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Frontotemporal Dementia-Like Syndrome Following Recall of Childhood Sexual Abuse

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Numerous psychopathological syndromes have been attributed to posttraumatic stress, both at the time of the trauma and many years later. To date, however, there is little literature on pseudodementia as a delayed traumatic stress response. The authors present a case history of a 50-year-old woman who developed severe cognitive impairment following retrieval of previously forgotten memories of childhood sexual abuse. Her cognitive condition deteriorated rapidly and dramatically. Neuropsychological assessment and clinical presentation led to a diagnosis of frontotemporal dementia (vs. corticobasal degeneration). Detailed neurologic and medical evaluations could not identify any underlying physical cause. Her condition progressively worsened over 9 months, at which point memantine, an N-methyl-D-aspartate receptor antagonist, was begun. The patient regained full functioning over the next year. Although an organic cause could not be ruled out, it was likely that recovery of traumatic memories was contributory to the patient's condition, as ongoing psychotherapy had begun 1 year into the course. If additional cases with similar presentations are reported, such cases would corroborate the notion that persistent, severe, and reversible cognitive impairment constitutes a previously unrecognized and atypical posttraumatic response.

Multiple psychopathological syndromes have been attributed to posttraumatic stress, both at the time of the trauma and many years later. Such pathological sequela include depression and dissociation (Briere & Elliott, 2003); personality disorders (Cohen et al., 2014; Johnson, Cohen, Brown, Smailes, & Bernstein, 1999); and substance abuse, suicidality, and eating disorders (Mullen, Martin, Anderson, Romans, & Herbison, 1996). To date, however, there is little literature on pseudodementia as a delayed traumatic stress response. A Medline literature search pairing the key words dementia/pseudodementia with posttraumatic stress disorder/stress disorder: posttraumatic yielded 44 articles, most about posttraumatic stress disorder (PTSD) among elderly or demented patients. These articles suggest PTSD to be a risk factor for the development of dementia in late life (Yaffe et al., 2010). Of note, although the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; American Psychiatric Association [APA], 2013) replaced the diagnosis of dementia with the diagnosis of neurocognitive disorder, the vast majority of the literature predates *DSM-5* and thus uses the term dementia. Likewise, *DSM-5* did not propose a new term for pseudodementia. Therefore, we retain the term dementia in this report.

Of the articles we located, only one pertained to pseudodementia as a sequelae of traumatic stress in younger adults. This report described a 45-year-old male who exhibited aphasia, amnesia, disorientation, trembling, and behavioral disorders over a period of 1 year, but had negative results on both medical and neurologic examination. These symptoms rapidly resolved after watching a movie about a victim of childhood sexual abuse, at which point he recovered his own memory of childhood sexual abuse. His presentation then shifted to one of clear PTSD and comorbid major depression (Montefiore, Mallet, Levy, Allilaire, & Pellisolo, 2007). Thus, it is possible that prolonged, but reversible cognitive deterioration in an otherwise healthy young or middle-aged adult may represent a rare and previously unrecognized response to traumatic stress.

Case

The patient gave informed consent for this report. Additionally, the Mount Sinai Beth Israel IRB exempted this report from institutional review board approval. The case was a 50-year-old woman who developed severe cognitive impairment following retrieval of previously forgotten memories of childhood sexual abuse by her father. Her cognitive condition, diagnosed as frontotemporal dementia versus corticobasal degeneration after a thorough neurologic evaluation, progressed for approximately 9 months, but then gradually reversed over the ensuing year with the initiation of memantine, an N-methyl-D-aspartate (NMDA) receptor antagonist. The patient regained full functioning. A

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Table 1
Time Line for Course of Illness

