Short Review

Mild Behavioral Impairment

Overview and Aspects of Forensic Psychiatry

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Abstract: Socially inappropriate behavior accompanies and modulates delinquency across the lifespan. In contrast to young people, the emergence of such traits among older individuals could indicate incipient neurodegenerative disease. Before developing a neurocognitive disorder, subtle behavioral changes may reflect a disintegration of neural networks involved in impulse control or social cognition. Whereas psychiatric evaluation often considers a comprehensive cognitive assessment, unremarkable results may discourage clinicians from recognizing brain disease underlying behavioral disturbance. We first provide an overview of its manifestations and neural correlates and the interrelations with mild cognitive impairment (MCI) before demonstrating how to investigate and diagnose mild behavioral impairment (MBI). Finally, we show how to appreciate MBI in geriatric forensic psychiatry.

Keywords: mild behavioral impairment, neurodegeneration, geriatric forensic psychiatry

Introduction

In the course of neurodegenerative diseases, we conceptualize mild cognitive impairment (MCI) as a possible predementia stage, itself preceded by the presence of neuropathology not (yet) interfering with cognition. Although clinicians may intuitively associate the presence of neuropsychiatric symptoms with a later-stage major neurocognitive disorder, there is an approximately 30% prevalence of depressive symptoms, apathy, and irritability already in patients with MCI (Lyketsos et al., 2002). However, mild behavioral impairment (MBI) could even be the first sign of a neurodegenerative disease, before cognitive deterioration (Creese et al., 2019). Initially introduced to describe early behavioral changes in patients with frontotemporal lobar degeneration (Scholzel-Dorenbos, 2006; Taragano & Allegri, 2003), we now know that MBI is not limited to these patients and can be considered a risk factor for developing dementia across a wider spectrum of neurodegenerative disorders, particularly among individuals without cognitive impairment (Taragano et al., 2009). In addition to other risk factors, MBI may therefore enrich patient samples at particularly high risk for cognitive decline (Creese & Ismail, 2022; Ismail et al., 2021).

The definition of specific criteria for MBI (Ismail et al., 2016) enables clinicians to consider a standardized approach to investigating heterogeneous neuropsychiatric symptoms in the absence of dementia (Martin & Velayudhan, 2020). Furthermore, MBI subcategories recognize the social domain of intrapersonal behavior changes (Ismail et al., 2016). This is interesting because how an individual interacts with the environment or other people could extend beyond unusual behavior and elicit caregiver burden (Sheikh et al., 2018) into violating social norms and delinquency. However, recognizing the neuropathology (Liljegren et al., 2019) underlying or modulating socially inappropriate and criminal behavior implicitly requires acknowledging the entire temporal continuum of neurodegenerative diseases, thus including individuals not (yet) suffering from cognitive changes.

This narrative review presents the range of MBI symptoms, their neural correlates, and how to investigate and operationalize them. It emphasizes forensic geriatric psychiatry. Irrespective of its legal appreciation within different judicial systems, MBI should be recognized in older individuals with criminal behavior.

Literature Review

We first conducted a PubMed search for the search term "(mild behavioral impairment[Title/Abstract]) OR (mild behavioural impairment[Title/Abstract])," which yielded 122 results as of December 2022. We additionally investigated the search term "((neuropsychiatric symptoms[Title/Abstract]) OR (behavioral symptoms[Title/Abstract]) OR (behavioural symptoms[Title/Abstract]) OR (non-cognitive symptoms)[Title/Abstract])) AND ((mild cognitive impairment[Title/Abstract]) OR (prodromal dementia[Title/Abstract]))" and examined the reference lists of previous reviews focusing on MBI (Creese & Ismail, 2022; Ismail et al., 2018; Jiang et al., 2022; Taragano et al., 2008).

