks of treatment before determining whether the drug is beneficial, depending on the duration of treatment.[41,43,44] Approximately 66% of patients are expected to respond to this treatment protocol.

The usual starting dose of fluoxetine is 20 mg once daily. An effect is expected within two to four weeks. Patients who do not respond to treatment may receive additional dose escalations of 10 to 20 mg daily every two to four weeks as tolerated until an effective dose is reached. The maximum dose is 60 mg per day.

One of the reasons why fluoxetine is preferred in the treatment of IED is a 12-week randomized trial comparing fluoxetine with a placebo in 100 patients with IED and a comorbid personality disorder (obsessive-compulsive, paranoid, or borderline).[41,43] Significantly more moderate to major improvements were observed in patients receiving fluoxetine compared to placebo (66% versus 29%). Other studies also indirectly support the use of fluoxetine to treat IED. A meta-analysis of randomized trials (3992 patients treated for various psychiatric disorders) found that significantly less impulsive aggressive behavior occurred in patients receiving fluoxetine than placebo (0.2% versus 0.7%).[45] In other randomized controlled trials, it was found that fluoxetine significantly reduced impulsive and aggressive behavior in patients with borderline personality disorder and in patients with a history of causing domestic violence and alcohol use disorder.[46,47]

In an open-ended study with 8 participants diagnosed with IED or cluster B personality disorder, it was observed that participants showing impulsive aggression decreased in aggression and irritability with the use of citalopram.[48] In another case series study, a positive response to antidepressant monotherapy using sertraline or venlafaxine was observed in 5 of 10 patients with IED, and a positive response to valproic acid or lithium was observed in 7 of 10 patients.[49]

b. Approach in the treatment-resistant patient

In IED, some patients do not respond to an SSRI within 6 to 12 weeks of starting the medication. (Response is defined as a significant improvement in the number, intensity, and frequency of symptoms, as well as stabilizing the safety of the patient and those around them.) For these treatment-resistant patients, it is recommended to taper the SSRI for one to two weeks, discontinue it, and switch to a drug from a different group, such as phenytoin, carbamazepine, or oxcarbazepine.

Phenytoin: The starting dose of phenytoin is 100 mg three times daily or 200 mg in the morning and 100 mg in the evening, depending on tolerability and compliance.[50,51] The 12-hour serum drug level should be checked two weeks after the first dose and one week after any dose change. In the majority of studies, the drug was kept at 300 mg per day, although there are no data correlating serum levels with efficacy in reducing impulsive aggression. However, patients who do not respond after two to three weeks may benefit from increasing the dose by 30 mg per day each week to 400 mg per day.

Carbamazepine, Oxcarbazepine: The starting dose of oxcarbazepine is 150 or 300 mg daily. It is increased by 150 to 300 mg daily every two to four days to a target dose of 1200 to 2400 mg daily, as tolerated.[52] The dose is divided into two doses per day. Carbamazepine is usually started at a dose of 200 mg per day in divided doses. The dose is increased by 200 mg daily every five days to reach a target dose of 800 to 1800 mg per day as tolerated. Although there are no data linking serum levels to efficacy in reducing impulsive aggression, extended-release formulations may provide more stable serum

levels. A systematic review of four randomized trials showed that oxcarbazepine and carbamazepine were superior to placebo in impulsive aggressive behavior.[53]

In IED, some patients do not respond to oxcarbazepine within 6 to 12 weeks of starting the drug. For these treatment-resistant patients, it is recommended to reduce oxcarbazepine by 300 to 600 mg every two to three days, discontinue it, and switch to a drug from a different group, such as lamotrigine, topiramate, valproic acid, or lithium. The rate of patients responding to the medication change applied in this way can be up to approximately %50.[20,44]

Lamotrigine: The starting dose of lamotrigine is 25 mg daily for the first two weeks. In the third and fourth weeks, the dose is increased to 50 mg per day in divided doses. The dose can then be titrated between 25 and 50 mg daily, once a week for each increase. This slow titration reduces the risk of Steven Johnson Syndrome, a potentially life-threatening side effect characterized by a skin rash. The target dose is 50 to 200 mg per day. In an 8-week randomized controlled trial comparing lamotrigine and placebo in 27 patients with borderline personality disorder, lamotrigine proved superior.[54]

Topiramate: The starting dose of topiramate is 50 mg per day, taken divided in half. The dose is increased by 50 mg daily each week to reach a target dose of 200 to 300 mg daily, as tolerated. Two randomized controlled trials conducted over eight weeks in patients with borderline personality disorder have demonstrated the superiority of topiramate over placebo.[55,56]

Valproate: The initial dose of valproate is 250 mg twice daily, which is increased by 250 mg daily as tolerated to reach an effective dose.[57] The maximum dose is 30 mg/kg/day. Although there are no data linking serum levels to efficacy in reducing impulsive aggression, some authorities target 12-hour serum drug levels of 80 to 120 mcg/mL to maximize efficacy.

