Association of Chronic Fatigue Syndrome and Acute Psychotic Episode: Is It Coincidental?

Sir: We present a case of a woman who had suffered from chronic fatigue syndrome (CFS) for several years and was admitted for an acute psychotic episode. This association has rarely been described.

Case report. Ms. A, a 43-year-old mother of 2 children, was admitted in January 2006 with delusion and hallucinations following a period of exacerbated fatigue. She was afraid that her children would be abducted by the devil and tried to protect them. She begged her children not to get near the walls of her house for fear that the devil could erupt from the walls and take them.

Ms. A first experienced persistent fatigue 3 years before admission. Prior to this, she had been a very active woman. She had to stop working and was able to participate in only very few activities during the day. She attributed her fatigue to the overwhelming task of educating her hyperkinetic 9-year-old son.

She had a depressive episode of several months' duration 10 years before admission, following an abortion of a pregnancy involving a malformed child. This episode had subsided without relapse. She had infectious mononucleosis 20 years before admission. A polysomnographic test 2 years before admission showed many awakenings interrupting Ms. A's sleep pattern. She was then diagnosed with chronic fatigue syndrome according to the criteria of Holmes[1] and Fukuda.[2] Antidepressive medication was prescribed; it alleviated the secondary depressive symptoms but had no impact on her fatigue complaint.

During Ms. A's hospitalization, her blood analysis results were unremarkable, excluding common organic causes of fatigue. Results of her neurologic examination at admission were normal. Her brain computed tomography (CT) scan showed frontal cortical atrophy, but neuropsychological tests failed to show major cognitive impairments.

Olanzapine was prescribed at the dosage of 15 mg/day, and her symptoms gradually subsided. She was discharged 1 month after admission, totally free of her psychotic symptoms. Her neuroleptic treatment was changed to 10 mg of aripiprazole because of excessive weight gain. Aripiprazole was as effective as olanzapine but allowed her to return to her usual weight. The treatment was gradually stopped after 1 year, with no recurrence of psychotic symptoms.

The association between CFS and psychosis has rarely been described. We are aware of only 2 other case reports. The first describes a 28-year-old man who developed CFS after mononucleosis and suffered afterward from a manic episode with psychotic characteristics.[3]

The second case report describes a 22-year-old patient who developed CFS after mononucleosis and was later diagnosed with schizophrenia.[4] His CT scan showed diffuse nonspecific atrophy and widening of sulci, especially in the cerebellum and frontal lobes. His condition had rapidly improved with classical neuroleptics and did not seem to relapse afterward.

Epstein-Barr virus is clearly associated with the development of CFS,[5,6] even if we still do not understand the pathophysiology involved. Several case reports of psychotic episodes following Epstein-Barr virus infection, but without CFS symptoms, have also been described.[7–9] Mononucleosis could be responsible both for the development of CFS and for the psychotic disorder, if the Epstein-Barr virus remains latent and is reactivated in times of greater stress or immunologic weakness.[10] The frontal atrophy in our patient as well as in the 22-year-old patient described earlier could be a sequela of an Epstein-Barr virus–induced central nervous system insult. Systematic evaluation of psychotic symptoms should be recommended in patients presenting with mononucleosis.

The authors report no financial or other relationship relevant to the subject of this letter.

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Atypical Neuropsychiatric Presentation of Addison's Disease: A Case Report

Sir: Addison's disease is a potentially life-threatening endocrine condition with known neuropsychiatric presentations. Anglin and colleagues' review[1] of the existing literature reported disturbances of mood, motivation, and behavior as the common presentations. Psychosis was associated with severe conditions and Addisonian crisis. Rare

presentations included self-mutilation and catatonia.[1] We report a case of Addison's disease with episodic neuropsychiatric presentations.

Case report. Mr. A, a 43-year-old man, presented to us in April 2007 with forgetfulness, motor restlessness, rearranging household articles repeatedly, and talking excessively for 1 day. He had experienced an episode in January 2007 of perceptual abnormalities of seeing his body transform into a tube containing multiple revolving discs and visual and command auditory hallucinations, which resolved spontaneously in 1 day. His family and personal history and a systemic examination revealed no abnormalities. Results of serum electrolytes, liver and renal function, and blood glucose tests; hemogram; brain magnetic resonance imaging; and electroencephalogram were within normal limits.

