The incidence of vasospasm after intracerebral haemorrhage

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ABSTRACT

Purpose: Cerebral vasospasm occurs frequently after aneurismal subarachnoid haemorrhage (SAH) and is a dangerous complication. Only a few cases of cerebral vasospasm after intracerebral haemorrhage (ICH) have been reported. To determine the incidence of vasospasm, the authors of this study evaluated the participants' digital subtraction angiographies (DSA) after these patients had experienced ICH.

Materials and methods: Sixty patients with ICH (26 women and 34 men between 20 and 69 years of age, mean age 49.6 years ± 13.9 SD) who underwent cerebral arteriography were included in this study. Cerebral vasospasm was graded as mild (up to 25% of vessel narrowing), moderate (26-50% of vessel narrowing), and severe (more than 50% of vessel narrowing).

Results: Vasospasm of the ipsilateral middle cerebral artery (MCA) to the ICH was found in 13 patients (21.6%), the ipsilateral anterior cerebral artery (ACA) in 4 patients, and the posterior cerebral artery (PCA) in one patient. Two patients had a spasm of the contralateral MCA. Severe MCA spasm was found in 3 patients, moderate in 5, and mild in 5. All cases of ACA and PCA spasms were assessed as mild.

Conclusions: Cerebral vasospasm is a rather frequent finding in patients who have just experienced ICH. Therefore, practitioners need to assess and monitor the status of the cerebral vasculature in these patients.

Key words: cerebral vasospasm, intracerebral hemorrhage, digital subtraction angiography.

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INTRODUCTION

Cerebral vasospasm contributes substantially to the poor outcome of patients after subarachnoid haemorrhage [1-3]. The development of vasospasm is a complex process in which various blood-derived components interact with the vascular wall [1,2,4]. Intracerebral haemorrhage (ICH) can also result in the contamination of cerebrospinal fluid with blood, which has the potential to affect the basal cerebral arteries and cause vasospasm [5-7].

The problem of vasospasm in patients with intraparenchymal haemorrhage, however, has been addressed in only few case reports and small case series [8-13]. Thus, the purpose of this study was to estimate the occurrence, severity and anatomical distribution of cerebral vessel narrowing in 60 patients who had experienced ICH.

MATERIALS AND METHODS

In this retrospective study, we analysed medical charts and imaging data of 60 consecutive patients in whom digital subtraction angiography of intracranial vessels (DSA) was performed after spontaneous supratentorial ICH. The patients were qualified for angiography in 3 different neurological wards and brought to this reference angiographic laboratory in order to detect a potentially operable source of intracerebral bleeding. There were 26 women and 34 men, 20 to 69 years of age.

The initial CT scans were reviewed independently by 2 neuroradiologists. The volume of intracerebral hematoma was measured using the AxBxC/2 method that has previously been described [14]. The amount of midline shift of the pineal gland was measured in millimeters. Significant subarachnoid hemorrhage was not observed on CT in any of the patients.

DSA performed with an Argos 2M angiography machine (Mecall S.R.L., Lissone, Italy) using Seldinger's method. The lumen of the middle (MCA), anterior (ACA) and posterior (PCA) cerebral arteries were assessed to a precision of 0.1 mm using electronic calipers on the digital display. The degree of narrowing by comparing the diameter of an affected artery to its pre-narrowed segment or to the diameter of the most distal segment of the parental vessel was determined [15]. Narrowing classified as mild when the vascular lumen was found to be decreased by 25% or less, moderate - when the decrease was greater than 25% to 50% and severe - when the vessel was narrowed by more than 50%. The diameter of the vessels was determined by two independent neuroradiologists, using previously published standards as a reference [15]. In cases of disagreement, a third neuroradiologist was asked to determine the status of the vessels.

Data relating to location of ICH, its volume and grading of pineal shift were recorded on a worksheet of the statistical package Systat 10 for Windows. Categorical variables were analysed with Chi-square test and *t*-test was used for continuous variables. A probability of less than 0.05 was considered statistically significant.

RESULTS

Table 1 shows characteristics of intra-cerebral haemorrhage in two groups of patients: those with diagnosed cerebral vasospasm and those with no vasospasm. No significant differences were found between all the parameters in these two groups, except the score on the Glasgow Coma Scale, which was 12 ± 2 for patients with vasospasm and 13.6 ±1.9 for the patients free of this condition ($t = 2.6$, $p < 0.05$).

