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REVIEW

Optimal management of pediatric obsessive-compulsive disorder

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Abstract: The last two decades have seen an increase in evidence supporting behavioral and pharmacologic treatments of pediatric obsessive-compulsive disorder, a debilitating anxiety disorder that affects about 1% of youth. However, dissemination of knowledge about these treatments to pediatric health care providers and families of affected children has been less successful. Following best practice guidelines, specific evidence for cognitive-behavioral therapy with exposure and response prevention and pharmacotherapy with serotonin reuptake inhibitors are presented. A discussion of clinical features and their impact on treatment delivery and empirically based suggestions for overcoming these barriers are also presented. Future directions for enhancing treatment implementation and dissemination are discussed.

Keywords: obsessive-compulsive disorder, children, treatment, cognitive-behavioral therapy, serotonin reuptake inhibitors, antidepressant medication

Introduction

Pediatric obsessive-compulsive disorder (OCD) is a chronic, disabling anxiety disorder characterized by obsessions and/or compulsions that are associated with significant distress and/or interference in functioning of daily life.[1] Obsessions are recurring, upsetting intrusive thoughts or images that cause disabling distress. Common obsessions include fears of contamination, sexual behavior (eg, fears of doing something sexually inappropriate), aggressiveness (eg, fears of harming another person or their self), or religious taboos (eg, concerns about committing blasphemy or offending a religious figure); and symmetry/exactness and hoarding/saving obsessions.[2,3] The distressing nature of obsessions motivates compulsions, which are repetitive, overt or covert behaviors performed to temporarily relieve the angst associated with obsessions. Common compulsions include cleaning, checking, repeating behaviors, reassurance seeking, covert rituals (eg, counting, praying), and hoarding/collecting.[2,4,5] Unlike adults with OCD, children are not required to have insight into their obsessive-compulsive symptoms,[1] although most children have some degree of insight.[6,7]

Prevalence

Clinically significant obsessive-compulsive symptoms occur in about 1% of children and adolescents,[8,9] with as many as 4% of youth exhibiting subclinical symptoms.[10] Nearly 80% of adult OCD cases have initial onset in childhood,[11,12] which typically runs a protracted course without the initiation of appropriate treatment. The sex distribution of childhood OCD is approximately 3:2 of boys to girls, which is due in

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part to a higher frequency of tic disorders in boys.[2] However, the sex ratio roughly equalizes by adolescence/early adulthood.[3,13]

Impairment

The scope of impairment associated with OCD is unique among the anxiety disorders[14] due to the time occupied by and distress associated with symptoms, avoidance, and psychiatric comorbidity.[15] Youth with OCD experience significant impairment in school, home, and family functioning,[16–18] and reduced quality of life.[19] Specific domains of impairment noted include impaired functioning in school/academic domains (eg, getting to school/class on time, reading, listening, concentrating on schoolwork, doing homework, and taking tests), home life (eg, getting dressed, getting ready for bed, bathing/grooming, and watching television/listening to music), and social relationships (eg, difficulty making new friends, going to movies, and eating in public places).[16] For example, a child may spend hours and hours redoing otherwise simple homework assignments due to the compulsion to write and rewrite letters, words, and sentences on writing assignments.

Assessment

Clinicians should routinely screen for the presence of obsessions and/or compulsions or repetitive behaviors in children and adolescents during clinical interviews with the family and utilize simple probing questions. For example, when probing for questions regarding obsessions, a clinician may ask, "Do you have worries that just won't go away even when you want them to stop, such as having concerns about germs or chemicals, having thoughts over and over of bad things happening to yourself or others, or worries about having to do things correctly?" When probing for compulsions, a clinician may ask, "Do you do things over and over or have habits you can't stop, such as washing your hands, checking things, touching objects the same number of times, feeling that you have to tell on yourself repeatedly, or always asking if you did the right thing or did it correctly?"[20] If screening questions suggest that obsessive-compulsive symptoms may be present, further probes should be conducted in the interview and/or with psychometrically sound measures that assess the time occupied by the symptoms, and the level of distress and functional impairment (eg, the Children's Yale-Brown Obsessive Compulsive Scale).[21] Furthermore, a full psychiatric evaluation is needed to assess mental status, comorbid disorders, and medical/family/developmental/school history to assess fully the clinical presentation, health status, and possible factors that may complicate treatment outcome.[21]

In order to inform differential treatment when assessing OCD symptoms, clinicians should be aware of important differences between obsessions versus developmentally normative worries, and OCD symptoms versus other psychiatric conditions such as tic disorders. For example, unlike OCD symptoms, tics are sudden, repetitive, stereotyped motor movements or phonic productions that may appear voluntary, which can be distinguished from OCD symptoms by their relative brevity, lack of purpose, and typically involuntary nature. It is critical that clinicians are able to differentiate the two conditions, because each condition has unique recommended practice guidelines for treatment.

