The sailor who won't follow orders

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Mr. L is rehospitalized 9 times in 3 months but repeatedly fails to adhere to outpatient treatment. What is the cause of his exasperating behavior?

CASE An unlikable patient

Mr. L, age 56, is admitted to the psychiatric unit at our Veterans Affairs Medical Center for active suicidal ideation; he has a history of self-injurious behaviors that include mutilation and overdose. He also has a history of alcohol dependence and multiple inpatient psychiatric admissions. He has never married and conflicts with his siblings—in whose home he has been staying—have led to frequent homelessness.

On presentation, Mr. L meets DSM-IV-TR criteria for borderline personality disorder, alcohol dependence, and bipolar disorder, current manic episode. He is unable to correctly assess social cues and lacks empathy—he speaks indifferently of the sequelae stemming from removal of his twin brother's "golf ball"-sized benign brain tumor.

His affect is silly and shallow. He also shows signs of haughtiness, disinhibition, grandiosity, and confabulation. For example, he says that while in the Navy he had 82 sexual exploits and developed a drug that cured herpes.

Dr. Henry is staff psychiatrist, Dr. Neptune is co-director of the inpatient psychiatry service, Dr. Chapman is a research scientist and director of education of the War-Related Illness and Injury Study Center, Dr. Schwartz is a research psychologist, Dr. Rosse is chief of psychiatry, and Dr. Deutsch is the associate chief of staff for mental health, Washington DC Veterans Affairs Medical Center. In addition, Drs. Henry and Neptune are clinical instructors, Dr. Schwartz is associate professor, Dr. Rosse is professor of psychiatry, and Dr. Deutsch is professor and associate chairman for clinical neurosciences, department of psychiatry, Georgetown University School of Medicine, Washington, DC.

We start Mr. L on divalproex, 1,500 mg/d, and quetiapine, titrated to 200 mg/d. After 3 days he is discharged, but this begins a cycle of repeated suicide gestures and readmissions—9 within the next 3 months. Each time he is discharged, Mr. L fails to follow through on treatment recommendations and is indifferent to our staff's annoyed reactions.

What strategy would you try next?

a) add an antidepressant to Mr. L's regimen
b) reevaluate his longitudinal history for evidence of functional decline
c) refer him for outpatient psychotherapy
d) refer him for electroconvulsive therapy

The authors' observations

Some of our staff members regard Mr. L's suicidal gestures as manipulative and feel angry and demoralized by his poor adherence to outpatient treatment plans. Their negative countertransference might have impacted how they evaluated Mr. L through repeated admissions and discharges. During Mr. L's ninth admission, we decide to reevaluate his longitudinal history for clues to his noncompliant behavior.

HISTORY Undocumented injury

Mr. L says he began drinking alcohol at age 16. He reports that he has grown marijuana

How would you handle this case?

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Despite repeated hospitalizations, Mr. L fails to follow treatment recommendations and is indifferent to our staff's annoyance

but has not smoked it since 1991. He denies using heroin or other drugs.

Mr. L reports that he completed a bachelor's degree in chemical engineering and served as a lieutenant in the Navy from 1973 to 1976, working in the radiation health/medical division within the medical service corps. He says he completed a master's degree in public health and held several industrial hygiene and radiation safety jobs. His last employment was approximately 3 years ago.

Mr. L states that he suffered a head injury in 1975 after falling off a ladder on a Navy ship. He describes losing consciousness for a brief but uncertain duration. He reports that he has developed a seizure disorder since this fall and a history of amnesia secondary to past seizures. His medical records contain no witnessed seizures. Mr. L also says he was hospitalized a few years ago and placed on a ventilator for 7 days for an undetermined reason.

What further testing would you order?

a) specialized electroencephalography (EEG) studies, including sleep deprivation and continuous video EEG monitoring
b) functional MRI with cognitive challenges to evaluate regions of diminished cerebral blood flow and metabolic activation
c) neuropsychological testing to probe memory and executive functions in particular
d) all of the above

The authors' observations

Based on Mr. L's report of a possible traumatic brain injury (TBI), we order a neurologic evaluation. A year earlier, MRI of the brain without contrast demonstrated minimal, nonspecific periventricular and subcortical, punctuate hyperintensities on flair and T2 weighted sequences that are nonspecific. Overall, the impression was "diffuse involutional changes and mild nonspecific periventricular and subcortical white matter hyperintensities," which might reflect covert vascular brain injury.

Mr. L's neurologic workup and EEG are essentially normal, except for abnormal tandem gait. CT indicates mild generalized atrophy and an area of low attenuation in the left temporal region that could represent an old infarct or cyst. MR angiography is interpreted as normal. Overall, these data suggest that Mr. L's cognitive deficits are not the result of focal brain pathology.

