Obsessive-compulsive disorder in children and adolescents: Epidemiology, pathogenesis, clinical manifestations, course, assessment, and diagnosis

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INTRODUCTION

Obsessive-compulsive disorder (OCD) is a severe, prevalent and most often chronically debilitating disorder characterized by repetitive, ritualistic, and distressing thoughts, ideas, and behaviors over which a person typically has very little if any control.

Research suggests approximately 50 percent of all cases have their onset in childhood and adolescence [1,2]. Obsessions and compulsions in children are more likely to change/evolve as well as wax and wane, compared to the course of the disorder in adults.

This topic reviews the epidemiology, pathogenesis, clinical manifestations, course, assessment and diagnosis of OCD in children and adolescents. The treatment of OCD in children and adolescents is discussed separately. The epidemiology, pathogenesis, clinical manifestations, course, assessment, diagnosis, and treatment of OCD in adults are also discussed separately. (See "Obsessive-compulsive disorder in children and adolescents: Treatment overview" and "Management of obsessive-compulsive disorder in adults" and "Obsessive-compulsive disorder in adults: Epidemiology, clinical features, and diagnosis" and "Obsessive-compulsive disorder in adults: Psychotherapy".)

EPIDEMIOLOGY

Obsessive-compulsive disorder (OCD) affects more than three million people in the United States. Estimates of the disorder's United States lifetime prevalence in both pediatric and adult populations have ranged from 1 to 3 percent [3-5]. Research suggests that approximately 50 percent of all cases have their onset in childhood and adolescence [1,2]. A United States epidemiologic study of a nationally representative sample reported that 21 percent of OCD cases had onset by age 10 years [3], with a mean age of onset for pediatric OCD between 9 and 11 years in boys and 11 and 13 years in girls [6]. Pediatric OCD appears to be more common in males than in females, in contrast to adults where the male-female ratio of OCD is approximately 1:1 [7,8].

A 1999 nationwide epidemiologic study of 10,438 5 to 15 year olds in the United Kingdom estimated a weighted overall prevalence of OCD at 0.25 percent (95% CI 0.14-0.35) [9]. The children with OCD were more likely to be from lower socio-economic class and of lower intelligence compared to normal controls.

Comorbidities — More than half of pediatric patients with OCD have been found to have at least one comorbid psychiatric disorder [10,11]. In a review of 11 epidemiological studies, children and/or adolescents with OCD have been found to have higher rates of co-occurring mental disorders compared to subjects without OCD [1,8,12-18]:

- Any psychiatric disorder, 63 to 97 percent
- Mood disorder, 13 to 70 percent
- Anxiety disorder, 13 to 70 percent
- Disruptive behavior disorder, 3 to 57 percent
- Tic disorder/Tourette syndrome, 13 to 26 percent
- Speech/developmental disorders, 13 to 27 percent
- Enuresis, 7 to 37 percent
- Pervasive developmental disorder, 3 to 7 percent
- Eating disorders, particularly in adolescents [19,20]

Co-occurring mood disorders and psychosis have increased prevalence in adolescents with OCD; co-occurring ADHD and non-OCD anxiety disorders have been found to occur at higher rates in children with an early age of onset of OCD [21]. Separation anxiety disorder in some cases is a precursor to OCD onset in childhood.

PATHOGENESIS

Etiology — The etiology of obsessive-compulsive disorder (OCD) is unknown; however, research suggests that multiple genetic risk factors lead to changes in cellular function or altered neurotransmitter signaling. These molecular and cellular changes perturb fronto-striatal-thalamic circuitry (FSTC), resulting in OCD.

Genetic studies — Twin and family studies provide compelling evidence that genetic factors are critically involved in the transmission and expression of OCD. Two controlled family studies of pediatric patients with OCD found the lifetime prevalence of OCD to be significantly higher in familial cases compared with control relatives [22,23]. Family studies of pediatric OCD patients demonstrate that pediatric patients usually have a more familial form of the disorder that may be etiologically more homogeneous or more strongly associated with genes of major effect [24].