Pediatric acute-onset neuropsychiatric syndrome

While assessing for the presence of obsessive-compulsive symptoms, clinicians should be aware of a subgroup of children with OCD who experience abrupt, dramatic onset of significantly severe and distressing obsessive-compulsive symptoms or severely restricted food intake which does not follow the pattern of gradual onset typically seen in youth with OCD known as Pediatric Acute-onset Neuropsychiatric Syndrome (PANS). These youths also exhibit additional neuropsychiatric symptoms (eg, anxiety, emotional lability and/or depression, irritability, aggression and/or severely oppositional behaviors, developmental regression, rigid or repetitive behaviors, tics, worsening fine motor skills, and compulsive urination) and show a marked deterioration in school performance and adaptive functioning.[22,23] Furthermore, symptom onset in a subgroup of these children is associated with an infectious trigger. Youth with PANDAS (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections) have prepubertal, acute onset of episodic (relapse-remitting) obsessive-compulsive symptoms or tics that have a temporal association with Group A streptococcal infections.[25] They also display neurologic abnormalities such as motor hyperactivity and choreiform movements.[22–25] In comparison, youth who display obsessive-compulsive symptoms or tics after any infection, not limited to Group A streptococcal infections, are broadly characterized as having PITANDS (Pediatric Infection-triggered Autoimmune Neuropsychiatric Disorders) with sudden, recurrent, prepubertal clinically significant obsessive-compulsive and/or tic symptoms that are triggered or exacerbated by a bacterial or viral infection (eg, influenza, varicella, Group A streptococcus).[25] Although evidence for the neuropsychiatric syndromes based on infections is not without controversy, and

definitive data do not exist at this time regarding treatment for PANS, clinicians should treat infections in the child as dictated by pediatric practice guidelines. Preliminary data and clinical accounts suggest that immune-targeted therapies, including acute antibiotic treatment, intravenous immunoglobulin, and plasmapheresis, may show efficacy in treating obsessive-compulsive symptoms.[26,27] There are also some data suggesting that cognitive-behavioral therapy (CBT) may be effective.[28,29] Collectively, these treatment data are preliminary, and more rigorous testing is required to determine conclusive treatment guidelines for PANS.

Treatment

Two treatments have demonstrated empirical support for the treatment of pediatric OCD, ie, CBT with exposure and response prevention (see Table 1 for a summary of randomized controlled CBT trials) and pharmacotherapy (serotonin reuptake inhibitors, including selective serotonin reuptake inhibitors and the tricyclic antidepressant, clomipramine. See Table 2 for a summary of randomized, placebo-controlled trials). CBT has demonstrated larger treatment effects (d = 1.45–1.98) than serotonin reuptake inhibitor monotherapy (d = 0.48–1.13),[30,31] and boasts greater symptom reduction (53%–65% versus 26%–30% as measured by the Children's Yale-Brown Obsessive Compulsive Scale)[32,33] and remission levels (24%–82% versus 10%–45%).[32,34–37] Given this evidence, expert consensus guidelines recommend that children with OCD should first be treated with CBT alone for mild and moderate cases, or together with serotonin reuptake inhibitor therapy for severe presentations.[20,38]

Cognitive-behavioral therapy

CBT for OCD typically incorporates three components, ie, psychoeducation, exposure and response prevention (ie, presentation of the feared stimuli while resisting engaging in compulsions), and cognitive therapy.[39] CBT for OCD is typically delivered weekly over the course of 12–14 weeks but can be applied in an intensive treatment format (ie, daily psychotherapy sessions) with comparable efficacy in both specialty clinics and traditional outpatient settings.[40] Clinicians begin CBT by educating parents and children about the nature of OCD and its treatment. The child is taught to identify and understand his/her anxiety symptoms and why treatment is necessary. To convey the treatment rationale at a developmentally appropriate level, clinicians often use metaphors (eg, "OCD is like a bully who makes you do things you do not want to do"), visual imagery (eg, "imagine how scared you felt when you watched your first scary movie. Now imagine watching the movie 100 times. You will probably feel less scared than when you watched the movie the first time. This is similar to exposure therapy. You face your fears numerous times and over time, your anxiety will go down"), or other examples to help make OCD more concrete.[35,41]

