Neuroradiology (1998) 40: 6–10

H. Urbach
J. Zentner
L. Solymosi

The need for repeat angiography in subarachnoid haemorrhage

Received: 14 March 1997
Accepted: 2 May 1997

H. Urbach (✉) · L. Solymosi
Department of Neuroradiology,
University of Bonn,
Sigmund-Freud-Strasse 25,
D-53127 Bonn, Germany
Tel. +49-228-287-6518;
fax +49-228-287-5598

J. Zentner
Department of Neurosurgery,
University of Bonn,
Sigmund-Freud-Strasse 25,
D-53127 Bonn, Germany

Abstract This study was designed to assess the necessity for a second angiogram study in patients in whom initial angiography after primary subarachnoid haemorrhage (SAH) was negative. During a 12-year period, 122 of 694 patients (17.5 %) had negative initial angiograms. CT, available for 98 patients, showed a preponderance of subarachnoid blood in the perimesencephalic cisterns in 50 of 73 patients (68.5 %) in whom blood was visible on CT. Angiography, repeated in 67 patients, revealed an aneurysm in 4 (6 %): 2 had an aneurysm of the anterior communicating artery, 1 of the posterior inferior cerebellar artery, and 1 of the P2 segment of the posterior cerebral artery. CT showed subarachnoid blood in the interpeduncular and ambient cisterns in this last case, and a preponderance of subarachnoid blood outside the perimesencephalic cisterns in the remaining 3 patients.

Key words Subarachnoid haemorrhage · Angiography · Computed tomography

Introduction

Even after detailed angiography subarachnoid haemorrhage (SAH) of unknown origin represents a significant proportion of cases of verified subarachnoid bleeding; figures given mainly range between 15 % and 20 % [1–5]. In these patients CT often reveals a preponderance of subarachnoid blood in the perimesencephalic cisterns [6–9]. Our aim in this retrospective study was to evaluate the results of a second angiogram and to correlate the findings with the distribution of subarachnoid blood on the initial CT studies.

Patients and methods

From January 1983 to February 1995 a total of 694 patients were admitted to our department of neurosurgery with an SAH verified by CT and/or lumbar puncture. Patients with head injuries, tumours, infections, or intracranial haematomas and those on anticoagulant therapy were excluded. After four-vessel angiography there remained 122 patients in whom no cause for SAH was detected. CT studies, available for 98 patients, were reviewed by a neuroradiologist with regard to the maximum subarachnoid blood. The following sites were defined: perimesencephalic (pontine, interpeduncular and ambient cisterns), quadrigeminal cistern, chiasmatic cistern, sylvian fissure, frontal interhemispheric fissure, and diffuse. All 122 patients were advised to have a second angiogram, generally 6 weeks after the initial study. However, because many patients were referred from distant cities, angiography was repeated in only 67 patients. The initial and repeat angiograms were reviewed by a neuroradiologist and correlated with the distribution of blood on the initial CT.

Results

There were 74 men (61 %) and 48 women (39 %), aged 16 to 76 years (mean 51 years). CT was performed in 78 of 98 cases in the first 3 days after the haemorrhage, and in 20 patients thereafter. Of these 98 patients 73 had subarachnoid blood on CT, the site of which is shown in Table 1.

Table 1 Preponderance of subarachnoid blood on CT in 73 patients with negative initial angiogram

Perimesencephalic cisterns[a]	50	(68 %)
Isolated	17	
Combined with other sites	33	
Quadrigeminal cistern	2	(3 %)
Suprasellar cistern	1	(1 %)
Sylvian fissure	2	(3 %)
Frontal interhemispheric fissure	3	(4 %)
Diffuse	15	(21 %)

[a] Pontine, interpeduncular and ambient cisterns

Repeat angiography, performed in 67 patients, revealed an aneurysm in 4 (6 %). Two patients with SAH of Hunt and Hess grade III had initial CT with diffuse blood in the basal cisterns and maximal blood in the frontal interhemispheric fissure, respectively. As the first angiograms did not show the anterior communicating artery (ACoA) clearly, the studies were repeated within 10 days, and in each case an ACoA aneurysm was visible. In one patient with grade III SAH and diffuse blood, two cut-film angiograms were negative. After an episode of rebleeding 3 years later, digital subtraction angiography revealed a small aneurysm of the left posterior inferior cerebellar artery (Fig. 1). We assume that this aneurysm would have been visible on earlier repeat angiography. One patient had a grade III SAH with blood in the interpeduncular and ambient cisterns on the left. The first angiogram was negative. CT and MRI 6 weeks later showed a dense high signal structure in the interpeduncular cistern on the left, and a second angiogram revealed a small aneurysm on the P2 segment of the left posterior cerebral artery (Fig. 2).