Segregation analyses provide compelling data that a major gene is important in transmitting OCD. A major susceptibility locus in a proportion of families with OCD was identified in five complex segregation analyses [25-28]. A large segregation analysis of OCD supported an autosomal dominant or co-dominant model, and rejected a recessive model [27]. The only segregation analysis of families that examined only pediatric OCD patients provided evidence for a major gene in families with OCD when age at onset was incorporated into the model [26]. When simple Mendelian models were compared, the dominant model provided a better fit to the data than the other Mendelian models.

A genome-wide linkage analysis of OCD in seven families of pediatric OCD patients identified a region on chromosome 9 with suggestive evidence for linkage. The glutamate transporter *SLC1A1* on chromosome 9 was implicated as a positional candidate gene for pediatric OCD by two research groups working independently [29,30]. Family-based association studies also implicated the 9p24 region in OCD by identifying two microsatellite markers (D9S288 and GATA62F03) flanking the *SLC1A1.* Studies by other research teams have had findings implicating the glutamate transporter gene (*SLC1A1*) [6,31,32] and the glutamate receptor gene (*GRIN2B*) [33].

Possible autoimmune subtype — Immune responsivity to infections with group A beta-hemolytic streptococcus (GABHS) is believed to result in basal ganglia inflammation and resultant OCD, tic and/or ADHD symptoms [34] (see "PANDAS: Pediatric autoimmune neuropsychiatric disorder associated with group A streptococci"). Immune-related dysregulation may also play a role in the pathophysiology of pediatric patients with OCD without a pediatric acute-onset neuropsychiatric syndrome diagnosis [35-37].

Increases in psychological stress lead to the upregulation and proliferation of "immature" circulating monocytes, which can enter the brain and have an enhanced capacity to release proinflammatory cytokines [38]. These proinflammatory cytokines act to propagate the neuroinflammatory response and may also affect brain function and the metabolism and availability of different neurotransmitters [39].

An analysis of monocyte composition in whole blood comparing the percentage of total monocytes, proinflammatory CD16+ monocytes, and monocyte subsets (ie, classical [CD16-], intermediate [low-expressing, CD16+], and nonclassical [high-expressing, CD16+] subsets) in a sample of 102 OCD patients versus 47 control participants found significantly higher percentages of total monocytes, proinflammatory CD16+ monocytes, and of CD16+ expressing intermediate and nonclassical monocyte subsets in OCD versus control participants [35].

A follow-up analysis exploring the functionality of monocytes via the production of proinflammatory cytokines was conducted by exposing a fixed number of monocytes that was identical for each sample to lipopolysaccharide (LPS) to stimulate an immune response. Although the number of monocytes between groups was the same, higher levels of granulocyte-macrophage colony stimulating factor, interleukins 6, 8, and 1 beta, and tumor necrosis factor alpha were found in OCD participants versus healthy controls after exposure to LPS [35]. No differences were observed in basal levels of monocyte activity and cytokine concentrations between OCD participants and controls in samples not exposed to LPS, suggesting an abnormal immune response in OCD participants that can become hyper-activated in response to immune triggers, such as psychological stress.

Perinatal complications — There is some evidence to suggest that perinatal events are associated with a higher risk of OCD later in life. Perinatal trauma is significantly higher in tic-related OCD, which is more common in males. Higher rates of perinatal trauma have been reported in males with early onset OCD [40]. Drug exposure in utero has been found to predict increased severity of tic-related OCD [41]. Pediatric OCD patients with comorbid Tourette syndrome have been shown to be approximately five times more likely to have been exposed to alcohol, nicotine, and caffeine in utero and approximately eight times more likely to have been delivered by forceps [42]. Pediatric OCD patients may also be more likely to have mothers who became ill during pregnancy necessitating medical intervention [43].