The largest randomized controlled trial failed to prove the superiority of valproate over placebo for 12 weeks in 116 patients with IED.[57] However, other smaller studies suggest that valproate may reduce impulsive aggressive behavior.[53]

Lithium: The starting dose of lithium is usually 300 mg two or three times daily. As tolerated, the dose should be increased by 300 to 600 mg every one to five days. The aim is to achieve a therapeutic serum level, which usually occurs with a dose of 900 mg to 1800 mg per day. The target serum level is between 0.8 and 1.2 mEq/L and should generally not exceed 1.2 mEq/L. In a 12-week randomized controlled trial comparing lithium with a placebo in 59 prisoners with chronic impulsive aggressive behavior, lithium proved superior.[58]

2. Psychotherapy

Psychotherapy in IED patients is generally planned to teach these individuals to recognize and manage their moods and moments of anger. Because it is assumed that the fact that these patients are unaware of their increasing anger leads to an unbearable accumulation in the process and that a sudden and inappropriate outburst of anger follows to discharge the accumulation. Group therapy is also known to be helpful in these patient groups.[59] The "exposure" method, which is frequently used in anxiety disorders, was examined in a non-controlled pilot study for anger treatment. In the process, people developed tolerance to anger-triggering scenarios and benefited from the treatment.[59] Another study compared relaxation training alone and combined cognitive behavioral therapy and relaxation training in drivers with anger control problems. While there was no significant improvement in the general anger level in both groups, it was found that the anger levels observed while driving decreased.[60] When this study was repeated in the following years with vehicle drivers with higher anger levels, both methods provided significant improvement in general anger level. Since subjects who received only relaxation training and subjects who received both relaxation training and cognitive therapy benefited at the same level, it has been shown that only relaxation training can be considered sufficient

in treating subjects who are likely to become angry while driving.[61] Mindfulness training is a meditation technique and is another option that can be preferred during the treatment of sudden outbursts of anger without planning in patients diagnosed with IED. This technique teaches the individual how to shift attention from the current situation that causes anger to a more neutral, unrelated part of the body (such as the palms of the hands or soles of the feet) or a more irrelevant situation.

3. Cognitive Behavioral Therapy

Impulsive aggressive behaviors can be controlled with CBT.[42] CBT teaches patients how to manage triggering stimuli in the everyday environment. Specific techniques used in CBT applied in IED include:

1. Cognitive restructuring (changing faulty assumptions and dysfunctional thoughts; the patient is encouraged to examine and evaluate the validity of assumptions and thoughts in the light of all available evidence).
2. Relaxation exercises (e.g., progressive muscle relaxation exercises involving deep breathing as well as tensing and relaxing different muscle groups while imagining situations that cause anger)
3. Training in coping skills (e.g., rehearsing responses such as role-playing potentially provocative situations and avoiding them)
4. Relapse prevention (educating patients that recurrence of impulsive aggressive behavior is common and should be seen as a "slippage" rather than a failure)

CBT is most effective with highly motivated patients who value a problem-solving approach. On the contrary, it is contraindicated in patients who cannot learn the specific techniques taught (e.g., patients with moderate or severe cognitive deficits).[42]

CBT can be delivered in a group or individual format. Patients typically receive 8 to 16 sessions of therapy, but some treatment plans may require 20 sessions, each lasting approximately 60 minutes. Skills taught in therapy are practiced between sessions.

In a 12-week randomized trial comparing group CBT, individual CBT, and a control group condition in 45 patients with intermittent explosive disorder not receiving pharmacotherapy, patients receiving CBT had a clinically large and statistically significant reduction in impulsive aggressive behavior compared with the control group; minor differences between group and individual CBT were observed, which were not significant.[62]

Conclusion

Intermittent explosive disorder is characterized by recurrent episodes in which the person is unable to resist aggressive impulses. These episodes may result in damage to other people or property. Although the frequency and severity of the attacks vary, the degree of aggression observed is disproportionate to the stressor that brought about the current situation. Even though many studies have been conducted to determine the prevalence of the diagnosis, very different results have been found. It can be said that this is due to the lack of a "gold standard" diagnostic tool and the fact that research methods are not organized in a way that distinguishes social diversity, so further studies are needed in this field. Aggressive behaviors and cognitive impairment are at the center of the clinical features. These symptoms can be observed for many years in diagnosed individuals. In addition to SSRIs such as fluoxetine, many different options, such as phenytoin, carbamazepine, and lamotrigine, can be used for the treatment of the disorder, especially in treatment-resistant cases. Psychotherapy combined with pharmacotherapy will also be appropriate in terms of treatment, as it is found to be more effective than pharmacotherapy alone.

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