Sleep Medicine 56 (2019) 104–110

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Insomnia in pediatric obsessive–compulsive disorder: prevalence and association with multimodal treatment outcomes in a naturalistic clinical setting

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ARTICLE INFO

Article history:
Received 19 September 2018
Received in revised form
28 November 2018
Accepted 8 December 2018
Available online 30 January 2019

Keywords:
Obsessive-compulsive disorder
Insomnia
Cognitive-behavior therapy
Children
Adolescents

ABSTRACT

Objective: Little is known about the prevalence and impact of insomnia on clinical outcomes in youth with obsessive–compulsive disorder (OCD). This study aimed to investigate this subject.
Patients/methods: A total of 193 patients from a specialist pediatric OCD clinic completed a range of diagnostic and clinical measures, including the Insomnia Severity Index (ISI). Patients scoring above a previously validated cut-off on the ISI (score ≥9) were compared to the rest of the sample on socio-demographic and clinical characteristics. In a subsample of 143 (from the initial 193) patients who were treated at the clinic, a mixed-model analysis of variance (ANOVA) was used to compare the outcomes of multimodal OCD treatment in the insomnia ($N = 60$) vs no insomnia ($N = 83$) groups. The primary outcome measure was the clinician-administered Children's Yale–Brown Obsessive–Compulsive Scale (CY-BOCS) at post-treatment and at three-month follow-up.
Results: The psychometric properties of the ISI in our sample were excellent. At baseline, 42% (81/193) of the sample scored above the ISI cut-off for clinical insomnia. These participants had significantly higher OCD severity, higher rates of psychiatric comorbidities, more severe depressive symptoms, poorer general functioning, and were more likely to take sleep medications, compared to those who scored below the ISI cut-off. In the treated subsample, while the insomnia group remained more severely affected through the three time-points, both groups improved similarly on the CY-BOCS at post-treatment and at three-month follow-up.
Conclusion: Insomnia is relatively common in pediatric OCD and is associated with more severe psychopathology. However, with adequate multimodal, evidence-based treatment, these patients can improve as much as those without insomnia.

1. Introduction

Sleep is a biological process necessary for the maintenance of both physical and mental health. Reduced sleep in healthy children has been linked to impairment in cognitive and emotional functioning [1]. Shortened sleep duration has been associated with low performance on intelligence quotient measures [2] and with impairment in verbal and nonverbal cognitive abilities [3] in healthy school-age children. An interaction between sleep and psychopathology also seems to exist, and disrupted sleep has been shown to affect the course, severity, and prognosis of different psychiatric disorders in children [4]. For example, pediatric patients diagnosed with a major depressive episode who also experienced sleep disturbances were found to be more severely depressed than those without sleep disturbances [5], and poor sleep in children with autism was associated with higher parent ratings of hyperactive-impulsive behaviors, as well as oppositional behaviors [6]. Insomnia is also common in youth with anxiety disorders, and may be associated with more severe anxiety symptoms and more

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https://doi.org/10.1016/j.sleep.2018.12.024
1389-9457/

interference in family functioning [7]. Greater amounts of rapid eye movement sleep have been related to more somatic complaints in children with generalized anxiety disorder, when compared to healthy controls [8].

Notably, little is known about sleep difficulties in general, and insomnia in particular, in young people with obsessive–compulsive disorder (OCD), a relatively frequent and debilitating psychiatric condition that tends to start at a young age [9]. The scarce literature available suggests that adults with OCD show alterations in some objective sleep patterns. Specifically, adults with OCD have shown shorter sleep duration, increased wake after sleep onset, and late sleep onset and offset, compared to healthy controls [10,11]. Regarding the latter, a high prevalence of delayed sleep phase disorder has been found in OCD samples [11]. Furthermore, delayed bedtimes have been associated with a higher prevalence of obsessive-compulsive symptoms in non-clinical samples [12].