Neuroimaging

Structural — Regions of the cortico-striatal-thalamic circuits have been implicated in the pathogenesis of OCD. Reduced striatal gray matter, which was most pronounced in patients with predominant checking and/or aggressive obsessions and compulsions but not in patients

with other obsessions and compulsions, eg, contamination, washing, etc. was reported in pediatric OCD patients versus healthy controls [7,44,45]. Increased anterior cingulate cortex gray matter (primarily in young patients) and orbital frontal cortex gray matter has been observed in pediatric OCD [45-47]. Psychotropic-naïve pediatric patients with OCD had larger thalamic volumes when compared with age and sex-matched controls [48,49]. Left amygdala-right amygdala volume ratios were increased in medication-naïve pediatric patients with OCD. Increased corpus callosum area in pediatric OCD patients versus age- and sex-matched healthy pediatric controls [7] as well as increased right but not left orbital frontal white matter has been observed in pediatric OCD [50]. Limbic-hypothalamic-pituitary-adrenal (LHPA) axis abnormalities have also been reported in OCD [51-55]. Using volumetric MRI, reduction in pituitary volume, primarily in males not females, has been reported in pediatric OCD patients versus healthy pediatric controls [56].

White matter alterations have been observed in pediatric OCD patients. Diffusion tensor imaging has shown increased fractional anisotropy, a potential measure of white matter fiber density, axonal diameter, and myelination in several white matter tracts including the corpus callosum (splenium and genu, and splenium respectively), the cingulum, and the inferior fronto-occipital fasciculus (right and left respectively) versus healthy pediatric controls [57,58]. In a related study, fractional anisotropy found increases in several white matter tracts, including the corpus callosum (anterior corpus callosum and splenium), anterior cingulum bundle, and the internal capsule [59]. All three studies reported a positive correlation between increased symptom severity and increased fractional anisotropy in either the splenium [57,58] or the anterior cingulum bundle [59].

Functional — Functional neuroimaging studies in adults with OCD have consistently implicated increased caudate and orbital frontal activity in OCD [33,60-64]. Using functional magnetic resonance imaging (fMRI), a study reported developmental alterations of fronto-striatal-thalamic connections in OCD [65]. Using fMRI to examine brain network interactions during a working memory task found hyperactivation profiles in dorsal prefrontal cortex, parietal lobe, and dorsal anterior cingulate cortex, but not in the striatum or thalamus, in pediatric patients with OCD [66]. Increased dorsal anterior cingulate cortex modulation of cortical, striatal, and thalamic targets was observed, which remained consistent regardless of task difficulty.

Neurochemistry — Serotonin and key metabolites (N-acetyl-aspartate, choline, creatine/phosphocreatine, and glutamate) have been implicated in the neurochemistry of OCD [67-75]. Reduced left and right medial thalamus N-acetyl-aspartate was reported in psychotropic-naïve pediatric OCD patients versus controls with reduced left medial thalamic N-

acetyl-aspartate levels correlating inversely with increased OCD symptom severity [76]. Psychotropic-naïve pediatric patients with OCD had significantly larger left and right medial thalamic choline levels compared with both healthy pediatric controls and psychotropic-naïve pediatric MDD patients who did not differ. Increased left and right medial thalamic creatine/phosphocreatine as well as increased left caudate creatine/phosphocreatine has been reported in psychotropic-naïve pediatric OCD patients compared with healthy pediatric controls [69,71]. Reduced anterior cingulate glutamatergic concentrations and increased left caudate glutamatergic concentrations were found in psychotropic-naïve pediatric OCD patients compared with healthy pediatric controls [69,74].

CLINICAL MANIFESTATIONS

The characteristic symptoms of obsessive-compulsive disorder (OCD) are obsessions and/or compulsions that are time-consuming, cause clinically significant distress or impair functioning. Obsessions are repetitive and persistent thoughts, images, or urges.

Obsessions are not pleasurable or experienced as voluntary. They are intrusive, unwanted, and cause marked distress or anxiety in most individuals. A person suffering from OCD attempts to ignore, avoid, or suppress obsessions, or may try to neutralize them with another thought or action (eg, performing a compulsion).

Compulsions (or rituals) are repetitive behaviors (eg, washing, checking) or mental acts (eg, counting, repeating words silently) that may be performed in response to an obsession or according to rules that may be applied rigidly. Compulsions are not rationally connected to the feared event, or are clearly excessive.

