Psychogenic dystonia and peripheral trauma

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ABSTRACT

Dystonia in association with peripheral trauma is a well-described clinical syndrome. The syndrome goes by many names—"traumatic" dystonia, "fixed" dystonia, peripherally induced dystonia, or complex region pain syndrome (CRPS) dystonia. We reviewed the role of peripheral trauma in the development of dystonia, focusing on 4 subtypes—cervical dystonia, focal limb dystonia, CRPS dystonia, and psychogenic dystonia. We show that peripheral trauma inducing, provoking, or precipitating structural changes within the CNS leading to dystonia is not an accepted concept, and current evidence supporting a pathophysiologic mechanism is virtually nonexistent. A better approach to this clinical syndrome is to define it as fixed abnormal posturing that is most commonly psychogenic. While symptomatic treatment of pain and spasms with medication can be beneficial, early psychological evaluation and patient-specific treatment is important. Modalities such as physical and occupational therapy should be utilized early. Finally, it should be emphasized that like many psychogenic movement disorders, it remains a highly disabling and distressing disorder. ***Neurology***® **2011;77:496–502**

GLOSSARY

CRPS = complex region pain syndrome; ***DSM-IV*** = *Diagnostic and Statistical Manual of Mental Disorders,* 4th edition; **GABA** = γ-aminobutyric acid.

The relationship between peripheral trauma (an injury to a peripheral nerve, root, or a soft tissue injury) and the subsequent development of dystonia is poorly understood.[1,2] It is frequently reported that it is "well accepted" that peripheral trauma can induce, provoke, or trigger dystonia.[3,4] Numerous case reports, case series, and reviews document patients who developed dystonia after minor peripheral trauma, including minor surgical procedures or with prolonged immobilization such as casting. The type of trauma associated with the development of dystonia is diverse (table 1). The lack of guidelines regarding a timeframe for the interval between trauma and the development of dystonia makes it difficult to standardize the diagnosis. Jankovic proposed that development of dystonia within 1 year of peripheral injury to the affected body part was acceptable. Chaos has followed, but Jankovic's criteria, despite being arbitrary and not validated, are often accepted.[5-7] However, the accumulated evidence does not support the concept that peripherally induced dystonia is a physiologic disorder, but rather a form of psychogenic movement disorder. We review the literature examining this syndrome and current concepts surrounding its etiology and pathophysiology. There is ample evidence supporting the hypothesis that an organic peripherally induced dystonia does not exist, and that posttraumatic abnormal posturing is more likely a psychogenic movement disorder.

CLINICAL CHARACTERISTICS

Trauma and cervical dystonia: A starting point. Cervical dystonia is the most commonly cited example of peripherally induced traumatic dystonia.[8] In 1991, Troung et al.[9] reported a series of 6 patients with cervical dystonia precipitated by neck trauma. They described a characteristic syndrome, atypical for nontraumatic cervical dystonia, of marked limitation of neck motion, severe neck spasms with pain, and absence of sensory tricks. In 1993, Goldman and Ahlskog[10] presented a similar series of 5

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Disclosure: Author disclosures are provided at the end of the article.

Table 1 The diversity of "dystonia" occurring after peripheral trauma[a]

A sampling of reported types of peripheral trauma "inducing" dystonia[1,45]	Anatomic locations of "dystonia" preceded by peripheral trauma[1]
Muscle strains	Cervical dystonia
Oromandibular dystonia[50]	Shoulder dystonia[49]
Contusions	Limbs
Burns	Eyelids
Lacerations	Trunk
Electric shocks	
Fixed immobilization[46]	
Minor surgical procedures	
Lipoma resections[47]	
Tonsillectomy[48]	
Laminectomy[49]	
Minor dental procedures	

[a] Most cited cases are single case reports or small series.

patients with cervical dystonia precipitated by trauma with similar findings, noting particularly the sustained muscle contractions and poor response to treatment.

In 1998, Tarsy[11] reported the largest series of patients with cervical dystonia associated with trauma. In individuals developing cervical dystonia within 4 weeks of acute neck trauma, there were differences compared to patients with nontraumatic cervical dystonia. Patients with nontraumatic cervical dystonia or dystonia which developed 3–12 months after trauma had gradual progression of motor symptoms, frequently had sensory tricks, and had minimal pain associated with the dystonia. This group also had better neck mobility despite abnormal postures. This was in sharp contrast to patients who developed cervical dystonia within 3 months of injury. These patients had significantly more pain and a fixed, immobile posture of the neck with reduced mobility. None of the patients with acute onset dystonia reported sensory tricks, and treatment response was poor.