Clinical Presentation and Diagnostic Criteria

One can conceptualize MBI as a means of describing the late-life onset of psychiatric symptoms as an early manifestation of neurodegenerative disease (Ismail et al., 2016), though it should not be considered in the presence of other neurological or psychiatric disorders (such as epilepsy, major depressive disorder, schizophrenia, and substance use disorder) that would better explain these symptoms (Ismail et al., 2016; Taragano et al., 2009). Clearly, a thorough diagnostic assessment is necessary that includes the investigation of biological markers. The first diagnostic criteria for MBI were a) the presence of a major change in patient behavior, b) this change occurring later in life (>60) and is persistent (>6 months), c) no complaint of cognitive impairment by patient/informant, d) normal occupational and social functioning, e) normal activities of daily living, and f) absence of dementia (Taragano et al., 2009). Taragano and colleagues highlight examples of behavioral changes, such as agitation, anxiety, apathy, depressive symptoms, disinhibition, impulsivity, lack of empathy, perseverant behavior, and others (Taragano et al., 2009). They state that the MBI-associated symptoms should not meet the criteria for MCI, whereas one should determine the absence of dementia based on preserved daily functioning, which does not exclude a measurable cognitive impairment as long as the patient does not lose independence or experience a subjective cognitive deficit (Taragano et al., 2009). Because of the rather complex or potentially ambiguous relationship between MBI and MCI following these assumptions, the criteria for MBI were updated a few years later (Ismail et al., 2016). The International Society to Advance Alzheimer's Research and Treatment (ISTAART) criteria for MBI (Table 1) acknowledge the possible breadth of neuropsychiatric symptoms within five major domains: motivation, affective regulation, impulse control, social cognition, and perception/thought content. These new criteria for MBI explicitly allow for the concurrent presence of MCI (Ismail et al., 2016) and lower the minimal patient age from 60 to 50 years. The ISTAART MBI criteria require a functional (social, occupational, or interpersonal) impairment attributable to noncognitive symptoms. Development of such symptoms after dementia onset would exclude the MBI diagnosis (Ismail et al., 2016). However, it remains a challenge – and sometimes may not be possible at all – to clearly attribute a functional impairment to neuropsychiatric symptoms or a cognitive deficit. It is also difficult to rule out subsyndromal psychiatric disorders with late-life onset or personality changes in adaptation to a given social or environmental milieu (Ismail et al., 2016). Repeated assessments may be helpful, although some etiologies could share common mechanisms. Clinicians should keep in mind that sensory deprivation, such as hearing loss (Gosselin et al., 2022) and frailty (Fan et al., 2020), also modulate the occurrence of MBI in older people, and that there are sex differences in these associations to the detriment of men (Gosselin et al., 2022; Guan et al., 2022).

Decreased Motivation

Apathy is the most frequent neuropsychiatric symptom across all stages of Alzheimer's disease (Lyketsos et al., 2011). It is associated with reduced metabolic activity in the bilateral anterior cingulate gyrus and medial orbitofrontal cortex (Marshall et al., 2007). Marshall and colleagues point to important reciprocal connections of the orbitofrontal region to the thalamus, which may be important in assigning internal relevance to external stimuli. Positive treatment effects on apathy with cholinesterase inhibitors (Wynn & Cummings, 2004) or selective serotonin reuptake inhibitors (Takemoto et al., 2020) suggest a neurotransmitter imbalance also related to cognition and mood. Recent data suggest an early involvement of temporal brain regions in MBI development. Matuskova and colleagues (2021) show an association of MBI with entorhinal cortex and hippocampal atrophy, the earliest sites of neuronal damage during Alzheimer's disease before developing dementia (Braak & Braak, 1991). This also aligns with an association of MBI and tau pathology in these regions (Johansson et al., 2021) and in blood plasma (Ghahremani et al., 2023).