In pediatric OCD samples, children have shown reduced total sleep time (TST) and longer awake periods after sleep onset [13], and adolescents with OCD have been reported to present reduced TST, reduced non-REM sleep time, and longer sleep onset latencies when compared to healthy controls [14]. The small number of studies assessing subjective sleep disturbances in children with OCD have yielded mixed and inconclusive results. An observational study comparing 185 children and adolescents with OCD, 177 non-OCD patients from a child psychiatric outpatient clinic, and a group of 1369 healthy controls of the same age found significantly higher rates of sleep problems – reported by parents and measured by selected items from the Child Behavior Checklist (CBCL) – in the OCD and in the psychiatric outpatient groups, compared with healthy controls [15]. The same study also showed that, in the OCD group, other comorbid symptoms measured by the CBCL (eg, thought problems, somatic symptoms, anxious/depressed, and aggressive behavior), predicted sleep problems to a greater extent than OCD itself [15]. In another cohort study including 66 children with OCD, the number of sleep-related problems measured by a composite of items extracted from the CBCL, the Multidimensional Anxiety Scale for Children, and the Children's Depression Inventory was positively related to OCD severity, measured by the Children's Yale–Brown Obsessive–Compulsive Scale (CY-BOCS) [16]. By contrast, another case–control study including 30 patients with OCD and 30 matched controls did not find a correlation between the sleep disturbances measured by the parent-reported Sleep Disturbances Scale for Children and OCD severity [17]. A larger study including 269 children and adolescents with OCD showed that more than two-thirds of the sample had at least one mild sleep problem, using a sleep composite score based on the CBCL [18]. This is also the only observational study to date to explore whether sleep difficulties are associated with clinical outcomes in pediatric OCD. Specifically, the authors reported that sleep problems at baseline predicted worse outcomes of cognitive-behavior therapy (CBT) for OCD [18]. This is a potentially important finding that requires replication because it may indicate that patients with sleep difficulties may require additional interventions to fully benefit from CBT, the mainstream treatment for pediatric OCD.

One important limitation of previous studies in pediatric OCD is that they have primarily employed parent-reported measures, which were often derived from other scales, rather than specifically developed and validated sleep scales. In an attempt to overcome some of the limitations of the existing literature and extend our understanding of the prevalence and impact of sleep difficulties in this group, this study aimed to: (1) explore the extent to which pediatric patients with OCD referred to a specialized clinic suffer from clinical insomnia, according to a self-reported, sleep-specific measure; (2), compare the demographic and clinical characteristics of pediatric OCD patients with and without insomnia; and (3) explore whether insomnia is associated with poorer multimodal treatment outcomes in this patient group.

2. Methods

2.1. Clinical setting

All study participants were recruited from a specialist pediatric OCD and related disorders outpatient clinic in Stockholm, Sweden. The clinic receives referrals from Child and Adolescent Mental Health Services (CAMHS) and pediatric services primarily across the entire Stockholm region, and occasionally from other Swedish regions and Nordic countries. All patients and their parents/legal guardians routinely fill in questionnaires before their first appointment with the multidisciplinary clinical team, comprised of child psychiatrists, clinical psychologists, and nurses. This information is then used to conduct a more focused and efficient face-to-face diagnostic assessment. In the first 3-h appointment, detailed sociodemographic and clinical information is gathered from the patients and their parents, and clinical diagnoses are made according to ICD-10 and DSM-5 criteria [19,20] using semi-structured instruments, including the Mini-International Neuropsychiatric Interview for Children and Adolescents (MINI-KID) [21]. After the assessment, patients are either offered treatment at the clinic or referred to more appropriate services. For all patients undertaking treatment at the clinic, all clinical measurements are repeated at post-treatment and at several fixed follow-up times, set at 3, 6, and 12 months after the end of the treatment. All patients assessed at the clinic are routinely asked for consent to participate in research studies.

2.2. Participants

Participants were 193 children and adolescents consecutively referred to our specialist OCD and related disorders clinic between January 2015 and March 2018, meeting ICD-10 and DSM-5 criteria for OCD, and who consented to using their routinely collected clinical data for research purposes. Table 1 shows the sample characteristics at baseline for all the study participants. The sample (107 girls, 55.4%) had a mean age of 13.6 years (standard deviation (SD) = 2.4; range 6–17). The self-reported mean age of onset of their OCD was 10.8 years (SD = 2.8; range 3–17). The mean CY-BOCS score was 22.6 (SD = 4.4; range 11–33), indicating moderately severe OCD. Almost half of the sample (N = 93, 48.2%) had additional psychiatric diagnoses. One hundred and forty-three participants (74.1%) received treatment for their OCD at the clinic, while the remaining patients were referred elsewhere.

All young people and their parents gave written consent to participate in the current study, which was approved by the Regional Ethical Review Board in Stockholm (reference number 2015/1977–31/4).