Month	Event
0	1st Psychotic episode
1	Olanzapine tapered
5	Sertraline tapered
6	2nd psychotic episode
8	Ziprasidone replaces Olanzepine
12	Start of psychotherapy
24	Pt recalls abuse by father (in somatosensory flashbacks)
25	Nightmare about abuse
30	Husband reports cognitive deterioration; B12 low, CRP high
32	B12 and CRP levels normalized
36	1st neuropsychological testing
39	Memantine 20 mgs initiated
40	Notable improvement
45	Per husband, 85% recovery
46	Starts volunteer work
47	2nd neuropsychological evaluation
47	Diagnosis of dementia withdrawn by neurologist, memantine d/c'd
60	Return to full time employment
120	Still functioning well, fully recovered

Note. Pt. = patient; CRP = C-reactive protein ; d/c'd = decompensated.

timeline for the course of the patient's illness is presented in Table 1.

The patient initiated psychiatric treatment at age 48 years following abrupt onset of a psychotic episode, characterized by paranoid ideation about a conspiracy against her that was revealed in a system of coded signals on her computer. She feared for her family and was afraid of being tortured. These symptoms followed several weeks of reduced sleep, which the patient attributed to menopause, along with reduced concentration, fatigue, and depressed mood. The patient recovered from her psychosis within 2 days after initiation of 5 mg daily of olanzapine, a second-generation antipsychotic, which was prescribed by her internist. She did not require hospitalization. A full medical and neurologic work up (computed tomography, magnetic resonance imaging [MRI], electroencephalogram [EEG], blood work) revealed no abnormalities. The patient's internist referred her to a psychiatrist (DB) who diagnosed her with major depressive disorder with psychotic features. He retained her on olanzapine and then augmented the regimen with the antidepressant sertraline, 100 mg daily. After a few weeks, the olanzapine was tapered off with no negative effect. After about 6 months of full response, with neither depressive nor psychotic symptoms evident, sertraline was tapered and discontinued, but the patient decompensated within a few weeks. As before, her psychotic symptoms included paranoia, confusion, and feelings of terror. This time she was hospitalized, treated with olanzapine and then discharged the next day, having quickly returned to baseline. After discharge, her outpatient psychiatrist restarted sertraline at 100 mg daily along with olanzapine 5 mg daily. She also was prescribed clonazepam 0.5 mg every night. After 2 months, because of weight gain, ziprasidone 40 mg was substituted for olanzapine. After this second psychotic episode, the patient was referred for psychotherapy with a psychologist (LJC).

Outside of longstanding, low-grade depression and pastoral counseling received 16 years prior to her first psychotic break, the patient had no history of psychiatric treatment or functional impairment. She was married for over 20 years, raised two children, completed graduate education, and had performed well in a professional career. Her family history, however, was positive for schizophrenia in both her maternal grandfather and her paternal uncle. In her first psychotherapy session, she disclosed that her father was currently in prison for sexually molesting her nephew. Upon further questioning, she revealed that three of her siblings recalled being sexually abused by their father although she had no memories of being abused herself. In consultation with her psychiatrist, the psychologist then reconceptualized the case as a brief psychotic disorder in the context of PTSD. The therapist focused on managing anxiety, recognizing triggers that might elicit psychotic symptoms (e.g., extended contact with the nephew her father molested), and coping with the ongoing family stressors related to her father's incarceration. Such stressors included her father's impending parole hearing, her mother's financial and residential concerns, and her mother's equivocation as to her father's guilt. Although the therapist deliberately avoided exploring the possibility of past trauma in the patient's own history for fear of further decompensation, the possibility of the patient's own sexual molestation did come up in the context of discussing her current family stressors as well as the possible causes of her two psychotic episodes. Nevertheless, the therapist also thoroughly addressed the risks of decompensation if exploration of trauma were to elicit excessive anxiety. Throughout the treatment, top priority was given to anxiety-management rather than potentially anxiogenic interventions.

