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Review

Onset, time course and prediction of spasticity after stroke or traumatic brain injury

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ABSTRACT

Objective: To describe spasticity from the onset of acquired brain injury, time course over the first year and factors associated with prediction of the development of spasticity.
Methods: Recent relevant literature known to the authors, along with a complementary search yielding a total of 9 articles, represented the base for this scoping review.
Results: Spasticity can be seen in the first week after brain injury and is more common in the upper than lower extremity. The severity of upper-limb impairment is a major factor in the development of spasticity during the first year after stroke. The prevalence of severe spasticity seems to increase during the first year. The combination of reduced arm motor function and spasticity in an early phase (4 weeks post-stroke) is an important predictor of the development of severe spasticity after 12 months. Spontaneous reduction in spasticity was seldom reported but may occur, especially in mild forms of spasticity.
Conclusion: Signs of spasticity can often be noted within the first 4 weeks after brain injury and is more common in the upper than lower extremity. Impaired sensorimotor function is a predictor. These findings highlight the importance to follow up patients with increased risk of developing severe spasticity to be able to start adequate spasticity treatment and prevent the negative consequences of spasticity. Understanding spasticity onset and progression also provides a basis for the development of effective therapies.

1. Introduction

Spasticity is a common feature of upper motor-neuron syndrome and thus can occur after stroke or traumatic brain injury. It is characterized by a velocity-dependent increase in tonic stretch reflexes (“muscle tone”) with exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex [1] The spasticity itself is only a phenomenon and the important issue is to evaluate whether the consequences of the spasticity are problematic for the patient.

Spasticity can result in reduced motor function and general mobility and is associated with increased incidence of falling [2], which all may limit the potential success of rehabilitation. Spasticity is more common in the upper than lower extremity [3]. Upper-extremity spasticity is associated with reduced arm function and reduced independence [4,5]. Spasticity in upper, lower or both extremities at 1 year after stroke seems to result in significantly reduced independence in activities in daily living [6]. After a stroke, the prevalence of spasticity in the upper limb varies from 4 to 38% [6–9] in the first 12 months and from 25 to 46% in patients with initial impaired arm function [10,11].

The prevailing idea is that early recognition of spasticity is of importance for later outcome. The identification of predictors that can help the clinicians identify patients at risk of post-stroke spasticity is important for preventing, if possible, the onset of spasticity, decrease its development rate or limit its progression [12,13]. Early treatment is believed to yield a better outcome [14]. Therefore, it would be helpful to know which factors are important for identifying patients at high risk of severe spasticity, especially during the initial acute phase and while the patient is still in the hospital. However, the onset of spasticity is highly variable and may occur in the short-, medium-, or long-term period after the stroke or brain injury.

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To provide good care [15], we need better knowledge of the pathophysiology and evolution of the condition over time. The aim of this review was to discuss the onset and time course of spasticity as well as possible predictors that can help identify the patients at risk of spasticity developing after stroke or traumatic brain injury.

2. Methods

A literature search of PubMed was performed with the following terms: natural history OR clinical course OR prevalence AND stroke OR brain injury AND spasticity. The results were limited to studies focusing on predicting spasticity up to 12 months after the onset of stroke or acquired brain injury. Relevant literature known to the authors along with this complementary search represented the base for this scoping review. The authors are aware that assessment of spasticity with the Modified Ashworth Scale (MAS) is only an epiphenomenon rather than the cause or consequence of the functional condition. However, in the studies, the MAS was a common descriptor of spasticity. The original MAS score categories (0, 1, 1+, 2, 3 and 4) are often converted to integers from 0 to 5, and the latter scoring was used in the current review. Spasticity is generally defined as MAS $\geq$ 1. Wissel et al. [9] defined mild spasticity as MAS = 1, moderate as MAS = 3, and severe as MAS $\geq$ 4. Opheim et al. [11,16] defined severe spasticity as MAS $\geq$ 2. However, Lundström et al. [7] defined disabling spasticity as spasticity causing disability that requires intervention.

