Disoriented and forgetful

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Ms. P, age 53, was diagnosed with Fabry's disease 5 years ago and now presents with memory problems, disorientation, and delusions. Is her medical disorder causing these mental status changes?

CASE Disoriented and delusional

Ms. P, a 53-year-old registered nurse, is admitted to the inpatient psychiatric unit with confusion, markedly disorganized thought processes, delayed verbal responsiveness, mood lability, and persecutory delusions. Shortly before hospitalization, Ms. P traveled approximately 360 miles from her daughter's home with a male companion. Noting changes in her mental status, the man brought Ms. P to the local hospital. She was then transferred to our facility.

At admission, Ms. P is not oriented to time. She denies auditory or visual hallucinations and does not display psychomotor agitation or retardation. She reports her mood as sad and her affect is mildly labile. Insight and judgment are considered poor.

Five years ago, Ms. P and her mother were diagnosed with Fabry's disease (FD) based on genetic analysis. Both women are carriers for the mutations and Ms. P's mother was found to have almost absent galactosidase activity.

What is the most likely cause of Ms. P's presentation?

a) delirium
b) dementia secondary to a general medical condition
c) major depressive disorder
d) dysthymic disorder
e) personality disorder

The authors' observations

FD is an X-linked recessive glycolipid storage disease caused by deficient activity of the lysosomal storage enzyme α-galactosidase A. The disorder affects both men and women and leads to progressive intracellular accumulation of globotriaosylceramide and other related glycosphingolipids.[1,2] The earliest FD symptoms—burning pain and acroparesthesias—typically appear in childhood (***Table 1, page 60***).[2] FD often is misdiagnosed in women because women tend to display neurologic symptoms later than men, with typical symptom onset in the teenage years.[3,4] Often, these symptoms are confused with psychiatric disorders or vague neurologic or pain syndromes.[5] In patients with no family history of FD, accurate diagnosis may not be made until adulthood.

Laboratory, dermatologic, and genetic tests can accurately determine the presence of FD.[1] However, because multiple organ systems are involved, initially attributing symptoms to FD is challenging, particularly in women.[1,3,5] For men, diagnosis

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How would you handle this case?

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Table 1

Typical signs and symptoms of Fabry's disease

Typical time at onset	Signs/symptoms
Childhood and adolescence (age ≤16)	Neuropathic pain
	Ophthalmologic abnormalities (cornea verticillata and tortuous retinal blood vessels)
	Hearing impairment
	Dyshidrosis (hypohidrosis and hyperhidrosis)
	Hypersensitivity to heat and cold
	Gastrointestinal disturbances and abdominal pain
	Lethargy and tiredness
	Angiokeratomas
	Onset of renal and cardiac signs (eg, microalbuminuria, proteinuria, abnormal heart rate variability)
Early adulthood (age 17 to 30)	Extension of any of the above
	Proteinuria and progressive renal failure
	Cardiomyopathy
	Transient ischemic attacks, strokes
	Facial dysmorphism
Later adulthood (age >30)	Worsening of any of the above
	Heart disease (eg, left ventricular hypertrophy, angina, arrhythmia, and dyspnea)
	Transient ischemic attacks, strokes
	Osteopenia and osteoporosis

Source: Mehta A, Beck M, Eyskens F, et al. Fabry disease: a review of current management strategies. QJM. 2010;103(9):641-659, by permission of Oxford University Press

Clinical Point

Neurologic symptoms of Fabry's disease often are confused with psychiatric disorders or vague neurologic or pain syndromes

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can be established by measuring plasma or urinary globotriaosylceramide or plasma α-galactosidase A in addition to genetic analysis. In women, genetic analysis is a better diagnosis strategy because elevations in globotriaosylceramide or α-galactosidase A may not be prominent. An algorithm for diagnosing and assessing patients with FD has been proposed.[2]

HISTORY Cognitive deterioration

Ms. P has had psychiatric symptoms such as depression and anxiety since childhood. However, 3 years ago she started to experience psychological and cognitive deterioration. Medical records indicate that Ms. P described memory and concentration problems over the previous few years. She also reported pain, weakness, and numbness in her left leg after surgery for a work-related back injury, for which she received a financial settlement through workers' compensation. Shortly thereafter, Ms. P separated from her third husband, moved in with her parents, and found work as a psychiatric nurse. She was dismissed after 6 weeks because she could not learn the electronic medical record system and had difficulty with memory and attention. Her performance on the Mini-Mental State Exam[6] at that time was 28 out of 30, which was within normal limits.