The study by Tarsy[11] demonstrated that cervical dystonia associated with trauma has unique clinical features that differed from nontraumatic dystonia. O'Riordan and Hutchinson,[12] in 16 patients with abrupt onset of cervical dystonia occurring within 4 weeks of neck trauma, also emphasized the unique clinical features of the syndrome. Replicating Tarsy's results, they found that patients with cervical dystonia occurring within hours to days of trauma had a similar syndrome—fixed, painful muscle contractions of the neck that were poorly responsive to therapy and lacking sensory tricks. These findings let them conclude that Jankovic's criteria of trauma occurring within 1 year of development of dystonia was "excessive." Sa et al.,[13] in a study of 16 patients with traumatic cervical dystonia, reported a high incidence of features suggestive of a psychogenic movement disorder such as nondermatomal sensory loss, give-way weakness, and an inconsistent gait disturbance. They proposed that "the disorder be referred to as 'posttraumatic painful torticollis' rather than characterize it as a form of dystonia." Psychological factors were prominent and 11 patients had psychological conflict, stress, or both channeled into somatic symptoms on standardized personality inventories. The authors wrote that psychological factors "play a critical role in the pathogenesis and maintenance of the disorder." These initial studies in posttraumatic cervical dystonia provided insights into the relationship between trauma and dystonia, with significant clues indicating a psychogenic etiology. These insights are relevant when studying peripheral trauma–induced focal dystonia not involving the head and neck.

Focal limb dystonia associated with trauma. In 1993, Bhatia et al.[14] reported 18 patients who developed fixed painful limb dystonia triggered by trivial peripheral injuries. Twelve patients had leg involvement, and 6 arm involvement. Like patients with traumatic cervical dystonia, these patients had fixed postures of the limbs, prominent pain, and poor response to therapy. Many developed pain and abnormal postures that spread to other limbs. These unusual features, compared to other forms of primary focal limb dystonia, led the investigators to name the entity "causalgia-dystonia syndrome." In 2004, a pivotal study by Schrag et al.[15] presented the largest series of patients with fixed dystonia. Of 103 patients, 41 were followed prospectively. Over 90% of the patients had focal limb involvement and 63% reported peripheral trauma to be the trigger. In this population, hallmark clinical features included a fixed immobile limb posture with frequent association of pain in the body part that had the trauma. As in traumatic cervical dystonia, sensory tricks were infrequent. After injury to the given limb, common postures included sustained foot inversion and plantar flexion, wrist and finger flexion, or sustained trapezius elevation. Psychogenic features were common in the cohort. The authors report 37% of patients fulfilled criteria for psychogenic dystonia, 29% fulfilled criteria for somatoform disorder, and 24% had characteristics of both. When compared to control patients with classic forms of dystonia, the group with fixed dystonia had significantly higher rates of dissociative and affective disorders (all psychiatric diagnoses were based on *DSM-IV*). Overall treatment

Table 2 Clinical features of trauma-induced dystonia compared to idiopathic, CRPS dystonia, and psychogenic dystonia

Clinical feature	Idiopathic focal or genetic dystonia	Trauma-induced "dystonia"	CRPS dystonia	Psychogenic dystonia
Associated pain	Occasional	Common	Severe	Common
Fixed postures	Rare	Common	Common	Common
Muscle hypertrophy	Not uncommon, particularly cervical	Uncommon	Uncommon	Uncommon
Sensory tricks	Common	Rare	Rare	Rare
Onset of symptoms	Slow	Abrupt	Abrupt to subacute	Abrupt to subacute
Response to treatment	Good for focal dystonia[a,c]; moderate for genetic dystonia[b,c]	Poor[a]	Poor[a]	Poor[c]

Abbreviation: CRPS = complex region pain syndrome.
[a] Treatment with botulinum toxin.
[b] Treatment with anticholinergics, dopaminergic therapy, deep brain stimulation.
[c] Psychological treatment and rehabilitation.

response was poor; however, a few patients had improvement and even remission. The authors concluded "the best outcome was seen in patients who underwent multidisciplinary treatment which incorporated rehabilitation with physiotherapy and occupational therapy, as well as psychotherapy and psychiatric treatment." The findings from Schrag et al. were replicated by McKeon et al.[16] in 2008, who studied the clinical spectrum of 36 patients with focal lower extremity dystonia. Like patients reported by Schrag et al., 10 of 36 patients evaluated by McKeon et al. were diagnosed with posttraumatic dystonia and those patients consistently demonstrated fixed painful postures and typically inversion of the affected foot.