Although there is demonstrated continuity in the presentation of OCD in children and adults, the nature of obsessions and compulsions differ between youths and adults [77]. In adults the male:female ratio of OCD is approximately 1:1, while pediatric OCD is more common in males than in females [7]. Boys typically have an earlier age of onset (7 to 9 years) of OCD than girls (11 to 13 years), although it can occur earlier in both genders.

Detecting obsessions can be more difficult in children with OCD compared to adults with the disorder. While compulsions can usually be observed, very young children may be unable to describe obsessive thoughts verbally or describe their aim in carrying out a compulsive action. Insight into the excessiveness or absurdity of the symptoms is characteristic in adults, but can be more difficult to identify particularly in younger children with OCD [78].

Developmental considerations should be considered when examining a child's expression of obsessions and compulsions. Children with OCD are more likely than adults with OCD to present with compulsions without obvious obsessive symptoms [6]. Obsessions and compulsions in children are more likely to change/evolve as well as wax and wane. Religious and sexual obsessions appear to be more common in adolescents than in children, while hoarding is seen more often in preadolescent children than in adolescents with OCD [79-81].

Families of pediatric patients with OCD may have high levels of anxiety. Associated anxiety in the child, parents and/or siblings can serve as a vicious cycle and exacerbate the child's condition.

In contrast to OCD in adults, OCD in children does not appear to increase the risk of suicide. Depression commonly co-occurs with OCD, however, and its presence is associated with an increased risk of suicide.

COURSE

Obsessive-compulsive disorder (OCD) that begins in childhood often persists into adulthood, frequently with a waxing and waning course. A meta-analysis of 521 children/adolescents with OCD in 22 longitudinal studies found that, in 40 percent of cases, full OCD persisted over the period subjects were followed (range = 1 to 15.6 years); in 60 percent of cases either full OCD or subthreshold OCD symptoms persisted [82].

The meta-analysis also found that earlier age of onset of OCD, increased duration of illness, and inpatient OCD treatment were associated with greater persistence of the disorder [82]. The prognosis for pediatric OCD appears to be better than the prognosis for adult onset OCD [83-85].

Childhood OCD, in the absence of adequate treatment, is a serious and disabling disorder with a chronic but fluctuating course for the majority of patients [86]. Symptoms may get somewhat better for months or even years, only to get worse again before returning to a lower level of severity [87].

OCD in childhood can lead to impairment in long-term functioning, including problems with separation-individuation from parents and occupational achievement as adults [82]. In a retrospective cohort study of 17 children with OCD (mean age of onset 9.6 years), 10 received a follow up assessment, which found that 7 of 10 still suffered OCD symptoms [88]. Of seven subjects who were over 16 years, all seven were employed; two reported difficulty keeping a job.

ASSESSMENT

In conducting a psychiatric assessment for possible obsessive-compulsive disorder (OCD), the child and his or her parents should be asked about the presence or absence of each characteristic of OCD described in DSM-5 diagnostic criteria for the disorder. Assessment of these symptoms and behaviors over the child's lifetime is particularly important in OCD, because obsessions and compulsions can wax and wane and change during the child's developmental maturation. (See 'Diagnosis' below.)

All patients should undergo a medical history and physical examination as part of a diagnostic assessment. Neurologic examination should assess for signs or symptoms indicating possible deficits or injury in brain regions implicated in OCD (eg, basal ganglia, frontal cortex, thalamus) and rule out other medical/neurologic conditions as a cause.

Inquiries about repeated streptococcal infections, rheumatic fever, or Sydenham chorea are important, particularly if they coincide with onset/exacerbation of obsessive and compulsive symptoms, because group A beta hemolytic streptococcal infections have been associated with OCD in some children. Assessment, diagnosis, and treatment of these conditions are discussed separately. (See "Acute rheumatic fever: Clinical manifestations and diagnosis" and "Sydenham chorea" and "Group A streptococcal tonsillopharyngitis in children and adolescents: Clinical features and diagnosis", section on 'Diagnosis'.)