Throughout the first 18 months of psychotherapy, while the patient remained nonpsychotic, her affect became progressively more odd, anxious, and detached, with a somewhat frozen facial expression frequently apparent. Although there were periodic eruptions of paranoid ideation accompanied by intense anxiety, the patient maintained insight into her symptoms and could regroup with reassurance and clonazepam as needed. Such incidents occurred approximately monthly and typically lasted several hours. One year after initiating psychotherapy, the patient came to the conclusion that she had in fact been molested by her father. This occurred in the context of a visit from the nephew who had been abused by her father; these visits often triggered anxiety for her. Although she had no fully coherent memories, she described what seemed like memory fragments, consisting of sensory–emotional experiences of pain,

unfamiliar white discharge in her underwear, surprise, and confusion. She told the therapist that she now believed definitively that she had been abused by her father and that it was important that the therapist believe her. Approximately 5 months later (and 17 months after initiating psychotherapy), she reported her first nightmare about the abuse. The dream did not consist of actual memories, but rather of symbolic images, such as that of a young child, about age 4, lying dead on the floor. In the dream, it was clear to the patient that the child had been abused.

Six months after her initial recognition of her own abuse and 3 weeks after her first abuse-related dream (18 months after initiating psychotherapy), her husband noted his wife to be confused, distractible, disoriented, and increasingly unable to complete daily tasks. Her husband described her behavioral changes in a letter to her psychiatrist, noting that she exhibited repetitive behaviors (taking napkin after napkin at a food stand, saying hello to the cat over and over again), wandering, confusion regarding time and money, misplacement of items (laundry in wastebasket instead of hamper), inability to complete procedural tasks correctly (cooking, folding laundry, setting the table), and general passivity and inattention.

At the first therapy session following this report, she was administered several psychometric instruments. She scored 26 out of 30 on the Mini Mental State Exam, scoring 0 out of 3 for recall and also exhibiting perseveration on a drawing task. Scores from 24–27 may be indicative of dementia in highly educated individuals (APA, 2000). She scored 5 on the Beck Anxiety Scale (normal or no anxiety), 4 on the Beck Depression Scale (minimal range) and 7 out of 38 on the Cornell Scale for Depression in Dementia (below clinical cutoff of 8; APA, 2000). Although her mood was euthymic, if not cheerful, and she reported no delusions, her cognition progressively deteriorated. Eventually, she took a medical leave from her job, obtained disability payments, and required 24-hour supervision. She was unable to fold laundry, use the stove safely, or even dress herself appropriately. She evidenced gross loss of motor coordination, with difficulty chewing and walking. She also lost weight, losing 7% of her normal body weight, despite denial of loss of appetite. She was spatially disoriented and wandered away when not supervised. She was verbally and motorically perseverative as well as somewhat disinhibited, displaying overly familiar behavior with her therapist.

Due to these sudden changes in mental status, her psychiatrist referred her to her internist who ordered a full medical and neurologic work up. MRI revealed mild sulcal enlargement, but no other abnormalities. Bloodwork, performed within one month of cognitive decline, revealed low normal B12 level (296 pg/mL; normal range = 200–900 pg/mL) and strongly elevated C-reactive protein (CRP; 27.5 mg/L; normal < 4.9 mg/L). Oral B12 was started by her internist and within weeks her B12 level was above the upper limit of normal (1000 pg/mL). CRP at this point was also within the normal range (2.97 mg/L). There was, however, no clinical change. Spinal tap was negative. Neurologic examination revealed hyperreflexia, but no pathological reflexes. The EEG was negative and she evidenced no loss of consciousness, periods of nonresponsivity, or diffuse spasmodic motor activity suggestive of seizures. Neuropsychological testing, performed 6 months after onset of cognitive decline, showed severe impairment in memory, visuospatial, language, and executive domains and suggested frontotemporal dementia, behavioral type (see Table 2). Throughout this period, her psychiatrist maintained her on a daily dose of sertraline 100 mg, ziprasidone 80 mg, and clonazepam 0.75 mg.