In patients with MCI, apathy is also highly prevalent, from approximately 15–40% in population-based studies and clinical samples, respectively (Lyketsos et al., 2002; Martin & Velayudhan, 2020; Zhang et al., 2012). In their meta-analysis, Pan and colleagues (2022) show a 19% pooled prevalence of decreased motivation in patients with MCI, which is approximately twice the prevalence of this behavior change compared to patients suffering only from subjective cognitive deficits or cognitively normal subjects. As an illustration of the elevated risk for conversion to dementia because of MBI, apathy has been shown to increase the risk of developing Alzheimer's disease in patients with MCI (Palmer et al., 2010). The strength of this association, when compared with depressive symptoms, could be because of additional cerebrovascular pathology, including cerebral amyloid angiopathy, associated with apathy (Nakamura et al., 2013; Richard et al., 2012; Smith et al., 2021).

Geda and colleagues (2014) extend the research of neuropsychiatric symptoms to cognitively normal older people. The authors demonstrate an increased incidence of MCI in cognitively normal individuals with nonpsychotic psychiatric symptoms, including irritability and apathy. It is also

Table 1. ISTAART research diagnostic criteria for MBI (Ismail et al., 2016)

1. Changes in behavior or personality observed by patient, informant, or clinician, starting later in life (age ≥50 years) and persisting at least intermittently for ≥6 months. These represent a clear change from the person's usual behavior or personality as evidenced by at least one of the following:
   a) Decreased motivation (e.g., apathy, aspontaneity, indifference)
   b) Affective dysregulation (e.g., anxiety, dysphoria, changeability, euphoria, irritability)
   c) Impulse dyscontrol (e.g., agitation, disinhibition, gambling, obsessiveness, behavioral perseveration, stimulus bind)
   d) Social inappropriateness (e.g., lack of empathy, loss of insight, loss of social graces or tact, rigidity, exaggeration of previous personality traits)
   e) Abnormal perception or thought content (e.g., delusions, hallucinations)
2. Behaviors are of sufficient severity to produce at least minimal impairment in at least one of the following areas:
   a) Interpersonal relationships
   b) Other aspects of social functioning
   c) Ability to perform in the workplace
   d) The patient should generally maintain his/her independence of function in daily life, with minimal aids or assistance.
3. Although comorbid conditions may be present, the behavioral or personality changes are not attributable to another current psychiatric disorder (e.g., generalized anxiety disorder, major depression, manic or psychotic disorders), traumatic or general medical causes, or the physiological effects of a substance or medication.
4. The patient does not meet the criteria for dementia syndrome (e.g., Alzheimer's disease, frontotemporal dementia, dementia with Lewy bodies, vascular dementia, other dementia). MCI can be concurrently diagnosed with MBI.

Note. ISTAART = International Society to Advance Alzheimer's Research and Treatment; MBI = mild behavioral impairment; MCI = mild cognitive impairment.

interesting to compare hazard ratios for apathy (2.26, 95% CI = 1.49–3.41) and hippocampal volume (1.8, 95% confidence interval [CI] = 1.4–2.20) in predicting incident cognitive impairment in a comparable research setting (Geda et al., 2014; Kantarci et al., 2013). Pink and colleagues show an additive interaction between apathy and the apolipoprotein E ε4 (APOE4) allele, the most important genetic risk factor for sporadic Alzheimer's disease, in predicting incident dementia (Pink et al., 2015), whereas we (Donix et al., 2010) previously highlighted the contributions of the APOE4 risk allele to hippocampal thickness in cognitively normal older people.

