online © ML Comm
**Journal of Movement Disorders** 2010;3:39-41
pISSN 2005-940X / eISSN 2093-4939

**ORIGINAL ARTICLE**

# Four Cases with Peripheral Trauma Induced Involuntary Movements

**Eun Joo Chung**[a]
**Sang Jin Kim**[a]
**Won Yong Lee**[c]
**Jong Seok Bae**[a]
**Eung Gyu Kim**[a]
**Sung Hwa Pang**[b]

[a]Departments of Neurology and
[b]Neurosurgery, Inje University College of Medicine, Busan, Korea
[c]Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

**Background and Purpose:** Although peripheral trauma induced movement disorders have been rarely reported, diagnostic criteria for peripherally induced movement disorders (PIMD) have been established. Because preexisting subclinical movement disorders, or secondary gain for compensation and legal purposes are difficult to confirm, differential diagnosis for physicians still remains difficult. **Case Reports:** We present four patients developed movement disorders after relatively various intervals after traffic accident. Three patients of them showed tremor and one patient presented propriospinal myoclonus. In this report, we investigate whether peripheral trauma can lead to movement disorders and describe the relationship between peripheral injury and movement disorders in four cases. **Conclusions:** Injury was serious enough to develop involuntary abnormal movements with pain and the latency between injury and the onset of movements in all of cases was less than 1 year. Thus, our cases showed temporal and anatomical correlation between injury and the onset of movement disorder, strongly supporting the cause-and-effect relationship by previous diagnostic criteria for peripherally induced movement disorders.

**Journal of Movement Disorders 2010;3:39-41**

**Key Words:** Peripheral injury, Tremor, Propriospinal myoclonus.

Despite a number of reports have described peripherally induced movement disorders (PIMD),[1-4] the cause and effect relationship between peripheral injury and subsequent movement disorder remains controversial.

We encountered four patients who developed movement disorders at various intervals after peripheral injury. Three of these patients showed tremor and the remaining patient presented with propriospinal myoclonus. We analyzed our cases with regard to points in the established diagnostic criteria.

## Case

### Case I

A 24-year-old man driver was involved in a collision with another car, and he was thrown forward and backward. He immediately developed pain of neck and left shoulder. He felt a tingling sensation and paresthesia in the left arm. He later noted of tremor of left hand, which was exacerbated by action and was also present at rest and while sleeping. The patient had no family history of tremor.

Upon neurological examination 2 months after the accident, left arm and shoulder movements were limited by pain and left hand grip revealed slight weakness. Deep tendon reflexes (DTR) were normal. There were no any parkinsonian motor symptoms. The tremor was less than 6 Hz with predominantly of the action type combined with resting type. During the performance of right hand grip, the hand tremor was neither ameliorated nor reduced. Nerve conduction studies and electromyography (EMG) revealed the left C5-8 radiculopathy. However, cervical spine and shoulder magnetic resonance imaging (MRI) were normal. Despite treatment with propranolol, his symptoms were not improved.

**Received** April 28, 2010
**Accepted** September 17, 2010

**Corresponding author**
Eun Joo Chung, MD, PhD
Department of Neurology,
Inje University College of Medicine,
633-165 Gaegeum-dong, Jin-gu,
Busan 614-735, Korea
**Tel** +82-51-890-6766
**Fax** +82-51-895-6367
**E-mail** strokerchung@yahoo.co.kr

· The authors have no financial conflicts of interest.

 

### Case II

A 15-year-old student presented with a history of right radius fracture due to a fall 3 years earlier. She immediately developed pain and weakness in the right wrist, and then a cast was placed for 1 month. Upon removal of the cast, the patient noticed action tremor in the right hand that resolved at rest. Brain MRI was normal. There was no past medical or family history of tremor.

The patient complained neurological examination showed an action-type tremor of approximately 9 Hz in the right hand. The tremor became slightly worse upon distraction. Except mild weakness of grasp and wrist extension on the right hand, there was no other motor and sensory abnormality on both upper limbs. Both biceps and triceps jerk were normal. Propranolol treatment did not improve the tremor.

