PAIN IN PATIENTS WITH SPINAL CORD INJURY

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ABSTRACT :

Pain is one of the most commonly encountered and one of the most disabling problems among spinal cord injured victims. The incidence of this intractable complaint has been reported to occur in 11 to 94% of patients with spinal cord injury (SCI) in different studies. Although the literature concerning SCI pain includes a broad number of studies, case reports and reviews, a concensus has not been reached regarding the classifications, descriptions or general features of this type of pain. Moreover, despite recent advances, the precise mechanism underlying SCI pain, the pathophysiology and the related sources are also still incompletely understood. In this paper, the authors have attempted to review the taxonomy, types, mechanisms, psycho-social outcomes and management of pain experienced by these patients.

Key Words: *Spinal cord injury, pain.*

INTRODUCTION

Most of the individuals with spinal cord injury (SCI) are likely to experience phantom-like sensations and may suffer chronic pain. Intractable chronic pain may become the most disabling sequela in some cases and may lead to chemical dependency, severe depression and even suicide (18). Chronic pain superimposed on SCI can virtually drain the individual of strength, motivation and will. For the SCI survior who already faces functional loss, severe pain can further restrict even the diversional activites that are available (1). It was reported that persons with SCI who suffer chronic pain complain that pain is the most disabling problem they face, interfering with daily routine and vocational activites (21). In a study of 98 persons an average 2.3 years post–injury, Lundqvist et al (13) discovered that severe pain was the only complication they tracked that related to lower quality of–life–scores. In a prospective longitudial study designed to isolate factors which might predispose a person to depression two years after sustaining SCI, Craig et al (5) reported that the experience of pain two years post–injury and feeling out of control of one's life prior to hospital discharge were predictive of depression two years post–injury.

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INCIDENCE

The incidence of hain has been reported to occur in 11 to 94% of patients with SCI in different studies (6, 8, 14, 17). In a study designed to question the most common complications and the social functions of 79 SCI patients after discharge from the Rehabilitation Department, the commonest complication were found to be pain (64.4%), urinary tract infections (62.2%), spasticity (60%), and pressure sores due to wheelchair activities (58% (10). In another study among 353 SCI patients, almost two–thirds of subjects reported significant pain with a predominance of neurogenic–type pain. It was concluded that neurogenic pain and neurological deterioration require more attention as these symptoms are usually ominous by virture of their impact on qualty of life (12).

CLASSIFICATION AND POSSIBLE MECHANISMS

Although a number of excellent classification schemes have been proposed, the overlap between them is extensive and there is not currently a commonly accepted classification scheme for SCI pain. The terms central pain, deafferentation pain, dysesthetic pain, central dysesthetic syndrome and spinal cord pain among others have all been used in the literature to describe more or less the same phenomenon: burn-

ing diffuse pain below the level of injury (16). Staas et al. (19) divide neuropathic pain following SCI into that experienced about the level of injury and that experienced more distally. The authors state that nerve root pain is sharp or lancinating or may have an electric shock quality. On the other hand, phantom body pain or spinal cord pain is felt distal to the lesion. This pain is described as sharp, burning, cold, tight and electric. Davidoff associates (6) suggest that most authors prefer the term diffuse dysesthetic pain which occurs distal to the level of SCI and this is the most common type of pain after SCI. The perception of pain according to Donovan (7), is a subjective and even an emotional experience which defines precise measurements and this fact in most cases precludes a clinician to find an adequate way of gathering evidence that proves the patient's description of discomfort is due to factors that exist at or below the level of lesion.

The term deafferentation pain has also been used by many authors which has a description different from nociceptive pain, and refers to pain occurring in any region of the body that has been deprived of afferent nervous impulses. This kind of pain is caused by lesions of nerves, dorsal roots, spinal cord, brainstem or cerebral cortex. The central nervous system mechanisms responsimle for deafferentation pain are only poorly understood and this state is also reflected by the unavailability of specific therapy (11).

According to Balazy (1) chronic pain after spinal cord injury is felt to be central in origin. This is neuropathic pain that is generated within the central nervous system and continues long after peripheral sources of acute painful pathology have healed. The author suggests that the anatomical changes after SCI cause an alteration in the normal neurophysiology of the spinal cord tissue. These alterations include abnormal burst firing, sprouting, altered neuropeptide concentration, ephaptic spread from sympathetic fibers to pain fibers and disturbed inhibitio of descending inhibitory pathways that terminate in various layers of the dorsal horn known as the substantia gelatinosa or dorsal root entry zone. On the other hand, Bullitt (2) uses the term "plasticity" to refer to the ability of neurons to change such as growing new processes, forming new synapses or altering the expression of neurotrasmitters; the term "regeneration" to indicate the regrowth of a transected axon toward its original target and the term "sprouting" to refer to the growth of uninjured or minimally damaged neurons that may make new connections with a deafferented neuronal pool. The author suggests that although there is a general consensus about the extent and pattern of sprouting in many regions of the deafferented brain, there is less agreement on the anatomical plasticity of the spinal cord (2).