2.3. Measures

All measures listed below were completed at baseline by all study participants (N = 193). Additionally, for the sample of patients treated at the clinic (N = 143), assessments were also performed at post-treatment and at three-month follow-up. Data from the 6- and 12-month follow-ups were not used in the current study as many patients are still in follow-up.

The Insomnia Severity Index (ISI) is a self-report instrument developed for the measurement of insomnia. It targets not only the symptoms but also the consequences of insomnia, as well as the degree of disturbance caused by the sleep impairment. It comprises seven Likert-type items ranging from 0 to 4, with a maximum total

Table 1
Baseline demographic and clinical characteristics of pediatric obsessive–compulsive disorder patients with and without clinical insomnia ($N = 193$).

Demographics	All participants ($N = 193$)		Clinical insomnia ($N = 81$)		No clinical insomnia (N = 112)		Statistic	p
	N	%	*N*	%	*N*	%	Chi-squared	
Girls	107	55.4	51	63.0	56	50.0	3.70	0.157
Family history of OCD	73	37.8	31	38.3	42	37.5	0.01	0.913
Any comorbid mental disorder	93	48.2	49	60.5	44	39.3	8.47[b]	0.004
ADHD	45	23.3	21	25.9	24	21.4	0.53	0.466
ASD	38	19.7	17	21.0	21	18.8	0.15	0.700
Depression	25	13.0	19	23.5	6	5.4	13.66[c]	0.000
Bipolar disorder	1	0.5	1	1.2	0	0	–	0.420[a]
Anxiety disorders	14	7.3	7	8.6	7	6.3	0.40	0.527
Tic disorders	12	6.2	4	4.9	8	7.1	0.39	0.531
Previous CBT treatment	88	45.6	31	38.3	57	50.9	3.02	0.082
On pharmacological treatment	84	43.5	42	51.9	42	37.5	3.94[b]	0.047
SRIs	53	27.5	23	28.4	30	26.8	0.06	0.805
Other antidepressants	1	0.5	1	1.2	0	0	–	0.420[a]
Antipsychotics	11	5.7	6	3.1	5	2.6	–	0.531[a]
Melatonin	26	13.5	21	25.9	5	4.5	18.57[c]	0.000
Antihistamines ($N = 192$)	18	9.4	9	11.1	9	8.0	0.53	0.468
Zolpidem	1	0.5	1	1.2	0	0	–	0.420[a]
Benzodiazepines ($N = 192$)	1	0.5	1	1.2	0	0	–	0.420[a]
ADHD medication	24	12.4	11	13.6	13	11.6	0.17	0.682
Others	1	0.5	1	1.2	0	0	–	0.420[a]
	Mean	SD	Mean	SD	Mean	SD	Student's *t*	*p*
Age at assessment	13.6	2.4	13.9	2.4	13.3	2.4	−1.86	0.064
Age of OCD onset ($n = 180$)	10.8	2.8	11.1	2.7	10.6	2.8	−1.09	0.279
Insomnia measure	Mean	SD	Mean	SD	Mean	SD	Student's *t*	*p*
ISI total	7.6	5.9	13.4	4.0	3.5	2.7	−19.63[c]	0.000
OCD measures	Mean	SD	Mean	SD	Mean	SD	Student's *t*	*p*
CY-BOCS								
CY-BOCS obsessions	11.2	2.3	11.6	2.1	10.9	2.3	−2.25[b]	0.026
CY-BOCS compulsions	11.3	2.4	11.9	2.1	10.9	2.5	−2.90[c]	0.004
CY-BOCS total	22.6	4.4	23.6	4.0	21.8	4.5	−2.72[c]	0.007
OCI-CV								
OCI-CV total	18.7	7.7	21.6	7.7	16.7	7.1	−4.56[c]	0.000
Other clinical measures	Mean	SD	Mean	SD	Mean	SD	Student's *t*	*p*
CDI-S ($N = 186$)	6.1	4.3	8.5	4.1	4.3	3.5	−7.48[c]	0.000
CGI-S ($N = 192$)	4.3	0.7	4.4	0.7	4.2	0.7	−2.02[b]	0.045
CGAS	50.7	5.9	49.4	5.7	51.6	5.8	2.57[b]	0.011

