Evaluation of Neurologic Deficit Without Apparent Cause: The Importance of a Multidisciplinary Approach

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Received December 6, 2006; accepted April 17, 2007

Abstract

Background/Objective: A patient presenting with an acute neurologic deficit with no apparent etiology presents a diagnostic dilemma. A broad differential diagnosis must be entertained, considering both organic and psychiatric causes.

Methods: A case report and thorough literature review of acute paraplegia after a low-energy trauma without a discernible organic etiology.

Results: Diagnostic imaging excluded any bony malalignment or fracture and any abnormality on magnetic resonance imaging. When no organic etiology was identified, a multidisciplinary approach using neurology, psychiatry, and physical medicine and rehabilitation services was applied. Neurophysiologic testing confirmed the absence of an organic disorder, and at this juncture, diagnostic efforts focused on identifying any psychiatric disorder to facilitate appropriate treatment for this individual. The final diagnosis was malingering.

Conclusions: The full psychiatric differential diagnosis should be considered in the evaluation of any patient with an atypical presentation of paralysis. A thorough clinical examination in combination with the appropriate diagnostic studies can confidently exclude an organic disorder. When considering a psychiatric disorder, the differential diagnosis should include conversion disorder and malingering, although each must remain a diagnosis of exclusion. Maintaining a broad differential diagnosis and involving multiple disciplines (neurology, psychiatry, social work, medical specialists) early in the evaluation of atypical paralysis may facilitate earlier diagnosis and initiation of treatment for the underlying etiology.

J Spinal Cord Med. 2007;30:509–517

Key Words: Hysterical paralysis; Atypical paralysis; Conversion disorder; Malingering; Spinal cord injuries

INTRODUCTION

The patient presenting with an apparent neurologic deficit that does not correlate with objective clinical or radiographic findings presents a diagnostic dilemma. It is incumbent on the physician to eliminate the possibility of an organic etiology responsible for the patient's symptoms. In the subset of patients in whom no neurologic abnormality can be found, a psychogenic etiology must be explored. Frank malingering and factitious disorders need to be excluded.

A meticulous physical examination coupled with the appropriate imaging studies has a high sensitivity for the identification of a neurologic lesion. The sensitivity of plain films alone for the evaluation of cervical spine fractures has been reported to range from 92% to 96%, with a specificity of 85% to 94% (1). Computed tomography (CT) imaging has a reported sensitivity of 96% to 100% and specificity of 90% to 100% for the diagnosis of fracture in the cervical spine (1). In the case of bilateral lower extremity paralysis in the absence of a known injury to the spine, a complete evaluation of the neural axis should include magnetic resonance imaging (MRI) of the brain, cervical, and thoracolumbar spinal cord. Metabolic disorders, which may affect the conduction of the neural pathways, must also be considered.

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Given the clinical urgency of expeditiously identifying and initiating treatment of a possible spinal cord injury, the emphasis of the initial evaluation is to identify organic causes of the neurologic deficit (2,3). A traumatic event is a significant psychologic stressor, and the clinician must also be aware that psychiatric maladies may present with acute loss of neurologic function (4). Disorders of mood can be associated with elevated symptom reporting and reduced functioning in the setting of a general medical condition. Mood may serve to increase symptom perception and reporting. Watson and Pennebaker (5) found that a mood disposition of negative affectivity was reflected in self-report stress scales and correlated with health complaint scales. How an individual evaluates or appraises their mood has been shown to affect both distress and symptom and illness reporting (4–6).

With the clinical presentation of atypical paralysis, conversion disorder is a frequently considered diagnosis. While frequently cited, somatoform disorders are relatively rare compared to disorders of mood that may also account for atypical symptom reporting (4). The DSM-IV diagnostic criteria necessitate that psychologic factors adversely affecting a general medical condition be excluded before diagnosing a somatoform disorder (4).

Conversion disorder has a reported rate of 0.011% to 0.5% (4) and is 2 to 10 times more common in women (7,8). Patients presenting with a conversion disorder tend to be of lower socioeconomic status and range from 10 to 35 years of age (1). The lifetime incidence of patients presenting with a somatization disorder is 0.1% to 0.5% and with conversion disorder is 0.01% to 0.3% (4,9).