Discussion

The prognosis of aneurysmal SAH markedly differs from that of nonaneurysmal SAH [10–13]. Approximately half of patients with a ruptured aneurysm present grades III–V on admission [10, 14]. Unoperated ruptured aneurysms carry a high risk of rebleeding, estimated as 20–50 % during the first 2 weeks, with a total mortality of approximately 50 % during the first 6 months after haemorrhage. The overall morbidity is high; only a third of patients are reported as having a good physical and psychological outcome [10]. Patients with SAH of unknown origin, on the other hand, are generally in good condition, approximately 90 % presenting in H & H grades I and II [9, 14–17]. The incidence of rebleeding is low [8, 18–20], and about 80 % recover to their previous functional capacity [2, 17]. A frequent finding in SAH of unknown origin is a relatively small amount of blood visible on CT [6–8]. In addition, the distribution of blood is remarkable. Up to two thirds of patients with SAH and a negative angiogram are reported to show a preponderance of blood in the perimesencephalic cisterns [3, 7, 9, 17]. This contrasts with aneurysmal SAH, in which haemorrhage suggesting an origin from a posterior circulation aneurysm were found in only approximately 10 % of patients [9]. In our series subarachnoid blood was visible on CT in 73 % of patients with SAH of unknown origin, 71 % of whom showed maximal blood in the perimesencephalic cisterns.

When we have a patient with SAH and negative four-vessel angiography, the question arises of whether the angiogram is falsely negative. This raises the question of whether a second angiographic study is necessary. The rate of false-negative angiograms is not clear: in most series it is less than 2 % [4, 8, 21, 22], and some groups did not encounter any false-negative studies [9, 14, 19, 21, 23–25]. Ruptured cerebral aneurysms may not be detected because of spontaneous thrombosis, destruction of the aneurysm by haemorrhage, or inadequate angiographic technique; it is also claimed that arterial spasm may disguise an aneurysm [26]. Dissecting aneurysm of the intracranial vertebral or basilar artery, known to present with SAH [27] in up to 4.5 % of cases [28] may resemble spasm; most angiograms in this condition show a fusiform dilatation of the vertebral or basilar artery with irregular proximal and/or distal narrowing. It is suggested that angiography be repeated whenever features such as spasms, haematoma or brain oedema is present. However, in our series 8 % of patients showed distinct spasm on the initial angiogram, all with maximal blood outside the perimesencephalic cisterns: angiography, repeated in these cases, showed that the spasm had subsided, but again, no aneurysm was found.

In the four patients in whom an aneurysm was found on repeat angiography, we have to focus on different aspects. In three patients the angiogram was suboptimal. In the two patients with ACoA aneurysms the first angiogram did not show the ACoA complex clearly, and in one other patient the aneurysm was probably not found due to a technical problem. With the use of digital subtraction angiography nowadays superimposition of bony structures is less problematic.

While the distribution of blood was strongly suggestive for a ruptured aneurysm in these three patients, the diagnosis of SAH of unknown origin would not have been uncommon in the patient with perimesencephalic bleeding. As we showed, however, perimesencephalic subarachnoid blood is compatible with an aneurysm on the P2 segment. Thus, although in many cases perimesencephalic haemorrhage corresponds with a normal angiogram, this bleeding pattern does occur in patients with a ruptured aneurysm of the posterior circulation. Rinkel et al. [9], who investigated 37 patients showing a preponderance of blood in the posterior basal cisterns (15 patients with a posterior circulation aneurysm and

