The clinical picture of cerebral vasospasm after subarachnoid haemorrhage (SAH)

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Introduction

It is now generally accepted that vasospasm is one of the major complications in cases with subarachnoid haemorrhage (SAH). Vasospasm is an angiographic diagnosis. Vasospasm should only be regarded to be present, when there is clear evidence of focal constriction of a cerebral vessel compared either with the same vessel at another point in its course, with other branches of the circle of Willis, or with the comparable vessel of the other side [17]. The narrowing of the cerebral arteries first produces neurological symptoms due to ischaemia when the lumen reduces to less than 50% of original caliber [14]. These focal neurological deficiences may be produced by the local type of vasospasm [1, 3, 8, 12], whereas diffuse general vasospasm is seen in patients with disturbed consciousness generally giving a grave prognosis [13, 16]. Fluctuating neurological deficit and fluctuating levels of consciousness may be potentially attributable to vasospasm, but also other mechanisms may be involved [18]. The following retrospective study was designed to evaluate the relationship of preoperative angiographic vasospasm and neurological findings. In addition, we attempt to determine if there is a typical clinical picture of vasospasm, which may allow the clinical assumption of its appearance.

Case material and methods

This paper is based on the analysis of 182 patients with spontaneous SAH, who were admitted to our clinic during the last three years. Patients were treated by standardized therapy (bedrest, analgetics, ε-aminocapronic acid, antiepileptics, dexamethasone and stool softening). Blood pressure was lowered by medication in those cases with extreme hypertension. Thirthy patients died before arteriography due to their poor condition. The remaining 152 patients underwent cerebral arteriography when clinically stable, independent of their neurological status. The arteriograms revealed the presence or absence of significant arteriographic vasospasm in one or more vessels. All patients underwent CT-scan examination and the evidence of an intracerebral haematoma or a cerebral infarction was noted.

At the time of arteriography and during the interval before aneurysm surgery, before death or recovery respectively, the neurological status of these patients was graded according to the level of alertness (alert, somnolent, soporose, comatose) and whether focal neurological deficits (including slight, moderate, severe hemiparesis and hemihypaesthesia) were present. Disturbances of ocular movements or the occurence of unequal pupils were not recorded. In addition, we divided patients with signs of brain stem impairment into 4 stages and patients with bulbar brain syndrome [5] into two stages including lateralizing signs [6]. Patients were classified into 4 categories – death, severe disability, moderate disability and good recovery [7]. The category severe disability includes patients in an apallic state [4].

Results

Patients with evidence of arteriographic vasospasm

The angiograms of cerebral vessel obtained preoperatively in our 152 patients within 3 weeks after SAH showed evidence of vasospasm in 40 (26%) cases. Vasospasms were accompanied by neurological focal signs in 26 (65%) cases. Vasospasm was identified angiographically on each day after bleeding including the day of SAH and the next 5 days. After the 5th day post SAH angiographic evidence of vasospasm has shown a more consistent relationship to focal neurological signs. The lowest relationship was seen on the first day after SAH (fig. 1). The CT-scan revealed intra-cerebral haematoma in 8 cases. The average age was 47 years varying from 20 to 71 years.

Patients with focal neurological deficit without evidence of angiographic vasospasm

51 patients (34%) of our cases (total number 152) demonstrated focal neurological signs, but showed no vasospasm in their arteriograms. Most of these arteriograms

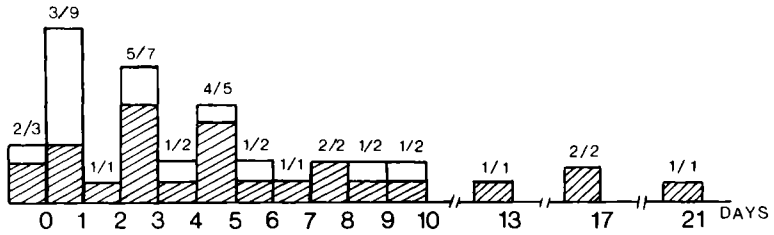

Fig. 1 The incidence of vasospasm per number of days from the attack of SAH until cerebral vessel angiography. The white columns indicate the number of angiograms with evidence of vasospasm, the dark columns demonstrate the focal neurological deficit found at the time of arteriography. Note the early appearance of vasospasm and the low relationship among vasospasm and focal neurological deficit on the first day after SAH.

were taken within the first 5 days after SAH. Neurological deficit was found more frequently during this time-interval in cases without angiographic evidence of vasospasm. However CT-scans carried out on the same day, or one day before or after arteriography, demonstrated an intracerebral haematoma in 17, cerebral infarction in 5 cases. After the 5th day neurological signs were more frequently seen in relation to angiographic vasospasm (fig. 2). The average age of this group was 51 years varying from 17–89 years.