Psychogenic paralysis defines a form of conversion disorder (conversion disorder with motor symptom or deficit) in which a patient presents with loss of motor and sensory function subsequent to a psychiatric stressor (4,10). Associated symptoms can include loss of vision and hearing, pseudo-seizures, ataxia, and genitourinary and gastrointestinal dysfunction (7). The severity of motor symptoms generally exceeds that of sensory loss; rectal tone and reflexes are usually retained (8,11). In a small study, 12 of 14 patients diagnosed with hysterical paralysis exhibited either diminished rectal tone, diminished rectal sensation, or diminished deep tendon reflexes. However, all 14 retained their bulbocavernosus reflex (12). Rectal tone has a volitional component, and rectal sensation is to a degree a subjective finding based on patient reporting. Similarly, deep tendon reflexes may be subjected to a degree of voluntary control based on antagonistic muscle contraction. The bulbocavernosus reflex is considered to be involuntary. Recovery of function in the setting of psychogenic paralysis is rapid and complete, with more than 90% of patients showing complete recovery within 1 month (7,11).

When diagnosing a conversion disorder, there is always the concern of missing an organic cause. Slater and Glithero (13) reported rates of missed organic causes

as high as 35%, suggesting the diagnosis of conversion disorders was a “delusion.” A recent review by Stone et al (14) based on 27 studies concluded that the average rate of misdiagnosis was significantly lower, only 4% on average. The now routine use of MRI and CT scans to evaluate the neural axis has dramatically increased the diagnostic sensitivity for detecting subtle organic findings. Stone et al (14) concluded that the lower rates of misdiagnosis are largely because of improvements in study quality and improved definitions of “hysteria.”

A thorough psychologic assessment is necessary when considering the possibility of conversion or somatoform disorders. DSM-IV criteria for conversion disorder (reviewed below) require that psychologic factors be associated with the onset or exacerbation of symptoms. Psychologic conflicts may be symbolically resolved by the symptoms (primary gain), and there may also be an associated secondary gain as well. Identifying these psychologic factors and assessing internal and external motivations is mandatory to approach a diagnosis, particularly when the differential diagnosis involves malingering.

The DSM-IV presents an efficient decision tree for the evaluation of differential diagnoses when considering somatoform disorders. Before reviewing this algorithm, it is necessary to first define the major disorders.

Conversion Disorder

Conversion disorder is a motor-sensory deficit after a traumatic event without an organic etiology (4). Although historically, conversion disorder has been associated with the patient exhibiting “la belle indifference,” a characteristic lack of normal concern about their deficit (15), the patient may also present in a dramatic fashion. There is some overlap with somatization disorder; however, conversion disorder is distinguished by its relatively acute nature and narrower symptom complex. The diagnosis of conversion disorder requires that psychologic factors be associated with the onset or exacerbation of symptoms. The American Psychiatric Association DSM-IV-TR (4) defines diagnostic criteria for a conversion disorder as follows:

- At least 1 symptom or deficit of sensory or voluntary motor function suggesting neurologic or general medical condition.
- The symptoms are not limited to pain or sexual dysfunction, do not occur exclusively during the course of somatization disorder and can not be explained by another mental disorder.
- Appropriate study does not identify a neurologic or general medical condition or the direct effects of substance use that can fully explain symptoms.
- A stressful event precedes the onset of symptoms, suggesting that psychologic factors are involved.
- The patient does not consciously or intentionally feign the symptoms to assume a voluntary sick role (factitious disorder) or for secondary gain (malingering).

- It is not a culturally sanctioned behavior or experience.
- The condition warrants medical evaluation, causes clinically significant distress, or impairs social, occupational, or personal functioning.

Malingering

Malingering is the intentional presentation of false or exaggerated symptoms for external gain (4). Symptoms are often discordant with the mechanism of injury, and subjective complaints generally do not correlate with physical findings. The patient may have a medicolegal history. There may be a marked lack of cooperation with the medical evaluation and subsequent poor compliance with the plan of treatment. The key differentiating factor that distinguishes malingering from somatoform disorders is the intentional fabrication of symptoms for external gain.

Factitious Disorder

The patient with a factitious disorder differs from the malingeringer in that the motor-sensory deficits are under voluntary control to play the role of the “sick person (4).” The motivation is consequently more complex than that of the malingeringer, because there are no external incentives.

Somatization Disorder

The manifestation of physical symptoms as the expression of an underlying emotional distress is characteristic of somatization disorder (4). In contrast to malingering and factitious disorders, the patient with somatization disorder believes in his/her symptoms and experiences real discomfort. Symptoms begin before the age of 30 years, persist for several years, and result in significant impairment; the symptoms are not intentionally produced. Pain must be related to at least 4 different sites or bodily functions, there must be at least 2 gastrointestinal symptoms, 1 sexual symptom, and 1 pseudoneurologic symptom.