A subsequent episode in early July 2007 was characterized by crying spells, depressive ruminations, and insomnia lasting 3 days, and an episode in late July 2007 was characterized by increased speech, decreased need for sleep, and elated affect lasting 7 days.

All episodes were abrupt in onset, preceded by headache and vomiting of 2 days' duration, and resolved spontaneously without medications. Mr. A was asymptomatic between episodes.

A thyroid function test revealed low triiodothyronine and thyroxine levels and normal thyroid-stimulating hormone levels. A complete metabolic investigation revealed low serum cortisol (baseline 2.54 µg/dL). The short Synacthen test indicated possible adrenal failure, as serum cortisol level (13.09 µg/dL) did not increase to expected values (18 µg/dL) at 60 minutes post-Synacthen challenge. The patient was started on replacement treatment with thyroxine 50 µg/day and prednisolone 10 mg/day and has remained asymptomatic to date.

Although neuropsychiatric manifestations are common in Addison's disease, our understanding of its pathophysiology remains unclear. Hyponatremia,[2] hypoglycemia,[3] Hashimoto's thyroiditis,[1] and increased endorphin production[4] have been hypothesized to underlie these presentations.

In our patient, decreased glucocorticoid production may possibly explain the presentations. Henkin and Daly[5] have proposed that decreased glucocorticoid production results in increased neural excitability and increased conduction velocity along peripheral axons while prolonging conduction across synapses. This probably delays the arrival of signals from the periphery to the central nervous system, resulting in a deficit between perception and integration of sensory signals, and is a possible mechanism for hallucinations and psychosis. The reasons behind the atypical presentations, however, remain unclear.

The authors report no financial or other relationship relevant to the subject of this letter.

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Low Cholesterol, Delinquency, and Suicidality

Sir: It is well known that incarcerated adolescents are at high risk of suicide.[1] We suggest that lower than normal cholesterol levels might be a biological marker of both a violent behavior leading to subsequent arrest and suicidality.

Mounting evidence suggests a relation between low cholesterol and violence, aggression, and hostility.[2,3] In particular, a statistically significant association has been shown between serum total cholesterol concentration below the 25th percentile (< 145 mg/dL) and aggressive behaviors among non–African American children from a national sample of noninstitutionalized, school-aged children.[4] Lower than median cholesterol levels have also been associated with the onset of conduct disorder during childhood among male criminals.[5]

On the other hand, lower than average cholesterol levels seem to indicate a population at risk for parasuicide or completed suicide,[6] and follow-up studies have found that individuals with lower cholesterol levels have an increased risk of completing suicide.[7] Indeed, the inheritance of defects leading to low cholesterol levels could predispose individuals to violent and suicidal behavior.[8,9]

Therefore, low cholesterol may be a risk factor for delinquency and suicidality or a risk marker for genotypes that predispose to such behaviors.

The authors report no financial affiliations or other relationships relevant to the subject of this letter.

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Diagnosis of Nonorganic Monoplegia With Single-Pulse Transcranial Magnetic Stimulation

Sir: Single-pulse transcranial magnetic stimulation (spTMS) is used in clinical practice to evaluate the integrity of the corticospinal tracts.[1] Here, we describe how spTMS assisted in proving that a patient with plegia of an upper limb and ipsilateral ptosis was malingering.

Case report. A 35-year-old woman was admitted to the neurology department in August 2007 due to plegia of the upper limb and ptosis on the right, which had been present for the previous 4 years but had worsened suddenly in the last 24 hours. Neurologic examination disclosed monoplegia (Medical Research Council power grade 0 on the right upper limb). There were no signs of spasticity or sensory deficits. Plantar responses were flexor. Right ptosis was present, but it was atypical, giving the impression of a voluntary contraction of the mimic muscles of the left, rather than that of a pyramidal lesion.

Magnetic resonance imaging (MRI) of the brain and cervical spinal cord, nerve conduction studies, electromyogram, and lumbar puncture had been performed 3 times previously. The results were unremarkable. Repeat brain and cervical MRI were again normal. We therefore re-evaluated the patient, focusing on potential nonorganic causes of her clinical picture (hysteria or malingering). Indeed, the patient's husband reported that the symptoms had worsened several times, always in periods of family distress. Given this, we proceeded to spTMS evaluation.