Although the patient demonstrated marked cognitive deterioration, she remained fully oriented, maintained full conversational capacities, and displayed reasonable insight into her condition. She was thus able to participate in and benefit from supportive psychotherapy. Therefore, psychotherapy continued throughout the period of her cognitive impairment. After the patient was unable to travel by herself, her husband accompanied her and sat in on all sessions. At this point, therapy focused entirely on her current condition, with discussion of her symptoms, her own and her family's adjustment to her condition, and practical solutions to logistical problems. There was no discussion of past trauma or even of problems in her extended family related to her father's incarceration. To provide support for her husband, he was referred to another psychotherapist for supportive psychotherapy during this very trying time.

Nine months after the onset of cognitive deficits, the patient's neurologist started her on donepezil 10 mg daily and then switched to memantine 20 mg daily. Both medications are cognitive enhancers: donepezil an acetylcholinesterase inhibitor and memantine a glutaminergic inhibitor acting on the NMDA receptors. Within weeks of starting memantine, she showed unmistakable cognitive improvement, the first time she had shown any improvement in her cognitive or functional status, let alone abatement of her progressive deterioration. Four months later (and 14 months after onset of cognitive deficits), her condition continued to improve and she asked to reduce her antidepressant. Five months after she started improving (15 months after onset of cognitive symptoms), her husband judged her 85% recovered. By 6 months after initial improvement, she was able to attend therapy sessions unaccompanied by her husband and had started volunteer work. Her mental status had returned to normal. Moreover, compared to her initial presentation, her affect was more full range and less constricted. One month later and 17 months after onset of cognitive disorder, she was evaluated by her neurologist who noted that she was remarkably recovered. He withdrew the diagnosis of frontotemporal dementia, as dementias are not reversible, but did not propose an alternative diagnosis. He stopped the memantine to no ill effect. Ziprasidone, sertraline, and clonazepam were sequentially tapered to discontinuation around the same time by the psychiatrist. Neuropsychological re-evaluation revealed significant global improvement, with no evidence of cognitive impairment, although there remained some relative decrements in visual memory and verbal fluency.

Table 2
Neuropsychological Performance at Three Time Points After Onset of Cognitive Impairment

	6 Months		18 Months		30 Months	
Test	Score	%tile	Score	%tile	Score	%tile
General						
WAIS-R Similarities	8.0	25.1	—	—	12.0	74.9
WAIS-R Digit span	9.0	37.1	—	—	13.0	84.1
WAIS-R Digit symbol-coding	6.0	9.2	7.0	15.9	12.0	74.9
Attention						
Trails A	30.0	68	28.0	75	23.0	87
Stroop word (*T* Score)	49.0	46.0	65.0	93.3	67.0	95.5
Stroop color (*T* Score)	29.0	1.8	52.0	57.9	49.0	46.0
Memory						
WMS-III Logical memory I	4.0	2.3	10.0	50.0	13.0	84.1
WMS-III Logical memory II	4.0	2.3	11.0	62.9	14.0	90.8
WMS-III Logical memory % retained	36		93		100.0	
WMS-III Visual reproduction I	21.0	7	—	—	—	63
WMS-III Visual reproduction II	3.0	1	—	—	11.0	5
WMS-III Vis. reproduction % retained	14	—	—	—	33	—
Rey-Oesterreith immediate recall	1.0	—	—	—	18.0	—
Rey-Oesterreith delayed recall	1.0	0.5	—	—	14.5	49
SRT[a] long-term storage	—	<1.0	—	90	—	81
SRT consistent long-term retrieval	—	1	—	73	—	52
Language						
Animals	—	19	—	85	—	81
Boston naming test total *Z* score	−2.6	0.5	−0.7	24.5	0.5	67.4
Visuospatial						
Rey-Oestereith copy	15	0.3	—	—	36	52
Benton Visual Retention Test/Matching	7.0	28	10.0	99	9.0	65
Executive						
Stroop color word *T* score	21.0	0.1	48.0	42.1	52.0	57.9
Stroop interference *T* Score	33.0	4.5	42.0	21.2	47.0	38.2
Trails B	Incom[b]	Incom[b]	47.0	90	49.0	90

[a]Selective Reminding Test. [b]Patient was unable to complete the test.