Table 1. Initial computer tomography results

Characteristics	**Patients with cerebral vasospasm, (N = 13)**	**Patients without cerebral vasospasm, (N = 47)**
ICH volume (ml)		
Mean (SD)	12.4 (2,2)	11.3 (2.46)
Median	13 (9-16)	11 (8-18)
ICH location		
Deep cerebral	4 (30.8%)	21 (44.7%)
Lobar	9 (69.2%)	26 (55.3%)
Pineal shift (mm)		
None	9 (69.2%)	33 (70.2%)
1-5 mm	4 (30.8%)	14 (29.8%)
> 5mm	0	0
Mean (SD)	1.39 (1.94)	0.98 (1.66)
Median	0 (0-5)	0.0 (0-5)

The mean age of the patients with cerebral vessel narrowing was 48.8 ± 11.8 and did not differ significantly from those in whom the cerebral vessels were not affected ($t=1.4$, $p>0.05$). Cerebral vessel narrowing was found in seven out of 26 women and in six out of 34 men ($Chi^2 = 0.9$, $p>0.05$).

The DSA was usually delayed in patients who were initially in deep coma (GCS 3-8). As a consequence, the time from the insult to DSA varied from two to 26 days, average 9 ±8 days (SD). The median GCS score of the patients at the time of DSA examination was 14 (range 8 to 15). In 16 patients (26.7%), DSA revealed an aneurysm of the middle cerebral artery, in two patients (3.3%), bleeding was caused by an arterio-venous

malformation (AVM) and in the remaining 42 (70.0%), no vascular lesion was found.

Narrowing of at least one of the major cerebral arteries was found in 13 out of 60 patients (21.6%, seven women and six men). Narrowing predominantly affected the MCA on the side of bleeding (Table 2).

Table 2. Clinical characteristics of patients with angiographically diagnosed narrowing of cerebral arteries. Patient No 12 had a history of drug abuse (amphetamine)

Patient	Age	Sex	GCS score	Angiographic finding	Affected artery	Degree of vasospasm	GOS
1	57	F	15	No underlying cause	MCA	mild	5
2	46	M	13	No underlying cause	MCA	mild	5
3	69	M	14	No underlying cause	MCA	moderate	3
4	43	F	11	MCA aneurysm	MCA	moderate	4
5	64	F	11	No underlying cause	MCA	moderate	4
6	51	M	10	MCA aneurysm	MCA MCA# ACA	severe mild mild	4
7	35	M	11	No underlying cause	MCA ACA	severe mild	4
8	50	F	14	No underlying cause	MCA	mild	5
9	43	M	11	No underlying cause	MCA	moderate	4
10	45	F	11	AVM fed by MCA and PCA	MCA ACA	moderate mild	5
11	63	F	13	No underlying cause	MCA	mild	4
12	29	M	8	No underlying cause	MCA MCA# ACA ACA# PCA PCA#	severe moderate mild mild mild mild	4
13	39	F	14	No underlying cause	MCA	mild	5

MCA- middle cerebral artery, ACA- anterior cerebral artery, PCA – posterior cerebral artery, # - narrowing of an artery on the side contralateral to bleeding, GCS- Glasgow Coma Scale, F– female, M- male, AVM – arterio-venous malformation, GOS – Glasgow Outcome Scale.

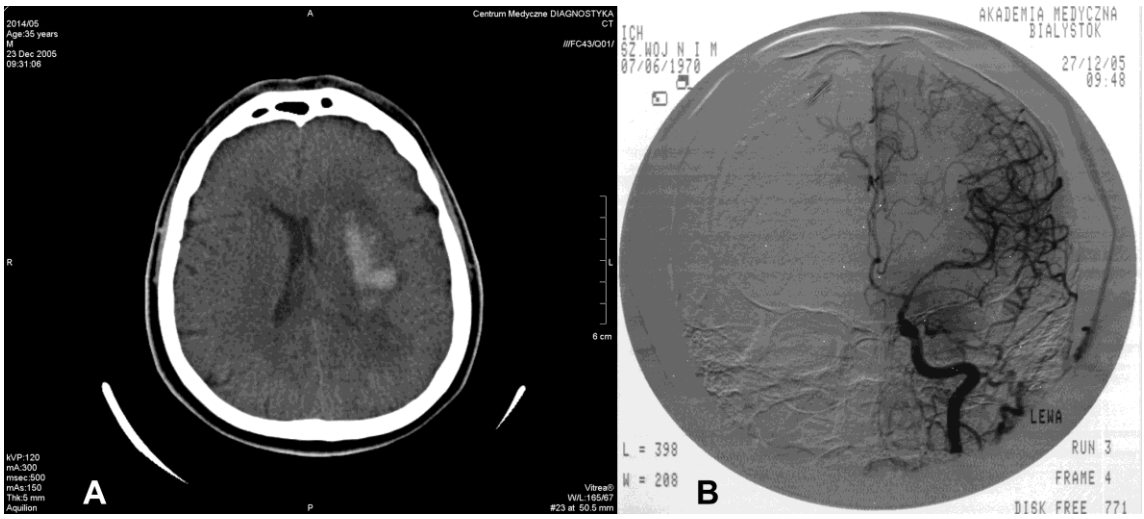

Figure 1. CT scan (a) and cerebral angiography (b) demonstrate spontaneous intracerebral hematoma in the basal ganglia of the left hemisphere and vasospasm of the left middle cerebral and anterior cerebral arteries in a 35 years - old male.