Single-pulse TMS was delivered with a Magstim Rapid-2 stimulator (Magstim Company Ltd., Whitland, United Kingdom). The hand motor cortex was targeted with a 70-mm figure-of-8 coil. Stimuli were delivered at 100% stimulator output. Motor-evoked potentials (MEPs) were recorded from the abductor digiti minimii (ADM) muscles bilaterally. The central motor conduction time (CMCT) was calculated as CMCT = (MEP latency) – (peripheral conduction time).[1]

The CMCT was within normal limits (right ADM = 6.8 ms, left ADM = 6.7 ms). During the examination, the patient was distressed by the fact that she was not accustomed to the procedure and did not know its diagnostic potential. She was also surprised by the movement of her right upper limb in response to the magnetic pulses, and she persistently questioned us as to the meaning of our findings. On the basis of the normal CMCT, we reassured her that things were much better than she thought so far, that her symptoms would improve, and that she could accelerate her recovery by cooperating with the attending physicians.

During the following day, the patient's right upper limb was still plegic, yet the ptosis was disappearing during conversation and reappearing when she was relaxed. Furthermore, she announced to her husband that the spTMS evaluation indicated a grave prognosis, contrary to what we had told her.

On the basis of this evidence, we diagnosed malingering (according to DSM-IV-TR criteria) and referred the patient to the psychiatry department for further evaluation.

Single-pulse TMS is a relatively new technique that is not used frequently in everyday practice; thus, patients are unaccustomed to it. As a result, it can prove useful in the management of malingerers simulating corticospinal lesions by providing neurophysiologic evidence on the integrity of the corticospinal tracts and by producing effects that surprise the patients.

The authors report no financial affiliations or other relationships relevant to the subject of this letter.

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Rash and Desquamation Associated With Risperidone Oral Solution

Sir: Antipsychotic agents are known to cause adverse cutaneous reactions in approximately 5% of the individuals for whom they are prescribed,[1] and there have been some reports of dermatologic disorders associated with conventional antipsychotics.[2–5] Although atypical antipsychotics cause fewer dermatologic symptoms than typical antipsychotics,[6] some recent reports have associated olanzapine[7–9] and clozapine[10–13] with skin lesions.

To our knowledge, there have not been any reports of adverse cutaneous reactions associated with risperidone or risperidone oral solution. We present a case in which adverse cutaneous reactions were developed after the initiation of risperidone oral solution treatment and soon disappeared after discontinuation of this antipsychotic agent.

Case report. Mr. A, a 37-year-old man with DSM-IV bipolar I disorder, was referred to our inpatient clinic in July 2007. His first manic episode had developed at the age of 23 and he had experienced several manic episodes since then. Mr. A was

euphoric and irritable. His speech was rapid. He continuously complained about politicians, and said that God had given him an enormous power to punish them. His sleep and appetite were severely disturbed.

We started treatment with risperidone oral solution 2 mg at bedtime, lithium 900 mg/day, diazepam 15 mg/day, zolpidem 10 mg at bedtime, and procyclidine hydrochloride 5 mg at bedtime.

On the third day of treatment, Mr. A complained of the facial flushing, and we could find rashes under both eyes.

On the fourth day of treatment, risperidone oral solution was titrated to 4 mg/day, and lithium was titrated to 1200 mg/day due to his persisting manic symptoms. Other medications were maintained at previous doses.

On the fifth day of treatment, the rashes had spread over Mr. A's whole face and neck, and areas of desquamation had developed over his face.

On the sixth day of treatment, risperidone oral solution treatment was stopped, and quetiapine 150 mg/day was started and titrated up to 600 mg/day to manage his manic symptoms.

On the second day after risperidone oral solution treatment was stopped, Mr. A's skin lesions had completely disappeared. Lithium 1200 mg/day was maintained and serum lithium level was 0.73 mmol/L.

Some studies have indicated that several cutaneous adverse effects, which may be dose dependent, are associated with lithium treatment.[14,15]

In our case, although lithium was coadministered with risperidone oral solution, the immediate improvement of the skin lesions after stopping risperidone oral solution suggests that the adverse skin reactions were because of risperidone oral solution rather than lithium.

The combined effect of risperidone oral solution and lithium on the skin cannot be completely excluded, however, and further studies are needed.