The cognitive portion of treatment involves identifying and challenging obsessional thoughts and replacing them with more realistic thoughts in a developmentally appropriate manner. For example, a child who experiences obsessions about contracting an illness if s/he touches a door knob might be encouraged to think about all the times when other people touch door knobs and do not get sick. Other cognitive tools, such as reframing OCD as something outside of the child (ie, externalizing OCD) and "bossing back" OCD (ie, having children tell OCD that they do not have to complete compulsions to make anxiety dissipate) may help children resist symptoms.[35,39]

Exposure with response prevention is the primary component of CBT and is based on the behavioral theory that anxiety is maintained by both classical and operant conditioning.[42] Through a combination of verbal transmission, direct conditioning, social learning, prior conditioning, and/or genetic factors,[43] neutral ideas or images are paired with feelings of heightened arousal (eg, anxiety) that motivate rituals which temporarily relieve distress. As a result, the rituals are maintained through a process known as negative reinforcement. In treatment, the child is gradually exposed to distress-provoking stimuli through the use of a stimulus hierarchy and prevented from engaging in the compulsion that would allow anxiety/distress to reduce naturally over time.[41] Children and their families are then assigned specific exposure tasks to conduct as homework to practice and generalize the skills learned in the clinical setting to the outside environment. Family involvement is central to reinforce treatment goals, promote generalization, and reduce family accommodating behaviors.[44,45]

Pharmacotherapy

In clinical trials, four selective serotonin reuptake inhibitors (paroxetine, fluoxetine, fluvoxamine, and sertraline) and a tricyclic antidepressant (clomipramine) have demonstrated efficacy relative to placebo in the treatment of pediatric OCD (see Table 2). Only fluoxetine (age 7 years and older, 10–60 mg/day), fluvoxamine (age 8 years and older, 25–200 mg/day), sertraline (age 6 years and older, 25–200 mg/day), and clomipramine (age 10 years and older, 25–200 mg/day) are approved by the Food and

Table 1 Randomized controlled trials of cognitive-behavioral therapy for pediatric OCD

Reference	Design and sample	Outcome measures	Results	Limitations
Barrett et al[32]	Individual family-based CBT (CBTF) versus CBTF group versus waitlist 14 weeks n = 77 Age 7–17 years	ADIS-P CY-BOCS CDI SAS MASC FAD NIMH GOCS DASS-21	• Mean reduction in total CY-BOCS scores from baseline to end of study: individual CBTF (65%) > group CBTF (61%) > waitlist (0%) • Significant improvement after 6 weeks on ADIS-P (ie, without a diagnosis of OCD): individual CBTF (88%), group CBTF (76%) > waitlist 0% • Individual and group CBFT conditions reduced anxiety and depression • Remission at 3-month follow-up: individual CBFT 82%; group CBFT 76% • Treatment gains maintained after 6 months of follow-up	• Parent diagnostic interventions only • Participant sample was not representative of a diverse race and ethnic population
Freeman et al[35]	Family-based CBT versus family-based relaxation treatment (RT) 14 weeks n = 42 Age 5–8 years	CY-BOCS CGI-I	• In the completer sample, mean reduction in CY-BOCS total scores from baseline to end of study: CBT (50%) > RT (23%) • For the completer sample, CBT had a large effect ($d = 0.85$) • In the completer sample, 69% of children in CBT group achieved a clinical remission compared with 20% in RT group • In the completer sample, treatment responders as measured by ratings of very much or much improved on the CGI-I at the end of treatment: CBT (69%), RT (40%)	
Piacentini et al[37]	CBT with exposure therapy and structured family intervention (FCBT) versus psychoeducation and relaxation training (PRT) 14 weeks n = 71 Age 8–17 years	ADIS-C/P CY-BOCS CGI-I COIS-R	• Mean reduction in total CY-BOCS scores from baseline to end of study: FCBT (46.2%) > PRT (32.0%) • FCBT within-group effect size ($d = 2.37$) • PRT within-group effect size ($d = 1.80$) • Between-group effect size ($d = 0.40$) • Faster decline in CYBOCS scores over time in FCBT group as compared with PRT group • Clinical remission rates were 42.5% for FCBT versus 17.6% for PRT • FCBT had significantly higher response rates than PRT on the CGI-I (57.1% versus 27.3%) • At 1-month follow-up and 6-month follow-up, the FCBT responders maintained their positive rates (81% and 73%, respectively) and the PRT responders maintained their positive rates (60% and 75%, respectively) as measured by the CY-BOCS	• Same therapist provided both treatments, which may have caused some elements of FCBT to carry over to PRT
POTS I[36]	RCT CBT versus sertraline versus CBT + sertraline versus medical management + placebo 12 weeks n = 112 Age 7–17 years	CY-BOCS ADIS-C NIMH Global Severity Scale	• Mean reduction in total CY-BOCS scores from baseline to end of study: CBT alone (46%), sertraline (30%), combined (53%) > placebo (15%) • Clinical remission as measured by total CY-BOCS score ≤ 10: CBT + sertraline, 53.6% ($d = 1.40$); CBT alone, 39.3% ($d = 0.97$); sertraline alone, 21.4% ($d = 0.67$); placebo, 3.6%	• CBT alone response differed between sites

