Communications

Depression and Conversion Hysteria in Chronic Inflammatory Polyradiculoneuropathy

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Chronic inflammatory polyradiculoneuropathy (chronic or relapsing Guillain-Barré syndrome) is a demyelinating disorder of nerve root and peripheral nerve with principally motor symptoms.[1] Sensory disturbances or ataxia may be prominent initially or subsequently.[2] Although the efficacy of various therapies in acute inflammatory polyradiculoneuropathy is unclear, the chronic form is responsive to steroid treatment.[3] Presentations of seemingly functional character are infrequently described, even though the physiological alterations in the disorder can produce signs and symptoms commonly associated with functional disorders.[4-6] This report illustrates the importance of considering underlying medical illness as a precipitant of apparent conversion symptoms, and inflammatory polyradiculoneuropathy as a reversible cause for motor and sensory complaints of seemingly psychogenic origin.

Case Report

A 29-year-old schoolteacher lost her position soon after the difficult birth of her first child. During the subsequent month she developed sadness, fearfulness, irritability, and difficulty in sleeping, and noted intermittent leg weakness and clumsiness in walking. She fell several times while descending stairs, and noted waxing and waning paresthesias in her feet and legs. She became increasingly concerned about her ability to care for her family, and suffered anorexia and weight loss. These symptoms worsened after she was defeated for re-election to an office in her teachers' union, and she experienced intermittent weakness and paresthesias in her arms, periodically dropping objects from her hands. Increasing efforts to continue her daily activities caused worsening of weakness and sensory complaints, and she developed feelings of fatalism and loss of control. Neurologic examination results were unremarkable, but physical findings suggested thyroiditis, for which she was given a brief course of methylprednisolone sodium succinate, with resolution of symptoms for two weeks. She reported hopelessness and suicidal ideation when her symptoms returned, and she was referred for neurologic and psychiatric evaluation. Her past medical and psychiatric history was unremarkable, and family history was noncontributory.

Her general examination was unremarkable except for mild obesity and fullness and slight tenderness in the neck, without palpable thyromegaly. Neurologic examination showed subjective difficulty in smiling and pursing the lips, although spontaneous facial movements were normal. Giving-way weakness of ratchety quality was present in all muscle groups. Sensation was intact, but she reported tingling paresthesias in her feet and hands. Her gait was stumbling and histrionic, and coordination was inconsistently impaired. Deep tendon reflexes, absent at the ankles, were otherwise symmetric but hypoactive. Her affect was labile, alternating between sardonic hostility and outbursts of tearful despair. Her conversation was tangential and occasionally inappropriate, and associations were at times loose. She refused

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to answer questions concerning orientation and mentation.

Complete blood count, blood chemistries, thyroid function tests, Westergren sedimentation rate, serum B_{12} and folate levels, serum protein electrophoresis, urinalysis, chest roentgenogram, electrocardiogram, electroencephalogram, visual and brain stem auditory evoked potentials, and computed tomographic brain scan were normal. Serologic tests for syphilis, hepatitis, rheumatoid factor, and antinuclear antibodies were negative, as was porphyrin screening.

Psychiatric interview indicated massive denial but suggested feelings of ambivalence about parenthood, inadequacy in job performance, and unmet dependency needs. Marital and family dysharmony was present, with particular conflict between the patient's clergyman husband and her parents, who were ministers of a different denomination. Her parents strongly disapproved of her decision to join her husband's church and had communicated little with her since that time. Minnesota Multiphasic Personality Inventory (MMPI) scales indicated a withdrawn and socially introverted orientation but no evidence of specific psychopathology. The psychiatric impression was adjustment disorder with depressed mood and a probable conversion disorder as the origin of the patient's symptoms.

Nerve conduction findings were as follows: very high stimulus threshold and very slow conduction velocity to right ulnar nerve stimulation, prolonged right median nerve distal latency and very high stimulus threshold, absent right median and ulnar sensory nerve evoked potentials, very high right peroneal nerve stimulus threshold with very slow conduction velocity and prolonged distal latency. Partial ulnar nerve conduction block between elbow and wrist and partial peroneal nerve conduction block between knee and ankle were present. Electromyography showed occasional fibrillation potentials, motor unit potentials of normal size and shape, reduced motor unit potential recruitment, and a marked increase in discharge frequency. The findings were consistent with acquired chronic demyelinating peripheral neuropathy, which was confirmed by sural nerve biopsy. The patient was given a course of high-dose methylprednisolone sodium succinate, and dysphoria, weakness, incoordination, and paresthesias subsided.

Discussion

This patient developed subjective weakness, incoordination, and sensory disturbance in the setting of acute situational stresses. She experienced transient improvement with steroid therapy, but had evidence on psychiatric examination of depression and conversion hysteria, and had a plausible psychodynamic explanation for the development and progression of her symptoms. She had giving-way weakness of apparently functional character and had absent ankle jerk reflexes, but was not otherwise areflexic. The initial clinical impressions of functional illness were substantially modified by electrophysiological evidence of chronic demyelinating polyradiculoneuropathy, which was confirmed by biopsy and successfully treated with steroids.

Previous series of patients with this disorder have emphasized occasional atypical presentations involving sensation or coordination, and preserved reflexes or extensor plantar responses have rarely been reported.[2,3] No hysterical or depressive features have heretofore been described, but the potential for psychological disturbance in consequence of severe neurologic deficit has been emphasized in the acute form.[7] A marked dissociation between clinical manifestations and electrophysiological indices of demyelination and remyelination has been demonstrated[8] and was encountered in this patient. It is likely that the severe symptoms in the absence of objective neurologic findings were a reflection of physiological alteration, and signs of nonorganic weakness and incoordination may have been due to aberrant peripheral nerve impulse transmission.

Impaired conduction of rapid impulse trains has been demonstrated in partially demyelinated peripheral nerve,[4] and increase in firing rate of demyelinated axons has been shown to induce conduction block.[5] Such nerve fibers are also more susceptible to sudden and intermittent conduction block with small environmental changes.[6] An aberrant response to increased firing rate could explain the patient's intermittent and ratchety motor symptoms and exacerbation of symptoms with effort. A similar process in partially demyelinated central axons could be responsible for the seemingly functional symptoms and signs occasionally encountered in multiple sclerosis.[9]

The patient's complaints may have been aggravated by significant situational stresses, but their response to detection and treatment of inflammatory polyradiculoneuropathy suggests a primary neurologic origin. In any case, concomitant medical illness has been found to be one of the most frequent precipitating causes of depression in primary care practice,[10] and reaction to underlying illness is often admixed with reaction to situational factors. Her MMPI findings, with clinical scale values within normal limits, also suggest a primary organic origin for her symptoms.[11] Any factors suggesting underlying organic disorder in such patients must be weighed carefully, inasmuch as up to 20 percent of diagnoses of conversion hysteria may be retrospectively proven wrong by the eventual recognition of a physical illness that was the cause of the initial symptoms.[12] As yet unrecognized physical illness should be considered as the cause of conversion symptoms generally, and chronic inflammatory polyradiculoneuropathy should be considered particularly in the etiology of subjective weakness, paresthesias, and incoordination of otherwise obscure cause. Such "functional" illness may, in fact, represent altered function of the central or peripheral nervous system.

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