Affective Dysregulation

The affective dysregulation domain of the ISTAART research diagnostic criteria for MBI (Table 1) encompasses several symptoms, such as anxiety, dysphoria/euphoria, or irritability. Regarding mood changes, depressive symptoms are almost as frequent as apathy in patients with Alzheimer's disease (Lyketsos et al., 2011). This fact has stimulated research into whether depressive symptoms are a prodromal feature of dementia or a risk factor, or whether affective and neurodegenerative symptoms share common mechanisms (Bennett & Thomas, 2014; Kida et al., 2016; Singh-Manoux et al., 2017). Furthermore, cognitive symptoms are a salient and frequent feature of depression (Rock et al., 2014), particularly in old age; the atypical presentation of a depressive episode in older patients and those with cognitive deficits (Taylor, 2014; Vida et al., 1994) could present a challenge for establishing a correct diagnosis. However, more closely related to MBI is the differentiation of major depressive disorder, which would exclude an MBI diagnosis from subsyndromal affective changes not developing into major depression. Such a diagnosis is undoubtedly difficult and requires longitudinal assessments or informant reports. Although metabolic changes (Alexopoulos & Morimoto, 2011) or cerebrovascular disease (Jellinger, 2021; van Sloten et al., 2015) could be contributing to affective changes, neurodegeneration may alter serotonergic and noradrenergic pathways to the frontal lobe and the hippocampus, respectively (Zubenko et al., 2003). Subclinical depressive symptoms in cognitively healthy older adults are associated with lower structural and functional integrity in a frontolimbic network as well as, for example, gray matter volume reduction in the hippocampus and glucose hypometabolism in the hippocampus and the medial and dorsolateral prefrontal cortex (Touron et al., 2022). Cerebral amyloid and tau deposition, hippocampal atrophy, and genetic risk of Alzheimer's disease modulate the relationship between depressive symptoms and cognitive function (Karlsson et al., 2015; Rubin-Norowitz et al., 2022).

Martin and Velayudhan (2020) highlight depressive symptoms as the most frequent neuropsychiatric phenomenon in patients with MCI. The authors emphasize how the complex and possibly bidirectional relationship between MCI and depressive symptoms as well as different assessment methods may contribute to conflicting data (Martin & Velayudhan, 2020), with prevalence rates ranging from approximately 20% in population-based samples to 80% in the clinic (Lyketsos et al., 2002; Rozzini et al., 2008). Irritability is less frequent, although quite prevalent among patients with MCI, with about half of the

aforementioned prevalence rates in the respective settings (Geda et al., 2004; Martin & Velayudhan, 2020; Zhang et al., 2012). It is nevertheless associated with the progression of MCI (Forrester et al., 2016). The same was found for anxiety (Somme et al., 2013), which occurs with a prevalence rate of approximately 12% in a population-based sample, although these individuals also qualified for specific diagnoses within the anxiety spectrum, such as generalized anxiety disorder (Mirza et al., 2017). This again illustrates the difficulty in recognizing and documenting subsyndromal symptoms that represent a change in personal behavior below diagnostic thresholds.

Impulse Dyscontrol

Impulsivity (e.g., agitation, disinhibition, obsessiveness) is another frequent neuropsychiatric symptom across the spectrum of neurodegenerative disorders. Saari and coworkers (2022) characterize impulse dyscontrol as difficult to operationalize, although closely connected to the symptoms of irritability, agitation, and rigidity. Impulse dyscontrol has been observed in approximately 17% of cognitively normal older people and 34% of patients with MCI (Mortby et al., 2018). In population-based samples, the frequency is about 11% in patients with MCI (Lyketsos et al., 2002; Martin & Velayudhan, 2020). Utilizing structural magnetic resonance and diffusion tensor imaging, Gill and colleagues find evidence for brain changes typical for Alzheimer's disease associated with impulse dyscontrol, even before cognitive decline (Gill et al., 2021). They detect atrophy in the medial temporal lobe, and impairments in white matter integrity extend to the fornix, superior fronto-occipital fasciculus, cingulum, and uncinate fasciculus. The authors suggest a fronto-striatal network dysfunction underlying impulse dyscontrol in advance of dementia and highlight the cingulum as a potential neuroimaging marker (Gill et al., 2021). The pathological changes in brain regions involved in agitation (Gill et al., 2021; Rosenberg et al., 2015) may correspond with an increased risk for further cognitive decline in patients with MCI (Forrester et al., 2016). In line with previous data, Gill and colleagues (2021) demonstrate how white matter microstructural changes precede gray matter atrophy in preclinical neurodegeneration (Zhuang et al., 2013). In a meta-analysis, impulse dyscontrol was the most prevalent MBI domain after affective dysregulation, with a pooled prevalence of approximately 30% among patients with MCI and 25% among cognitively normal older people (Pan et al., 2022). Applying their original MBI criteria, Taragano and colleagues (2018) show that MBI is associated with a higher risk for conversion to frontotemporal dementia than to Lewy body dementia or dementia because of Alzheimer's disease (approximately 45%, 28%, and 27%, respectively). Using ISTAART domain-specific MBI data, Gill and colleagues (2020) demonstrate the importance of emotional dysregulation and impulse dyscontrol for correctly classifying Alzheimer's disease from neuroimaging and behavioral data.