Franklin et al[59]	Medication management (MM) versus medication management plus CBT instructions (MM + I-CBT) versus medication management plus CBT (MM + CBT) 12 weeks n = 124 Age 7–17 years	CY-BOCS ADIS-C NIMH Global Severity Scale CGI-S	• MM + CBT was superior to the other two groups on all outcome measures (CY-BOCS, NIHM-GOCS) • Responders (defined as having at least a 30% reduction in CY-BOCS scores): MM + CBT (68.6%) > MM + I-CBT (34.0%) > MM (30.0%) • Treatment effect sizes at week 12 (as measured by CY-BOCS scores): MM + CBT versus MM: $d = 0.85$ MM + I-CBT: $d = 0.16$	• Participant sample was not representative of a diverse race and ethnic population
Williams et al[90]	RCT CBT versus waitlist 12 weeks n = 21 Age 9–18 years	ADIS-C CY-BOCS CDI MASC OCI CRAS CRIQ	• Mean reduction in total CY-BOCS scores from baseline to end of first phase (3 months): CBT (48%) > waitlist (7%) • CBT group had significantly reduced scores on CY-BOCS ($d = 2.62$) • CBT group decreased on OCI, CDI and MASC	

Note: d = Cohen's d effect size.

Abbreviations: RCT, randomized controlled trial; ADIS-C, Anxiety Disorders Interview Schedule for Children; ADIS-P, Anxiety Disorders Interview Schedule for Parents; CDI, Child Depression Inventory; CGI-I, Clinical Global Impression-Improvement Scale; CGI-I, Clinical Global Impression-Severity Scale; COIS-R, Child Obsessive Compulsive Impact Scale-Revised; CRAS, Children's Responsibility Attributions Scale; CRIQ, Children's Responsibility Interpretations questionnaire; CY-BOCS, Children's Yale-Brown Obsessive Compulsive Scale; MASC, Multidimensional Anxiety Scale for Children; NIMH Global Severity Scale, National Institute of Mental Health Global Severity Scale; OCD, obsessive-compulsive disorder; OCI, Obsessions and Compulsions Inventory.

Drug Administration (FDA) for the treatment of pediatric OCD (United States FDA Center for Drug Evaluation and Research, personal communication, December 28, 2011). In a meta-analysis, Geller et al[47] examined study designs, outcome measures, and medication efficacy across 12 clinical trials of selective serotonin reuptake inhibitors (paroxetine, fluvoxamine, fluoxetine, and sertraline) and clomipramine, finding a modest aggregate effect size relative to placebo ($d = 0.46$; 95% confidence interval 0.37–0.55). Clomipramine was significantly more effective in reducing obsessive-compulsive symptoms than paroxetine, fluvoxamine, fluoxetine, and sertraline, which were not significantly different from one another. However, clomipramine is not recommended as the first line of pharmacotherapeutic treatment for pediatric OCD due to side effects (eg, dry mouth, dizziness, fatigue, tremors, and cardiac complications).[20,46,47]

Pharmacologic monotherapy typically demonstrates a 30%–40% reduction in obsessive-compulsive symptoms,[36,47] leaving many youth with clinically significant residual symptoms.[47–49] Symptom remission rates for children treated with serotonin reuptake inhibitors alone are modest but positive, with one study finding only 21.4% of children no longer meeting criteria for OCD.[36] While generally safe, serotonin reuptake inhibitors are associated with some adverse effects eg, headaches, abdominal pain, nausea, fatigue, and somnolence;[51–53] most notably, concern about behavioral activation resulted in a black box warning requiring physicians to monitor serotonin reuptake inhibitor usage carefully due to reports of increased risk of suicidal ideation and suicidal-like behaviors.[54–57]

