

Giant aneurysm causing frontal lobe syndrome

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Summary. A 67-year-old patient with a completely thrombosed giant aneurysm of the anterior communicating artery is described. The only neurological findings were a disturbance of stance and gait and slight bilateral grasp reflexes. He had had the features of a frontal lobe psychotic syndrome for more than 12 years. Computed tomography demonstrated a primarily hyperdense calcified suprasellar space-occupying lesion. Magnetic resonance imaging proved valuable in establishing the correct diagnosis, which was confirmed post mortem.

Key words: Giant aneurysm – Frontal lobe syndrome – Cranial computed tomography – Magnetic resonance imaging

Introduction

Intracranial giant aneurysms (GIAs) are defined as aneurysms measuring more than 2.5 cm in diameter [8]. Their overall frequency has been cited as approximately 5–13% of all intracranial aneurysms [10]. They constitute a special group which do not usually cause subarachnoid haemorrhage (SAH). Instead, they are commonly detected as slow-growing, space-occupying mass lesions [4]. Although cranial computed tomography (CT) facilitates early diagnosis, there may still be confusion with other cerebral mass lesions, such as meningioma, craniopharyngioma or pituitary adenoma [5]. Angiography will usually establish the correct diagnosis, but it can fail when there is complete thrombosis in the aneurysm, which occurs in 13–20% of cases [12]. The introduction of magnetic resonance imaging (MRI), which uses signal differences resulting from blood flow within the lesion, has proven to be more specific in detecting a GIA [2, 3, 11].

We present here a case of an unusually large completely thrombosed giant aneurysm of the anterior communicating artery (ACoA). For more than 12 years the 67-year-old patient suffered from a progressive frontal lobe syndrome without any focal neurological defects. The clinical presentation and radiological features of this unusual case, including angiography, CT and MRI and post-mortem findings, are discussed with reference to the current literature.

Case report

A 67-year-old Caucasian male was transferred to the hospital for surveillance after acute head trauma. The history was

mainly contributed by his wife: She had noticed a personality change in her husband throughout the last 12 years with increasing deteriorating in the last 3 years. Furthermore, her husband had developed a progressive loss of memory involving especially short-term memory. Spontaneous movements and speech had been markedly reduced and he had recently lost control of bowel and bladder function. On higher mental function examination the patient was alert, but responded slowly to questioning and simple commands, occasionally incorrectly. He appeared disoriented, particularly with respect to time and place. Naming, spelling and repetition were all intact, but verbal communication was markedly reduced. He was unable to remember three test objects after 5 min. The patient displayed little spontaneous behaviour and had to be strongly prompted to produce a reaction to our commands. He showed no change in affect, but also no disinhibition of behavior.

Examination of the skull revealed tenderness over the right temporal region. The patient had a right-sided periorbital hematoma. No nuchal rigidity was present. Pupillary responses and extraocular movements were intact, without nystagmus. The optic disks were flat with sharply defined margins; the fundi appeared to be pale. The other cranial nerves were normal. Tendon reflexes were symmetrical; no pathological reflexes were present. There was no abnormality of muscle bulk, tone of power, and sensation was normal to light touch, pinprick and vibration. Slight grasp reflexes of both hands were present. The patient was unable to stand upright without help. His steps were shortened to a shuffle, and he was not capable of walking.

The electroencephalogram showed a normal alpha rhythm without focal slow activity or epileptic discharges. On the plain skull radiographs the sharply defined line of a right temporal fracture and in addition, a paramedial curvilinear calcification in the suprasellar region were seen. There were no signs of raised intracranial pressure.