Metabolic Conditions

Metabolic derangements can alter the normal neurologic processes. Thyroid toxicity and hypokalemia can cause periodic paralysis (16–18). Autoimmune disorders such as myasthenia gravis may cause weakness or frank paralysis. Other diseases such as Guillain-Barré may generate weakness or paralysis by their demyelinating effect on the peripheral and central nervous system.

The DSM-IV presents an efficient decision algorithm (4) for the differential diagnosis for the evaluation of potential somatoform disorders. Psychologic factors can exacerbate both a patient’s perception of symptoms of a general medical condition and reporting of those symptoms. Therefore, a psychologically exacerbated general medical condition should be considered and excluded. If the reported symptoms can not be explained by a general medical condition, the intentionality of the

symptoms and the presence of external incentives (narcotics seeking, seeking housing, avoidance of work, etc) should be appraised. If symptoms are intentionally produced for external incentives malingering is the probable diagnosis. The intentional production of symptoms in the absence of external incentives is indicative of a factitious disorder. A history of multiple physical complaints should raise the suspicion of somatization disorder. Isolated symptoms or deficits affecting voluntary motor and/or sensory function suggest a conversion disorder.

As outlined, multiple psychiatric conditions (conversion disorder, somatization disorder, factitious disorder, and malingering) can present with neurologic deficit. Clinical reports frequently refer to paralysis secondary to conversion disorder as hysterical paralysis. For this discussion, the term psychogenic paralysis will be used to refer to paralysis attributed to psychologic cause.

DIAGNOSTIC TESTS FOR THE EVALUATION OF ATYPICAL PARALYSIS

The diagnosis of a psychiatric disorder is by necessity a diagnosis of exclusion. When examining a patient with significant neurologic complaints (ie, paralysis), clinical findings that indicate the preservation of neural pathways (normal reflexes, rectal sensation and tone, bulbocavernosus reflex) should raise the index of suspicion for a psychologic etiology. However, adequate radiographs should be performed to exclude bony injury or spinal malalignment, and an MRI, myelogram, or CT should be obtained to evaluate for any abnormality of the entire neural axis.

Somatosensory-evoked potentials (SSEPs) and motor-evoked potentials (MEPs) evaluate the latency of sensory signals to the cortex and cortical signals to the muscles. The presence of normal SSEPs and MEPs shows a functioning cerebral–spinal axis and would suggest an inorganic component to the paralysis. While very sensitive and effective for identifying psychogenic paralysis, neurophysiologic testing is costly and may not be available at all institutions (12).

Single photon emission CT (SPECT) is a nuclear medicine tomographic imaging technique that defines changes in cerebral blood flow in response to sensorimotor stimuli. Vuilleumier et al (19) have reported that cortical activation is symmetrical in both premotor and sensory regions, explaining why conversion disorder with motor deficit patients present with normal neurophysiologic studies. Vuilleumier et al, however, described a striking decrease in subcortical activation in the regions of the contralateral thalamus and basal ganglia in all conversion disorder with motor-deficit patients. They hypothesize that the sensorimotor dysfunction seen in conversion disorder with motor deficit is a result of decreased activation of the basal ganglia and thalamus and a subsequent disruption of the striatothalamocortical loop. Further evidence of the subcortical nature of this

disease is shown by the decreased contralateral caudate activity. The level of decreased caudate activity has been found to be a predictor of recovery: greater caudate dysfunction is associated with decreased recovery from hysterical symptoms at follow-up visits (19).

Functional MRI (fMRI) identifies regions of the brain that have changes in blood flow during the execution of specific tasks. During cortical activation at the initiation of a specific task, there is a concomitant increase in local blood flow with a corresponding local reduction in deoxyhemoglobin. Deoxyhemoglobin has different magnetic properties compared with oxygenated hemoglobin (20). With certain fMRI imaging sequences, the alteration in the relative ratios of oxygenated and deoxygenated hemoglobin can be imaged, and it is hypothesized that these correlate with neural activity.

Marshall et al (21) described a fMRI study on a patient with left-sided paralysis and the absence of any structural or metabolic neural lesions. With voluntary attempted movement of the affected limb, there was activity in the right anterior cingulate and orbitofrontal cortex, structures that are responsible for inhibiting volitional movements. Marshall et al hypothesized that the activity in those regions suggested overinhibition of specific neural pathways as a causal relationship for conversion disorder with motor deficit (21). Marshall et al (21) also note that the advantages of fMRI over positron emission tomography (PET) include the absence of preimaging injections of radioactive isotopes, a single scan vs multiple acquisitions, and reduced imaging times. Additionally, fMRI is clearly more ubiquitous and has higher resolution capabilities than PET, although it is labor intensive and dependent on patient effort in cooperating with a given task.