Augmenting agents

Between 40% and 70% of OCD patients treated with a serotonin reuptake inhibitor alone do not respond adequately to treatment,[49,52,58–60] and full symptom remission from serotonin reuptake inhibitor monotherapy is infrequent.[36,49,50] An adequate dose of serotonin reuptake inhibitor therapy is defined as a minimum of 10 weeks of serotonin reuptake inhibitor therapy at maximum recommended or maximum tolerated doses, with no change in dose for the preceding three weeks. Adequate dose for CBT is defined as at least 10 sessions, which includes at least eight sessions of exposure/response prevention.[20] Patients who remain symptomatic after adequate serotonin reuptake inhibitor intervention are often augmented with off-label atypical antipsychotic medications, but systemic efficacy data are lacking. Although few experimental studies exist, case reports and open trials of augmenting agents have reported benefit (clonazepam,[61]

Table 2 Pharmacotherapy randomized placebo-controlled trials in pediatric OCD

Reference	Age, years	Medication (dosage)	Study design	Results
DeVeaugh-Geiss et al[91]	10–17	Clomipramine (75–200 mg/day)	Randomized placebo-controlled 8 weeks n = 60	• Mean reduction in total Y-BOCS scores from baseline to end of study: clomipramine (37%) > placebo (8%) • Ratings of very much or much improved on Patient Self-Rating Scale at the end of treatment: clomipramine (53%) > placebo (8%) • Participants continued onto an open-label phase and efficacy was maintained after one year of treatment • Dropout: 6 (4 clomipramine, 2 placebo)/60 subjects
Geller et al[58]	7–17	Fluoxetine (20–60 mg/day) (mean 24.6 mg/day)	Randomized placebo-controlled 13 weeks n = 103	• Treatment responders (defined as having at least a 40% reduction in CY-BOCS scores): fluoxetine (49%) > placebo (25%) • Effect size (as measured by the total CY-BOCS score, $d = 0.50$) • Ratings of very much or much improved on CGI-I at end of treatment: fluoxetine (55%) > placebo (18.8%) • Dropout due to lack of efficacy: 18 (10 fluoxetine, 8 placebo)/103 subjects
Geller et al[52]	7–17	Paroxetine (10–50 mg/day) (mean 23.0 mg/day)	Randomized placebo-controlled 10 weeks, flexible dose n = 207	• Mean reduction in total CY-BOCS scores from baseline to end of study: paroxetine (36%) > placebo (21%) • Treatment responders (defined as having at least a 25% reduction in CY-BOCS scores): paroxetine (61%) > placebo (42%) • Ratings of very much or much improved on CGI-I at end of treatment: fluoxetine (45%), placebo (35%) • Dropout due to lack of efficacy: 19 (5 paroxetine, 14 placebo)/207 subjects
March et al[49]	6–17	Sertraline (25–200 mg/day) (mean 167 mg/day)	Randomized placebo-controlled 12 weeks n = 187	• Treatment responders (defined as having at least a 25% reduction in CY-BOCS scores): sertraline (53%) > placebo (37%) • Ratings of very much or much improved on CGI-I at end of treatment: sertraline (42%) > placebo (26%) • Drop-out due to lack of efficacy: 5 (3 sertraline, 2 placebo)/187 subjects
Riddle et al[50]	8–17	Fluvoxamine (50–200 mg/day) (mean 165 mg/day)	Randomized placebo-controlled 10 weeks n = 120	• Mean reduction in total CY-BOCS scores from baseline to end of study: fluvoxamine (24.6%) > placebo (13.6%) • Effect size (as measured by total CY-BOCS score, $d = 0.68$) • Treatment responders (defined as having at least a 25% reduction in CY-BOCS scores): fluvoxamine (42.1%) > placebo (26.0%) • Ratings of very much or much improved on CGI-I at end of treatment: fluvoxamine (29.8%) > placebo (17.5%) • Dropout: 46 (19 fluvoxamine, 27 placebo)/120 subjects

Note: d = Cohen's d effect size.
Abbreviations: CGI-I, Clinical Global Impression-Improvement Scale; CY-BOCS, Children's Yale-Brown Obsessive Compulsive Scale; OCD, obsessive-compulsive disorder.

risperidone,[62] haloperidol,[63] aripiprazole,[64] and riluzole[65]). However, these medications have been associated with adverse effects (eg, excessive sedation, dizziness, increased appetite, weight gain)[61,63,65–67] and their off-label use should only be considered after other more safe and tolerable alternatives have been tried.[68] To this end, recent data indicate the efficacy of CBT augmentation for youth with OCD who only partially respond to SRIs.[59]

Factors that influence treatment outcomes

There are multiple factors that may contribute to the efficacy and sustainability of positive treatment outcomes, such as family accommodation, insight and motivation, and comorbidity.