The ipsilateral ACA was affected in four patients, while the PCA was assessed as

pathologically narrowed in only one patient. Multi-segment narrowing was found in four patients. In one patient with a history of drug abuse (amphetamine), all the major cerebral arteries were very narrow and the transit time of the dye was delayed. Narrowing of the ACA and PCA was mild in all four patients.

Narrowing of the MCA was classified as mild in five patients, moderate in another five and as severe in three patients. All patients with diagnosed vasospasm were transferred to the ICU; normovolemia was established and colloids and nimodipine administered. In spite of this, in two patients (3 and 7) with MCA narrowing (one with severe and one with a moderate degree of vasospasm) a subsequent MRI showed hyper-intensive regions in the watershed areas of the MCA, consistent with a diagnosis of localized ischemia. The volume of intracerebral hematoma in these patients was 14 and 16 ml as calculated from the dimensions of blood collections evident on CT scans.

DISCUSSION

A MEDLINE database search indicated that more than 90% of publications related to "stroke" pertain to ischemic stroke and only 3% to ICH. Intracerebral haemorrhage is a particularly devastating type of stroke and little is still known about the sequence of pathologic events leading to secondary brain damage. Mass effect, the toxic influence of the extravasated blood and systemic complications are traditionally considered to be major contributors to poor outcome [6,7,16]. Nevertheless, in patients with ICH, blood comes into direct contact with the cerebrospinal fluid (CSF) and/or diffuses along the Virchow-Robin perivascular spaces, which according to most authors, remain in direct contact with the CSF [5, 12,17,18]. Hemoglobin degradation products released from lytic erythrocytes inhibit the endothelium–dependent relaxation of cerebral arteries and may play an important role in the development of vaso-constriction [3,18]. In our patients after ICH, cerebral vessel narrowing of varying severity was determined in as many as 21% of cases. This finding suggests that cerebral narrowing is a typical feature of ICH pathophysiology, though less prevalent than that following SAH [1,2].

Our estimate is based only on a single DSA examination performed at different time intervals from the ICH onset. Vasospasm usually develops seven to 14 days after SAH [1,2]. Based on these findings it may be extrapolated that the real incidence of vasospasm is underestimated in our study, as our patients were not monitored during the entire hospital stay. Furthermore, DSA was performed in patients who were in a better clinical condition, as angiography was undertaken mainly in order to diagnose potentially operable cerebral vascular malformations. It might be expected that a more systematic follow-up of the status of the cerebral vasculature would reveal an even higher incidence of narrowing of the cerebral arteries among patients with ICH. In most instances of ICH, cerebral vessel narrowing seems to be caused by spasm.

Nevertheless other causes should also be considered such as atherosclerosis and vessel hypoplasia. Differential diagnosis between the narrowing caused by atherosclerosis and vasospasm may be attempted on the basis of anatomical appearance. Hypoplasia most often involves the A1 and P1 segments. Making a distinction between vasospasm and hypoplasia in these segments requires a more dynamic monitoring of the status of the cerebral vasculature to determine changes over time. DSA is not an appropriate method for this purpose as it is an invasive procedure and carries a small risk of serious complications, including stroke. Nevertheless, given their current techno-logical refinement, MRA and CT angiography are ever increasingly used for the primary diagnosis of cerebral vascular malformations and their current fidelity is high enough to attempt differential diagnosis of major cerebral vessels narrowing [19, 20]. Recently, many reports have been published evidencing that transcranial color coded duplex sonography is a good alternative for diagnosing and monitoring cerebral vasospasm [15,21-23].

The occurrence of cerebral vasospasm in patients with spontaneous ICH is a problem of utmost clinical importance. Despite the intensive care extended to all our patients with disclosed cerebral vessels narrowing, two patients with MCA spasm developed brain ischemia in the territory supplied by this artery (one out of three patients with MCA narrowing classified as "severe").

The volume of hematoma was moderate in these patients and the foci of infarction were localized in the watershed area of the MCA, far from the intracerebral blood collection. From this it may be concluded that the brain ischemia was more likely to have been caused by MCA vasospasm and not by the "mass effect" of the hematoma itself.

CONCLUSIONS

This study suggests that cerebral vasospasm is a rather frequent finding in patients who have experienced ICH. Therefore, practitioners need to assess and monitor the status of the cerebral vasculature in these patients.

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Conflicts of interest

The authors have declared no conflicts of interest.

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