3. Results

Results from 7 studies (9 articles), identified for this scoping review, were extracted and summarized (Table 1). Several studies assessed spasticity at various times post-stroke, but no studies regarding the onset or development of spasticity in individuals after traumatic brain injury were found.

3.1. Onset of spasticity

The onset of spasticity has been addressed in only a few studies [10,11]. Sommerfeld et al. [10] reported that at baseline assessment (mean: 5.4 days post-stroke) 21% of patients with a first-ever stroke (n = 95) and 26% of those with hemiparesis (n = 77) showed spasticity according to the MAS (score > 0). At 3 months, 19% of all patients (n = 95) demonstrated spasticity and for those with remaining hemiparesis (n = 64), the frequency was 28%. These numbers indicate no increase in prevalence from 5 days to 3 months in the entire group and only slight increase in a subgroup with remaining hemiparesis. Most patients with spasticity had a MAS score of 1 or 2 and no patient had a score of 4 and 5 at baseline, and only 1 patient had a score of 4 after 3 months.

Opheim et al. [11] presented data from a study of patients with a first-ever stroke; 117 patients with impaired arm function on day 3 were consecutively included and assessed at 6 occasions over the first year, and 76 patients were assessed at 12 months. Upper-limb spasticity in elbow flexion/extension and wrist flexion/extension was assessed with the MAS. "Any spasticity" was considered a MAS score $\geq$ 1 and "severe spasticity" a MAS score $\geq$ 2 in any of these muscles. In total, 25% had spasticity in at least one of the muscle groups assessed at day 3, 44% (n = 48) after 4 weeks, 38% at 3 months and 46% (n = 35) after 12 months after onset. These results suggest that the onset of spasticity occurs as early as 3 days post-stroke in approximately half of patients in whom spasticity develops, and that in the second half, the spasticity will evolve during the first month. At day 3, only 6 patients showed severe spasticity, and patients with spasticity always had spasticity in elbow flexor muscles.

In the study by Lundström et al. [3], at baseline (2–10 days post-stroke), 4% (2 of 49) of patients had spasticity (MAS score $\geq$ 1). Spasticity was further observed in 27% (13 of 48) of patients at 1 month and 23% (11 of 47) at 6 months. Wissel et al. [9] found spasticity (MAS score $\geq$ 1) in 25% of participants (n = 94) at 2 weeks post-stroke.

3.2. Time course of spasticity

Opheim at al. [11] showed that in a sample of patients with impaired upper-limb function at 3 days post-stroke, 19 (17%) changed from no spasticity to having spasticity at day 10, and 5 had a changed status in the opposite direction. From day 10 to week 4, these numbers were 15 and 9, respectively. This finding suggests that even when the presence of spasticity increases among patients with arm paresis during the first 4 weeks post-stroke, for some, the early assessed spasticity resolves after the first 4 weeks. The study by Lundström et al. [3] found spasticity (MAS score $\geq$ 1) in 27% of participants (n = 48) at 4 weeks post-stroke, with 4% exhibiting more severe degrees of spasticity (MAS score $\geq$ 3) [3]. Again, the study by Opheim et al. [11] reported a higher prevalence of spasticity after 4 weeks, 44% in participants with impaired upper-extremity function at onset. At a median of 6 weeks (1–3 months)

Table 1
Studies included in the review in alphabetical order.