Thirty months from the onset of cognitive impairment and 20 months after initial improvement, she returned to work in a position with slightly less responsibilities and less pressure. Five years later, she continued to function well in both her professional and family responsibilities.

Approximately 4 years after initial cognitive improvement, however, there was one recurrence of attenuated psychotic symptoms accompanied by anxiety. While at work, she became acutely paranoid, fearing that people were talking about her. With reassurance from her husband, she was able to calm down within hours. The trigger appeared to be an encounter with her mother, whom she had not seen for 2 years. Her mother's continued relationship with her father and continued denial of his molestation of his children had been a source of great distress for her. Of note, she had no symptomatic response to an unrelated violent incident that occurred around the same time.

Discussion

This case raises significant diagnostic challenges. Despite two full neurologic and medical workups, no underlying physiological pathology could be found. Yet at the peak of her cognitive impairment, this patient manifested the full picture of frontotemporal dementia or what would now be called major frontotemporal neurocognitive disorder in *DSM-5*. Motor abnormalities led to an alternative diagnosis of corticobasal degeneration. Moreover, the psychotic and mood symptoms preceding her dementia-like episode are not uncommon correlates of neurocognitive disorders (Sadock & Sadock, 2003). The full resolution of her cognitive symptoms and her sustained recovery over 5 years is the strongest argument against a true neurocognitive disorder. Nonetheless, we could not entirely rule out organic pathology as many subtle brain abnormalities are not always detectable on standard neurologic

evaluation. For example, anti-NMDA receptor encephalitis can present with psychiatric disturbances including psychosis, depression, and anxiety as well as cognitive impairment, although typically in the context of seizures (Torgovnick et al., 2011). Moreover, the patient did show markedly elevated CRP levels in the early period of cognitive impairment. CRP, a nonspecific marker of inflammation, is elevated in both peripheral and CNS inflammatory states (Ahmad, Ali, Fakhir, & Chandra, 1991; Ramlawi et al., 2006). Even though the patient's cognitive deficits remained long after her CRP value normalized, the fact that the patient improved with memantine, an NMDA receptor blocker, adds support to a diagnosis of encephalitis. B12 deficiency is also linked to neurodegenerative illness (Moore et al., 2012), but the patient's B12 deficiency, if it existed at all, was mild and was rapidly corrected with oral B12 supplementation within 2 months after onset of cognitive impairment and approximately 8 months prior to any signs of improvement.

Moreover, the patient's clinical presentation cannot be separated from her history of trauma, such that it is highly likely that her trauma history is contributory if not wholly causative. Her father had sexually abused almost all of her siblings and even his grandson. Although she did not initially recall her own abuse history, it is highly plausible that she also would have been a victim of her father's molestation. Further, she did eventually experience a series of likely memory fragments of sexual trauma as well as a subjective conviction that she had indeed been sexually assaulted by her father. Finally, the recurrence of paranoid ideation following renewed contact with her mother, but not in the context of a violent incident at work supports the contributory role of her childhood trauma. It is of interest that her first psychotic episode occurred when her oldest daughter was leaving for college. The patient felt that the sleep disruption, caused by both the onset of menopause as well as helping her daughter prepare her college applications, was a significant contributing factor. As much of her delusional content involved her fear that her family was in mortal danger and that she could not protect them, it is also quite possible that the departure of her daughter from the protective family fold may have precipitated posttraumatic anxiety.