After her parents died 3 years ago, Ms. P lived with her daughter, who became her primary caregiver and legal guardian. Ms. P's daughter notes that her mother's impulsive and risky behaviors grew more pronounced. Ms. P went on shopping sprees and became sexually promiscuous.

Ms. P's psychiatric history includes childhood sexual abuse, hospitalization for a suicide attempt at age 19, and courses of psy-

chotherapy and pharmacotherapy. In addition to FD, Ms. P's medical history consists of coronary artery disease, type 2 diabetes mellitus, hypercholesterolemia, obesity, arthritis, back pain, fibromyalgia, and gastroesophageal reflux disease. Her family history is notable for alcohol abuse (both parents and a brother), lung cancer (mother), myocardial infarction (father), and Alzheimer's disease (father).

The authors' observations

Because α-galactosidase A is ubiquitous throughout the body, in addition to neurologic symptoms, FD involves multiple organ systems, with possible dermatologic, renal, gastrointestinal, cardiac, and cerebrovascular dysfunction. Despite growth in FD research, including the Fabry Outcomes Survey,[3] the psychosocial and neuropsychiatric implications of the disease remain unclear.[7] Behavioral presentations are idiosyncratic and unstable over time, depending on the structures impacted by progressive glycosphingolipid accumulation. Premature cardiovascular events (onset between age 30 and 40 for women), greater incidence of ischemic stroke or transient ischemic attack (7% to 30%), and frequent evidence of white matter lesions put FD patients at greater risk for developing presenile vascular dementia.[1,3] Nearly all male FD patients with dementia show some evidence of stroke or transient ischemic attack; cognitive functioning has not been well explored in female patients.[4] In a heterogeneous sample of 15 FD patients age 7 to 61, Segal et al[8] noted deficits in attention, processing speed, and executive function .75 to 1.95 standard deviations below normative means. No patients in this study had a history of stroke or transient ischemic attack; neuroimaging studies were not reported. Kolodny and Pastores[9] suggested multiple mechanisms for cognitive disruption, suggesting that mild dementia late in the disease course could be secondary to diffuse leukomalacia, multiple strokes, or possibly to lipid storage in hippocampal and frontal lobe neurons.

Psychiatric comorbidity

Psychiatric illness, such as depression or a personality disorder, may be comorbid with FD, although pathologic mechanisms remain unclear.[7,10,11] Hypothesized mechanisms include:

- psychosocial stress from chronic disease
- white matter changes
- disruption of impaired L-arginine-nitric oxide pathways.[7,12]

Crosbie et al[13] noted that FD patients presented with greater psychological distress as measured by the Minnesota Multiphasic Personality Inventory-2 than patients with Gaucher disease or chronic heart disease. However, no significant differences were found between patients with FD and those diagnosed with a pain disorder. In the Segal et al study, out of 11 adult FD patients, 4 were diagnosed with major depressive disorder, 1 with schizophrenia, 2 with schizotypal personality disorder, and 1 with borderline personality disorder.[8]

EVALUATION Brain abnormalities

Head CT scans (conducted 2 years ago and 6 months ago) revealed prominent cortical sulci likely caused by underlying volume loss, especially in bifrontal areas. A brain MRI performed 2 months ago indicated a moderate degree of subcortical atrophy in bilateral frontal and parietal regions. These radiology findings suggest mild to moderate frontal atrophy, mild degree of white matter changes, and slightly enlarged ventricles. An EEG showed background slowing and lack of an alpha rhythm, indicative of cerebral cortical dysfunction.