In addition to the primary disorder, the child should be examined for co-occurring psychiatric disorders, particularly those that occur at an increased rate in OCD. (See 'Comorbidities' above.)

The patient's family history should be reviewed for OCD given genetic studies showing that a positive family history is associated with an elevated risk of the disorder in children. (See 'Genetic studies' above.)

There are currently no genetic or neuropsychological tests or brain imaging findings with diagnostic or prognostic utility in OCD.

Rating scales — Valid and reliable scales can be used to identify symptoms and behaviors characteristic in OCD, and to monitor changes in the severity of obsessions and compulsions over time. The best studied, standardized scale is the Children's Yale-Brown-Obsessive-Compulsive Scale (CY-BOCS) [89]. This is a clinician rated scale; the clinician administering the scale should receive training from a mental health clinician experienced in the assessment of childhood OCD.

While the CY-BOCS remains the most commonly used clinician rating scale, the use of brief screening questionnaires, such as the Obsessive-Compulsive Inventory – Child Version [90,91], the OCD subscales of the Child Behavior Checklist (CBCL-OCS) [92] and the Revised Children's Anxiety and Depression Scale (RCADS) [93], may also be useful for identifying OCD symptoms during an initial assessment [94]. These screening tools can be administered quickly and inexpensively and without the need for additional clinician training. Both the CBCL-OCS and RCADS are also offered as parent and youth self-report versions, which can help more accurately identify both internalizing and externalizing OCD symptoms, that otherwise may be reported inaccurately, or may not be reported at all [95].

DIAGNOSIS

Diagnosing pediatric obsessive-compulsive disorder (OCD) can be difficult. Intrusive thoughts, doubts, or images are almost universal in the general population and their content can be indistinguishable from clinical obsessions [96]. Such intrusions occur, by definition, outside the volition of the person experiencing them and tend to interrupt other mental activity because they are inconsistent with the person's usual value system [97].

DSM-5 criteria — The diagnosis of OCD is based on DSM-5 diagnostic criteria [98]:

- A. Presence of obsessions, compulsions, or both:
  - Obsessions as defined by:
    - 1. Recurrent and persistent thoughts, urges, or images that are experienced, at some time during the disturbance, as intrusive and unwanted, and that in most individuals cause marked anxiety or distress.
    - 2. The individual attempts to ignore or suppress such thoughts, urges, or images, or to neutralize them with some other thought or action (ie, by performing a compulsion).
  - Compulsions as defined by:
    - 1. Repetitive behaviors (eg, hand washing, ordering, checking) or mental acts (eg, praying, counting, repeating words silently) that the individual feels driven to perform in response to an obsession, or according to rules that must be applied rigidly.

- 2. The behaviors or mental acts are aimed at preventing or reducing anxiety or distress or preventing some dreaded event or situation; however, these behaviors or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive.

  - Note: Young children may not be able to articulate the aims of these behaviors or mental acts.

- B. The obsessions or compulsions are time-consuming (eg, take more than 1 hour per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

- C. The obsessive-compulsive symptoms are not attributable to the physiological effects of a substance (eg, a drug of abuse, a medication) or another medical condition.

- D. The disturbance is not better explained by the symptoms of another mental disorder, for example:

  - Excessive worries, as in generalized anxiety disorder
  - Preoccupation with a defect in appearance, as in body dysmorphic disorder
  - Difficulty discarding or parting with possessions, as in hoarding disorder
  - Hair pulling, as in trichotillomania (hair-pulling disorder)
  - Skin picking, as in excoriation (skin-picking) disorder
  - Stereotypies, as in stereotypic movement disorder
  - Ritualized eating behavior, as in eating disorders
  - Preoccupation with substances or gambling, as in substance-related and addictive disorders
  - Preoccupation with having an illness, as in illness anxiety disorder
  - Sexual urges or fantasies, as in paraphilic disorders
  - Impulses, as in disruptive, impulse-control, and conduct disorders
  - Guilty ruminations, as in major depressive disorder
  - Thought insertion or delusional preoccupations, as in schizophrenia spectrum and other psychotic disorders
  - Repetitive patterns of behavior, as in autism spectrum disorder

Specifiers

- Patient’s degree of insight into the illness

- With good or fair insight – The individual recognizes that obsessive-compulsive disorder beliefs are definitely or probably not true or that they may or may not be true.
- With poor insight – The individual thinks obsessive-compulsive disorder beliefs are probably true.
- With absent insight/delusional beliefs – The individual is completely convinced that obsessive-compulsive disorder beliefs are true.