Our frustration over Mr. L's repeated readmissions for suicidal gestures led us to seek outside evaluation and consultation from a senior psychiatrist for assistance with discharge and treatment planning. Unlike our staff, the consulting psychiatrist did not harbor strong negative feelings toward the patient.

Mr. L's history of deterioration in psychosocial functioning prompted this psychiatrist to perform a thorough mental status examination that focused on cognitive elements and request formal neuropsychological testing.

EVALUATION Cognitive deficits

During mental status examination, Mr. L has difficulty recalling 3 items and uses a memory strategy to assist himself. He fails to recollect in reverse order the last 5 U.S. presidents. He spells "world" backward, but has difficulty repeating 6 digits forward and 4 backward. He is unable to do serial 7 subtractions from 93 to 65 correctly. He adequately copies interlocking pentagons and draws a clock with the correct time. He achieves a score of 28/30 on the Folstein Mini Mental State Exam, missing the date by 4 days and recalling 2 of 3 words.

These results suggest Mr. L has difficulty with attention and working memory, short-term memory, fund of general information and long-term memory, and ability to perform simple calculations. Most important, they indicate the need for further study, especially a neuropsychological test battery.

Mr. L's abnormal neuropsychological test results are summarized in the ***Table***. He manifests concretization of thought. His loss of conceptual fluidity is documented formally by measures of perseverative errors and categories completed on the Wisconsin Card Sorting

Table

Abnormal findings on Mr. L's neuropsychological testing

Cognitive domain	Test	Score	Interpretation
Mental status and effort			
Mental status	MMSE total score	28/30	2 of 3 items recalled after delay
Orientation	MMSE orientation questions	9/10	Date off by 4 days
Premorbid IQ estimate	WRAT-4 Reading Standard	66th percentile	Within normal limits. Inconsistent with educational attainment, but could be impacted by temporal lobe findings
Verbal memory			
Immediate memory	RBANS Immediate Memory Index (List and Story Learning)	1st percentile	Severe impairment
Delayed memory	RBANS Delayed Memory Index	1st percentile	Severe impairment
Recognition memory	List Learning	<1st percentile	Severe impairment
Visuospatial memory			
Delayed memory	RBANS Figure Recall	3rd percentile	Severe impairment
Executive functioning			
Cognitive flexibility	Trails B	10th percentile	Severe impairment based on educational attainment
	WCST	<1st to 12th percentile	Low scores: Nonperseverative errors, perseverative errors, and categories completed

* Tests of mental status effort, visuomotor processing speed, confrontation naming, visuospatial function, attention, and executive functioning fluency/initiation were within normal limits

MMSE: Mini Mental State Exam; WRAT: Wide Range Achievement Test; RBANS: Repeatable Battery for the Assessment of Neuropsychological Status; WCST: Wisconsin Card Sorting Test

Test (WCST). These findings support a diagnosis of acquired dementia.

The authors' observations

Mr. L's history, cognitive testing, head imaging, and behavioral observations suggest that several pathogenic factors contribute to his impaired functioning. First, he describes a TBI of unknown severity occurring in 1975. Although brain scans did not show evidence of midline shift or encephalomalacia, a direct blow to the head after falling from a height combined with possible post-injury seizures suggests a TBI of at least moderate severity.

Second, Mr. L describes an incident in which he required inpatient respiratory assistance. Although the precipitating medical event was unclear, anoxia or hypoxia is likely. A recent CT revealed low attenuation in the left temporal region that could represent an infarct.

Third, a recent MRI demonstrates periventricular and subcortical hyperintensities consistent with small-vessel disease. Finally, the patient's 30-year history of alcohol dependence likely contributed to his cognitive difficulties, particularly in memory and abstract problem-solving. Laboratory testing ruled out Wernicke's encephalopathy.

continued

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Neuropsychological testing results support a diagnosis of acquired dementia

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Impairment in memory and cognitive flexibility are common after a traumatic brain injury

Box 1

Injury-related personality changes can go undetected

Mr. L reported suffering a head injury from falling off a ladder. Personality changes that result from traumatic brain injury (TBI) of the sudden deceleration type—even when mild—are frequently referable to the frontal lobe, especially focal orbital and/or ventromedial damage of the prefrontal cortex.[1-5] This is because of the physical proximity of the sphenoid wing to the orbitofrontal region and effects of shearing.