Family accommodation

Many family members accommodate their child's obsessive-compulsive symptoms (eg, providing objects for rituals, following routines to minimize anxiety, providing extra assistance with homework and chores, providing reassurance) to minimize their child's distress and to decrease symptom-related impairment.[44] Although well intentioned, accommodation maintains obsessive-compulsive symptoms in the same way as rituals (eg, anxiety reduction) and prevents the child from learning that the feared consequences usually do not occur, resulting in greater impairment, symptom severity, and negative family dynamics.[44,69,70] Furthermore, family accommodation can serve as a treatment barrier, preventing the child from experiencing the natural habituation of anxiety and reinforcing obsessive-compulsive symptoms.[71,72]

Insight and motivation

Children with poor insight and low motivation may not respond optimally to treatment due to limited symptom resistance and poor adherence to treatment recommendations (eg, taking medication, participating in CBT).[67,73] Poor insight is associated with more severe obsessive-compulsive symptoms, higher levels of internalizing behaviors, greater impairment in school, social, and family contexts, and poor treatment response to CBT and pharmacotherapy.[7,67,71,74,75] For children who refuse or have low motivation to engage in CBT, medication treatment may be considered.[20] Behavioral approaches to increase a child's motivation to continue treatment include the creation of a reward program early in treatment[20,35] and CBT supplemented with motivational interviewing.[76] Motivational interviewing is a therapeutic intervention used to decrease ambivalence and promote motivation to change one's behavior. It may be particularly helpful for youth who may be less willing to follow instructions or lack the self-confidence to engage in exposures in and outside of the sessions.[76]

Comorbidity

Comorbid conditions are common in children with OCD, with about 74%–80% meeting criteria for another psychiatric illness.[77–79] In addition to being directly related to a more severe clinical presentation, comorbid conditions interfere with treatment outcome. In CBT, comorbid conditions may reduce treatment adherence and participation in sessions.[78,80–82] Furthermore, comorbid disorders can adversely affect response rates to serotonin reuptake inhibitors and result in greater rates of relapse.[81,83,84] With paroxetine, for example, the full sample response rate of 71% decreased significantly when examining responses for comorbid subgroups (attention deficit/hyperactivity disorder, 56%; chronic tic disorder, 53%; oppositional defiant disorder, 39%).[83] Consequently, a child who has a comorbid condition such as major depression, disruptive behavior disorder, or attention deficit hyperactivity disorder may require a different treatment approach considering how the comorbid disorder might affect presentation and/or treatment course.[20,82,85–87] For example, if depression could impact the course of treatment, sequentially providing evidence-based depression therapies (eg, antidepressant medication, CBT, or interpersonal psychotherapy) may be indicated. Similarly, those with uncontrolled attention deficit hyperactivity disorder may benefit from concurrent pharmacologic intervention.[88]

Conclusion

Pediatric OCD is an impairing disorder that runs a chronic course without treatment. For mild to moderate cases of pediatric OCD, CBT is recommended as the first line of treatment. For moderate to severe cases of pediatric OCD, serotonin reuptake inhibitor medication in addition to CBT is recommended.[20] However, as discussed in this review, treatment may vary on a case-by-case basis dependent upon factors such as family accommodation, insight, motivation, and comorbidity. Clinical indications that signal a need for a higher level of care (ie, intensive or residential OCD-specific care) include limited response to past treatments, persisting lack of motivation and insight, and severe symptomology.[20] Although CBT is recommended per established guidelines, dissemination of these guidelines is lagging.[20] Fewer than 10% of adults being treated with CBT receive CBT with exposure therapy,[89] although this has greatly improved over

the past decade. While both CBT and the serotonin reuptake inhibitors demonstrate efficacy, many youth are partial responders; thus, future efforts need to examine innovative methods for maximizing treatment outcomes and remission rates, as well as examining treatment moderators to individualize treatment.

Disclosure

The authors report no conflicts of interest in this work.

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