Patients without neurological deficit and without angiographic vasospasm

Out of 152 cases 61 patients (40%) had neither focal neurological symptoms nor signs of vasospasm in the arteriogram. The average age was 49 years varying from 15–75 years. In these cases the CT-scan demonstrated an intra-cerebral haematoma in 3, cerebral infarction in 1 case.

Level of alertness

20 (50%) patients with angiographic vasospasm were alert. 14 patients (38%) were somnolent, 2 (5%) were soporose and 6 (15%) were comatose. In addition, symptoms of brain stem impairment (midbrain or bulbar brain syndrome) was found in 10 cases (25%). In cases with neurological deficit but without vasospasm 21 patients (41%) were classified to be alert, 17 (33%) to be somnolent, 4 (8%) to be soporose and 9 (18%) to be comatose. 8 patients (17%) were judged to show symptoms of the midbrain or bulbar brain syndrome. Patients without evidence of neurological focal deficit and angiographic vasospasm were alert in 50 (82%) cases. Somnolence was

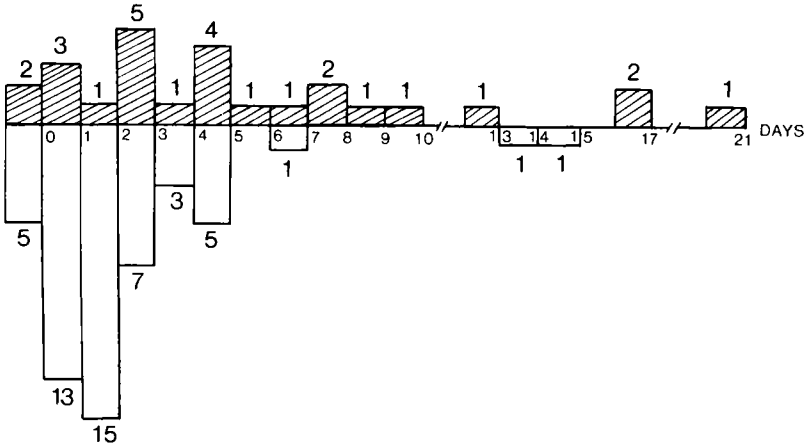

Fig. 2 White columns (downwards) demonstrate the incidence of focal neurological deficit in patients without angiographic vasospasm. Dark columns (upwards) show the incidence of focal neurological deficit in cases with vasospasm. Neurological deficit without angiographic vasospasm is seen more frequently during the first 5 days after SAH.

found in 9 (15%), sopor in none, and coma in 2 (3%) cases. Symptoms of central herniation were found in 4 (15%) cases. Except in cases without focal neurological and without angiographic vasospasm, there was no clear difference in the level of alertness in patients with focal neurological signs whether they had the angiographic finding of vasospasm or not (fig. 3).

Final results (fig. 4)

Out of 40 patients with angiographic vasospasm 20 (50%) had a good recovery, 3 (8%) remained moderately well, 4 (10%) severely disabled. 2 of the 4 patients classified as severly disabled developed an apallic syndrome. Both showed postoperatively severe generalized vasospasm in angiography. 13 patients (32%) died. Of 51 patients with focal neurological signs and without angiographic vasospasm 16 (32%) recovered well. 13 (25%) were moderately disabled and 6 (12%) were severely disabled. 2 of these patients were in an apallic state, 1 after severe post-