Social Inappropriateness

Social inappropriateness, such as lack of empathy, loss of insight, or social tact, are signs of impaired social cognition that may emerge because of neurodegeneration, sometimes prior to cognitive decline (Desmarais et al., 2018). Recognizing and interpreting verbal and nonverbal social stimuli, e.g., facial expressions, speech prosody, or gestures, is a prerequisite for understanding and adequately responding to the emotions and intentions of others (Desmarais et al., 2018). Having a theory of mind refers to mentalizing the emotional and affective states of others, which is closely linked to metacognition, the ability to reflect on one's actions and think about one's own thoughts (Frith & Frith, 2012). Kessels and colleagues (2021) show that patients with amnestic MCI perform worse than healthy older people in recognizing anger, disgust, or fear from facial expressions. They also perform worse on answering questions requiring the ability to infer the thoughts and feelings of others. Although this could reflect social cognition deficits in prodromal Alzheimer's disease, others highlight the utility of using facial expression recognition to differentiate behavioral variant frontotemporal dementia from other entities (Gossink et al., 2018). In their review on social inappropriateness in neurodegenerative disorders, Desmarais and coworkers (2018) refer to lesion data and functional magnetic resonance imaging studies for how to appreciate the neural basis for social cognition. They point to different frontal cortices, such as the orbitofrontal cortex, the dorsolateral prefrontal cortex as well as the amygdala and the cingulate cortex as important areas within the different networks involved, but they also mention neurochemical hypotheses, e.g., dopaminergic or serotonergic dysfunction underlying social cognition impairment (Abu-Akel, 2003; Amodio & Frith, 2006).

Impairments in social cognition could range from subtle changes, such as making less eye contact or struggling to comprehend sarcasm, to abnormal social behavior leading to arrests and criminal charges (Desmarais et al., 2018). Whereas prominent behavioral changes may be typical for behavioral variant frontotemporal dementia and its prodromal stages (Rascovsky et al., 2011), social inappropriateness in Alzheimer's disease is usually less severe and more likely an effect secondary to cognitive decline (Desmarais et al., 2018; Lindau et al., 2000). Woolley and colleagues (2011)

show the risk of misclassifying neurodegenerative disease presenting with behavioral symptoms, which could be especially relevant for MBI without a recognizable cognitive impairment.

Abnormal Perception or Thought Content

MBI presenting as abnormal perception or thought content refers to delusions or hallucinations. It is the least prevalent MBI-associated phenomenon in patients with MCI (approximately 5%) and in cognitively healthy older people (approximately 2%) (Pan et al., 2022). Hallucinations could be present in patients with MCI before developing Lewy body dementia (Gan et al., 2022; Liu et al., 2021) or vascular dementia (Peters et al., 2013). Delusions and hallucinations do not predict MCI in cognitively normal people (Geda et al., 2014). However, in patients with MCI because of Alzheimer's disease or associated with Lewy bodies, the presence of hallucinations predicted future cognitive decline (Hamilton et al., 2021). This illustrates the rather low specificity of such abnormal perception or thought content in cognitively unimpaired individuals, which may change with the development of other symptoms within the temporal spectrum of neurodegeneration associated with alpha-synuclein. Pathophysiological mechanisms for the development of delusions or hallucinations range from dopaminergic dysbalance to brain structure changes in regions associated with visual processing and in the limbic system (Burghaus et al., 2012).

