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Sensory Syndromes

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Abstract

Somatosensory deficit syndromes represent a common impairment following stroke and have a prevalence rate of around 80% in stroke survivors. These deficits restrict the ability of survivors to explore and manipulate their environment and are generally associated with a negative impact on quality of life and personal safety. Sensory impairments affect different sensory modalities in diverse locations at varying degrees, ranging from complete hemianesthesia of multiple modalities to dissociated impairment of somatosensory submodalities within a particular region of the body. Sensory impairments induce typical syndromal patterns which can be differentiated by means of a careful neurological examination, allowing the investigator to deduce location and size of the underlying stroke. In particular, a stroke located in the brainstem, thalamus, and the corticoparietal cortex result in well-differentiable sensory syndromes. Sensory function following stroke can be regained during rehabilitation even without specific sensory training. However, there is emerging evidence that specialized sensory interventions can result in improvement of somatosensory and motor function. Herein, we summarize the clinical presentations, examination, differential diagnoses, and therapy of sensory syndromes in stroke.

Perception and interpretation of somatosensory information is a general requirement for human life. Mental registration and interpretation of sensory stimuli is dependent on information from several sensory systems. Hence, the somatosensory network is closely interlinked to all other sensory structures, including the higher functional areas in the brain, and the motor system. Due to this tight relationship, somatosensory function is not only susceptible to damages to somatosensory brain areas, but also vulnerable to impairments of other brain systems. Accordingly, most stroke survivors suffer several somatosensory deficits (body senses such as touch, temperature, pain, and proprioception). Reported prevalence rates appear to vary between 65 and 100% [1–4]. Even in patients diagnosed with pure motor stroke via neurological examination, sensory dysfunction was found in 88% of cases [3]. Impaired sensory function is often notably underdiagnosed, though it hinders the ability to explore and manipulate one's environment and negatively affects the quality of life as well as personal safety. Therefore, correct diagnosis and appropriate treatment of sensory syndromes has raised much attention in recent years.

Clinical Presentations

Stroke affects one or more of the sensory modalities in varying degrees, ranging from complete hemianesthesia of multiple modalities to dissociated impairment of somatosensory submodalities within a particular body region.

Studies report an impairment of elementary sensory modalities such as touch, pressure, pain, vibration, and temperature in 53–64% survivors after stroke [2]. Impaired proprioception occurs at a similar frequency [2, 5]. Moreover, the majority of studies consistently regard stereognosis (tactual object recognition) as being the most common somatosensory impairment following stroke [2, 6]. However, pure or predominant somatosensory symptoms in stroke patients are also not uncommon. These are reported as representing the most frequent lacunar syndromes [7–9]. In most cases, the lacuna was found in the thalamus [10–12], but also brainstem [13, 14], capsular [15], and parietal [6] lesions are described as causing predominantly somatosensory symptoms. The degree of impairment of sensory function correlates closely to stroke severity (NIHSS score) and extent of lesion [2, 5]. In contrast, larger strokes almost always result in non-sensory symptoms due to the tight relationship of the somatosensory network to other systems.

Although somatosensory impairment varies widely according to location of stroke within the CNS, the investigator can suspect stroke site via particular patterns of presentation:

Sensory impairment due to brainstem stroke results mostly from small infarcts or hemorrhages in the medulla or pons. Lateral brainstem strokes in medulla and pons often cause a loss of pain and temperature sensation. Most lateral, they affect the ipsilateral face and the contralateral lower body (type I of Stopford's classification [16]). Mediolateral lesions can affect only the upper part of the body (type II), while large strokes in both of these regions can result in a combination of crossed and unilateral pattern (type III).

Paramedian infarcts are found more frequently in the pons than in the medulla and affect elementary sensations (most often vibration and position sense) and often show dominance in the cheiro-oral or leg region. Somatosensory symptoms of the facial or perioral region due to paramedian pons lesions frequently occur bilaterally. Aside from sensory deficits, most patients also suffer dizziness and gait ataxia [13].

Sensory impairment owing to thalamic stroke is predominantly caused by lacunar infarcts in the ventroposterior nucleus of the thalamus, again mostly affecting elementary sensations showing a faciobrachiocrural distribution. A typical constellation of symptoms for thalamic strokes include numbness and paresthesia, although dysesthesia and pain also commonly occur. The latter can develop directly following stroke, or subacutely a few days later (2–15 days) [12].

Corticoparietal stroke mainly involves discriminatory modalities of sensation like proprioception, stereognosis, or texture recognition which are usually limited to one or two parts of the body, sparing the trunk [6, 17]. In particular, a combination of impaired discriminating modalities with a preserved vibration sense can be considered as being characteristic for cortical strokes [6]. Since this pattern arises mainly due to lesions in the superior-posterior parietal cortex, it is referred to as the cortical sensory syndrome. However, a lesion in the inferior-anterior parietal cortex (parietal operculum, posterior insula) can mimic a thalamic sensory syndrome and is designated as the pseudothalamic syndrome [6]. A corticoparietal stroke resulting in a pseudothalamic syndrome cannot be differentiated from a thalamic stroke on the basis of sensory deficits alone. In cases of left hemispheric parietal strokes, neuropsychological dysfunctions usually involve language impairment, while right hemispheric lesions lead to visuoconstructive and visuospatial disturbances [6, 18–20]. In addition, somatosensory impairment due to cortical strokes is accompanied by some motor dysfunction in over 90% of cases [21].

