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ORIGINAL REPORT

PREVALENCE OF SPASTICITY AFTER ANEURYSMAL SUBARACHNOID HAEMORRHAGE

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Objective: **The prevalence of spasticity after stroke is approximately 20%. There is, so far, little information in the literature on the development of spasticity after aneurysmal subarachnoid haemorrhage. The objectives of this study were to estimate the prevalence of spasticity after aneurysmal subarachnoid haemorrhage and to identify possible risk factors in the acute phase.**

Methods: **A total of 87 patients were assessed for spasticity with the Modified Ashworth Scale after 6 months. A multivariate logistic regression model was used to evaluate risk factors.**

Results: **Spasticity was present after 6 months in 19 (22%) of the patients, but was treated pharmacologically in only 1 case. Worse clinical status at admission carried a high risk for spasticity (odds ratio (OR) 10.2; 95% confidence interval (CI) 2.4–43.2), followed by the presence of infection (OR 7.4; 95% CI 1.6–33.8) and vasospasm (OR 4.8; 95% CI 1.2–19.0) during the intensive care phase.**

Conclusion: **Spasticity after aneurysmal subarachnoid haemorrhage occurred with the same prevalence as after other stroke. Risk factors for spasticity were worse clinical condition at admission and the occurrence of infection and vasospasm during the intensive care period. Pharmacological treatment was not commonly used.**

Key words: subarachnoid haemorrhage; aneurysmal; stroke; muscle spasticity; prevalence; rehabilitation; risk factors.

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INTRODUCTION

Aneurysmal subarachnoid haemorrhage (aSAH) is a type of stroke cause by sudden bleeding from a ruptured intracranial aneurysm into the subarachnoid space. It is a devastating disease with an incidence rate of 10–12 per 100,000 person-years and only 60% survival rate (1, 2). Patients surviving aSAH have difficulties in returning to normal working life, even in cases classified as favourable outcomes according to established follow-up protocols. Often this is due to cognitive and neuropsychological difficulties (3).

Spasticity is one of many impairments that can hamper rehabilitation after stroke. It is often associated with exaggerated reflexes and clonus. Spasticity is classified as a sign of upper motor neurone (UMN) syndrome, which is a clinical phenomena observed after lesions of cortical motor areas or the corticofugal descending tracts. Preventing and treating spasticity after aSAH could help to reduce the costly and quality-of-life-reducing consequences of this condition (4). This is especially important since patients with aSAH tend to be younger at diagnosis, and the loss of productive life years is comparable to that of cerebral infarction (2, 5).

With the exception of 2 studies limited to severely injured patients (6, 7) there are little published data on the prevalence of spasticity after aSAH. Studies on spasticity after other stroke show a prevalence of 17–22% (8–11), although some studies with different settings report a prevalence of up to 43% (12, 13). Furthermore, little is known about to what extent patients with spasticity after aSAH receive treatment for the condition.

The risk factors for spasticity are not well documented. aSAH causes damage to the brain through the initial global ischaemia, but also by repeated secondary insults giving rise to multiple complications, such as vasospasm, intracerebral haemorrhage, hydrocephalus, infections, and brain ischaemia. Patients with aSAH experience a variety of these conditions, making the patients suitable for the study of risk factors for spasticity.

The objectives of the present study were to estimate the prevalence of spasticity 6 months after aSAH, to identify risk factors in the acute phase for spasticity after aSAH, and to investigate the extent to which patients receive pharmacological anti-spastic treatment.

METHODS

Study population

Patients aged 18 years or over, admitted with aSAH to the Department of Neurosurgery at Uppsala University Hospital, Sweden and with no previous history of subarachnoid haemorrhage or spasticity were eligible. The Department of Neurosurgery at Uppsala University Hospital has a catchment area of 2 million inhabitants. Assessment was performed at the intensive care unit. The recruitment period was from 6 January 2010 to 13 July 2011. The aim was to include 100 patients. The sample size was based on previous research on spasticity after stroke (8–15). It was assumed that 20% of the patients would develop severe paresis, and of those, 70% would develop spasticity. In addition, 30% of the patients without severe paresis would develop

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spasticity. With the above proposed sample size these approximations would provide a power of 90% to detect a difference between the 2 groups at a significance of 5%, which was considered acceptable.