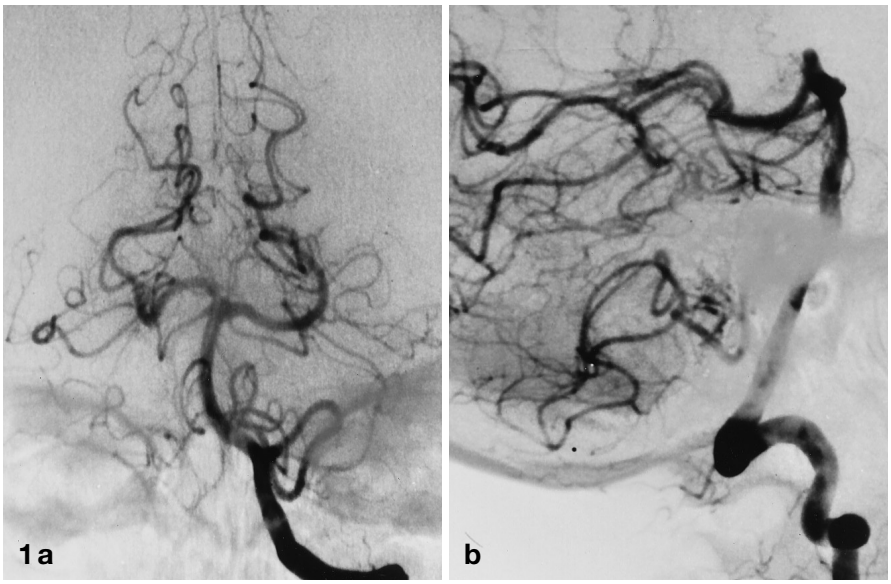


Fig. 1 Conventional left vertebral arteriogram in Towne's (**a**) and lateral (**b**) projections. No aneurysm is visible, but PICA origin is not visible in the lateral projection due to superimposition of the petrous bones. Digital subtraction angiogram in Towne's (**c**) and lateral (**d**) projections 3 years later after a further episode of haemorrhage. A small aneurysm at the PICA origin is visible in both projections. There is diffuse arterial spasm

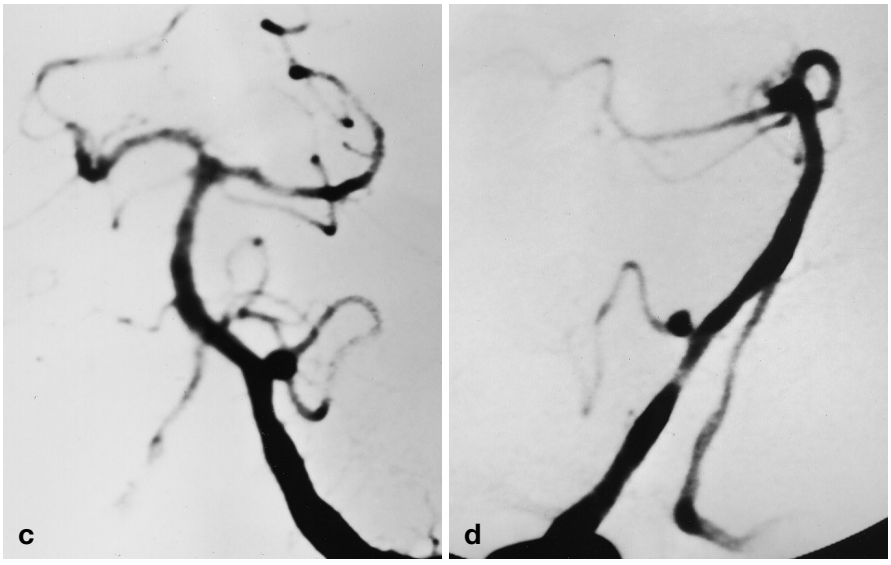
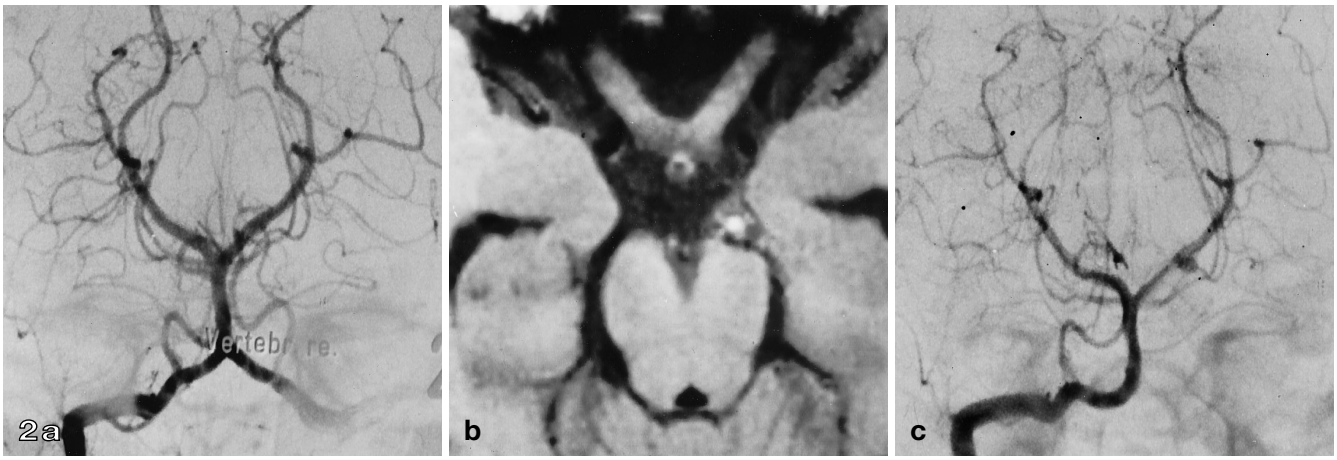