ADHD, attention-deficit/hyperactivity disorder; ASD, autism spectrum disorders; CBT, cognitive behavioral therapy; CDI-S, Children's Depression Inventory – Short Version; CGAS, Children's Global Assessment Scale; CGI-S, Clinical Global Impression – Severity; CY-BOCS, Children's Yale-Brown Obsessive–Compulsive Scale; ISI, Insomnia Severity Index; OCD, obsessive–compulsive disorder; OCI-CV, Obsessive–Compulsive Inventory – Child Version; SD, standard deviation; SRIs, serotonin reuptake inhibitors.
[a] Fisher test.
[b] Significant at 0.05.
[c] Significant at 0.01.

score of 28. The ISI is considered suitable for screening purposes and for measuring change over time [22]. The optimal cut-off score for clinically significant insomnia in adolescents has been established to be 9 [23]. In this study, we used the Swedish translation of the ISI, which has been adapted for use amongst young patients [24]. This version has shown adequate internal consistency and good correspondence with other measures of insomnia [24]. Using the previously established cut-off in young people [23], the OCD sample was divided into a clinical insomnia group (ISI scores $\geq$9) and a no clinical insomnia group (ISI scores <9).

The CY-BOCS is a clinician-rated, semi-structured interview used to assess the severity of the OCD symptoms [25]. It consists of five severity items for both obsessions and compulsions, with a total OCD severity score ranging from 0 to 40. The CY-BOCS has shown good reliability as well as good convergent and discriminant validity [26] and is the gold standard outcome measure in clinical trials of OCD.

The Obsessive–Compulsive Inventory – Child version (OCI-CV) is a self-reported measure of OCD symptom severity in children and adolescents. In addition to a total score, this multidimensional measure includes the following subscales: doubting/checking, obsessing, hoarding, washing, ordering, and neutralizing. Both the total score and the subscales of the OCI-CV have good internal consistency and test–retest reliability, as well as good convergent and discriminant validity [27].

The Children's Depression Inventory – Short Version (CDI–S) is a 10-item short version of the CDI assessing the presence and severity of depressive symptoms in children and adolescents [28].

The Clinical Global Impression-Severity (CGI-S) is a one-item clinician rating of symptom severity. It is rated on a seven-point scale ranging from 1 (no symptoms) to 7 (extremely severe symptoms) [29]. The Clinical Global Impression-Improvement (CGI-I) is a one-item clinician rating of symptom severity change from the baseline assessment. This scale ranges from 1 (very much improved) to 7 (very much worse) [30].

The Children's Global Assessment Scale (CGAS) is an adaptation of the Global Assessment Scale (GAS) designed to reflect the lowest level of functioning of a child or adolescent during a certain period of time. It ranges from 1 (more impaired) to 100 (best functioning) [31].

Additionally, following a consensus statement of international experts [32], 'treatment response' was defined as a 35% or greater drop on the CY-BOCS and a CGI-I score of 1 or 2, and 'remission' as a score of ≤12 on the C-YBOCS and a CGI-S rating of 1 or 2 [32].

2.4. Treatment

All patients treated at the clinic were offered a course of CBT and a subset of cases were additionally treated with medication, when deemed clinically relevant. The CBT was protocol-driven and focused on exposure with response prevention (ERP), with parental involvement [33,34]. Treatment was delivered by clinical psychologists with extensive experience in treating pediatric OCD. The treatment protocol included the following key components which, in most cases, were delivered in 12–14 sessions: psychoeducation about OCD and anxiety (two sessions), development of an ERP hierarchy and graded ERP (10 sessions), and relapse prevention (two sessions) [33,34]. However, the number of sessions could be adapted depending on the patient's severity and response to the intervention. Usually sessions lasted 1 h and were conducted weekly. Alternately, complex cases could be offered intensive (eg, several hours per day) clinic-based or home-based sessions (for homebound patients). Parental involvement in the treatment was encouraged in all cases, and the reduction of parental accommodation was often a specific treatment target. Homework tasks were assigned between sessions, which mainly consisted of encouraging daily practice of ERP tasks.

Patients with comorbid autism spectrum disorders (ASD) followed a modified protocol including additional parental education, a longer psychoeducation phase, use of concrete visual materials, use of examples and metaphors linked to the young persons' special interests, a higher number of therapy sessions and the involvement of additional therapists, when required [35]. No specific protocol-driven interventions were directed towards insomnia, although the clinic's treatment protocol for OCD cases with comorbid ASD includes an ancillary sleep hygiene module which is incorporated in the psychoeducation sessions. This module includes general sleep hygiene recommendations, such as encouraging patients to be outdoors and active during the day, not using smartphones or other electronic gadgets before bedtime, dimming the light about 1 h before bedtime, going to bed at the same time every day, or avoiding drinking coffee, tea, or energetic drinks before going to bed.

