MOVEMENT DISORDER IN PERIPHERAL NERVE INJURIES

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Abstract. The conventional wisdom and classical description of movement disorders point to the central nervous system (CNS) as the originator of movement disorder, it is becoming obviously that peripheral nerve injuries can also initiate and contribute to movement disorders. Even though such conditions are exceptional and rare as compared to the CNS movement disorder, there is evidence that the peripheral nervous system (PNS) can also contribute to movement disorder.

Descriptors. central nervous system (CNS), complex regional pain syndrome (CRPS I and CRPS II), movement disorder, myoclonic jerks, Parkinson Disease (PD), reflex sympathetic dystrophy (RSD), tremor.

INTRODUCTION

The first report of peripheral origin of movement disorder was in 1888 when Gowers suggested that movement disorders can be produced not only by central but also by peripheral nervous system injuries(1). In the following decades, the concept was challenged. However in the 1980's and 1990's the concepts have become more accepted and reported(2-9). In the case of neuropathic pain of complex regional pain syndrome (CRPS I) also known as reflex sympathetic dystrophy (RSD) the efferent dysfunction of the sympathetic system is likely to be accompanied by motor paresis (10). The stimulation of the sympathetic nerve fibers, on the other hand, may cause an anti fatigue phenomenon which is referred to as Orbeli Phenomenon(11). It becomes obvious that a dysfunction of the sympathetic system can cause the two extremes of weakness and tremor on one end, and strengthening of the muscle(Orbeli Phenomenon) on the other(11).

MOVEMENT DISORDER

Movement disorders are common in most RSD patients(12). As, Schwartzman emphasizes the “movement disorder is frequently ascribed to hysteria and pain” (13).

The condition is seen in complex regional pain syndrome (CRPS I) (RSD) as well (CRPS II) (causalgia) (14).

The movement disorder due to peripheral trauma may be in the form of Parkinsonian Tremor(15).

In the extensive studies carried out by Jankovic, movement disorders in RSD have been accompanied by tremor and dystonia (6, 15).

In, Schwartzman and Kerrigan study of 200 patients with RSD, subtle dystonia and movement disorder were seen in 10 patients(16).

Animal experiments by Basbaum and colleagues have demonstrated the importance of damage and degeneration especially in the dorsal horn of the spinal cord due to a noxious stimulus(17,18). This secondary abnormality in the central nervous system(spinal cord) may play a role in movement disorder seen in neuropathic pain syndromes.

Pathological, peripheral nervous system input can also cause reorganization of other CNS structures such as the spinal cord, basal ganglia, nucleus gracilis, and thalamus(19,20,21). Obviously such secondary disorganization cerebral nuclei are compatible with secondary development of movement disorder accompanying peripheral nervous system damage.

PARKINSON DISEASE

James Parkinson, in his original thesis of "shaking palsy" has referred to injuries in the medulla as the cause of Parkinson Disease (PD)(the disease named after him)(22). Peripheral nerve injuries have the potential of causing PD(2,23,24).

Cardoso and Jankovic reported on 28 patients suffering from PD purely due to peripheral nervous system injury(15). The Parkinsonian tremor developed as early as two months and as late as one year after trauma. Even though the Parkinsonian tremor was initiated by peripheral nerve injury, PET scan studies showed a dysfunction in the striatum and in caudate nuclei in some of these patients. L-DOPA treatment did not help control the Parkinsonian-type tremor.

Cardoso and Jankovic also reported the frequent occurrence of Parkinsonism tremors in nine of eleven RSD patients who were treated with application of plaster cast(immobilization) to the involved extremity (4).

MECHANISM OF DEVELOPMENT OF TREMORS IN RSD

In 1984 Marsden and colleagues first reported that tremors can occur in RSD(25). Schott and Scherokman have also reported tremors in RSD(8,9). These reports appeared in the literature from 1984 through 1986. Later, Jankovic and van der Linden wrote their classic report on this subject in 1988(4).

Jankovic and van der Linden reported the development of tremors can start from one day to nine months after injury to the peripheral nerves (4).

In a study of twenty-one patients suffering from RSD, Deuschl and colleagues reported a fasciliation and enhancement of physiologic tremors(26). Cardoso and Jankovic have reported tremors in other peripheral injuries with neuropathic pain as well(27).

Obviously the peripheral nerve damage in and of itself is incapable of starting and perpetuating tremor, dystonia, or hypertonicity. On the other hand, the tremor is usually accompanied by certain degree of flexor spasm and tendency for dystonia. Goldman and Ahlskog, as well as Dewey and colleagues - both groups of authors from the Mayo Clinic have emphasized that post traumatic dystonia secondary to a cervical sprain is (28,29):

(i). Not psychogenic

(ii). The condition is on the basis of "centrally driven post traumatic muscle spasms" (28,29).