Ms. P's α-galactosidase A level was within normal limits; however, normal enzyme levels frequently are reported in symptomatic and asymptomatic female FD patients.[14] A dermatology consult confirmed the presence of skin findings characteristic of FD (ie, multiple cherry red papules extensively distributed throughout Ms. P's chest, abdomen, and back, as well as upper and lower extremities).

continued

Clinical Point

In FD, behavioral presentations are idiosyncratic and unstable, depending on structures impacted by glycosphingolipid accumulation

Clinical Point

Psychiatric illness, such as depression or a personality disorder, may be comorbid with Fabry's disease

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Visit this article at CurrentPsychiatry.com for a summary of Ms. P's neuropsychological tests

Table 2

Symptoms that preceded Ms. P's admission

Time frame	Symptoms
24 months before admission	Depressed mood Decreased ability to manage independent activities of daily living (eg, finances, cooking) Minimal objective cognitive impairment
12 months before admission	Increased depression Mild to moderate decline in cognitive functioning Visual and auditory hallucinations Impulsivity/poor impulse control Irrational decision-making Increased risky behavior
6 months before admission	Severe cognitive decline with cognitive symptom exaggeration Psychiatric symptom exaggeration Disorganized thinking Continued risky behavior and poor decision-making

Ms. P completed 2 neuropsychological assessments separated by 5 months. For a summary of the results of these tests, see this article at CurrentPsychiatry.com. Both assessments revealed grossly impaired intellectual capacity, memory, processing speed, and motor functioning. During the assessment, Ms. P could understand all directions with minimal changes from standardized protocols. Ms. P became insistent that she would not be able to complete memory tasks successfully. She gave up prematurely on tasks, saying they were too difficult. She admitted to guessing on several items because she did not want to continue the task.

Ms. P's performance on tasks measuring effort and validity of a person's neuropsychological presentation was consistent with someone exaggerating neurologic symptoms. A person with true dementia may perform as poorly as Ms. P did. However, Ms. P's scores likely underestimated her level of functioning, even if she was experiencing dementia. Ms. P could not complete tasks individuals with severe dementia complete successfully, such as simple addition and subtraction and digit repetition. Ms. P recalled several recent and remote events, such as her breakfast menu and location of her first assessment, but could not recall words practiced multiple times. Although Ms. P's scores on a complex card-sorting task were in the impaired range, a detailed review of her pattern indicated that although Ms. P could not generate any correct sorting categories, she made few repetitive responses and errors. This pattern is consistent with someone who understands task requirements, but deliberately avoids answering correctly. This suggests that she retained some ability for hypotheses generation and problem solving; however, because she exaggerated her symptoms, specific deficits could not be determined.

How would you manage Ms. P?

a) explain to Ms. P what the results of her neuropsychological assessment suggest
b) start Ms. P on a cholinesterase inhibitor such as donepezil
c) initiate an atypical antipsychotic, such as risperidone
d) all of the above

The authors' observations

Ms. P presented with an interesting manifestation of neuropsychiatric symptoms in the context of FD; however, common cardiac and cerebrovascular features of the disease were not fully developed. Ms. P experienced progressive cognitive and behavioral changes for 2 years before her admission (***Table 2***), which may represent a prodromal period leading up to what appeared to be a frontally mediated dementia syndrome. Müller et al[15] described a patient with FD who displayed a behavioral profile similar to Ms. P's that included increasingly unstable mood for at least 3 years, borderline personality disorder features, and rapidly fluctuating mood. A case study reported that risperidone, 1 mg/d, used to treat psychosis in a male FD

patient caused extrapyramidal symptoms.[16]

Ms. P presented with no evidence of stroke or transient ischemic attacks, which is atypical for FD patients with cognitive impairment. However, neuroimaging did reveal frontal atrophy that may be associated with her impulse control deficits, risk-taking behavior, emotional instability, and poor judgment. Her cognitive testing was notable for impairment and exaggeration of symptoms consistent with personality disorder symptoms. Possible reasons for exaggeration include a desire to maintain the sick role or secondary gain related to obtaining disability income.

Ms. P's behavior pattern could be caused by dementia with frontal features, possibly secondary to FD, in combination with personality and psychiatric pathology.