The overlap between fixed dystonia associated with trauma and psychogenic limb dystonia is clear. Lang[17] reported 18 patients with documented or clinically established psychogenic dystonia. Eleven of 18 of these patients reported peripheral trauma as the trigger, and 13 had limb dystonia. In this group onset of dystonia was abrupt with rapid progression, fixed dystonic postures, and pain. Similar findings in children with fixed painful dystonia reveal a high prevalence of psychological features meeting the criteria for psychogenic dystonia.[18] In their original description of the causalgia-dystonia syndrome, Bhatia et al.[14] concluded "at present it is impossible to decide whether this distressing syndrome is a true functional disorder of the CNS, or is of psychogenic origin." Eighteen years later, the preponderance of clinical studies tip the scales toward a psychogenic origin.

CRPS dystonia. CRPS is a poorly understood pain disorder that is usually preceded by peripheral injury, prolonged immobilization, or surgery.[19] In patients with CRPS, dystonia is the most common movement disorder. Van Rijn et al.[20] studied 185 patients who developed CRPS after soft tissue trauma or prolonged immobilization and found that 65% (121 patients) had a movement disorder, with dystonia accounting for over 90% of those with any movement disorder. In 75% of the patients with CRPS dystonia, the affected limb was in a tonic or fixed posture. The temporal relationship between CRPS and dystonia is variable, and can range from days to 5 years. As Schott[21] observed, it is important to recognize that CRPS and fixed dystonia frequently co-occur, and often need to be managed together. As in posttraumatic limb dystonia and posttraumatic cervical dystonia, individuals with CRPS dystonia have many psychogenic features. Verdugo and Ochoa[22] studied 58 patients with CRPS and movement disorders (60% dystonia). All patients had "pseudoneurologic" signs, and none of 58 patients had structural nerve, spinal cord, or intracranial abnormalities on neuroimaging or electrophysiologic testing. They also reported that 16 of 37 patients (43%) responded to placebo nerve blocks.

There are no validated measures for clinical diagnostic criteria for dystonia associated with peripheral trauma. The clinical features of dystonia associated with trauma are markedly different from other forms of dystonia, and include fixed painful postures that markedly reduced mobility of joint movement, absence of sensory tricks, and co-occurrence of pain and CRPS (table 2). Poor response to treatment is frequently reported. The overlap between peripheral trauma–related dystonia and psychogenic dystonia is striking. Kumar and Jog[24] also recognized that the clinical syndrome of peripheral induced "dystonia" differed from "classic" dystonia in many ways, leading them to propose naming it "posttraumatic syndrome." Some reviews contend that "a few patients" with psychosomatic disease or malingering have clouded the picture toward an understanding of

posttraumatic dystonia, or that malingering and psychiatric factors are present in all movement disorders.[23,24] Based on the large case series reported, this is not the case. A substantial majority of patients with dystonia associated with peripheral trauma have a readily identified primary psychogenic disorder.

PATHOPHYSIOLOGY **Proposed physiologic mechanisms.** If there is a mechanism for peripheral trauma causing dystonia it is unknown. Frequently cited possibilities to account for this syndrome are more hypotheses-driven than supported by data. The pathophysiology of dystonia seems to involve abnormal motor processing within the CNS.[25] The question regarding peripheral trauma–induced dystonia is how a soft tissue or peripheral nerve injury can result in changes in basal ganglia or motor cortex and disrupt motor control and induce dystonia. The hypotheses generated to answer this question typically start with extrapolating data from other disorders, specifically focal task-specific dystonia and CRPS.[5,26]