Assessment

When investigating challenging (e.g., socially inadequate) behavior across the lifespan, one has to keep in mind that its possible diagnostic value may change with the emergence of new diagnostic classification systems. Therefore, the assessment of MBI according to the ISTAART research criteria allows us to primarily recognize and operationalize a behavioral impairment without making diagnostic inferences about a specific (neurodegenerative) disease. Furthermore, compared with the original MBI criteria (Taragano et al., 2009), the ISTAART research diagnostic criteria also establish a more clearly defined relationship with MCI in operationalizing behavioral changes (Ismail et al., 2016). The definition of separate domains within the MBI construct can be considered inclusive toward different neurodegenerative diseases, as it may encourage clinicians to also recognize less challenging impairments. Following this rationale and domain structure, Ismail and colleagues developed the MBI Checklist (MBI-C) (Ismail et al., 2017), which consists of 34 items across 5 domains, created to be used by family members and informants (Ismail et al., 2017). In contrast to the evaluation of neuropsychiatric symptoms in patients with dementia, e.g., using the Neuropsychiatric Inventory (Cummings, 1997), the MBI-C is specifically adapted to nondemented individuals. It is also sensitive for detecting MBI in patients with MCI (Mallo et al., 2018). In addition to further confirming the longitudinal validity of the MBI concept, it is yet to be shown whether there is an optimal cutoff score for prognostic purposes, and whether there are differences in risk based on domain scores (Ismail et al., 2017). The MBI-C is freely available in a growing number of languages; a German translation recently became available (Dibbern et al., 2023).

First-Time Criminal Behavior in Patients with Brain Disease

Although there are remarkable cases in which neuroimaging data were used as evidence in criminal court proceedings (Batts, 2009), it has still not been demonstrated that an altered mental state is associated with structural or metabolic brain changes to evaluate criminal liability. Lesion data unsurprisingly reveal the significance of specific brain regions for social interaction impairments, for example, when considering the role of the orbitofrontal cortex in decision-making and emotional processing (Bechara et al., 2000) or the importance of the amygdala for affective influences on perception (Anderson & Phelps, 2001). In line with "acquired sociopathy" in a patient with bilateral ventromedial frontal brain injury (Saver & Damasio, 1991), Darby and colleagues (2018) present a systematic mapping of brain lesions (orbitofrontal cortex, ventromedial prefrontal cortex, anterior temporal lobes) with a temporal association to criminal behavior. They demonstrate a resting state network of functionally connected brain regions associated with morality, value-based decision-making, and theory of mind, which is distinct from networks underlying different neuropsychiatric syndromes.

However, among older first-time criminal offenders, brain changes associated with neurodegenerative diseases may be modulating antisocial behavior more often than a brain tumor or focal injury. Liljegren and colleagues (2015) investigated criminal behavior in the medical records of almost 2,400 patients presenting to a university memory clinic. The authors found evidence of criminal behavior in approximately 37% of patients with behavioral variant frontotemporal dementia (e.g., theft, traffic violations, sexual advances, trespassing) and in 8% of patients

with dementia because of Alzheimer's disease (predominantly traffic violations). In a cohort study using medical record data of 220 Swedish patients with neuropathologically confirmed frontotemporal lobar degeneration or Alzheimer's disease, criminal behavior could be detected in approximately 42% and 15% of the patients, respectively (Liljegren et al., 2019). Compared with their previous data, the higher prevalence could signal the inclusion of police contacts because of socially inappropriate behaviors that would not lead to criminal charges. On the other hand, it is also possible that certain behaviors would not reach the legal system in the light of neurodegenerative disease, e.g., trespassing because of wandering or insulting someone, despite being criminal offenses or misdemeanors, depending on the legal appreciation across different judicial systems.