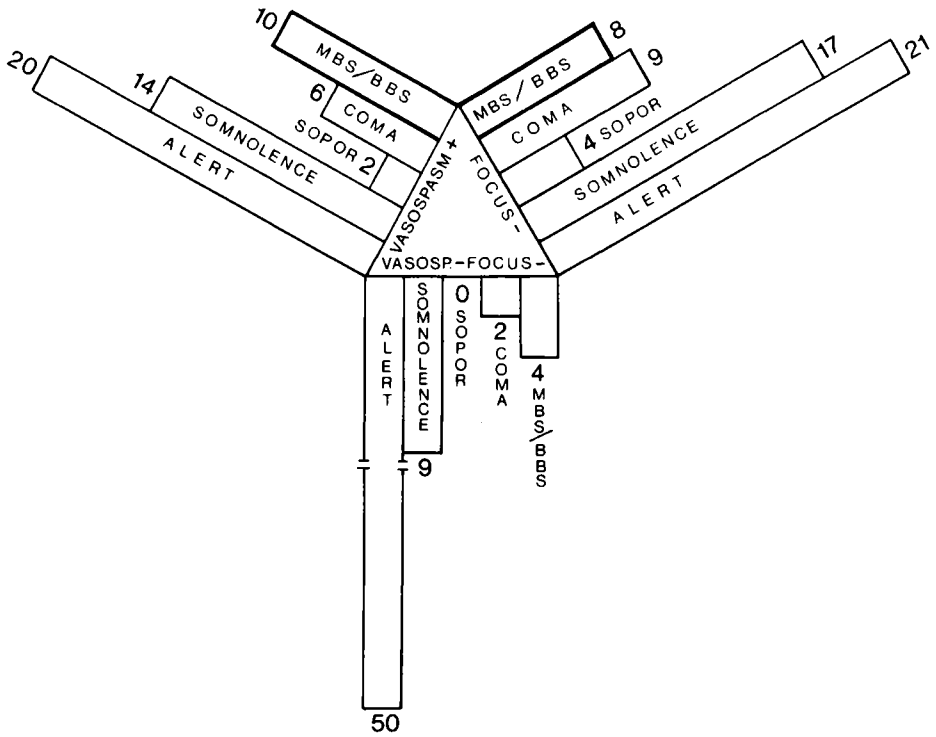

Fig. 3 Comparison of the levels of alertness in the different groups (patients with angiographic vasospasm, patients with focal neurological deficit without vasospasm, and patients without focal neurological deficit and without vasospasm). No clear difference in patients with focal neurological deficit alone compared to patients with angiographic vasospasm.

operative generalized vasospasm, the other developed this state pre-operatively without any signs of vasospasm in the arteriogram.

In contrast to these two groups 38 (62%) of the patients without neurological focus and without angiographic vasospasm recovered well. 9 (15%) showed moderate disability, 3 (5%) severe disability outcome and 11 (18%) died. 1 of the severely disabled patients had this outcome in form of an incomplete locked-in syndrome (re-bleeding, no operation), another remained appalic after severe post-operative generalized vasospasm.

The course of neurological symptoms (level of alertness, focal neurological deficit) between arteriography and surgery, death or recovery:

Patients with angiographic vasospasm: 14 patients showed no change of their symptoms, in 14 patients the severity of the neurological symptoms increased, in 8 patients a decrease of symptoms was noted. A fluctuating course (increase and decrease) was seen in 4 patients.

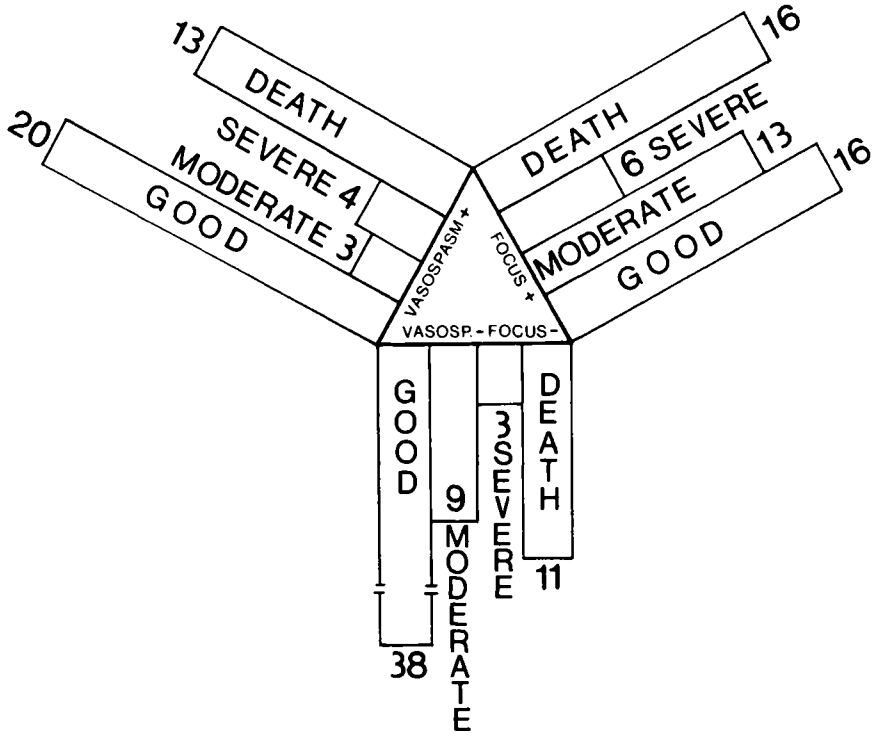

Fig. 4 Comparison of clinical outcome in the different groups (see Fig. 3). Good results were more frequently seen in patients with vasospasm (50%) and in patients without neurological deficit and without vasospasm (60%).