There is a robust literature documenting the variety of psychopathological sequelae to childhood maltreatment in general, and to sexual abuse in particular, including depression, dissociation (Briere & Elliott, 2003), personality disorders (Cohen et al., 2014; Johnson et al., 1999), substance abuse, suicidality, and eating disorders (Mullen et al., 1996). Additionally, there is increasing evidence of the somatic sequelae of early childhood trauma, including autoimmune disturbances (Kiecolt-Glaser et al., 2011). Therefore, it is possible that the stress from the recovery of traumatic memories could have triggered a pathological somatic process, such as encephalitis. Although the notion of suppressed memories of childhood abuse, and of sexual abuse in particular, has been highly controversial (Loftus & Ketcham, 1994), there is evidence that delayed recall of childhood trauma does occur (McNally, Perlman, Ristuccia, & Clancy, 2006; van der Hart, Bolt, & van der Kolk, 2005). Studies of patients with dissociative identity disorder, which is linked to severe and persistent childhood abuse (Dorahy et al., 2015), support the validity of suppressed traumatic memories that re-emerge decades after the initial trauma (van der Hart, Bolt, & van der Kolk, 2005). Interestingly, such memories are often in the form of somatosensory flashbacks, as reported in this case (van der Hart et al., 2005). The literature also supports the danger that premature exploration of traumatic memories, whether previously suppressed or not, can lead to intense exacerbation of psychiatric symptoms, including dissociation, self-injurious behavior, panic, sleep disorders, depression, and suicidality (Briere & Scott, 2006; Rothschild, 2000).

Given the atypical presentation in this case, it is important to consider other possible diagnoses. The patient was initially diagnosed with major depressive disorder (MDD) with psychotic features. It is worth considering whether her cognitive impairment reflected pseudodementia in the context of a severe major depressive episode. Although the patient did report depressed mood and symptoms consistent with a depressive diagnosis, such as fatigue and decreased sleep, appetite, and concentration in her first psychotic episode, she did not report depressed mood in subsequent episodes nor did she demonstrate sad affect, fatigue, pessimistic ideation, or somatic preoccupation after her first episode. In fact, her mood was quite cheerful throughout the period of her cognitive impairment. Overall, the most prominent affective symptom was anxiety, except during the period of cognitive impairment, at which point it was notably absent. Moreover, her very rapid recovery from both psychotic episodes was not consistent with MDD with psychotic features, which typically takes several months at least to resolve (Meyers, Flint, Rothschild, & Mulsant, 2009).

As she had a strong family history for schizophrenia, this diagnosis or some variant should also be considered. At age 48, her onset of psychotic symptoms occurred much later than the average age of onset for schizophrenia, which falls in the early 20s for males and late-20s to early 30s for females (Sadock & Sadock, 2003). Nonetheless, late-onset schizophrenia is not infrequent, particularly in women (see Harvey, 2005, for a review). Evidence against a diagnosis of schizophrenia included the short duration and rapid resolution of her two psychotic episodes, her sustained remission for at least 5 years without antipsychotic medication, and the high level of functioning she maintained after symptom remission.

Despite the unlikely diagnosis of schizophrenia, it is possible that she carried some genetic vulnerability to schizophrenic-like symptoms. Genetic studies suggest that schizophrenia is a polygenic disease, in which thousands of common alleles each contribute a very small effect (International Schizophrenia Consortium, 2009). Therefore, it is possible that the patient inherited a slightly elevated polygenic burden—a small amount of schizophrenia-related genes, and thus was rendered more vulnerable to psychotic symptoms in the context of stress, without necessarily developing the full disorder.

Another possible diagnosis was a dissociative disorder. Although the patient did not meet criteria for any specific dissociative disorders listed in either *DSM-IV* (APA, 1994) or *DSM-5* (i.e., depersonalization/derealization disorder, dissociative amnesia, or dissociative identity disorder), there may well have been a dissociative component to her presentation. Dissociation is strongly associated with trauma (Dalenberg et al., 2012) and dissociative experiences, as well as generally blunted emotional responsivity, is a criterion for PTSD in both *DSM-IV* and *DSM-5*. Moreover, the predominance of anxiety in her presentation is consistent with dissociative defenses, as one view of dissociation is as a defense against the terror experienced during traumatic incidents (Dalenberg et al., 2012). In this vein, it is possible that her long period of cognitive impairment may have represented an atypical dissociative response to new recall of traumatic memories.