PHARMACOLOGIC INTERVENTIONS

Hypnotic sedatives have been used by investigators in the identification of psychogenic paralysis. The resolution of psychogenic symptoms after the administration of sodium Amytal has been reported in multiple case studies (22,23). This short-acting barbiturate has sedative, hypnotic, and anticonvulsant properties. The intravenous administration of 8 mL of 2.5% solution administered over 15 to 20 minutes has been shown to provide an immediate, effective, and lasting cure of paralytic symptoms in the patient suffering from conversion disorder with motor deficit. Because conversion disorder is believed to represent the physical manifestation of a subconscious (unrealized) conflict, it is hypothesized that dissociative medication and serial interviews while the patient is sedated will facilitate the recognition and resolution of symptoms.

PHYSICAL EXAMINATION

There are a number of physical examination maneuvers that have been validated to aid in the identification of psychogenic paralysis.

Spinal Injuries Center Test

The Spinal Injuries Center (SIC) Test was described by Yugue et al (12) for the evaluation of conversion disorder with motor deficit involving the lower extremities. The patient is placed in the supine position and the physician passively lifts the patient's knees to a flexed position with feet flat on the bed. The physician releases the patient's knees; when the patient is able to maintain the flexed position, he is considered to be SIC test-positive. If the patient has severe paralysis, he will not be able to maintain his knees in the flexed position, and the leg on the paretic side will spontaneously drop into a flaccid extension. In this scenario, the SIC test result is considered negative.

Barre's Sign

Barre's sign may be used for the evaluation of hemiplegia. The patient is placed in the prone position with knees flexed at right angles. The examiner releases the knees and asks the patient to maintain the position; the limb on the affected side will fall into extension in the event of true paralysis. In a patient with psychogenic hemiparalysis, the affected limb will extend rapidly with no muscle contraction of the hamstrings (12).

Babinski Thigh-Trunk Test

When attempting to sit from a supine position, the natural tendency is for the lower extremities to tension in extension through contraction of the gluteal muscles to provide a stable base. In true hemiplegia, the affected lower extremity will involuntarily exhibit thigh flexion with attempted sitting, because the gluteal muscles are without strength and there is no extensor tone. With paraplegia, both extremities will exhibit thigh flexion. The patient with psychogenic paralysis will typically maintain both lower extremities in extension when attempting to sit, reflexively firing the gluteal muscles (24).

Hoover's Test

In the evaluation of hemiparesis, the examiner lifts the patient's legs slightly from the examination table while the patient is in the supine position. While the examiner supports both limbs from the heels, the patient is asked to lift the affected extremity; the absence of downward heel pressure in the contralateral (uninvolved) limb is indicative of apathy of intent. As with Barré's test, Hoover's test is of no use in the evaluation of paraplegia (12).

Recognition of underlying psychologic factors that may contribute to the presentation of an atypical neurologic deficit may obviate the need for further clinical studies and expedite the treatment of the patient's underlying psychologic issues or psychiatric disorder. While somatoform disorders have been described in numerous case reports of "hysterical paralysis" (7,8,10,11,13,25), their incidence is relatively rare (4).

The clinical team must consider how psychologic factors can affect both patient perception of and reporting of symptoms. We present a case of atypical paralysis evaluated at our regional spinal cord injury center and both discuss the relevant differential diagnosis and review the maneuvers and imaging modalities essential in making a correct diagnosis.

CASE ILLUSTRATION

A 37-year-old male pedestrian presented to a local hospital after being “pinned” between a motor vehicle and a wall. He was transported by ambulance to a local hospital, where on presentation he was found to have absent sensation and motor activity below his waist. No visceral or bony injuries were identified; however, he was transferred to a Spinal Cord Injury Center at a University Hospital for further evaluation of his neurologic deficit. Systemic steroids were initiated as per National Acute Spinal Cord Injury Studies (NASCIS) protocol.