2.5. Statistical analyses

Data were analyzed using SPSS version 25.0 for Windows. Chi-squared and Fisher tests were used for between-group comparisons of categorical variables and Student's *t*-tests for continuous variables.

The psychometric properties of the ISI were examined in the current sample. Internal consistency was evaluated using Cronbach's alpha. The construct validity was assessed in two ways. First, the factor structure of the ISI was examined using principal component analysis (PCA). Second, Pearson's correlations were used to explore the convergence/divergence of the ISI with the above-described instruments. Following Cohen's classification, large correlations were defined as greater or equal to 0.50, medium correlations between 0.30 and 0.49, and small correlations from 0.10 to 0.29 [36].

A mixed-model analyses of variance (ANOVA) was used to test for a differential effect of group (clinical insomnia/no clinical insomnia) on OCD symptom improvement. The significance level was set at $p < 0.05$ (two-tailed). Different sample sizes may have been used in the analyses due to missing data.

3. Results

3.1. Psychometric properties of the ISI

The mean ISI score at baseline was 7.6 (SD = 5.9). The ISI showed good internal consistency, with a Cronbach's alpha value of 0.85. The PCA revealed a single factor solution. This factor had an eigenvalue of 3.74, accounting for 53.4% of the variance (Supplementary Table S1; Supplementary Fig. S1). Table 2 shows the pattern of correlations between the ISI and other measures of OCD severity, depression, and general functioning, all in the small to medium range.

3.2. Comparison of patients with and without clinical insomnia at baseline

According to previously established cut-offs in young people [23], a total of 81 out of 193 participants (42%) were considered to meet criteria for clinical insomnia at baseline. Demographic and clinical characteristics of the groups with and without insomnia are shown in Table 1. There were no significant between-group differences in terms of sex, age, age at onset of OCD, family history of

Table 2
Correlational analyses between the Insomnia Severity Index and other study measures.

		ISI	CY-BOCS	OCI-CV	CDI-S	CGAS
CY-BOCS	Pearson correlation	0.20[b]	1			
	p	0.005				
	N	193	193			
OCI-CV	Pearson correlation	0.34[b]	0.21[b]	1		
	p	0.000	0.003			
	N	191	191	191		
CDI-S	Pearson correlation	0.45[b]	0.31[b]	0.36[b]	1	
	p	0.000	0.000	0.000		
	N	186	186	184	186	
CGAS	Pearson correlation	−0.19[b]	−0.59[b]	−0.18[a]	−0.39[b]	1
	p	0.010	0.000	0.013	0.000	
	N	193	193	191	186	193
CGI-S	Pearson correlation	0.13	0.74[b]	0.21[b]	0.28[b]	−0.50[b]
	p	0.066	0.000	0.004	0.000	0.000
	N	192	192	190	185	192

CDI-S, Children's Depression Inventory – Short Version; CGAS, Children's Global Assessment Scale; CGI-S, Clinical Global Impression – Severity; CY-BOCS, Children's Yale–Brown Obsessive–Compulsive Scale; ISI, Insomnia Severity Index; OCI-CV, Obsessive Compulsive Inventory – Child Version.
[a] Significant at 0.05.
[b] Significant at 0.01.

OCD, or receipt of previous CBT. However, the presence of any comorbid mental disorder was more frequent in the insomnia group ($p = 0.004$), specifically depression ($p = 0.000$).

Participants with clinical insomnia scored significantly higher on all measures of OCD severity (CY-BOCS, OCI-CV, and CGI-S) and depressive symptoms (CDI-S). They also had lower scores on the CGAS, indicating worse general functioning (Table 1). A higher proportion of patients with insomnia were on pharmacological treatment ($p = 0.047$); this finding was driven by the higher rate of melatonin prescriptions in the insomnia group, compared to the no insomnia group (25.9% vs 4.5%, respectively).

3.3. Treatment characteristics and outcomes of OCD patients with and without insomnia

A total of 143 patients had treatment at the clinic and provided outcome data. Sixty of the 143 treated patients (42%) scored above the ISI cut-off, corresponding to clinical insomnia. All received CBT, and those with insomnia ($N = 60$) received more CBT sessions than those without insomnia ($N = 83$; 14.7 vs 12.0 sessions, respectively; Student's $t = -2.16$, $p = 0.033$).