Management of aneurysmal subarachnoid haemorrhage

The patients with aneurysms received nimodipine for 3 weeks, had bed rest for 10 days and were kept normo-volaemic in order to avoid hypotension. The ruptured aneurysms were, in general, treated as soon as possible, either with surgical clipping or endovascular coiling. Patients with hydrocephalus and/or decreased consciousness received a ventricular drain. Unconscious patients received mechanical ventilation. Vasospasm was suspected when neurological deterioration that could not be explained by new haematomas, ischaemia or hydrocephalus occurred. Treatment of vasospasm included increased blood volume and blood pressure and improved blood rheology. Secondary insults (high intracranial pressure, low cerebral perfusion pressure, seizures, fever, hypoxia and hypo-/hyperglycaemia) were treated according to the neurointensive care unit's protocols for programmed care (16).

Risk factors for spasticity

The following admission parameters were collected for statistical analysis: the location of the aneurysm as anterior or posterior circulation, the clinical condition at admission measured with the World Federation of Neurosurgical Societies scale (WFNS) (17), the amount of blood visualized at the diagnostic computed tomography (CT) scan graded using the Fisher scale (18), paresis, age, and gender. WFNS classifies the clinical condition of the patient with SAH using a scale between 1 and 5; where 1 represents best clinical condition and 5 worse. The Fisher scale has 4 grades, of which 1 represents no blood detected and 4 intracerebral or intraventricular clots. We used the whole scale though its inter-rater reliability has been discussed (19). Anterior circulation includes aneurysms on the internal carotid artery, anterior cerebral artery and middle cerebral artery, and posterior circulation those on the basilar and vertebral arteries.

The treatment method for the aneurysm was recorded during the intensive care period as well as CT-verified brain ischaemia, intracerebral haemorrhage and acute hydrocephalus. Infections and vasospasm were recorded when they required medical treatment. WFNS and paresis were assessed by the attendant neurosurgeon, and these data were collected from the hospital's journal system. There was no standardized approach to measure paresis; paresis was defined as weakness in at least one limb at arrival at Uppsala University Hospital according to the neurosurgeon's assessment.

Spasticity, apart from the 6-month follow-up, was also assessed at inclusion. This assessment was, however, affected by the patient's medication and intubation to an extent, and therefore spasticity at the recruitment phase was not considered reliable as a potential risk factor for spasticity at 6 months and has not been included in the risk factor analysis.

Spasticity assessment at 6-month follow-up

A patient follow-up was conducted 6 months after the aSAH, either at the neurology department or in the patient's home. Spasticity was assessed using the Modified Ashworth Scale (MAS) (20). The upper limb (shoulders, elbows, wrists and fingers) and lower limb (hips, knees, and ankles) were assessed. The shoulders were examined in abduction and adduction with the elbow in 90º flexion. The other joints were examined in flexion and extension. Each joint was examined separately at different velocities with the patient lying in the supine position. The MAS grades the resistance of a relaxed limb to passive stretches into 6 grades. A normal muscle tone with no spasticity is graded as 0, a slight increase as 1 or 1+, a more marked increase as 2, a considerable increase as 3, and the most severe spasticity with a rigid limb in flexion or extension as 4. Patients with a MAS score of one or more in at least one joint were considered to have spasticity. Assessment was performed by clinicians trained in the examination of spasticity and use of the MAS. We assessed whether or not antispastic treatment had been administered during the first 6 months based on interview questions of the patients as well as medical records. To measure functional outcome, we used the modified Rankin Scale (mRS) (21), which is graded between 0 and 6 to assess disability after stroke, where 0 represents no symptoms at all and 6 is dead.