In primate models, performing repetitive tasks can lead to abnormal limb posture. Monkeys trained to perform repetitive tasks may develop a movement disorder resembling focal hand dystonia, loss of dexterity, and other disturbances of motor control. In these animals, disorganization of somatosensory cortex occurs.[27] In humans with a focal task-specific dystonia (writer's cramp), there is abnormal sensory cortex organization. These data support the hypothesis that performing repetitive tasks may result in aberrant organization of the somatosensory cortex in "susceptible" individuals.[28] These abnormal sensory representations are thought to influence motor control, causing overexcitability and functional changes within motor cortex and striatum. Despite evidence for plasticity within motor cortex in these disorders, the changes within motor cortex are poorly understood.[29] Moreover, studies in patients with cervical dystonia successfully treated with botulinum toxin demonstrated reversal of abnormal corticomotor representations over 3–5 years.[30] The current model to explain focal task-specific dystonia is that it is a disorder primarily of representation of sensory fields within sensorimotor cortex.[31] This model of focal task-specific dystonia based on sensory cortex plasticity is often extrapolated in the literature to explain peripheral trauma–induced dystonia. Instead of repetitive tasks causing changes in somatosensory cortex, pain, inflammation, or immobilization are hypothesized to be the triggers.[5]

Pain, inflammation, and immobilization are also hypothesized to trigger CRPS, and investigation into these possible etiologies is often used to explain peripherally induced dystonia.[24] There is evidence that neuropathic pain in CRPS is a multifactorial process that involves inflammatory changes, altered gene expression, and sometimes neural cell death. These changes at the level of the nerve and spinal cord are thought to result in maladaptive processing of nociceptive input in multiple levels of the CNS, including spinal cord, brainstem, and sensory cortex.[32] Furthermore, studies with fMRI (fMRI) comparing patients with CRPS to normal controls demonstrated increased activation within motor cortex of patients with CRPS. This increased activation was interpreted as motor cortex reorganization.[33] Many investigators have extrapolated the maladaptive nociceptive hypothesis for CRPS to explain the development of dystonia. They have embraced the possibility that abnormal alterations for processing of pain lead to aberrant motor processing within the CNS.[24] However, evidence supporting this hypothesis is limited. Substance P, a neuropeptide released during neuroinflammation, can amplify nociceptive withdrawal reflexes. It has been proposed that this may play a role in the flexion postures seen in patients with CRPS and dystonia. Nociceptive withdrawal reflex amplification, only seen in animal models, has led to the hypothesis that neuroinflammation may cause central disinhibition which results in amplification of pain and abnormal motor signals leading to dystonia.[23] The neurotransmitter γ-aminobutyric acid (GABA) has been a therapeutic target for treating dystonia. Small studies showing patients with CRPS dystonia respond to intrathecal baclofen, a GABA receptor agonist, have been used to support the hypothesis that neuroinflammation leads to amplification of motor signaling.[34,35]

Neurophysiologic studies in patients with CRPS dystonia have raised more questions than answers. There are 2 measurable neurophysiologic features of primary dystonia. The first is reduced excitability of inhibitory circuits in spinal cord, brainstem, and cortex. Impaired inhibition of motor signals is thought to lead to abnormal motor control and unwanted contraction of muscles in dystonia. The second feature is increased responsiveness to transcranial magnetic stimulation (TMS) which indicates increased plasticity in motor cortex.[36] In one study, 10 patients with CRPS and dystonia had findings that suggested impaired inhibition of sensory and motor processing at spinal cord and brainstem levels.[37] However, a recent report studying somatosensory evoked potentials with paired stimulation in 33 patients with CRPS and dystonia and 19 controls showed normal somatosensory processing at the cortical level. The authors concluded that their findings supported normal cortical proprioceptive processing, and favored a