After the replacement of risperidone oral solution with quetiapine, the skin lesions did not recur. We think that quetiapine is relatively safer than risperidone oral solution in view of the dermatologic adverse effect.

The majority of adverse cutaneous events are benign and easily treated.[1] However, these adverse events may impact compliance.

Our case suggests that risperidone oral solution may cause adverse skin reactions like rashes and desquamation and that quetiapine can be an alternative choice in such cases.

The authors report no financial or other relationship relevant to the subject of this letter.

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Reexamining the Elderly Patient's Presentation With Depression

Sir: We wish to comment on the well-written and well-designed article by Voils et al.[1] In our clinical practice in 2 of the major pain centers in the Midwest, we frequently see patients referred from primary care offices who are allegedly suffering from depression and who present with insomnia, anxiety, somatization, low energy, and some sadness. It is difficult for the patient to articulate the word *depression*, let alone acknowledge its presence. Anxiety, on the other hand, appears to be less vulnerable to the patients' self-esteem and easier for them to articulate. Another description manifest by the patient includes "I am under some stress."

Insomnia and somatization, noted on the Hamilton Rating Scale for Depression[2] as items 12 and 13, however, are seen prominently in our practice. Following months to years of the existence of depression, characteristics manifest by the patient are somatic and include headaches, neck pain, back pain, shoulder pain, hip pain, leg pain, arm pain, abdominal pain, and chest pain of noncardiac origin. Generally with these complaints, the primary care physician reacts in a diagnostic manner and orders radiographic testing, laboratory testing, and workups, including electromyograms and blood tests, in an attempt to document the physical pathology of pain complaints of the patient and to determine the etiology.

Patients often will react in a manner as to protect themselves and will not disclose what is true but will rather disclose that which they perceive would be favorable to the clinicians who will be evaluating the outcome of the structural patient

evaluation tools or forms of evaluated tools provided to the patients and/or families. Consequently, an additional medication focused at a new complaint or an attempt to abate the patient's pain complaints is often initiated with futile outcomes. It is not uncommon to see the patient change the somatic complaints from one body organ or system or location to that of another.

Clinical judgment provides an index of suspicion to discriminate psychopathology (hopelessness, worthlessness, loss of self-esteem, suicidal ideation, and guilt) from that which is revealed by the elderly patient presenting with disseminated somatic pain complaints. DSM-IV-TR provides multiple diagnoses which are imbedded with somatic complaints that reflect pain and psychiatric complaints. Elderly patients may not vocalize or express the psychiatric events underlying their geriatric presentation. Commonly, elderly patients elect to guise these somatic issues as a mask for their psychiatric issues (depression, anxiety, etc.). This masking presentation of somatic symptoms permits the opportunity for presentation into the health care system. To preserve the patient's self-esteem, we often address with equal vigor both the somatic complaints and the underlying psychiatric basis. Consequently, this joint focus of attention at resolution of both the presenting somatic complaints and the psychiatric basis facilitates a more successful and robust remission in the elderly patient. This effort is in realization that painful physical comorbidity may be less than adequately addressed and opens the patient-provider relationship to decremental changes in both their physical painful comorbidity and the consequent psychiatric sequelae.

In our clinical practice, we have noticed that the time when depression may be actually declared by the patient as a comorbidity is after he or she has established a fiduciary relationship of trust and confidence with the clinician. We often advise other clinicians, such as fellows and residents, that they "don't talk to the patients but talk with them, stop hearing them and begin listening to them." Such a clinical pearl assists in building a relationship of trust and confidence with the patient over time until the disclosure of depression may appear.

It is also appropriate to remember that when depression and comorbid anxiety exist, the anxiety may persist after the depression has reached remission. This may also occur with insomnia. Insomnia may not uncommonly coexist but additionally may be independent of depression and must be addressed once depression has achieved remission.

These observations are based on the hundreds of patients who are sent to a pain center from primary care physicians who appear to have reached academic and professional frustration in not being able to satisfy the patients' need for amelioration of their somatic complaints. More often than not, treatment has been focused on duration of somatic complaints as opposed to the patient's psychosocial and emotional issues that may remain undisclosed or disguised.

This letter was shown to Dr. Voils, who declined to reply.

—Editor

The authors report no financial or other relationships relevant to the subject of this letter.

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