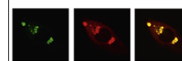


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Research Report

Cortical and white matter alterations in patients with neuropathic pain after spinal cord injury

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ABSTRACT

Neuropathic pain is one of the major problems of patients with spinal cord injury (SCI), which remains refractory to treatment despite a variety of therapeutic approach. Multimodal neuroimaging could provide complementary information for brain mechanisms underlying neuropathic pain, which could be based on development of more effective treatment strategies. Ten patients suffering from chronic neuropathic pain after SCI and 10 healthy controls underwent FDG-PET, T1-anatomical MRI and diffusion tensor imaging. We found decreases of both metabolism and the gray matter volume in the left dorsolateral prefrontal cortex in patients compared to healthy controls, as well as hypometabolism in the medial prefrontal cortex and gray matter volume loss in bilateral anterior insulae and subgenual anterior cingulate cortices. These brain regions are generally known to participate in pain modulation by affective and cognitive processes. Decreases of mean diffusivity (MD) in the right internal capsule including cerebral peduncle, pre- and post-central white matter, and prefrontal white matter as components of the corticospinal and thalamocortical tracts were demonstrated in patients. Further, lower MD value of prefrontal white matter was correlated with decreased metabolism of medial prefrontal cortex in patients. These results indicated that white matter changes imply abnormal pain modulation in patients as well as motor impairment. Our study showed the functional and structural multimodal imaging modality commonly identified the possible abnormalities in the brain regions participating pain modulation in neuropathic pain. Multifaceted imaging studies in neuropathic pain could be useful elucidating precise mechanisms of persistent pain, and providing future directions for treatment.

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1. Introduction

Neuropathic pain resulting from a primary lesion or dysfunction of the central nervous system (Nicholson, 2004) is a momentous problem in many patients with spinal cord injury (SCI); it has a prevalence of almost 50% among patients with SCI and leads to greater deterioration of quality of daily life than does motor impairment (Finnerup et al., 2003; Siddall et al., 2003). Despite a variety of pharmacological, neurosurgical, and behavioral therapeutic strategies, patients with neuropathic pain following SCI often fail to experience sufficient relief (Hulsebosch et al., 2009). Therefore, a better understanding of the mechanisms underlying neuropathic pain is required for designing more effective treatment strategies.

Several neuroimaging studies have reported reorganization of the brain in chronic neuropathic pain conditions (Schweinhardt and Bushnell, 2010; Tracey and Bushnell, 2009). A decrease in the thalamic blood flow contralateral to the symptomatic side (Iadarola et al., 1995) or hypometabolism in the posterior insula, anterior cingulate cortex (ACC), and pre- and post-central gyri (Egloff et al., 2009) were found to be indicators of functional change in the brains of patients with neuropathic pain. Recently, functional magnetic resonance imaging (MRI) studies have demonstrated the reorganization of the primary somatosensory cortex (Wrigley et al., 2009a) and disruptions of the default mode network (Baliki et al., 2008; Napadow et al., 2010) in patients with chronic pain. Anatomical MRI techniques have also shown that chronic pain is associated with structural changes in the brain. For instance, one study demonstrated a reduction of gray matter volume in the thalamus and dorsolateral prefrontal cortex of patients with chronic back pain (Apkarian et al., 2004a), whereas another study reported a loss of gray matter volume in the anterior insula and ventromedial prefrontal cortex in patients with complex regional pain syndrome (CRPS) (Geha et al., 2008). Furthermore, studies using diffusion tensor imaging (DTI), which allows investigation

of the microstructure and integrity of the white matter fiber tracts (Sundgren et al., 2004), have found that patients with neuropathic pain has significant anatomical changes in a number of brain regions associated with sensory perception and affective dimensions of pain, including the thalamus, prefrontal cortex, insula, amygdala, and posterior parietal lobule; moreover these changes were correlated with pain severity (Geha et al., 2008; Gustin et al., 2010; Lutz et al., 2008). This evidence suggests that chronic neuropathic pain is associated with structural and functional changes of both gray and white matter, which are involved in broad regions related to pain perception and modulation. However, the patterns of detected brain alterations may vary according to the type of neuropathic pain and the method of neuroimaging. Therefore, it is necessary to explore the brain changes occurring in a specific type of neuropathic pain using various brain imaging approaches.

In this study, we aimed to contribute to better understand the neural mechanisms underlying chronic neuropathic pain after SCI by using multiple imaging modalities such as fludeoxyglucose-positron emission tomography (FDG-PET), structural MRI, and DTI, which would enable to capture different functional and anatomical brain characteristics and their relationship with each other. We investigated whether these three different imaging modalities provide complementary information on the brain mechanisms of neuropathic pain following SCI.

2. Results

2.1. Patient characteristics

The characteristics of patients are listed in Table 1. The elapsed time since injury was about 1.5 years on average, ranging from 8 to 27 months. Seven patients were classified as having a complete spinal cord lesion (American Spinal Injury Association (ASIA) impairment scale, grade A), while the remaining three patients had preserved sensory function

Table 1 – Characteristics of patients with neuropathic pain after SCI.

Subjects	Gender	Age (y)	Level of SCI	ASIA	Months since injury	Mean pain intensity	BDI	Pain location
1	M	53	C5/C5	A	27	7	11	Both arms/hands
2	M	39	T5/T5	A	19	8	17	Both legs/feet
3	M	40	T11/T11	A	8	8	27	Both feet
4	F	48	C5/C7	B	21	7	15	Both lower back/buttocks/legs/feet
5	F	37	T6/T6	B	21	8	16	Both buttocks/legs/feet
6	M	40	T8/T8	A	18	8	30	Both elbows/hands/lower legs/feet
7	F	36	C6/C6	A	26	7	32	Both elbows/hands/lower legs/feet
8	M	40	T10/T10	A	11	8	16	Both legs/feet
9	M	34	C5/C5	A	11	7	4	Both arms/hands/knees/feet
10	M	31	C4/C4	B	22	8	13	Both forearms/hands/lower back

SCI, spinal cord injury; ASIA (American Spinal Injury Association) impairment scale, A=complete, no sensory or motor function in sacral segments S4-S5; B=incomplete, sensory but not motor function preservation below the neurologic level and extends through sacral segments S4-S5; BDI, Beck Depression Inventory; F, female, M, male.

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