The mainstay of FD treatment is enzyme replacement therapy (ERT), which addresses the underlying enzyme deficiency. Available research indicates that ERT may reduce symptom severity and slow disease progression; however, further studies are needed to determine if it will reduce outcomes such as stroke, ischemic heart disease, or renal disease.[2]

TREATMENT Persistent deficits

Ms. P is started on risperidone rapidly titrated to 4 mg/d for delusional thinking and behavioral disturbance. After initially improving, she develops delirium when risperidone is increased to 4 mg/d. She has visual hallucinations, marked confusion with disorientation, worsened short-term memory, and an unsteady, shuffling gait. Risperidone is tapered and discontinued and Ms. P's motor symptoms resolve within 2 days; however, she remains confused and delusional. We start her on quetiapine, 25 mg/d titrated to 50 mg/d, and her agitation and delusional thinking progressively decline. Memantine, titrated to 20 mg/d, and rivastigmine, started at 3 mg/d titrated to 9 mg/d, are added to address her cognitive symptoms.

Over several weeks, Ms. P's mental status slowly improves and her drug-induced delirium completely resolves. However, she has persistent cognitive impairment characterized by compromised short-term memory and poor insight into her medical and psychological condition. She maintains unrealistic expectations about her ability to live independently and return to the workforce. The treatment team recommends

Related Resources

- National Institute of Neurological Disorders and Stroke. Fabry disease information page. www.ninds.nih.gov/disorders/fabrys/fabrys.htm.
- National Fabry Disease Foundation. www.thenfdf.org.
- Rozenfeld P, Neumann PM. Treatment of Fabry disease: current and emerging strategies. Curr Pharm Biotechnol. 2011;12(6):916-922.

Drug Brand Names

Donepezil • Aricept
Memantine • Namenda
Quetiapine • Seroquel
Risperidone • Risperdal
Rivastigmine • Exelon

Disclosure

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

Clinical Point

Ms. P's performance on tasks measuring effort and validity was consistent with someone exaggerating neurologic symptoms

Bottom Line

Fabry's disease (FD) is an X-linked recessive glycolipid storage disease caused by deficient activity of the lysosomal storage enzyme α-galactosidase A. It often presents with cardiovascular, cerebrovascular, dermatologic, and neurologic symptoms. FD patients may display confusion, mental status changes, and memory and concentration problems that may be confused with dementia or other neuropsychiatric disorders.

that Ms. P's daughter pursue guardianship and that she receive around-the-clock supervision after discharge from the hospital.

Clinical Point

Ms. P presented with no evidence of stroke or transient ischemic attacks, which is atypical for FD patients with cognitive impairment

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ULTRA-RAPID CYCLING continued from page 52

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Table

Ms. P's neuropsychological assessment results

	June	November
Intellectual functioning		
Wechsler Adult Intelligence Scale-III		
FSIQ	60	
VIQ	68	
PIQ	56	
Ravens Colored Progressive Matrices		70
Premorbid intellectual functioning estimates		
Peabody Picture Vocabulary Test-2		89
Barona Demographic Estimate	104	104
North American Adult Reading Test	99	
Memory functioning		
Wechsler Memory Scale-III		
Immediate memory	45	
General delay memory	47	
Auditory recognition delay	55	
California Verbal Learning Test-II		
Trial 1 (immediate recall)	<60 (raw = 3)	
Trial 5	<60 (raw = 3)	
Total Words Learned	<60 (raw = 15)	
Short Delay Free Recall	<60 (raw = 2)	
Long Delay Free Recall	<60 (raw = 4)	
Executive functioning		
Trail Making Test A	88	88
Trail Making Test B	failed to understand	failed to understand
Wisconsin Card Sort-64		
Number of categories	<60 (raw = 0)	
Errors	81	
Percent conceptual level responses	74	
Perseverative responses	107	
Perseverative errors	108	
COWAT FAS	65	69
Category exemplar	69	80
Motor functioning		
Finger Tapping Dominant Hand	68	
Finger Tapping Non-Dominant Hand	62	
Invalidity/effort		
TOMM		
Trial 1	raw = 34	raw = 37
Trial 2	raw = 42	raw = 45
Recognition	raw = 44	
MSVT verbal		fail
MSVT nonverbal		fail

Scores provided are standardized (mean = 100; SD = 15). Raw scores are also provided when indicated.

COWAT: Controlled oral word association test; FSIQ: Full Scale IQ; MSVT: Medical Symptom Validity Test; PIQ: Performance IQ; TOMM: Test of Memory Malingering; VIQ: Verbal IQ