Study	No.	Time after stroke	Outcome measure	Spasticity prevalence
Lundström et al., 2008 [3,7]	140	First-ever stroke followed at 12 months	Spasticity: MAS (MAS $\geq$ 1); NIHSS; mRS; BI	Spasticity: 17% Disabling spasticity: 4% (by mRS 2-5; and a worse BI score)
Malhotra et al., [17]	30	First-ever stroke, baseline within 3 weeks, 6, 12, 24 and 36 weeks	EMG activity during passive wrist extension at 2 velocities. ARAT	Signs of spasticity (92%) were seen throughout the study period
Mirbagheri et al., [20]	20	First-ever stroke, baseline, 1, 2, 3, 6 and 12 months	Kinematis during extension and flexion, Spasticity MAS, Fugl-Meyer	NA
Opheim et al., [11] [16]	117	First-ever stroke Days 3 and 10; week 4; and 3, 6, and 12 months	Spasticity: MAS > 0 Fugl-Meyer, ARAT	25% at day 3, at 12 months in 46%, and 29% having severe spasticity (MAS score $\geq$ 2)
Sommerfeld et al., 2004 [10]	95	Acute first-ever stroke	MAS (Spasticity: MAS > 0)	Spasticity: 21% at 5.4 days; 19% at 3 months
Urban et al., 2010 [17]	211	Up to 6 months	MAS (Spasticity: MAS $\geq$ 1)	Spasticity: 42.6% Severe spasticity: 15.6% (MAS $\geq$ 3)
Wissel et al., 2010 [16]	94	Up to 4 months	MAS (Spasticity: MAS > 0)	Spasticity: at 2 weeks: 24.5%; at 6 weeks: 26.7%; at 4 months: 21.7% Severe spasticity: 9.6% (MAS $\geq$ 3)

BI: Barthel Index; MAS: Modified Ashworth Scale; mRS: modified Rankin Scale; NIHSS: National Institutes of Health Stroke Scale; ARAT: Action Research Arm Test; EMG: electromyography; NA: not available.

post-stroke, both the proportion of patients exhibiting spasticity and the degree of spasticity continued, with spasticity in 27% and severe spasticity (MAS score ≥ 4) in 9% ($n = 86$) [9]. Among participants with no arm function at the initial evaluation post-stroke, signs of spasticity were present in 93% ($n = 30$) after 6 weeks [17].

Spasticity appears to remain from 3 and 6 months after stroke [9,17]. At a median of 16 weeks post-stroke, spasticity was observed in 22% of patients, almost 10% with severe spasticity [9]. This slight decrease in the prevalence of any spasticity is explained by the normalization of muscle tone in several individuals with only minor or slight signs of spasticity at baseline, which supports that not all spasticity requires treatment [9]. However, because the number of individuals with moderate to severe spasticity (MAS score ≥ 3) increased to more than half of the total number with spasticity (MAS score ≥ 1) at later times [9], patients with moderate to severe MAS scores may benefit from early treatment of spasticity because it is often associated with impaired function.

More than 6 months after stroke, spasticity was still observed in 93% of participants (28 of 30) with no arm function at initial evaluation, including 5 with recovered arm function as assessed by the Action Research Arm Test (ARAT) at 9 months post-stroke [8]. The number of patients with severe spasticity seems to increase during the first year, as shown in the study by Opheim et al. [11], because the number of patients increased from 6 of 117 (5%) at day 3 to 22 of 76 (29%) at 12 months.

Although spasticity often seems to remain after the first year post-stroke, data on long-term follow-up (> 12 months) is not available. In one article [18], patients were included 1 to 5 years post-stroke at a physiotherapy clinic in Brazil. In the consecutive sample of 146 patients with prior ischemic stroke, spasticity was identified in 26%. In this study, spasticity was defined as MAS ≥ 1; however, no information on location of the spasticity was available and thus the location could include both upper- and lower-limb spasticity.

3.3. What predicts spasticity?

The prevailing idea is that the prediction of developing spasticity at later times is likely improved by using measures of spasticity early after stroke. We have some evidence that sensorimotor function during the first weeks post-stroke rather than spasticity itself might be a stronger predictor of later spasticity [16]. Opheim et al. [16] showed that sensorimotor function was the most important predictor both for any spasticity and severe spasticity 12 months post-stroke. Spasticity was a significant predictor of severe spasticity not earlier than 4 weeks post-stroke. The results indicate that the best prediction model for any spasticity was observed 10 days post-stroke (85% sensitivity, 90% specificity). The best prediction model for severe spasticity was observed 4 weeks post-stroke (91% sensitivity, 92% specificity). Prediction models based on assessments at admission showed low sensitivity for predicting spasticity at 12 months.