The evident response to memantine in light of the possible role of dissociation is particularly notable, raising the question of whether there is a glutamatergic role in dissociation and/or PTSD. Current research suggests that glutamate, as the major excitatory neurotransmitter in the brain, plays an important role in extinguishing traumatic memories (Myers, Carlezon, & Davis, 2011) and that memantine may indeed be effective in treating PTSD (Battista, Hierholzer, & Khouzam, 2007; Sani et al., 2012).

As noted above, this case of prolonged, but reversible cognitive impairment remains inconclusive with regard to diagnosis. Nonetheless, the two most likely diagnostic candidates appear to be some form of PTSD and encephalitis associated with excessive glutaminergic activity. Evidence for PTSD includes her history of childhood sexual trauma, onset of symptoms following both recovery of repressed memories and a trauma-related nightmare, and recurrence of paranoid symptoms years later following exposure to a trauma-related stimulus. Additionally her nightmare, anxiety, and paranoid symptoms can be conceptualized as the intrusive symptoms of PTSD, whereas her failure to remember her abuse until her late 40s along with her restricted affect may be conceptualized as avoidant or numbing symptoms. Therefore, based on *DSM-IV*, which was in use at the time of the patient's illness, the patient met criteria for PTSD with delayed onset. Nonetheless, this diagnosis may or may not explain the episode of cognitive impairment, which could also be attributed to encephalitis. Evidence for the encephalitis involving the glutaminergic system includes her apparent response to memantine, an NMDA receptor blocker. Memantine has been reported to reduce inflammation in the CNS in humans (Wu et al., 2009).

One possibility is that the stress of her PTSD precipitated an encephalopathy. Although we could find no reports of PTSD-precipitated encephalitis, an association between brain inflammation and PTSD has been found in a rat model of PTSD (Wilson et al., 2013). It is also possible that an incipient encephalopathy weakened the inhibition of her traumatic memories. There are multiple reports in the literature of a recurrence of PTSD symptoms from decades old trauma in elderly trauma survivors with neurocognitive impairment (Hamilton & Workman, 1998; Mittal, Torres, Abashidze, & Jimerson, 2001).

Finally, to what extent and in what way her acute psychotic episodes and her prolonged, but reversible cognitive impairment were part of the same pathological process is also not clear; however, it is reasonable to think they are related. There are several ways that the two conditions could be related to each other. It is possible that both the psychosis and the cognitive impairment reflected an atypical expression of severe PTSD. It is also possible that the psychosis was not a manifestation of PTSD, but rather an early marker of encephalitis. Finally, it is possible that the two conditions were unrelated to each other. Thus, although there is a high likelihood that the patient's PTSD was strongly contributory to her prolonged episode of cognitive impairment, we must draw any diagnostic conclusions with a strong note of caution.

To our knowledge there is only one other report of reversible but prolonged cognitive impairment as a likely result of childhood trauma (Montefiore et al., 2007). To the extent that additional cases with similar presentations are reported, this case would support the notion that persistent, severe, and reversible cognitive impairment may constitute a previously unrecognized and atypical posttraumatic response. If future research does identify such a posttraumatic syndrome, this would have significant clinical implications given the disturbingly large incidence of childhood sexual abuse and other forms of childhood trauma. More specifically, clinicians could evaluate for PTSD in patients presenting with atypical cognitive impairment without clear organic cause. Likewise, clinicians could evaluate for cognitive symptoms in patients with significant trauma histories. Finally, future research could investigate the efficacy of memantine in the treatment of PTSD and/or trauma-related cognitive impairment.

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