Ethics

The study was approved by the regional ethics review board in Uppsala. Patients were included after written informed consent either directly from the patients, or from next of kin.

Statistical analysis

Univariate analysis was used to compare possible risk factors for spasticity between patients with and without spasticity. Student's *t*-test, χ^2 test, Fisher's exact test, and Mann-Whitney *U* test were used, where appropriate. A risk factor with a $p < 0.05$ was considered significant. WFNS was dichotomized in good (WFNS 1–2) or worse (WFNS 3–5). The significant risk factors were selected for a multivariate logistic regression model using the backward stepwise (likelihood ratio) method. OR for spasticity were calculated and are presented with a 95% CI. Data was analysed using STATA 10.0 software for Microsoft Windows.

RESULTS

Prevalence, distribution and treatment of spasticity

Of 124 eligible patients during the inclusion period, 96 (77%) were enrolled in the study. Clinical demographic parameters are shown in Table I, showing that the study population seems to be representative of the whole consecutive eligible group. Eight patients died before 6 months and one was lost to follow-up. The remaining 87 patients were examined after 6 months. Nineteen patients (22%) had developed spasticity (Table II). Thirteen of those had mild spasticity, presented as MAS grade 1 or 1+. Seventeen (20%) had spasticity in the upper limb, 14 (16%) in the lower limb and 12 (14%) had spasticity in both

Table I. *Clinical characteristics of eligible patients and study cohort*

Characteristics	Eligible patients *n* = 124	Study cohort *n* = 96
Age, years, mean (SD)	59 (12)	59 (12)
Women, *n* (%)	83 (67)	63 (66)
WFNS, *n* (%)		
1	61 (49)	48 (50)
2	15 (12)	12 (13)
3	5 (4)	4 (4)
4	30 (24)	26 (27)
5	13(11)	6 (6)
Fisher scale		
1	2 (2)	3 (3)
2	29 (23)	23 (24)
3	56 (45)	44 (46)
4	36 (29)	26 (27)
Aneurysm location, *n* (%)		
Anterior circulation	105 (85)	83 (86)
Posterior circulation	19 (15)	13 (14)
Treatment, *n* (%)		
Endovascular coiling	83 (67)	62 (65)
Surgical clipping	37 (30)	34 (35)
No treatment	4 (3)	0

SD: standard deviation; WFNS: World Federation of Neurosurgical Societies scale.

Table II. *Distribution of patients according to Modified Ashworth Scale (MAS) scores*

MAS grade	Description	*n* (%)
0	No increase in muscle tone	68 (78)
1	Slight increase	6 (7)
1+	Slight increase	7 (8)
2	A more marked increase	3 (3)
3	Considerable increase	1 (1)
4	Rigid	2 (2)

the upper and the lower limb. Of the 19 patients presenting with spasticity, 2 had developed spasticity in every examined joint. Sixten had developed spasticity in the elbow, 13 in the knee, 7 in the wrist, 6 in the ankle, 5 in the hip, and 4 in the fingers. One patient had been treated with intramuscular botulinum toxin, and this was the only patient in the whole sample who had received pharmacological anti-spastic treatment. The patients with spasticity had a worse functional outcome, measured with mRS, compared with those without spasticity ($p < 0.001$) (Table III).

Risk factors for spasticity

Univariate analysis showed that patients with spasticity at 6 months were in significantly worse clinical condition at admission, had more blood on the CT scan, and more often had paresis at admission. Furthermore, they were more likely to have had vasospasm, intracerebral haemorrhage, hydrocephalus, and to have been treated for infections (Table IV) during the intensive care period. When entering these factors into a multivariate logistic regression model using backward selection we found that worse clinical condition at admission (high WFNS score), the presence of vasospasm and of infection were selected as independent risk factors for developing spasticity. WFNS carried the highest risk (OR 10.2; 95% CI 2.4–43.2), followed by infection (OR 7.4; 95% CI 1.6–33.8) and vasospasm (OR 4.8; 95% CI 1.2–19.0). CT-verified brain ischaemia was marginally not significant in the univariate analysis ($p = 0.057$), but was also tried in the same model. However, it was not found to be an independent risk factor. Age, gender and aneurysm location did not have a significant impact on the development of spasticity.