Fig. 2 Conventional right vertebral arteriogram in Towne's projection (**a**). No aneurysm is visible. Axial MRI 6 weeks later (**b**) shows a high-signal structure surrounded by a mass of intermediate signal in the left side of the interpeduncular cistern. A second angiogram (**c**) reveals a small aneurysm on the left posterior cerebral artery



22 with a normal angiogram), reported one basilar artery aneurysm with perimesencephalic bleeding. Kaim et al. [17] encountered in a series of 76 ruptured aneurysms one posterior communicating artery aneurysm with perimesencephalic bleeding. As in our series, this was detected only on a second angiogram.

Recent prospective studies concerning the complication rate of cerebral angiography revealed permanent neurological deficits in 0.5 % [29], 0.09 % [30] and 0.1 % [31]. About two thirds of these complications occurred in patients undergoing angiography because of ischaemic cerebrovascular disease [30, 31]. The rate of neurological complications nowadays is much lower than in earlier reports [32]. Dion et al. [31] and Mani and Eisenberg [32] showed that the duration of an angiogram increases the complication rate. Thus, the lower complication rate nowadays may in part be explained by shortening of the procedure with the use of digital subtraction angiography [30].

Changing our strategy of a repeat angiogram in every patient with negative initial angiogram, we would not have found a ruptured aneurysm in approximately 4.5 % of all patients and in 2 % of those with perimesencephalic bleeding. This figure would have been much lower if technically satisfactory investigations with digital subtraction angiography had been carried out initially. With an estimated mortality rate of unoperated ruptured aneurysm of about 50 % during the first 6 months after haemorrhage, omitting a second angiogram could have led to mortality rates of 2 % and 1 %, respectively, although one of the four patients survived 3 years before bleeding again. As these numbers seem to outweigh reported complication rates of cerebral angiography we recommend a second angiogram in every patient with primary SAH and a negative initial angiogram (particularly if the latter is not technically irreproachable) including these with perimesencephalic bleeding.