Thus, there is still some uncertainty about which measures should be used and when these measures should be obtained. The study by Lundström et al. [3] included 49 people with any paresis after a first-ever stroke and assessed them at baseline (2–10 days post-stroke) and 1 and 6 months post-stroke. At all occasions, spasticity (assessed by the MAS), global disability (assessed by the mRS) and stroke severity (assessed by the National Institutes of Health Stroke Scale [NIHSS]) were recorded to determine the occurrence of and risk factors for spasticity. Severe arm paresis (> 2 points on item 5 of the NIHSS) at baseline was associated with increased risk of spasticity ($P < 0.001$) as early as 1 month post-stroke. Being younger and smoking was also associated with increased risk of spasticity.

A large study ($n = 301$) by Urban et al. [8] found spasticity more frequent in patients with reduced sensory function than in those without sensory deficits and those with severe paresis. Spasticity was associated with lower Activities of Daily Living score and reduced quality of life.

In a study by Mirbagheri et al. [19], 21 patients with hemiparetic upper-extremity spasticity were assessed at 4 weeks post-stroke and followed during the first year by using an advanced technique to quantify neural and muscular changes associated with spasticity in the elbow flexors. Using mathematical modelling, the authors detected differences associated with the initial reflex stiffness magnitudes (high or low). Participants with high reflex stiffness at baseline showed reduced stiffness over time, as compared with those with moderate initial reflex stiffness, who showed increased reflex stiffness over the first year. Motor function at 4 weeks post-stroke was the strongest predictor of reflex recovery patterns, which indicates that high initial scores on sensorimotor function, assessed by the Fugl-Meyer Assessment (FMA), might predict declining levels of hyperreflexia over time and that a midrange FMA score might be associated with the development of hyperreflexia.

4. Summary of findings

Our understanding of the clinical course of spasticity places an important focus on the time course of development, particularly at the early stage, from immediately after stroke onset to 12 months, although spasticity prevalence seemed to peak at approximately 4 weeks after stroke. Although the goal of early management is long-term recovery in stroke patients, emphasis should also be placed on appreciating the difference between spontaneous recovery and improvement in performance in terms of the outcomes measured. Most patients showing spasticity post-stroke have signs of spasticity within the first 4 weeks post-stroke. Because the neurobiological background for the development of spasticity after a brain injury is similar whether caused by stroke or trauma, the finding from individuals with stroke are to a large extent generalizable to those with traumatic brain injury as well. Focal damage to the brain (stroke or traumatic) may result in upper motor-neuron syndrome, whereby, among other features, exaggerated deep-tendon reflexes including spasticity and clonus can be seen. Spasticity is more common in the upper than lower extremity. This was shown in studies reporting the frequency of spasticity in both upper and lower extremities.

The prevalence of severe spasticity seems to increase during the first year after stroke, but in some patients, the early assessed spasticity (MAS ≥ 1) disappears during follow-up.

Spasticity can lead to reduced function and increased dependency in daily living, as shown in the study by Bhakta et al. [4], in which reduction in spasticity was shown to reduce the burden for caregivers.

During the first 2 to 4 weeks post-stroke, the sensorimotor impairment rather than spasticity itself seem to be the strongest predictor of severe spasticity developing after 12 months.

The combination of reduced arm motor function and spasticity in an early phase (4 weeks post-stroke) is an important predictor of the development of severe spasticity after 12 months. Whether early spasticity interventions can stop or delay the development of spasticity is still unclear.

Spontaneous reduction in spasticity has seldom been reported but may occur, especially in individuals with mild forms of spasticity. We still lack data in this area.

These findings highlight the need to follow up patients with increased risk of severe spasticity developing to be able to start adequate spasticity treatment and prevent the negative consequences of spasticity. We still lack knowledge regarding the onset

and developmental course of spasticity. Thus, understanding spasticity onset and progression also provides a basis for the development of effective therapies.

Disclosure of interest

The authors declare that they have no competing interest.

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