As a result of this damage, patients lack insight into their accompanying cognitive and behavioral abnormalities, such as the egocentricity and impaired empathy shown by Mr. L. These changes might not be detected in clinical interviews and over brief periods.[2] Appreciating an acquired personality disturbance may require evaluating the patient's behavior over months or years.[2]

Mr. L's severe memory impairment and moderate to severe impairment in cognitive flexibility are commonly reported after a TBI of moderate severity. If an ischemic incident were the primary contributor, a lateralized pattern of cognitive dysfunction—which Mr. L does not exhibit—would be expected.

Although Mr. L likely has vascular dementia, his MRI findings do not indicate sufficient disease to account for his memory scores. Vascular dementia is associated with slow, stepwise cognitive deterioration, which is not consistent with severely impaired memory in a 56-year-old patient.

Finally, alcoholism is associated with cognitive difficulty in memory, visuospatial functioning, and abstract reasoning. Mr. L demonstrated significant difficulty in memory and abstract reasoning, but his visuospatial functioning was largely intact. In the absence of Wernicke's encephalopathy, chronic alcoholics generally do not show memory decrements in line with Mr. L's. His MRI results indicated only minimal ventricular and sulcal enlargement. Because atrophy is present in approximately 60% of chronic alcoholics, this finding provides evidence of a contribution, but the other contributory factors are associated with more definitive medical outcomes. Thus, alcoholism must be viewed as a secondary contributor to Mr. L's impaired functioning.

Taking into account all known contributors, TBI emerges as the primary diagnosis.

Consider neurologic injury

Recognizing and characterizing personality changes related to neurologic injury and disease is often problematic and unreliable, even when psychometrically validated instruments and structured diagnostic interviews are used (***Box 1***).[1-5] Mr. L's presentation differed from the more commonly reported "impulsive aggression" associated with closed head injury. Sequelae from TBI were contributing to his clinical presentation but was obscured by his shallow and silly affect, inability to accurately assess social cues, and lack of empathy.

In retrospect, Mr. L's seeking repeated inpatient psychiatric hospitalizations is consistent with poor planning and problem-solving skills. He has a limited repertoire of adaptive behaviors and has learned that suicidal gestures lead to admission and caretaking. These are important to him because he is frequently homeless. His lack of insight is seen in his unrealistic plans for employment in jobs requiring specialized technical skills.

Mr. L's case emphasizes the importance of considering brain injury as an etiologic factor in personality changes. It also highlights the complex—and seemingly nonoverlapping—functions and dysfunctions of the frontal lobe, including:

- source memory
- working memory
- sustained attention
- conceptual fluidity
- imaginative thinking
- impulse regulation
- planning and problem-solving skills.

Related Resource

• Silver, JM, McAllister TW, Yudofsky SC, eds. *Textbook of traumatic brain injury*. Washington, DC: American Psychiatric Publishing; 2005.

Drug Brand Names

Divalproex • Depakote
Quetiapine • Seroquel

Disclosure

The authors report no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products.

Deficits may be uneven, affecting one or several frontal lobe functions while sparing others.[1]

Documenting Mr. L's cognitive deficits and acquired dementia diagnosis changed our staff's perception of his behavior, enabling us to overcome negative countertransference (***Box 2***). We no longer regarded him as deliberately manipulative and refer him for appropriate treatment.

OUTCOME Residential placement

We realize Mr. L needs cognitive rehabilitation—including assistance with planning and problem solving—and arrange for his placement in a residential facility for this specialized rehabilitation. Mr. L receives supportive psychotherapy and cognitive remediation from a psychologist. He also is involved in incentive work therapy with a vocational rehabilitation specialist.

Box 2

How countertransference inhibited Mr. L's treatment

Countertransference can interfere with optimal workup and treatment of patients with character changes related to traumatic brain injury and neurodegenerative processes. When we interpreted Mr. L's suicidal gestures and hospitalizations as manipulative and deliberate, we failed to appreciate the limited number of things he could do to obtain a safe and protective environment. We also failed to recognize that his poor planning and problem-solving skills—as well as lack of insight into his illness—prevented him from adhering to outpatient treatment.

Originally, we attributed Mr. L's egocentricity, lack of empathy, and lack of adherence to axis II pathology.

Our staff's hostile feelings toward Mr. L led us to insufficiently consider his history—which is consistent with cognitive decline—during biopsychosocial evaluation and treatment planning. Mr. L's status as a frequently homeless, unemployed person reflects a sharp decline for a highly educated person who served as a Navy officer and performed radiation inspections on nuclear-powered vessels.

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Consider brain injury as an etiologic factor in personality changes

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Bottom Line

Consider traumatic brain injury as an etiologic factor in patients with personality changes. Imaging studies, mental status examination, and neuropsychological testing can help identify neurologic causes of cognitive and functional impairment. Be vigilant for adverse effects of negative countertransference when caring for noncompliant patients.