References

1. Alexander M, Dias P, Uttley D (1986) Spontaneous subarachnoid hemorrhage and negative cerebral panangiography. J Neurosurg 64: 537–542
2. Eskesen O, Sorensen EB, Rosenorn J, Schmidt K (1984) The prognosis in subarachnoid hemorrhage of unknown etiology. J Neurosurg 61: 1029–1031
3. Van Gijn J, van Dongen KJ, Vermeulen M, Hijdra A (1985) Perimesencephalic hemorrhage: a nonaneurysmal and benign form of subarachnoid hemorrhage. Neurology 35: 493–497
4. Juul R, Torbjorn A, Fredriksen TA, Ringkjob R (1986) Prognosis in subarachnoid hemorrhage of unknown etiology. J Neurosurg 64: 359–362
5. Kassell NF, Torner JC, Jane JA, Haley EC, Adams HP (1990) The International Cooperative Study on the Timing of Aneurysm Surgery. J Neurosurg 73: 37–47
6. Barlow P (1985) Incidence of delayed cerebral ischaemia following subarachnoid haemorrhage of unknown cause. J Neurol Neurosurg Psychiatry 48: 132–136
7. Ferbert A, Hubo I, Biniek R (1992) Non-traumatic subarachnoid hemorrhage with normal angiogram. Long-term follow up and CT predictors of complications. J Neurol Sci 107: 14–18
8. Giombini S, Bruzzone MG, Pluchino F (1988) Subarachnoid hemorrhage of unexplained cause. Neurosurgery 22: 313–316
9. Rinkel GJ, Wijdicks E, Vermeulen M, Ramos L, Tanghe H, Hasan D, Meiners L, van Gijn J (1991) Nonaneurysmal perimesencephalic subarachnoid hemorrhage: CT and MR patterns that differ from aneurysmal rupture. AJNR 12: 829–834
10. Säveland H, Sonesson B, Ljunggren B, Brandt L, Uski T, Zygmunt S, Hindtfeldt B (1986) Outcome evaluation following subarachnoid hemorrhage. J Neurosurg 64: 191–196
11. Sahs AI, Nibbelink DW, Torner JC (1981) Aneurysmal subarachnoid hemorrhage: report of the Cooperative Study. Urban & Schwarzenberg, Baltimore, pp 47–48
12. Sundt TM, Whisnant JP (1978) Subarachnoid hemorrhage from intracranial aneurysm: surgical management and natural history of disease. N Engl J Med 299: 116–122
13. Vilkki J, Holst P, Öhman J, Servo A, Hiskanen O (1990) Social outcome related to cognitive performance and computed tomographic findings after surgery for a ruptured intracranial aneurysm. Neurosurgery 4: 579–585
14. Cioffi F, Pasqualin A, Cavazzani P, Da-Pian R (1989) Subarachnoid hemorrhage of unknown origin: clinical and tomographical aspects. Acta Neurochir 97: 31–39
15. Hawkins TD, Sims C, Hanka R (1989) Subarachnoid haemorrhage of unknown cause: a long term follow-up. J Neurol Neurosurg Psychiatry 52: 230–235
16. Hayward RD (1977) Subarachnoid haemorrhage of unknown aetiology. J Neurol Neurosurg Psychiatry 40: 926–931
17. Kaim A, Mader I, Kirsch E, Radü EW, Steinbrich W (1995) Die perimesencephale Subarachnoidalblutung: klinische und computertomographische Aspekte. Fortschr Röntgenstr 162: 274–281
18. Andrioli GC, Salar G, Rigobello L, Mingrino S (1979) Subarachnoid hemorrhage of unknown etiology. Acta Neurochir 48: 217–221
19. Jain V, Hedge T, Easwaran R, Das B, Reddy G (1987) Benign subarachnoid haemorrhage of unknown aetiology. Acta Neurochir 86: 89–92
20. Kawamura S, Yasui N (1990) Clinical and long-term follow-up study in patients with spontaneous subarachnoid haemorrhage of unknown aetiology. Acta Neurochir 106: 110–114
21. Forster DMC, Steiner L, Hakason S, Bergvall U (1978) The value of repeat panangiography in cases of unexplained subarachnoid hemorrhage. J Neurosurg 48: 712–716
22. Sundbärg G, Brismar J, Ljunggren B (1982) Subarachnoid haemorrhage of unknown origin. A good-natured catastrophe. Acta Neurochir 66: 251
23. Beguelin C, Seiler R (1983) Subarachnoid hemorrhage with normal cerebral panangiography. Neurosurgery 13: 409–411

24. Brismar J, Sundbärg G (1985) Subarachnoid hemorrhage: epidemiology, diagnosis, management and outcome. Stroke 4: 591–594
25. Ishii R, Kuroki M, Tanaka R (1983) Subarachnoid hemorrhage of unknown cause: clinical study. Neurol Med Chir 23: 262–266
26. Suzuki S, Kayama T, Sakurai Y, Ogawa A, Suzuki J (1987) Subarachnoid hemorrhage of unknown cause. Neurosurgery 21: 310–313
27. Endo S, Nishima M, Nomura H, Takaku A, Okada E (1993) A pathological study of intracranial posterior circulation dissecting aneurysm with subarachnoid hemorrhage: report of three autopsied cases and review of the literature. Neurosurgery 33: 732–738
28. Sasaki O, Ogawa H, Koike T, Koizumi T, Tanaka R (1991) A clinicopathological study of dissecting aneurysm of the intracranial vertebral artery. J Neurosurg 75: 874–882
29. Heiserman JE, Dean BL, Hodak JA, Flom RA, Bird CJ, Drayer BP, Fram EK (1994) Neurological complications of cerebral angiography. AJNR 15: 1401–1407
30. Grzyska U, Freitag J, Zeumer H (1990) Selective cerebral intraarterial DSA. Complication rate and control of risk factors. Neuroradiology 32: 296–299
31. Dion JE, Gates PC, Fox AJ, Barnett HJM, Blom RJ (1987) Clinical events following neuroangiography: a prospective study. Stroke 18: 997–1004
32. Mani RL, Eisenberg RL (1978) Complications of cerebral arteriography: analysis of 5000 procedures. AJR 131: 867–869