spinal cord origin for dystonia.[38] Espay et al.[39] examined cortical and spinal inhibitory circuits and cortical activity with voluntary movement using TMS in patients with clinically definite psychogenic dystonia, patients with primary dystonia, and normal controls. Compared to the normal control group, patients with psychogenic and primary dystonia shared physiologic abnormalities, suggesting that abnormal cortical and spinal excitability may be a consequence of the sustained movement rather than the cause. The overall evidence in support of a proposed mechanism to explain peripheral trauma inducing dystonia is weak, controversial, and driven by expert opinion, speculation, and hypothesis.[40] For example, if recent studies showing normal cortical sensory processing in patients with CRPS and dystonia are accurate,[38] it would refute the proposed mechanism whereby peripheral trauma is thought to induce cortical plasticity and aberrant processing resulting in focal dystonia. If aberrant nociceptive inputs precipitate aberrant motor processing at the cortical, subcortical, or spinal cord levels, what is the explanation for patients without pain with soft tissue injuries and dystonia? Another problem is that current electrophysiologic measures of dystonia may be correlated with the consequence of the dystonic movement and not the underlying cause of the movement. Understanding the possible mechanisms (if any exist) continues to be challenging. The dearth of evidence for a pathophysiologic mechanism for peripheral trauma–induced dystonia may result from it not being an organic disorder.

TREATMENT AND PROGNOSIS The clinical syndrome remains difficult to treat and has a poor prognosis. Complications such as fixed contractures occur. A wide array of treatments including botulinum toxin, antispasticity medications, and occupational/physical therapy have been reported, and may occasionally be useful.[1] Psychological factors should be evaluated and addressed early.[15] Given the preponderance of evidence implicating a psychogenic etiology of peripheral trauma–induced dystonia, we recommend appropriate psychiatric and supportive treatment instead of an exhaustive and invasive neurologic workup, especially when fixed painful "dystonia" occurs within 1 month of peripheral trauma. It is clear that delay of treatment in psychogenic movement disorders contributes to poor long-term outcomes.[41] Despite treatment, most patients remain significantly disabled with a poor prognosis. In a study of 34 patients with fixed dystonia, Ibrahim et al.[42] reported that fewer than 25% improved, and only 6% had marked improvement to remission. One-third of the patients continued to deteriorate, and the average health status was poor. This is similar to disability seen in psychogenic movement disorders, which surprisingly has been reported to be similar to disability rates in progressive neurodegenerative diseases like PD.[43] Patients with dystonia associated with trauma are frequently involved with litigation surrounding their injury and disability. Scarano and Jankovic[44] evaluated 40 patients with posttraumatic movement disorders and reported that 19/40 had obtained legal representation. Patients with legal representation tended to be younger and were more likely to develop CRPS. An important role for the treating physician is to make the correct diagnosis and to prevent the patient from undergoing potentially harmful and costly unwarranted treatments (MRI, EMG, EEG, angiography, manipulations, and various infusions). There is often reluctance for neurologists to follow these patients but a reassuring physician who examines the patient and maintains contact can often prevent therapeutic misadventures.

DISCUSSION The syndrome of fixed painful dystonia after mild peripheral trauma is a well-described clinical syndrome, and frequently co-occurs with CRPS. Evidence supporting a pathophysiologic mechanism for peripheral trauma inducing or provoking dystonia is virtually nonexistent. However, the overlap between fixed painful dystonia and psychogenic movement disorders is marked. The literature clearly supports the view that fixed dystonia is a psychogenic movement disorder. This syndrome can be disabling and should be evaluated and treated with appropriate interventions. The role of the neurologist is crucial in appropriately evaluating and managing this disorder.

AUTHOR CONTRIBUTIONS

Dr. Hawley drafted the primary manuscript, performed applicable research and revisions, and assisted in the conceptualization of the review. Dr. Weiner had the primary conceptualization of the review, and assisted in analysis and interpretation of the data, as well as drafting and revising the manuscript.

DISCLOSURE

Dr. Hawley is an active duty medical officer serving in the United States Army. Dr. Weiner has served on scientific advisory boards for Santhera Pharmaceuticals and Rexahn Pharmaceuticals, Inc.; serves on the editorial boards of *Parkinsonism and Related Disorders* and *Neurological Reviews*, and as Editor of *Treatment Options in Neurology*; receives royalties from the publication of *Neurology for the Non-Neurologist* (6th edition, Wolters Kluwer/Lippincott, 2010), *Parkinson's Disease: A Complete Guide for Patients and Family* (2nd edition, Hopkins University Press, 2007), and *Handbook of Clinical Neurology Hyperkinetic Disorders* (Elsevier, 2011); has received research support from Novartis, Santhera Pharmaceuticals, and Boehringer Ingelheim; and has provided expert testimony and served as a subject matter expert in legal proceedings.

Received February 28, 2010. Accepted in final form April 19, 2011.

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