### Case III

A 62-year-old woman presented to our hospital after experiencing a traffic accident. She developed severe pain due to right elbow injury and fracture of the right mandibular condylar process. Approximately 6 months after the accident, she noted an insidious mild tremor in the right hand and jaw at both the active and resting states. The right hand tremor persisted during sleep.

Two years later, follow-up neurological examination revealed a predominantly action-type tremor of 6-7 Hz in the right hand, which was modulated by limb position, and a 4-5-Hz tremor in the jaw. Distractive maneuvers produced no change. Slightly parkinsonian motor symptoms including bradykinesia and rigidity on right hand were observed without rest tremor. Her past medical and family history did not include tremor. The cervical and brain computed tomography (CT) were normal except screws placed surgically in the right mandible after the accident. The jaw tremor was still presented and right hand tremor became less pronounced at the fourth year after the accident. Propranolol and clonazepam showed no therapeutic benefit.

### Case IV

A 45-year-old office cleaner was involved in a road traffic accident. Although she felt pain in the trunk, she did not show immediate neurological abnormalities. Ten days after the accident, she felt difficulty with gait or work due to jerking movements of the trunk. She had no family history of movement disorders.

Neurological examination showed jerking movements with flexion of the trunk, legs, and arms. Sensorimotor examination, and cervical and thoracic spine MRI showed no visible abnormalities. In the surface multi-channel EMG, the activity was firstly recorded in the rectus abdominis muscle driven by T10 and followed by a slow spread upward and downward. Despite treatment with valproate and clonazepam, her symptoms continued for over 6 months.

## Discussion

The proposed diagnostic criteria of PIMD was as follow : 1) the trauma is severe enough to cause local symptoms for at least 2 weeks or requires medical evaluation within 2 weeks after trauma ; 2) the initial manifestation of the movement disorder is anatomically related to the site of injury ; and 3) the onset of the movement disorder is within days or months (up to 1 year) after the injury.[3-5] Peripheral injury in our patients was sufficiently serious to result in the development of involuntary abnormal movements with pain within 2 weeks after trauma. The latency between injury and abnormal movements was less than 1 year, and the symptoms had an anatomical association with the site of the injury (Table 1).

Despite the pathogenesis of movement disorders by central mechanism, the concept of PIMD is also becoming widely accepted.[4] However, the mechanism by which PIMD remains obscure and controversial because of the artificial and simplistic composition of the published diagnostic criteria.[6] The concept that peripheral trauma can induce abnormal movements is based on clinical reports that peripheral trauma of varying intensity and type can induce a chronic movement disorder. However, this concept did not address the issue whether "unrelated" areas of the central nervous system (CNS) after previous severe closed head trauma can effect on the development of abnormal movements.[6] In principle, demonstrable lesions on brain imaging should not be present, or lesions on imaging should be in "unrelated" areas of the CNS.[6] However, in these instances, it difficult to see whether its lesions may be pre-disposing factors of abnormal movements. Another problem was that PIMD diagnosis was required to determine wheth-

**Table 1.** Summary of clinical features of patients with peripherally induced movement disorders

| Patient | Onset age/Sex | Type of injury | Latency between injury and movement disorder | Type of movement disorder |
|---|---|---|---|---|
| I | 24/M | Whiplash injury of neck | Immediate | Tremor of left hand, movement and rest |
| II | 12/F | Right radius fracture | 1 month | Tremor of right hand |
| III | 62/F | Right elbow injury, right mandibular fracture | 6 months | Tremor of right hand, jaw tremor |
| IV | 45/F | Truncal injury | 10 days | Prpopriospinal myoclonus |

er the symptoms had features suggesting a psychogenic etiology.[7] In this regard, there were no legal proceedings in any of our cases and patients showed show, at least, one of motor or sensory, or DTRs deficit, or NCV or EMG abnormal results, or structural abnormal findings on MRI. However, to make a strict diagnosis in outpatients suspected with PIMD, it is necessary to consider the individual symptoms in each case and the patient's medical history as well as diagnostic criteria. A lack of preexisting dysfunction of the CNS must be supported by a lack of other obvious possible causes of the movement disorder.[4] It required further checkup whether the trauma had unmasked an underlying subclinical movement disorder. In this point, our patients had been healthy without relevant family history until involuntary movements were developed. These findings enhanced probable diagnosis of PIMD in our cases.