Forty-five (31.5%) of the participants treated with CBT were also receiving medication for their OCD (mainly serotonin reuptake inhibitors). Of those, eight patients also received treatment with antipsychotics (mainly risperidone and aripiprazole) as an augmentation strategy. There were no significant differences in baseline OCD severity between those who received OCD medication, compared to those who did not (22.96 vs 22.13 on the CY-BOCS, respectively; Student's $t = -1.00$, $p = 0.321$). Those who received medication for their OCD scored similarly on the ISI at baseline to those that did not receive it (7.93 vs 7.53, respectively; Student's $t = -0.35$, $p = 0.730$).

Additionally, 24 of the 143 treated participants (16.8%) received medication for insomnia. Of these, 17 (11.9%) were on melatonin, 11 (7.7%) on antihistamines as sedatives, and one (0.7%) patient was on zolpidem. There were no significant differences in OCD severity at baseline between those who received sedative medication compared to the ones who did not (22.63 *vs* 22.34 on the CY-BOCS, respectively; Student's $t = -0.27$, $p = 0.786$). As expected, patients who received sedative medication scored significantly higher on the ISI (11.79 *vs* 6.82, respectively; Student's $t = -3.91$, $p = 0.000$).

The ISI, CY-BOCS, and OCI-CV mean scores for the groups with and without clinical insomnia at all time-points (baseline, post-treatment, and three-month follow-up) are shown in Table 3.

The mean percentage reduction in the total CY-BOCS score from baseline to post-treatment was 55.4% for the insomnia group and 62.5% in the non-insomnia group; corresponding reductions from baseline to the three-month follow-up were 62.8% and 71.8%, respectively. There was no significant correlation between the ISI score at baseline and the percentage reduction on the CY-BOCS from baseline to post-treatment ($r = -0.09$, $p = 0.294$) or from baseline to the three-month follow-up ($r = -0.15$, $p = 0.136$).

A total of 101 children and adolescents with available data in the three time points were used in a mixed-model ANOVA with time (baseline vs post-treatment vs three-month follow-up) as the within-subjects factor and group (with vs without clinical insomnia) as the between-subjects factor. The model revealed a main effect of time ($F(1.77, 174.88) = 345.61$, $p < 0.001$), indicating a significant reduction in the CY-BOCS total score over time. A significant main group effect ($F(1, 99) = 15.18$, $p < 0.001$) indicated that the insomnia group had more severe OCD symptoms, compared to the non-insomnia group. However, there was no significant time by group interaction effect ($F(1.77, 174.88) = 0.024$, $p = 0.966$), indicating that the two groups improved similarly with treatment (Fig. 1).

In order to investigate potential effects of the medication for insomnia, we next introduced medication for insomnia status as an additional factor in the mixed-model ANOVA. The results indicated significant main effects of time ($F(1.76, 170.43) = 176.76$, $p < 0.001$) and group ($F(1, 97) = 11.51$, $p = 0.001$), but no significant main effect of medication ($F(1, 97) = 0.785$, p=0.378), and no significant time by group ($F(1.76, 170.43) = 0.006$, $p = 0.989$), time by

Table 3
Mean Insomnia Severity Index (ISI), Children's Yale–Brown Obsessive–Compulsive Scale (CY-BOCS), and Obsessive–Compulsive Inventory – Child Version (OCI-CV) scores in treated study patients ($N = 143$) with and without clinical insomnia at baseline, post-treatment, and three-month follow-up.

	Clinical insomnia ($N = 60$)		No clinical insomnia ($N = 83$)		Statistic *t*-test	*p*
	Mean	SD	Mean	SD		
ISI total						
Baseline	13.4	4.0	3.5	2.9	−16.45[b]	0.000
Post-treatment	7.1	5.3	3.2	3.8	−3.97[b]	0.000
3-month follow-up	7.4	4.5	2.1	2.8	−6.56[b]	0.000
CY-BOCS total						
Baseline	23.9	4.1	21.3	4.6	−3.52[b]	0.001
Post-treatment	10.4	5.4	7.8	5.6	−2.85[b]	0.005
3-month follow-up	8.7	5.9	5.9	6.3	−2.27[a]	0.026
CY-BOCS obsessions						
Baseline	11.8	2.2	10.6	2.4	−3.11[b]	0.002
Post-treatment	5.4	2.8	3.9	2.9	−3.16[b]	0.002
3-month follow-up	4.4	3.0	3.0	3.2	−2.33[a]	0.022
CY-BOCS compulsions						
Baseline	12.1	2.1	10.7	2.5	−3.59[b]	0.000
Post-treatment	5.0	2.8	3.9	2.8	−2.36[a]	0.020
3-month follow-up	4.3	3.0	2.9	3.3	−2.09[a]	0.039
OCI-CV						
Baseline	21.5	8.2	16.3	6.9	−4.16[b]	0.000
Post-treatment	11.5	6.4	8.6	7.6	−2.07[a]	0.041
3-month follow-up	11.9	7.9	7.2	6.4	−3.20[b]	0.002

