In addition to the well-known cognitive impairment following traumatic brain injury (TBI), neuropsychiatric sequelae are often reported as well. Although not the most common neuropsychiatric consequence of TBI, obsessive-compulsive disorder (OCD) has been associated with TBI. However, diagnosing new onset OCD secondary to TBI is complicated by the potential for cognitive impairment secondary to TBI masquerading as OCD. In particular, memory difficulties and executive dysfunction may be confused as representing obsessions and compulsions. Research in this area, which could guide clinical practice, remains limited. In addition to using Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) criteria, neuropsychological testing and collateral interviews may help clinicians when considering differential diagnoses in this complex area of neuropsychiatry.

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Diagnosing OCD in the context of TBI is complicated due to the potential for overlapping symptomatology between conditions. Cognitive impairments associated with TBI clearly have the potential to mimic aspects of the clinical presentation of idiopathic OCD. For example, memory deficits following TBI may result in excessive checking of tasks, mirroring compulsions observed in OCD. Likewise, executive function impairments resulting in perseverative behavior may mimic the repetitive behavioral patterns typical of OCD. Similarly, poorer speed of information processing following TBI may be indistinguishable from obsessional slowness. Thus, disentangling whether particular symptoms better reflect the well-known cognitive impairments characteristic of TBI, or new-onset OCD, is challenging. Memory performance may be a more useful cognitive indicator to consider when diagnosing OCD secondary to TBI, and a diagnosis of OCD secondary to TBI should probably be questioned when OCD-type symptomatology occurs alongside significant deficits in memory.

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders outlines guidance for the diagnosis of neuropsychiatric comorbidities in TBI populations. According to DSM-V criteria, OCD symptomatology occurring secondary to a medical condition should be coded as “OCD due to Another Medical Condition.” Furthermore, it is possible to specify whether OCD-like symptoms, appearance preoccupations, hoarding symptoms, hair-pulling, or skin-picking symptoms predominate the clinical picture. As highlighted above, there is limited research evidence supporting a direct pathophysiological link between damage to specific brain structures and pathways following TBI, and subsequent OCD. Thus, medical indicators (e.g., functional and structural imaging techniques) may currently offer only modest utility in aiding diagnostic accuracy of OCD secondary to TBI. Accordingly, consideration of other factors not currently outlined in DSM-V may help to further confirm or reject a diagnosis of OCD post-TBI. For example, the fact that cognitive impairment commonly observed early post-injury has the potential to mirror OCD symptoms requires careful consideration.

Structured neuropsychological testing may help elucidate whether specific behaviors typically associated with OCD (i.e., excessive checking, compulsive and inflexible behavioral patterns) might better represent underlying cognitive impairment (e.g., memory, executive deficits) commonly observed following TBI. Moreover, as time since injury elapses and the temporal link between injury and OCD onset becomes more ambiguous, the potential impact of nonbiological factors upon clinical presentation should be considered. For example, environmental changes (e.g., change in living situation, disruption to established routines) or an increase in environmental demands (e.g., return to employment) may exacerbate post-injury memory difficulties and accompanying anxiety. Such environmental changes may lead to excessive checking, repetitive behaviors, and associated anxiety, potentially being mistaken for new-onset OCD. Indeed, anxiety post-TBI may be a psychological response to cognitive disability (e.g., memory difficulties) and the associated impairment in adaptive functioning, and not necessarily indicate the presence of “pure” OCD.
symptomatology. If OCD-like symptoms (eg, checking) after TBI are considered to actually reflect injury-related cognitive impairment (eg, memory), a diagnosis of “Major or Mild Neurocognitive Disorder due to TBI” should be considered.23

Despite much research in the area, in reality little is known about OCD secondary to TBI. Accurate prevalence data regarding OCD after TBI are lacking, as is our current understanding regarding key etiological mechanisms involved in the emergence of the disorder. The clinical phenomenology and cognitive functioning of TBI patients who report new-onset OCD are almost identical to idiopathic OCD samples, suggesting a possible association between both conditions, although the nature of this association remains unclear. DSM-V provides helpful guidance on diagnosing OCD in the context of a medical condition, but the potential for overlapping symptomatology between TBI and OCD makes reaching a diagnosis challenging. Ultimately, what is the relevance of what may appear to be an overly meticulous focus on diagnostic issues in a complex, multifactorial area of neuropsychiatric presentation? Accurate diagnostic formulation is the cornerstone of treatment plans. This is of course the case in brain injury rehabilitation also. “OCD after TBI,” which in fact represents cognitive impairment, may respond better to cognitive rehabilitation (including memory compensatory strategies), whereas “true OCD” associated with TBI, may require different treatment strategies, including pharmacological and psychological (for example, cognitive behavior therapy and response prevention) approaches.

Disclosures

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