On arrival to the emergency department, the patient reported inability to move his lower extremities with associated numbness and tingling. The patient complained of back pain and bilateral lower extremity numbness. A pain numerical rating score was not reported. He denied any change in bowel or bladder function. His medical history was significant for sleep apnea for which he used continuous positive airway pressure. The patient reported no medications and no known drug allergies. He smoked 1 pack of cigarettes per day for approximately 20 years. He stated occasional alcohol use and denied illicit drug use. The patient had no stated history of psychiatric disorders; further psychosocial information was not obtained. Pertinent findings on his physical examination included cranial nerves II–XII grossly intact and upper extremity strength of 5/5 bilaterally in all muscle groups. Both lower extremity iliopsoas/quadriceps/tibialis anterior/extensor hallucis longus/gastrocnemius muscle groups had 0/5 strength. Sensation to light touch and pinprick was intact above the umbilicus and T10; the patient denied any light touch or pinprick sensation to the T11–S1 dermatomes. No visceral or bony injuries were identified, and no abrasions or bruising were noted on examination. The patient denied perianal sensation but had normal rectal tone, volition, and bulbocavernosus reflex. The biceps/triceps muscle stretch reflexes were 2+/4 bilaterally, and his patellar/Achilles reflexes were 1+/4 bilaterally. Babinski sign was absent bilaterally. There was no clonus, and Hoffmann’s signs were negative bilaterally.

Because of the mechanism of injury and absence of lower extremity function, the patient was evaluated as per the institution’s spinal cord injury protocol. Plain radiographs of the entire spine, MRI of the cervical, thoracic, and lumbar spine, and a CT scan of the cervical, thoracic, and lumbar spine showed no bony or soft tissue abnormality.

Hospital Course

The patient was admitted to the trauma service for observation and continued evaluation, because imaging studies (x-ray, CT, and MRI) did not identify any abnormality. Both the Orthopaedic Spine and Neurosurgery services evaluated the patient initially in the emergency department and continued to follow the patient throughout his hospital course. The Rehabilitation Medicine service evaluated the patient as is standard practice for all patients admitted for evaluation of possible spinal cord injury on hospital day 1. Given the patient’s lower extremity weakness with no apparent etiology, it was the consensus of the Trauma, Orthopaedic Spine, Neurosurgery, and Rehabilitation Medicine services to consult both the Neurology and Psychiatry services. The Neurology service evaluated the patient on hospital day 2. Because of the inconsistencies between the patient’s clinical examination and imaging studies, it was the recommendation of the neurology service that SSEP testing and EMG analysis be performed, which showed no abnormalities. Social workers evaluated the patient on hospital day 2 to review the patient’s case; during the evaluation by social work, the patient indicated a desire to consult with an attorney. The psychiatry service evaluated the patient on hospital day 4 and felt that his apparent neurologic deficit may have been secondary to a conversion disorder. Laboratory results were within normal limits, and no metabolic abnormality was identified.

Given the absence of any objective findings to explain the patient’s neurologic deficit, it was felt that the patient’s symptoms were consistent with a psychogenic etiology. To study this problem further, an fMRI was obtained. Although the efficacy of fMRI in the evaluation of conversion disorders has not been confirmed, it was the opinion of the neuroscientists at our institution that the study may yield information of both diagnostic and academic importance. The nature of the fMRI procedure was explained to the patient, and he verbally consented to the study with an understanding of its investigational nature. The proposed goal of the fMRI was to determine whether cortical areas of activation correlated temporally with motor and sensory testing performed during the patient’s examination, potentially identifying a specific abnormality in somatosensory or motor processing.

The fMRI did not show activation in the contralateral motor cortex (appropriate site for his affected limb) when the patient was asked to move his foot (consistent with apathy of intent); we hypothesized that the presence of motor cortex activation without actual foot movement would have correlated with a true conversion disorder. Interestingly, activation was noted in the somatosensory regions of the cortex in response to foot stimulation. However, during the sensory task, the patient was notably agitated, possibly compromising the validity of the study (Figures 1 and 2). The patient’s agitation

Figure 1. fMRI images during actual vs imagined movements.

seemed to be secondary to claustrophobia and an associated anxiety about the confines of the MRI scanner. Agitation may have compromised the study because of both possible motion artifact and patient distraction during sensory stimulus. Currently, there are no established diagnostic criteria with respect to somatosensory fMRI findings in patients with conversion disorder (26,27). Whether the lack of motor activation in the presence of a motor task is indicative of malingering vs a conversion disorder was indeterminate. Lack of cooperation with diagnostic testing is an associated symptom of malingering.