SD, standard deviation.
[a] Significant at 0.05.
[b] Significant at 0.01.

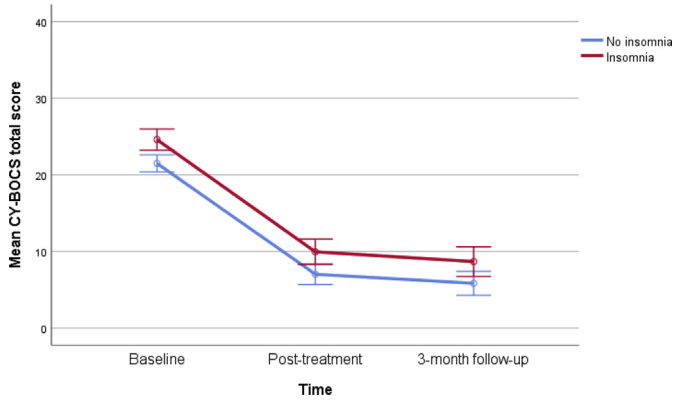

Fig. 1. Mean Children's Yale–Brown Obsessive–Compulsive Scale (CY-BOCS) total scores at each time point (baseline, post-treatment, and three-month follow-up), by insomnia status. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

medication ($F(1.76, 170.43) = 0.892$, $p = 0.400$), or time by group by medication ($F(1.76, 170.43) = 0.077$, $p = 0.904$) interactions.

Because we found significant differences between groups in depressive symptoms at baseline and in the number of sessions received, we ran a second model adjusting for these two variables. When adding these two covariates, the time by group interaction effect remained non-significant ($F(1.76, 170.99) = 0.233$, $p = 0.765$), but the main group effect disappeared ($F(1, 97) = 2.36$, $p = 0.128$).

In addition, we ran a sensitivity analysis excluding all those patients with comorbid ASD who undertook treatment at the clinic ($N = 23$; 16.1%). Results remained unchanged overall (time by group interaction effect: $F(1.71, 137.04) = 0.045$, p=0.936).

The proportion of treatment responders at post-treatment was 78.3% in the insomnia group and 79.3% in the non-insomnia group (Chi-squared = 0.02, $p = 0.893$). Similarly, the proportion of patients classed as being in remission at post-treatment was 56.7% in the insomnia group and 66.3% in the non-insomnia group (Chi-squared = 1.37, $p = 0.243$).

Of note, symptoms of insomnia, as measured by the ISI, improved during the course of the treatment in the whole group (effect of time: $F(1.68, 130.62) = 19.13$, $p < 0.001$). This improvement was more notable in the insomnia group than in the non-insomnia group (time by group interaction effect: $F(1.85, 142.23) = 22.89$, $p < 0.001$) (Table 3). When patients with comorbid ASD were removed, results remained overall unchanged (effect of time: $F(1.64, 110.16) = 15.86$, $p < 0.001$ and time by group interaction effect: $F(1.81, 119.51) = 21.56$, $p < 0.001$).

4. Discussion

This is, to our knowledge, the first study examining the prevalence of insomnia in a large sample of pediatric OCD patients using a self-reported, sleep-specific scale. We found that 42% of children and adolescents with OCD reached the cut-off for clinical insomnia on the ISI. Insomnia was not only common in our sample, but it was also associated with more severe psychopathology and lower general functioning. However, despite having higher OCD symptom severity throughout the study, patients with insomnia improved to a similar degree than patients without insomnia, and similar proportions were classed as treatment responders and remitters by the end of the treatment.