Patients with focal neurological deficit without angiographic vasospasm: 12 patients remained stable, 12 showed an increase, 21 a decrease of symptoms. A fluctuating course was seen in 6 patients.

Patients without angiographic vasospasm and without focal neurological deficit: 41 patients showed no significant change in alertness, 9 patients developed more severe symptoms, in 5 patients symptoms decreased. A fluctuating course was observed in 6 patients.

Incidence of aneurysms

35 aneurysms (multiple in 7 cases) were found in patients with angiographically demonstrated vasospasm. 29 were operated, 6 patients died before surgery. 38 aneurysms were detected in patients with focal neurological deficit without angiographic vasospasm. 29 patients were operated, 9 patients dies before operation. In patients without focal neurological deficit and without vasospasm there was angiographic evidence of 38 aneurysms was present (7 multiple). 33 patients were operated, 5 patients died before surgery. Most patients (91%) without angiographic evidence of an aneurysm in this group had a good recovery.

Discussion

There is no general agreement whether the relationship of vasospasm and development of cerebral infarction is a definite one. Schneck and Kricheff found that only 28% cases of cerebral infarction were related to spasm alone [15]. Millikan stated that 81 of his 198 patients with SAH had angiographic vasospasm, but only 45,7% showed abnormal neurological findings, whereas 52% of 117 patients without vasospasm had abnormal neurological signs [9]. Our findings confirm these previous reports. Focal neurological deficits and alterations of the level of consciousness were detectable in cases with angiographic vasospasm but also in cases without any evidence of vasospasm. 65% of our patients with vasospasm showed neurological focal deficits, but it occurred in 46% of our cases without angiographic vasospasm as well. The level of alertness was not clearly related to vasospasm. Similar changes in consciousness were seen in cases with focal neurological deficit without angiographic evidence of vasospasm. This may be especially true in patients with cerebral vessel angiography within the first 5 days after SAH. On the other hand delayed neurological signs may occur more frequently in the presence of vasospasm [2].

There was no relationship between the presence of angiographically demonstrated vasospasm in the pre-operative angiograms and the outcome. Both groups, patients with angiographic vasospasm and patients with focal neurological signs but without vasospasm had a similar outcome. This findings may support previous reports [9,

18] indicating a more suggestive relationship between pre-operative vasospasm and outcome. The fluctuating neurological signs, which were regarded as transient and reversible ischaemic neurological conditions especially seen after aneurysm surgery [18] were found pre-operatively in some of our cases and were not related to the presence of angiographic vasospasm. Other mechanisms such as changed vaso-reactivity, intravascular aggregation, microvascular occlusions may be important for these fluctuating symptoms [18]. The development of an apallic syndrome was a rare event. Most of them were seen after operation due to angiographically demonstrated severe generalized vasospasm. In contrast 2 of our patients with preoperative generalized vasospasm recovered well and never exhibited a clinical picture of an apallic syndrome. The observation of vasospasm in the first days after SAH is a rare event [8, 11] and in contrast to other reports, which generally deny these findings [10, 19]. The low incidence of focal neurological deficit in these cases may point to a transient diminution of the vascular caliber. Although there is a strong correlation between angiographic vasospasm and focal neurological deficit we have no evidence for a consistent clinical picture of vasospasm, which would allow a diagnosis of cerebral vasospasm by a clinical examination alone.

Summary

In a total of 152 patients with SAH, pre-operative cerebral vessel angiography was carried out. 40 patients demonstrated angiographic vasospasm. 65% of these patients showed focal neurological signs. In contrast also 46% of our patients without evidence of vasospasm in the arteriogram had focal neurological signs. Also the level of alertness was not clearly related to vasospasm, similar changes of alertness were seen in cases with focal neurological deficit without angiographic evidence of vasospasm. The outcome of patients was independent of the pre-operative vasospasm. Fluctuating neurological signs were present in 16 cases and were not related to vasospasm. An apallic syndrome was a rare outcome in our patients, most frequently seen after generalized post-operative vasospasm. Vasospasm was not a consistent sign of the clinical picture and can hardly be diagnosed by clinical examination alone.

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