Whereas delinquency in patients with Alzheimer's disease is primarily associated with cognitive dysfunction, loss of oversight, and anosognosia for these deficits in later stages of the disorder, it is more prevalent in dementia patients with greater frontal lobe pathology, e.g., because of frontotemporal lobar degeneration or vascular lesions (Liljegren et al., 2018, 2019). Patients with frontotemporal dementia experience impulse dyscontrol and affective dysregulation early in the course of the disease, and criminal behavior may even be the first sign (Liljegren et al., 2015).

MBI and Delinquency

In 2018, only 7.6% of all criminal suspects in Germany were >60 years old (75% male), whereas the most frequent offenses were theft, (negligent) bodily injury, insult, and fraud (Haussmann et al., 2022). The high frequency of first-time offenses among these individuals and the relationship with disinhibition are suggestive of an emerging psychopathology in contrast to challenging personality traits. Verhulsdonk and colleagues (2023) demonstrated greater impairment using the frontal assessment battery (FAB) among older forensic inpatients when compared with older prisoners. Liljegren and coworkers (2019) therefore recommend that older individuals exhibiting criminal or socially inappropriate behavior for the first time be screened for neurodegenerative disorders. When investigating a possible relationship between this behavior and brain pathology, forensic geriatric psychiatrists should be aware of the following aspects:

(1) Preserved cognition must not prevent clinicians from considering that neurodegeneration may possibly underlie MBI.
(2) MBI should be examined using a standardized assessment, complementing witness or caregiver reports, and personal impressions. The MBI-C (Ismail et al., 2017) is inclusive of different neurodegenerative diseases.
(3) Brain imaging should complement diagnostic assessments, although it may be without pathological findings in people with MBI. Whereas structural imaging may not (yet) capture focal atrophy, metabolic scanning (e.g., positron emission tomography) also does not definitively prove or exclude neurodegeneration. However, postmortem neuropathological findings would also not always correlate with clinical impairments.
(4) MBI must be recognizable beyond the legal context. A phenomenon associated with criminal behavior – for example, impulse dyscontrol – should be present in everyday situations, although possibly modulated or exacerbated by stressful conditions.
(5) Although a particular MBI domain could be prominent (or legally relevant), a symptom may be accompanied by others in the temporal course of the underlying disease.

Concerning facing criminal charges, Liljegren and colleagues (2019) find it problematic that some patients with frontotemporal dementia can understand the criminal nature of an action but proceed regardless. From a forensic psychiatric point of view, it is not, since most legal systems distinguish between the cognitive faculty to distinguish right from wrong and being able to act accordingly. It is plausible that MBI, in the absence of dementia syndrome, would not prevent individuals from knowing social or legal norms per se. In the rare event of abnormal perception or thought content, it is questionable whether it would still represent MBI, for example, if complex hallucinations directly impact behavior (e.g., commanding voices). These may qualify for other psychiatric diagnoses, although it could be difficult in this case to classify an organic psychosis without a measurable cognitive impairment or to diagnose a brief psychotic disorder despite the suspected neurodegeneration.

Among individuals without dementia, MBI could be associated with deficits in volitional control. However, the complete lack of control because of MBI is not a realistic scenario. In contrast to patients with dementia, unimpaired or relatively preserved cognition should allow for some degree of adaptation to different contexts and settings (e.g., family, police, courtroom). Constellating factors, such as substance use or advanced age, should be considered. Williams and colleagues (1999), for example, show an age-related change in inhibitory control, whereas Miao and coworkers (2021) highlight the influence of cerebrovascular lesions. In people with incipient neurodegenerative disease, these variables, among others, could be associated with or lower the threshold for MBI.

Conclusions

MBI could be present before developing cognitive impairment because of neurodegeneration. The spectrum of MBI symptoms includes impulse dyscontrol and social inappropriateness, which could be related to first-time criminal behavior in older people. Recognizing these deficits in the absence of a dementia syndrome requires knowledge of the affected brain networks and how to reliably diagnose MBI. Ultimately, MBI and its impact on volitional control may have prognostic and legal consequences.

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