Subsequent to the fMRI study, the patient showed progressive neurologic recovery, while also beginning to exhibit narcotic-seeking behavior (stating that he would be amenable to discharge from the hospital if provided with a prescription for narcotic pain medication). The

patient was noted to have unusual behavior with inconsistencies noted in serial physical examinations, as well as inconsistencies in his statements. On daily follow-up evaluation, it was the opinion of the psychiatry service that the patient was malingering. Malingering was diagnosed because the patient was believed to be intentionally fabricating his symptoms for the secondary gain of obtaining narcotics. He was discharged from the hospital on hospital day 10 with normal strength and sensation.

DISCUSSION

This case illustrates the diagnostic dilemma of evaluating the patient with paralysis of no apparent origin. While our patient ultimately seems to have been a malingerer, the clinical presentation and diagnostic evaluation is illustrative of the work-up of any patient with atypical paralysis.

Figure 2. fMRI images during right foot stimulation minus rest comparison (image columns 1–5) and fMRI Images during left foot stimulation minus rest comparison (image columns 6–10).

A conversion disorder was the working diagnosis of the psychiatry team evaluating our patient, and his case illustrates how difficult it can be to form an accurate psychiatric diagnosis in this difficult patient population. Ultimately, it was the patient's rapid and total recovery coupled with the realization that he was intentionally producing his symptoms and exhibiting narcotic-seeking behavior that led to a presumptive diagnosis of malingering.

Our patient's hospital course exemplifies the problematic nature of dealing with acute paralysis that is of a psychogenic nature. Particularly in the setting of a reported injury (in this case a pedestrian struck by a motor vehicle), any patient presenting with an acute loss of function must initially be assumed to have a potential organic disorder until proven otherwise. Diagnostic

imaging was used to exclude any bony malalignment or fracture as well as any spinal cord signal abnormality on MRI. When no apparent cause was found, a systematic multidisciplinary approach using the neurology, psychiatry, and physical medicine and rehabilitation services was applied. Neurophysiologic testing confirmed the absence of an organic disorder, and at this juncture, diagnostic efforts focused on identifying any psychiatric disorder to facilitate appropriate treatment for this individual.

In this case, considerable resources were used to evaluate symptoms that were intentionally fabricated by the patient. In retrospect, this patient presented diagnostic clues from the time of presentation (atypical acute paraplegia with no spine fracture or malalignment, inconsistent neurologic findings, and no physical findings

correlating with a reported traumatic injury of considerable force) that his paralysis may be psychogenic in nature. The physical examination maneuvers reviewed to identify psychogenic paralysis were not performed at initial evaluation after excluding spine fracture. We hypothesize that if a broad differential had been studied concurrently with the initial evaluation for traumatic injury and an effort had been directed to a more thorough evaluation of the patient's psychologic profile and identification of possible psychosocial issues, the diagnosis may have been found in a more expeditious fashion.

Psychologic factors should be considered in the evaluation of any patient with an atypical presentation of paralysis, along with psychosocial factors, because both can play a role in a patient's presentation of disease. A thorough clinical examination in combination with the appropriate diagnostic studies can exclude, with confidence, an organic disorder. While the incidence is relatively low, psychiatric disorders should be considered as part of a broad differential diagnosis. This case is an example of a clinical scenario in which a relatively rare psychiatric disorder (conversion disorder) was diagnosed before fully studying the patient's psychological factors—contributing to a prolonged and wide-ranging evaluation of symptoms that ultimately turned out to be a case of malingering. While it is appropriate to involve the medical and psychiatry services in the evaluation of the patient, an emphasis needs to be placed on effectively communicating and coordinating the care among multiple physician teams. A concerted effort should be made to coordinate care with the physical medicine and rehabilitation services and the physical and occupational therapists who work with them. Additionally, the social workers, occupational and physical therapists, and nurses spend significantly more time interacting with the patient than many members of the physician team. This extensive contact affords them the opportunity to form valuable opinions based on their observations of the patient's behavior. A true multidisciplinary approach necessitates the coordination and efficient exchange of information between all facets of the care team.

CONCLUSIONS

The pursuit of an accurate diagnosis in this challenging patient population will invariably lead the clinician on a tortuous path. Conversion disorder, somatoform disorder, and malingering remain diagnoses of exclusion, requiring a systematic and often exhaustive approach. These patients remain a distinct diagnostic challenge for the clinician assessing the atypical patient presenting with paralysis without apparent cause. The prompt identification of these patients, use of appropriate and validated physical examination maneuvers, and coordination of care and information exchange between all members of the care team may facilitate the expeditious care of these patients in a more time- and cost-effective manner.

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