Somatosensory impairment is more frequent in right hemispheric than in left hemispheric stroke [22]. Several studies report significant sensory impairment of the ipsilateral body side with an incidence of 17% following unilateral stroke [2, 3, 23]. The border zone of sensory symptoms on

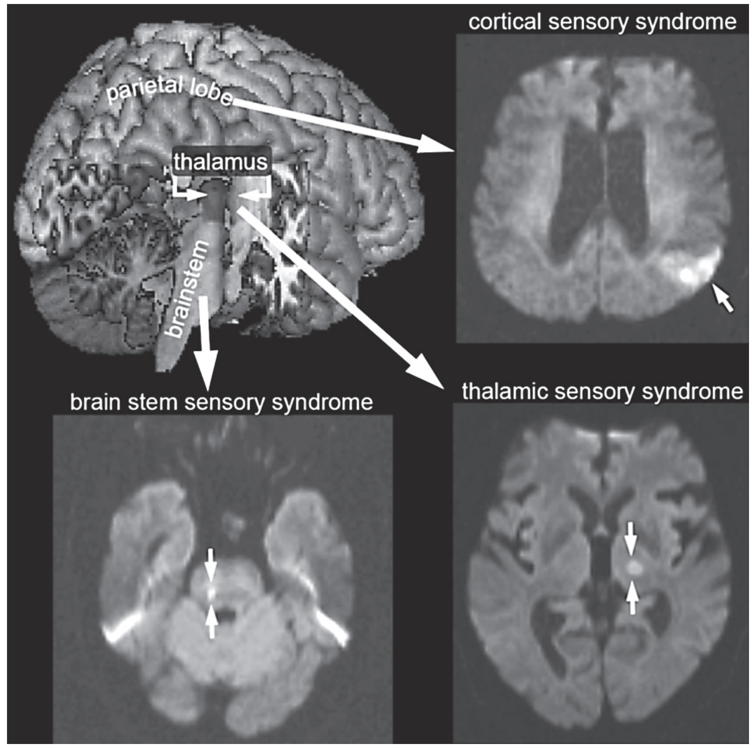

Fig. 1. MRI lesion examples (diffusion-weighted imaging) for different sensory syndromes.

the trunk and face are expected to be paramedian due to the 1–2 cm sensory function overlap of the intercostal nerves (fig. 1).

Diagnosis of Somatosensory Syndromes

Study results pertaining to the occurrence of somatosensory impairments after stroke vary widely [1, 2, 4, 24]. It is thought that the incidence is often underestimated since differential sensory modality assessments are limited in their scope. By assessing a single elementary somatosensory modality like touch, an impairment was found in only ~25–40% of stroke survivors, whilst a multimodal testing of elementary sensory modalities revealed dysfunction in ~60% of cases [2, 3]. However, good agreements between different body areas were found within each modality, indicating redundancy of testing between adjacent body regions [2]. The highest sensitivities for sensory impairments after stroke were found by testing discriminatory sensations, such as stereognosis, texture discrimination, position sense, and two-point discrimination. By such testing, impairments were found in 85–89% of stroke survivors [2, 3]. Many studies have described sensory functions using largely subjective scales such as 'absent' versus 'impaired' versus 'normal', thereby restricting interpretability and comparability between studies. New measures have been developed for improved standardized clinical somatosensory testing. The two most frequently used test batteries comprise the Nottingham Sensory Assessment (NSA) [25, 26] and the Rivermead Assessment of Somatosensory Performance (RASP) [26]. Both tests aim to identify sensory deficits after stroke and to monitor their recovery from stroke. The NSA employs eight quantifiable subtests that can be used in a clinical setting without additional instruments. However, inter-rater reliability is relatively poor even in the revised NSA [25]. There are attempts to further modify the NSA for a better inter-rater reliability [27]. In contrast, the RASP has good inter-rater reliability, but requires additional tests that are not common in clinical practice [26].

Therapy and Prognosis

The main complaint of stroke survivors is loss of somatosensory function. Most patients undergo rehabilitation to regain and relearn lost skills. In the past, rehabilitation training has focused mainly on motor recovery, whereas somatosensory recovery has received less attention. This is probably due to the assumption that loss of sensation is less important for motor recovery. Recent studies, however, have shown that impaired sensory function is associated with the quality of upper limb movement, force control, manipulation of fine-graded objects, and sensory ataxia [4, 23]. The resulting sum of impairments predict poor functional outcome after stroke, including independence in activities of daily life, and even mortality [4, 5]. These findings have increased interest in underlying mechanisms of somatosensory recovery after stroke. It has long been recognized that sensory function following stroke improves during typical rehabilitation training without specific sensory training [2, 28]. Thus, multiple studies were performed to investigate the effectiveness of specialized sensory rehabilitation training [29–32]. In general, these interventions used sensory discrimination tasks [1, 30, 33] and sensory stimulation approaches involving tactile [29], electrical [32], thermal [31], and magnetic stimuli [34]. There is emerging evidence that specialized sensory interventions can result in increased recovery of somatosensory function as well as an improvement of other impairments such as motor function [for review, see 35].

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