Melzack and Looser (15) proposed that loss of sensory input after SCI results in spontaneous firing of dorsal horn cells and other cells of the somatosensory projection system above the lesion for prolonged periods of time. The authors also suggested that this prolonged bursting activity can be modulated by somatic, visceral and autonomic inputs, by input from neural mechanisms that underlie personality and emotional variables and by brain stem descending inhibitory influences. Loss of input leads to loss of descending inhibition, a condition which makes it easier for non-noxious stimuli to trigger abnormal bursting patterns.

Tasker (20) summarizes the general features of central pain of spinal cord origin as follows:

– Spinal cord lesions commonly cause central pain.

– Lesions of a wide variety of pathologies, whether complete or incomplete, regardless of the cord level, can cause central pain.

– The pain may be local or remote and consist of a steady, usually burning or dysesthetic, and/or intermittant and lancinating component, with or without predisposition to the perineal area.

– In complete lesions pain may be accompanied by allodynia and/or hyperpathia.

– If nerve roots are also affected, deafferentation pain syndromes with or without hyperpathia or allodynia may occur in the distribution of these nerve roots.

– A band of hyperpathia or allodynia and/or pain may occur at the level of the sensory loss.

– The pathophysiology of central pain caused by a spinal cord lesion is unknown.

– The development of central pain is an individual idiosyncrasy, so that not everyone with apparently identical lesions goes on to develop the pain.

Lack of trials constituted of large series of patients in the literature due to low incidence and prevalance of SCI seems to limit the attempts for a widely accepted SCI pain classification scheme. Thus, a valid and widely accepted classification for SCI pain is still lacking despite the broad number of studies in this

field. Wegener and associates (21) proposed that multiaxial approaches assessing medical, psychosocial and behavioral/functional dimensions are necessary to measure adequately the impact of chronic pain in SCI patients. The authors also stated that psychosocial factors such as coping ability, perceived interference, personality styles and environmental resources will influence the degree of pain-related impairment. Cohen et al (4) suggested that in cases where chronic pain is secondary to a chronic medical catastrophe such as SCI, aspects of Mc Gill Pain Questionnaire and the Minnesota Multiphasic Personality Inventory assess the person's total medical disability and not just the meaning and impact of pain.

In addition to an available taxonomy, number of studies concerning the sources and the precise mechanism(s) underlying SCI pain also need to be increased. This is important because, approaches in the treatment of this kind of pain may only progress in the presence of new clues regarding the underlying mechanism(s).

MANAGEMENT

It is generally agreed that 10 to 33% of patients with SCI require medical or surgical intervention because of the severity of their pain (6).

Considering that the SCI pain is a central neuropathic pain, the treatment should be directed centrally. The goal of treatment in SCI pain is to resolve the abnormal central neurophysiological activity with centrally acting medications and centrally acting neurosurgical procedure. Peripherally acting treatment approaches never bring permanent relief of this kind of pain but they may also be considered as an adjunctive therapy in order to reduce peripheral noxious stimuli that can exacerbate central pain (1).

SCI pain is extremely difficult to treat and often is quite refractory to non-surgical procedures. But it is usually best to start the treatment conservatively and use more invasive surgical procedures later. One should always keep in mind that the most effective treatment program requires a multi-disciplinary team approach. The team usually consists of physicians with experience in pain management, rehabilitation nursing staff, psychologist and physical/occupational/recreational therapists (1, 3).

According to Donovan et al (7) treatment of spinal cord pain remains difficult. Pharmacotherapy is often inneffective except tricyclic antidepressants in some cases. The authors also suggested that permanent nerve blocks are contra-indicated when the patient's lesion is quite incomplete and epidural stimulation at high frequencies, for example 100 herz or more, has proved effective in some cases.

Fenollosa and associates (8) reported a "step program" which they used in the management of SCI pain. The program consisted of 4 steps and the waiting period between the stages was 35 days average if the former step failed. The 4 steps were:

1) amitryptiline + clonezepan + non-steroidal anti-inflammatory drug (NSAID)

2) amitryptiline + clonezepan + 5-OH-tryptophane + transcutaneous electrical nerve stimulation (TENS)

3) amitryptiline + clonezepan + spinal cord stimulation (SCS)

4) morphine by continous intratechal infusion.