Such movement disorders (tremor or dystonia) originate from a peripheral nerve injury with secondary pathologic input to CNS (especially the spinal cord). The prolonged neuropathic afferent input, in the long run, causes disturbance of plasticity in the spinal cord(30,31,32). The disruption of normal plasticity and inhibitory effect of the spinal cord on the neuropathic afferent input eventually results in the following phenomena (30,31,32) (Table I).

Blümberg and Jänig have reported tremor and other movement disorders in more than 80% of CRPS patients(33). Veldman, et al, has noted movement disorder in 95% of 829 patients(34). In our series of 824 patients, the incidence was 78%(35).

Application of a cast causes immobilization and stimulation of the deep mechanoreceptors (36). These "silent sleeping nociceptors" become activated with rest and inactivity(36). This, in turn, leads to pain, edema and movement disorder.

Table I. CNS modulation of plasticity secondary to neuropathic pain.

1. Windup: The summation of excitatory post-synaptic potentials (EPSP) originating from prolonged dorsal horn pain input and activating ventral horn os spinal cord efferent response (37,38).
2. Long Term Potentiation (LTP): Prolonged excitation of hippocampus secondary to prolonged afferent neuropathic pain input changing the memory function of the plasticity (32,39-43).
3. Secondary opening of NMDA receptor channels, CA^{++} and NA^{+} input to the post-synaptic cells, and efferent excitation(44-46)

MYOCLONIC JERKS

Myoclonic jerks are common forms of movement disorder in CRPS(35).

(i). They may be a manifestation of deafferentation and sensitization of spinal cord due to long-term afferent cytokines damage to the inhibitory granular cells in layers I and II(47). As such, they develop in later stages of the disease. Any form of immobilization (cast, wheelchair, etc.)contributes to this phenomenon(35).

(ii). The myoclonic jerks are seen in patients undergoing withdrawal of opioids (rebound phenomenon)(35).

(iii). In 38 of our 824 patients suffering from CRPS due to spinal cord injury myoclonic jerks were invariably noted. In addition, myoclonic jerks were present in 44 of 63 CRPS patients secondary to electrical injury (48,49). This may be due to electricity going through the path of least resistance (afferent c-fibers) and secondarily originating spinal cord dysfunction (12,35,48).

(iv). Myoclonic jerks are a long-term complication of limb amputation (10 of 11 amputees among our 824 patients)(35).

Myoclonic jerks are frequently mistaken for "pseudoseizures" due to the fact that the ictal events originating from spinal cord are too deep to present themselves on scalp video-EEG monitoring (50). In more severe cases, such as electrical injury complicated by CRPS, somatosensory evoked potential (SSEP) test identifies the spinal cord dysfunction as the originator of this form of myelogenic seizure (48,49,50).

TREATMENT

As underlined by Schwartzman, RSD tremors are usually a late development (stage III) of the clinical picture of RSD (13).

It is well known that stage III RSD is somewhat resistant to treatment. This is due to the fact that the chronic disturbance of plasticity of the CNS is due to the long-standing presence of the disease(18). However, nerve blocks, alpha I and alpha II blockers (Clonidine, Hytrin, and Dibenzyline) may be quite helpful in the management of the tremor. In our study of 82 patients with late stage RSD treated with an infusion pump, seven patients suffered from tremors. Six patients were reverting back to normal, and in one patient the tremor was markedly improved.

As noted by Cardoso and Jankovic, immobilization of the involved extremity is a major activation of the development of tremors in RSD. Removal of cast, braces and the discontinuation of using crutches or a wheelchair helps in the prevention and management of movement disorders in RSD patients(12,52).

Koltzenburg, has noted that inactivity and low level of afferent input from the periphery aggravates the pain and central plasticity changes of neuropathic pain. Mobilization and prevention of inactivity are essential in the treatment of this condition(36).

Torebjork, has demonstrated that adverse effect of ice in generation and perpetuation of neuropathic pain. The use of ice should be avoided (53).

The use of brachial plexus and regional blocks are beneficial in correcting such movement disorders (35).

CONCLUSION

Practically every form of movement disorder can be observed in peripheral nerve injuries, especially in CRPS patients. These are frequently in the form of dystonic movements and are mistaken for conversion reaction. It is true that any group of movement disorders, including seizure disorder, is contaminated by a minority of conversion reaction patients or malingerers (17% in the case of seizure disorder). This minority incidence does not prove that all movement disorder patients are "functional"(35).

Treatment with Klonopin and Baclofen which exert direct inhibitory effect on anterior and ateriolateral cells of the spinal cord is quite beneficial in RSD patients suffering from tremors.

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