Rupture of intracranial vascular lesions during arteriography

SA TSEMENTZIS, RP KENNETT, ER HITCHCOCK

From the Department of Neurosurgery, University of Birmingham, England, UK

SUMMARY Four cases with ruptured intracranial vascular lesions during angiography and confirmed radiologically are presented to emphasise a dangerous complication of the procedure.

Angiography of the cerebral vessels was first performed by Moniz in 1927.¹ Direct percutaneous puncture of arteries in the neck was introduced by Loman and Myerson in 1936² and since that time the procedure has been performed in many centres with a low incidence of complications. For instance Niozuma³ reported the incidence of hemiplegia as 0.72%, aphasia 0.4% and visual impairment 0.01% and Coddon⁴ reported permanent non-fatal complications in 0.8% of angiographies. Minor local complications may occur in 6.7%.

Thirty one cases of rupture of intracranial aneurysms during angiography have been reported.⁵⁻³⁵ The outcome of most of these has been catastrophic. We present three further cases of radiographically confirmed spontaneous haemorrhage from intracranial arterial aneurysms to re-emphasise the danger of this potential complication. We also present a case of haemorrhage from an arterio-venous malformation during arteriography which has not been previously described.

Case reports

Case 1

This 21-year-old right handed man was admitted on 29 April 1983, 2 months after the sudden onset of occipital headache after jumping into a shallow pit. He was initially thought to be suffering from "muscle spasm" and was treated conservatively. However, the pain remained present for 7 weeks when, during an exacerbation of the pain, he developed diplopia and vomiting. Examination at a peripheral general hospital on 22 April revealed a right oculomotor nerve palsy.

On transfer the positive physical findings were marked

Address for reprint requests: SA Tsementzis, Midland Centre for Neurosurgery and Neurology, Holly Lane, Smethwick, Warley, West Midlands, B67 7JX, UK.

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neck stiffness, bilateral Kernig's sign and a complete right oculomotor nerve palsy. The blood pressure was 120/85 mmHg. Angiography was performed on 2 May by percutaneous carotid arterial puncture. Lateral views of the cranial contents revealed a multi-loculated aneurysm of the right internal carotid artery with spasm of this vessel and the middle cerebral artery above. There was evidence of contrast material in the Sylvian fissure on the next control film and slow injection of contrast confirmed the rupture.

Immediately after the procedure the patient developed stridor and a decorticate posture. Despite intravenous dexamethasone the left pupil became dilated, respiratory arrest ensued and the patient died.

Post-mortem examination of the brain showed the dura to be moderately tense and the gyri flattened, especially on the right. A small area of subarachnoid haemorrhage at the tip of the temporal lobe was in continuity with a larger clot in the basal cisterns. There was a medium sized multi-loculated aneurysm at the junction of the right internal carotid and posterior communicating arteries with displacement of the right third cranial nerve. There was well marked tonsillar herniation and a medullary pressure cone into the foramen magnum.

Case 2

This 62-year-old right handed lady was transferred from a general hospital on the 27 May 1982, 12 days after the onset of severe occipital headache, nausea and vomiting. There had been no loss of consciousness.

On arrival she was drowsy and confused but able to recall correctly her age, date of birth, name of hospital and name of current monarch. There was severe neck stiffness but no other neurological signs.

Cerebral angiography was performed the next day (that is 13 days after the ictus) by direct carotid arterial puncture. This demonstrated a right posterior communicating arterial aneurysm projecting laterally. However, between taking the lateral and antero-posterior films there was evidence of rupture of the aneurysm with considerable right middle cerebral arterial spasm (fig 1). The pupils became large and unreactive. There was no response to noxious stimuli. The blood pressure rose to 220/140 mmHg. Endotracheal intubation and positive pressure ventilation

Fig 1

were required but the patient developed ventricular fibrillation and died. Post-mortem examination was not performed.

Case 3

This 56-year-old right handed lady was admitted to a district general hospital on the 27 May 1983 with a confusional state. Two days later she suffered a grand mal seizure and on the next a mild left hemiparesis was noted. There was no history of headache, photophobia or neck stiffness. Examination of the CSF on 31 May revealed xanthochromic fluid.

She was transferred to this centre on 4 June 1983 when the positive findings were mild neck stiffness, bilaterally positive Kernig's sign and marked external ophthalmoplegia. She was disorientated in time and place. A CT scan demonstrated a left sided frontal low density area but no subarachnoid blood.