Table III. *Association of functional status with spasticity*

Modified Rankin Scale	No spasticity *n* (%)	Spasticity *n* (%)
0	10 (15)	0 (0)
1	25 (37)	3 (16)
2	17 (25)	2 (11)
3	13 (19)	2 (11)
4	2 (3)	8 (42)
5	0 (0)	4 (21)

Patients with spasticity show higher scores on the modified Rankin Scale ($p < 0.001$).

Table IV. *Clinical characteristics of patients with and without spasticity at the neurointensive care unit*

Characteristics	Spasticity	No spasticity	*p*-value
Patients, *n* (%)	19 (22)	68	
Age, years, mean (SD)	61 (12)	56 (12)	>0.05
Women, *n* (%)	12 (63)	42 (62)	>0.05
WFNS, *n* (%)			<0.001
1–2	4 (21)	52 (76)	
3–5	15 (79)	16 (24)	
Fisher grade, *n* (%)			<0.01
1	0 (0)	3 (4)	
2	2 (11)	21 (31)	
3	8 (42)	31 (46)	
4	9 (47)	13 (19)	
Aneurysm location, *n* (%)			>0.05
Anterior circulation	17 (89)	58 (85)	
Posterior circulation	2 (11)	10 (15)	
Initial paresis, *n* (%)	8 (44)	7 (10)	<0.01
Vasospasm, *n* (%)	10 (53)	13 (19)	<0.01
Intracerebral haemorrhage, *n* (%)	9 (47)	16 (24)	<0.05
Hydrocephalus, *n* (%)	13 (68)	21 (31)	<0.01
Infection, *n* (%)	16 (84)	22 (32)	<0.001
Brain ischaemia, *n* (%)	11 (58)	23 (34)	>0.05

WFNS: World Federation of Neurosurgical Societies scale; SD: standard deviation.

DISCUSSION

It was found that 22% of the patients developed spasticity within 6 months of the aSAH, but only one patient had received pharmacological treatment. Worse clinical status at admission carried a high risk for spasticity (OR 10.2; 95% CI 2.4–43.2), followed by the presence of infection (OR 7.4; 95% CI 1.6–33.8), and vasospasm (OR 4.8; 95% CI 1.2–19.0) during the intensive care phase.

There are only a few studies on SAH for comparison. Blicher & Nielsen (6) studied a mixed sample with stroke, SAH, traumatic brain injury (TBI) and others, and found a prevalence of spasticity of 40% 1 year after SAH. Singer et al. (7) also studied a mixed sample of acquired brain injuries including SAH. After 20 weeks 65% had normal muscle tonus, 13% were predominantly spastic and 22% predominantly dystonic. However, these studies had patients that were selected to represent more severe injuries with admission Glasgow Coma Scale less than 10 and 12, respectively. In our study, no exclusion was done based on clinical condition, which could explain the lower rate of spasticity.

Our results are similar to most studies on stroke, showing a prevalence of spasticity between 17% and 22% (8–11, 15). Some studies report higher prevalence, up to 43% (12, 13), which may be due to differences in inclusion criteria or in the method of assessing spasticity. For example, Urban et al. (12) only included patients with central paresis, and Watkins et al. (13) used a slightly different definition of spasticity that combined the MAS and the Tone Assessment Scale.