- Presence or history of a tic disorder (see 'Comorbidities' above)

Differential diagnosis — The differential diagnosis of pediatric OCD can be challenging, particularly since comorbidity is the rule rather than the exception. (See 'Comorbidities' above.)

Normal development — It is imperative that normal developmental factors be ruled out. Certain repetitive, ritualistic behaviors can be seen at certain stages of the child's development. As examples, children developing normally may be fascinated with symmetry or superstitions for periods of time.

Early onset psychosis — Although patient insight into the unreal nature of his or her obsessions can distinguish OCD from psychotic disorders in adults, this is not necessarily true in children. The failure of treatment to respond to OCD treatment can be indicative of psychosis. (See 'Clinical manifestations' above and "Obsessive-compulsive disorder in children and adolescents: Treatment overview".)

Autism spectrum disorders — Obsessions and compulsive behaviors can occur as part of autism spectrum disorders. While OCD patients often experience their symptoms as ego-dystonic, typically, the repetitive and stereotypic behaviors seen in autism spectrum disorders are not found to be excessive or problematic [77].

Tic disorder — Some tics can be similar to compulsions. A complex motor tic, such as repeating actions a specific number of times, can be indistinguishable from compulsions [99,100]. To differentiate between the two, the clinician should explore whether there is simply an urge or uncomfortable feeling preceding the movement (as with tics), or if the action was preceded by a specific anxious thought, feeling of doom, or fear that something bad might happen which triggers the behavior, as with OCD.

Other — Other mental disorders have clinical features that may overlap with OCD on presentation, but can be distinguished from OCD based on other features of the disorder; these are described in Criteria D of DSM-5. (See 'DSM-5 criteria' above.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Obsessive-compulsive disorder and related disorders".)

SUMMARY AND RECOMMENDATIONS

- Obsessive-compulsive disorder (OCD) is a severe, prevalent and most often chronically debilitating disorder characterized by repetitive, ritualistic and distressing thoughts, ideas, and behaviors over which a person typically has very little if any control. (See 'Introduction' above.)

- OCD affects more than three million people in the United States. Estimates of the disorder's United States lifetime prevalence in both pediatric and adult populations have ranged from 1 to 3 percent. More than half of pediatric patients with OCD have been found to have at least one comorbid psychiatric disorder, including tic disorder, attention deficit hyperactivity disorder, and mood and anxiety disorders. (See 'Epidemiology' above.)

- Research suggests that multiple genetic risk factors lead to changes in cellular function or altered neurotransmitter signaling. These molecular and cellular changes perturb fronto-striatal-thalamic circuitry (FSTC), resulting in OCD. (See 'Pathogenesis' above.)

- The characteristic symptoms of obsessive-compulsive disorder are obsessions and/or compulsions that are time-consuming, cause clinically significant distress or impair functioning. Young children, compared to older children and adults, are less likely (and able to) describe obsessions in conjunction with observed compulsive actions, and less likely to have insight into the excessiveness or absurdity of their OCD symptoms. (See 'Clinical manifestations' above.)

- Research suggests that approximately 50 percent of all cases of OCD have their onset in childhood or adolescence. The prognosis for pediatric OCD appears to be better than the prognosis for adult onset OCD. (See 'Course' above.)

- It is particularly important that assessment for obsessions and compulsions evaluates children's lifetime, because OCD symptoms can wax and wane and change during the child's developmental maturation. (See 'Assessment' above.)

- The differential diagnosis of childhood OCD includes normal childhood development, early onset psychosis, autism spectrum disorders, and tic disorder. (See 'Diagnosis' above.)

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