The authors concluded that chronic pain in SCI patients may require a specialized approach since in some cases it is necessary to use aggressive therapies and it is very important to evaluate the type and location of the pain because some patients obtain sufficient relief using only physical therapy and NSAIDs.

Balazy (1) classifies treatment options for chronic SCI pain into 5 categories as 1) general health promotion and relief from exacerbating factors 2) non-narcotic pharmacologic 3) physical 4) surgical and 5) narcotic.

The first step in SCI pain management involves the care and education to improve the patient's general health and reduce factors that exacerbate chronic SCI pain. For this purpose, general medical problems and those complications common to patients with SCI such as pulmonary and genitourinary infections, autonomic hyperreflexia, spasticity, decubitus ulcers and heterotopic ossification should be treated.

Non-narcotic treatment includes centrally acting agents such as antidepressants, anticonvulsants and antipsychotics. Reports of the studies regarding the efficacy of these medications in providing SCI pain relief are controversial. The most commonly used antidepressants are the tertiary amines including amitryptiline, nortriptyline, doxepin and imipramin. The possible mechanism of central pain relief provided by these drugs is believed to be an interference with the re-uptake of the neurotransmitters such as dopa-

mine, norepinephrine and serotonin in the nerve synapses which results in a reduction of pain transmission in the afferent pain pathways and/or an increase in norepinephrine concentration in the descending pain inhibitory pathways. On the other hand, the most widely used anticonvulsants are carbamazepine and phenytoin. These drugs act by stabilizing the threshold against hyper-excitability of neurons and inhibit the spread of epileptiform activity in second order neurons involved in nociception. Neuroleptics are the least commonly used drugs in the treatment of SCI pain (1, 3, 6, 17).

Physical therapy in the management of SCI pain involves rehabilitation treatment of mechanical and musculoskeletal problems and the administration of various physical therapy agents such as transcutaneous electrical nerve stimulation (TENS) and warm or cool modalities. Treatment of mechanical and musculoskeletal problems are important because they can exacerbate chronic SCI pain. Range of motion and stretching exercises should be applied in order to reduce spasticity and prevent contractures, adhesive capsulitis and reflex sympathetic dystrophy. This rehabilitation program provides assistance and education in daily living activities and maximizes the patient's level of independence (1).

Neurosurgical treatments which should be considered if conservative management has failed consist of neuroablative and neuroaugmentative procedures. Nerve blocks help to distinguish peripheral from central pain and may also reduce peripheral noxious stimuli that can exacerbate central pain. Neuroablative procedures include sympathectomy, neurolyses, dorsal rhizotomy, cordectomy, mesencephalotomy and cingulotomy. All of these techniques are considered to be ineffective in the present time. Dorsal root entry zone (DREZ) surgery is an ablative procedure with a high success rate in releiving chronic SCI pain. Dorsal root entry zone, also known as substantia gelatinosa, is a column of small neurons in dorsal gray horns throughout the length of the spinal cord. Pain massage from primary sensory neurons is received by the substantia gelatinosa and may be subject to modification before transfered to secondary pain neurons or tract cells. After SCI, this modification fails to occur and secondary pain neurons or tract cells. After SCI, this modification fails to occur and secondary pain neurons gain abnormal electroneurophysiologic character (hyperexcitability). The effectiveness of the DREZ surgery is thought to be due to destruction of these abnormal secondary neurons in the dorsal horn (1).

Finally opioid treatment should be considered in those selected patients who failed to obtaion satisfactory relief from conservative and surgical treatment. Methadone is the drug of choise among opioids in the treatment of SCI pain (1, 8).

SUMMARY

Pain is one of the most important problems among patients with SCI. The literature concerning SCI pain includes a broad number studies, case reports and reviews, however a consensus has not been reached regarding the classifications, descriptions or general features of this type of pain. This intractable pain may become the most disabling sequela in some cases leading to chemical dependency, severe depression and even suicide. The pathophysiology and the underlying mechanism of SCI pain are also still incompletely understood. Chronic SCI pain is central neuropathic pain and requires centrally directed treatment. Treatment should be started with conservative medical and physical therapy and then should progress to neuroablative procedures and finally narcotics as warranted. The most effective treatment of date is the DREZ surgery. It should always be remembered that multiple treatments require a team of multi-disciplined professionals.

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