The definition of spasticity and the assessment methods have been debated (22). The most commonly used definition is probably that of Lance (23) from 1980: "… a motor disorder

characterised by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex as one component of the upper motor neurone syndrome." Lance's definition points out that spasticity is only one component of the UMN syndrome, and that spasticity is a sign at clinical examination, but not with regard to impact on motor ability or need for treatment. Our results indicate that high MAS scores in patients with aSAH are rare. Only one patient had received pharmacological treatment. This may be due to the low MAS scores in our group of patients. Other explanations could be the lack of awareness of problems associated with spasticity among healthcare personnel, or the side-effects of oral anti-spastic drugs. Treatment should be given when the spasticity causes problems for the patient's functioning or care provision. Therefore, the MAS alone is not sufficient to define the threshold above which spasticity is an undesirable impairment. Spasticity, especially in the lower limb, can sometimes be functionally useful in helping patients to walk, stand, and maintain posture.

Another difficulty with spasticity is how to measure this phenomenon. Spasticity occurs when losses of the upper motor neurones leads to decreased inhibition of lower motor neurones, which, in turn, causes an increase in nervous activity that manifests as spasticity (24). In order to compare our results with other studies we used the MAS and defined spasticity as a MAS score of one or more, which is the most commonly used definition in stroke studies. However, MAS does not differentiate between mechanical and neural components of the increased resistance to passive movements (25, 26). In addition, the performance of the MAS is not standardized. Neither the position of the limb nor the speed of the movement is specified.

Although aSAH is a haemorrhagic event, much of the pathophysiology is based on a number of ischaemic events. At the rupture of the aneurysm there is often a transient global brain ischaemia due to the high intracranial pressure. This is followed, after some days, by the development of vasospasm, which can lead to hypoperfusion and brain ischaemia. Furthermore, the brain's metabolism seems to be altered and increased in the days following the rupture (7), and events leading to further increases in the metabolism and/or substrate failure can then result in ischaemic lesions in spite of a normal blood flow. Infections increase the metabolism and frequently result in fever, which is one of the secondary insults on the neurointensive care unit's care targets. In our sample, we found that vasospasm and infections were independent predictors of spasticity. These are factors that compromise substrate delivery and increase the metabolic demand on the brain. This supports the theory that ischaemia is an important factor in the development of spasticity. In our study, however, CT-verified brain ischaemia was marginally not significant in the univariate analysis of our sample. The reason for this may be that CT is too crude a method to adequately quantify ischaemia, especially in the acute and subacute phase. Magnetic resonance imaging (MRI) would provide more reliable information, and should be used in further studies to identify risk factors in the acute phase for the development of spasticity.

Since the neurosurgical department in Uppsala admits all patients from a geographical uptake area, it is likely that selection bias is low. As there is no national guideline in Sweden for treatment of spasticity, patients were given standard care.

This study has several limitations. First, the spasticity assessment at inclusion was not considered reliable; a temporary reduction in the medications and removal of intubation would have enabled a more reliable assessment. This would have been of interest as it is not clear whether initial spasticity increases the risk of spasticity in the rehabilitation phase. Future research may assess spasticity already at the emergency unit, which may give a more reliable assessment than our assessment at the neurointensive care unit. Secondly, additional spasticity measurements would have added to the description of how spasticity in patients with aSAH changes over time. Furthermore, a larger sample size would have given a more reliable estimate of the prevalence of spasticity since the sample size in this study was based on estimates of spasticity after stroke other than aSAH. A multicentre set-up would have given a more representative picture of the prevalence of spasticity in aSAH patients regarding geographical differences, and intensive care differences and rehabilitation differences between centres. Also, the MAS assessment was performed by more than one person, which may have influenced the results, since the inter-rater and intra-rater reliability of the MAS has been discussed (27). Finally, physiotherapy provided to the patients was not studied. This would have been of interest, as the treatment of spasticity is primarily physical, and pharmacological treatment should be considered as a supplement (28).

In conclusion, spasticity occurred in 22% of patients with aSAH, which is in line with other studies on stroke. The predictors of spasticity were worse clinical condition at admission and the occurrence of infection and vasospasm during the intensive care period. Brain ischaemia might also be a risk factor; however, this should be investigated in future research in which MRI is used to quantify ischaemia. Pharmacological treatment of spasticity after aSAH seems to be rarely used in this sample.

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