Arch aortography was performed on 14 June. This demonstrated an anterior communicating arterial aneurysm pointing upwards and to the left. During the right carotid injection contrast was seen to move slowly up the artery and clinically at this stage she developed a complicated cardiac dysrhythmia. The angiogram showed marked

spasm of the right anterior cerebral artery. At the end of the procedure she was found to be comatose. The pupils were large and unreactive to light. She required artificial ventilation for 2 days before she died. A CT scan performed 4 hours after angiography demonstrated lateral ventricles filled with blood and contrast material. A post-mortem examination was not performed.

Case 4

This 9-year-old right handed boy was admitted 8 days after the onset of severe frontal headache. There was no photophobia or localising neurological symptoms.

On admission (19 August 1976) he was found to be alert, with normal higher nervous function and without focal neurological signs. There was moderate neck stiffness. The blood pressure was 130/70 mmHg. Examination of the CSF revealed diffusely blood stained fluid with supernatant xanthochromia.

Angiography was performed on the 25 August, 14 days after the onset of headache, via a femoral catheter. A small angioma was demonstrated lying in the lateral wall of the left lateral ventricle. The blood supply was from three branches of the middle cerebral artery. At the end of the procedure contrast material was seen to enter the left lateral ventricle and it was evident on two control films. The second of these, 5 minutes after the last injection of contrast, shows medium faintly in both lateral ventricles (figs 2, 3).

There was no neurological deterioration and the blood pressure remained stable. Craniotomy for excision of the angioma was performed on 2 September and the patient made a full and uneventful recovery.

Fig 2

Discussion

We noted in the introduction that the incidence of complications following cerebral angiography is low.

Fig 3

If we now consider the incidence of rebleed we find that the reported incidence is also low. For instance, Niozuma³ had one haemorrhage during angiography in 939 patients with saccular aneurysms (0.106%) and only one patient in the USA co-operative study of 5,484 suffered a rebleed during angiography (that is, an incidence of about 0.02%).²⁵ However, we have demonstrated four cases of rebleed radiologically since 1976 during which time 840 cerebral arteriograms for subarachnoid haemorrhage have been performed giving an incidence for this complication of 0.48%.

It would seem that there are three possible reasons for occurrence of repeated subarachnoid haemorrhage during cerebral arteriography.

First, coincidence may play a role. In 15 of the previously reported cases of rebleed during angiography the procedure was carried out within 2 days of the initial ictal event and it is impossible to know whether these represent a further haemorrhage or a continuation of the first. Of the remaining reported cases only two^{14,34} associated rebleeding with angiography in the period 7 to 10 days after the initial ictus which is recognised to be the time of peak incidence of spontaneous rupture,³⁵ that is the time when coincidence should have the maximum effect.

The second possibility is that the angiography itself is responsible for the rehaemorrhage. Lin *et al*¹⁴ observed a rise in the local brachial artery pressure transmitted to the carotid arteries distal to an injection of the contrast material. There is, however, no evidence that this rise in intraluminal pressure is transmitted to the aneurysmal sac to cause a thin wall to rupture or a clot to disperse.

Thirdly, a rise in arterial pressure during the induction of anaesthesia in our cases may have been responsible for the re-rupture, but again the correlation between peripheral hypertension and intracranial events is only conjectural.

The outcome of rebleed during angiography in the previously reported and our cases has been disastrous. Twenty-three of 34 cases (68%) died, five had residual neurological defects (four hemiplegia, one oculomotor nerve palsy) and only three (8.8%) remained neurologically intact.

The relative infrequency with which intracranial angiomas cause subarachnoid haemorrhage (in Richardson's series of subarachnoid haemorrhages 4.7% were caused by angiomas whereas 51.6% were caused by intracranial aneurysms)³⁸ may explain why the rupture of these lesions during angiography had not until now been described. Whether the favourable outcome of the patient with the ruptured angioma as compared to the other three aneurysmal cases who all died, reflects a general analogy between the two types of lesion under these circumstances, remains to be seen.

At the moment cerebral arteriography is the only method of accurate localisation of intracranial arterial aneurysms without which surgery cannot proceed and therefore its use will continue despite the potential complications. However, in the future investigations such as digital vascular imaging may prove to be safer yet provide the accurate information about aneurysms that is required before neurosurgery can be contemplated.

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