The prevalence of insomnia in children and adolescents with OCD had been examined in a handful of previous studies but their results were difficult to reconcile due to marked methodological differences and the use of a heterogeneous range of composite measures to measure sleep difficulties. For example, Jaspers-Fayer et al., [17] reported that 72% of their OCD participants scored above the cut-off for clinically disturbed sleep on the Sleep Disturbances Scale for Children, but this instrument is a broad sleep measure which captures a range of sleep-related problems, including insomnia. Similarly, Ivarsson et al., [18] found that the prevalence of mild sleep problems was 68.3% in a pediatric OCD sample, although this percentage included symptoms as heterogeneous as nightmares, talking in sleep, or 'sleeping more'. When looking at those sleep problems separately, the percentage of patients experiencing 'trouble sleeping' and 'sleeping less' was 40% and 29.5%, respectively; figures which are closer to those found in our study using a well-validated insomnia instrument. The psychometric properties of the ISI in our sample were excellent, with high internal consistency, a single factor structure, and construct validity.

Young patients with insomnia in our sample were more frequently on pharmacological treatment and had higher rates of comorbid mental disorders (specifically depression). They were more severely affected by obsessive–compulsive symptoms, depressive symptoms, and showed a lower level of global functioning when compared with patients without insomnia. We found a significant positive association between sleep impairment and the severity of obsessive-compulsive symptoms, although the correlation between the ISI and the CY-BOCS was modest. These results are in the same line as previous findings in young [13] and adult patients [37,38] that found positive associations between an objectively measured decrease in total sleep time [13,37] and a subjectively measured disturbance in sleep [38] with OCD severity. As it could be expected, the ISI's strongest correlation was with depressive symptoms, as measured by the CDI-S, despite the fact that this measure does not include any sleep-specific items.

Unlike Ivarsson et al. [18], who concluded that sleep problems might have a negative impact on CBT efficacy, we found that patients with insomnia were just as likely to respond to developmentally appropriate CBT as were patients without insomnia. The results remained unaltered after adjusting for baseline depression severity, number of CBT sessions, prescription of drugs for insomnia, and in a sensitivity analysis excluding patients with comorbid ASD who had received a specific sleep hygiene module as part of their treatment package.

There are some important differences between our study and that of Ivarsson et al., [18] that are worth considering when interpreting the results of both studies. First, Ivarsson et al., [18] reanalyzed data from a clinical trial evaluating CBT intervention for OCD using a strict protocol. Conversely, our study was conducted in a naturalistic setting where the multidisciplinary team had more flexibility to offer additional interventions, such as brief sleep hygiene advice or medication (including sedatives), or to deliver a larger number of CBT sessions (as was the case for the group with clinical insomnia), as required by the individual patient care plan. Thus, it may still be the case that insomnia interferes with the efficacy of pure CBT, but its detrimental effects on learning can be minimized if the treatment is delivered by a specialist multidisciplinary team in a naturalistic setting.

In our study, insomnia symptoms also improved significantly after treatment, although we cannot be sure whether this was due to the improvement in the OCD symptoms, additional interventions such as sleep hygiene advice or sleep medication, or a combination of these approaches. However, it is important to note that only a small proportion of cases in our sample (16.8%) received medication for their sleep problems.

This study addresses an important gap in the literature but it is not without limitations. First, the diagnosis of insomnia was made using the validated cut-off of a self-reported insomnia measure

[22,23], but structured diagnostic interviews for insomnia were not performed. Future studies would benefit from more structured assessment of diagnostic criteria for insomnia. Nonetheless, the clearly higher rate of sleep medication use in the insomnia group provided some concurrent validity to our grouping. Second, this study was conducted in a specialist clinic receiving referrals for relatively severe and/or treatment-refractory cases and, hence, the findings may not be generalizable to other samples and settings. However, many similarities were noted between the sample characteristics described here and in the previous literature. Third, results regarding the improvement in sleep symptoms should be interpreted cautiously due to the lack of control over potential sleep hygiene recommendations (not explicitly included in the OCD protocol) that the therapists could have provided during the treatment.

5. Conclusions

Insomnia is common among youth with OCD and its presence is associated with more severe obsessive–compulsive and depressive symptomatology. Yet, in our sample, insomnia did not seem to interfere with response to multimodal treatment for OCD, it should still be routinely evaluated and managed alongside the treatment of OCD.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2018.12.024.

Conflict of interest

D. Mataix-Cols and L. Fernández de la Cruz receive royalties for contributing articles to UpToDate, Wolters Kluwer Health. L. Sevilla-Cermeño was supported by a Fellowship from the Alicia Koplowitz Foundation. The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript; and decisions to submit the manuscript for publication.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2018.12.024.

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