PIMD are often associated with pain and other sensory phenomena, including the complex regional pain syndrome (CRPS).[3] The frequency of movement disorders in patients with CRPS has not been systematically studied. In a recent study, 58 patients, all with CRPS type 1 (and none with CRPS type 2) had abnormal movements characterized as "muscles spasms leading to dystonic posture" (60.4%), "coarse postural or action tremor" (15.5%), "irregular jerks" (8.6%), "dystonic spasms and irregular jerks" (8.6%), "dystonic spasms and postural tremor" (5.2%), and "episodic generalized choreiform movement" (1.7%).[8] A history of "significant" neck trauma, such as a whiplash associated with a motor vehicle accident, is reported by about 10-20% patients presenting to a neurologist with cervical dystonia.[9] In addition, peripherally induced tremor can be of resting and/or action type.[1,2,4] The pathophysiology of resting tremor is proposed to involve reorganization of the nigrostriatal system by peripheral trauma.[4] Neuronal networks in the thalamic ventralis lateralis nucleus, which receive input from the basal ganglia, may be also involved in the generation of resting- or action-type tremor.[4] Cases I and III showed predominantly action-type tremor with mild resting-type tremor, and case II presented with only action-type tremor.

Propriospinal myoclonus involves hyperexcitability of a specific myelomere, which is activated spontaneously or by various stimuli. This excitability spreads to the other myelomeres, probably through the slow propriospinal polysynaptic pathways.[10] In case IV, despite the lack of a definite neuroradiological spinal parenchymal lesion, surface EMG suggested a perimeter centering around the T10 vertebra as the origin of propriospinal myoclonus. These discrepancies may reflect dynamic compression by vertebral protrusion during only specific movement or functional spinal impairment by mechanical compression without neuroradiological evidence.[11]

It remains unclear why the responsible cortical-subcortical loop involves the central pathway after peripheral trauma. We concluded that PIMD was truly caused by peripheral-CNS interaction in spite of initiation by a peripheral generator.

## REFERENCES


1. Ellis SJ. Tremor and other movement disorders after whiplash type injuries. J Neurol Neurosurg Psychiatry 1997;63:110-112.
2. Hashimoto T, Sato H, Shindo M, Hayashi R, Ikeda S. Peripheral mechanisms in tremor after traumatic neck injury. J Neurol Neurosurg Psychiatry 2002;73:585-587.
3. Jankovic J, Van der Linden. Dystonia and tremor induced by peripheral trauma: predisposing factors. J Neurol Neurosurg Psychiatry 1988; 51:1512-1519.
4. Cardoso F, Jankovic J. Peripherally induced tremor and parkinsonism. Arch Neurol 1995;52:263-270.
5. Jankovic J. Can peripheral trauma induce dystonia and other movement disorders? Yes! Mov Disord 2001;16:7-12.
6. Weiner WJ. Can peripheral trauma induce dystonia? No! Mov Disord 2001;16:13-22.
7. Costa J, Henriques R, Barroso C, Ferreira J, Atalaia A, de Carvalho M. Upper limb tremor induced by peripheral nerve injury. Neurology 2006; 67:1884-1886.
8. Verdugo RJ, Ochoa JL. Abnormal movements in complex regional pain syndrome: assessment of their nature. Muscle Nerve 2000;23:198-205.
9. Tarsy D. Comparison of acute- and delayed-onset posttraumatic cervical dystonia. Mov Disord 1998;13:481-485.
10. Post B, Koelman JH, Tijssen MA. Propriospinal myoclonus after treatment with ciprofloxacin. Mov Disord 2004;19:595-597.
11. Manconi M, Sferrazza B, Iannaccone S, Massimo A, Zucconi M, Ferini-Strambi L. Case of symptomatic propriospinal myoclonus evolving toward acute "myoclonic status." Mov Disord 2005;20:1646-1650.