The unenhanced CT scan showed a hyperdense calcified suprasellar mass of 7.5 cm in diameter in the anterior fossa. There was minimal surrounding brain oedema and compression of the anterior horns of the lateral ventricles. Intravenous contrast medium gave no further enhancement. Intra-arterial cerebral angiography revealed a normal pattern of internal and external carotid arteries. Both anterior cerebral arteries were depressed and displaced to either side. Anteriorly the pericallosal artery curved laterally and appeared semicircular before returning to the midline at its junction with the posterior internal frontal artery (posteromedial frontal artery) (lateral view not shown). A small deposit of contrast medium

could be seen at the base of the aneurysm, there marking the former connection with the parent vessel. The capillary and the venous phase showed a central avascular zone and posterior displacement of the venous angle.

On T1-weighted MR images medium signal intensity was found throughout the whole area representing the thrombosed aneurysm without demonstrating a patent lumen. In contrast to these, T2-weighted images displayed hypointensities inside the capsule of the aneurysm representing deposits of haemosiderin and thrombus calcification.

The patient died of intercurrent pneumonia before a planned neurosurgical operation could be performed. Important post-mortem findings included a giant aneurysm of the ACoA with a maximum diameter of 7.5 cm and a total volume of more than 169 ml. It contained multiple layers of calcified thrombotic material without any residual lumen. No further neuropathological findings were made.

Discussion

GIA's are rare. They are found more commonly in the posterior cerebral circulation [4]. In one large series only 3 of 21 GIA's were located on the ACoA [14]. The most common presentation is that of an intracranial space-occupying lesion: headache, ocular pain and focal neurological signs involving the cranial nerves, particularly nerve VIII, followed by V, VI and IX in order of frequency [9]. GIA's of the anterior cerebral artery (ACA) and ACoA usually cause visual problems, especially bilateral hemianopia. A patient presenting with decreased intelligence as the only neurological abnormality has been documented [14]. In about 70% of cases, the duration of illness spans more than a year [13] with gradual progression of symptoms. A history of more than 12 years, as in the case discussed here, can be regarded as the upper end of the scale. The patient's characteristic disturbance of stance and gait has been described with bilateral frontal lesions [1, 6]. An inervatory apraxia for walking may be related to the magnetic syndrome seen in patients with frontal lobe affections [1]. The term describes a fixation on objects, which prevents the patient from shifting his centre of attention or activity. The patient's feet remain fixed to the ground when he tries to walk.

The prognosis of GIA's remains poor. The incidence of SAH for GIA's is usually around 30% [14]. Furthermore, ischaemic cerebral complications can result from compression due to the GIA itself or due to emboli emanating from the aneurysm. Therefore, surgical treatment or embolization are preferable even though the success rates are low (20–40%) [13].

Three different CT patterns have been documented in GIA's after the injection of contrast medium: (1) a well-defined hyperdense mass which enhances strongly and homogeneously and can possibly mimic a meningioma [9]; (2) a "target sign" which consists of an outer hyperdense zone corresponding to dense fibrous tissue layers surrounding an isodense area with central enhancement produced by a partly thrombosed aneurysm; (3) a primarily hyperdense calcified lesion without contrast enhancement resulting from complete thrombosis of the aneurysm as found in the case presented here (Fig. 1).

Intra-arterial angiography will only show the non-thrombosed areas of the aneurysm; the full size must be estimated

from the extent of displacement of surrounding vessels. It has also been suggested that local vasospasm and haemodynamic disturbances can prevent contrast filling of a residual aneurysmal lumen [7]. As shown in Fig. 2 there is no contrast enhancement inside the aneurysm, but contrast medium protrudes from the ACoA towards the base of the aneurysm. The ACAs are seen to be pushed widely apart. The absence of pathological vascularization combined with the considerable size of the mass lesion renders the diagnosis of a malignant tumour less probable.

CT and MRI studies of GIA's have demonstrated a similar mechanism of growth as that of encapsulated chronic subdural haematomas. Recurrent haemorrhage occurs from the vessels of the highly vascularized membranous wall of a giant aneurysm. The blood dissects between old thrombotic material and the surrounding membranous capsule, which leads to the expansion of the aneurysm by newly added layers of thrombus [8]. The cross-section shows the multiple layers of thrombotic material inside the fibrous capsule.

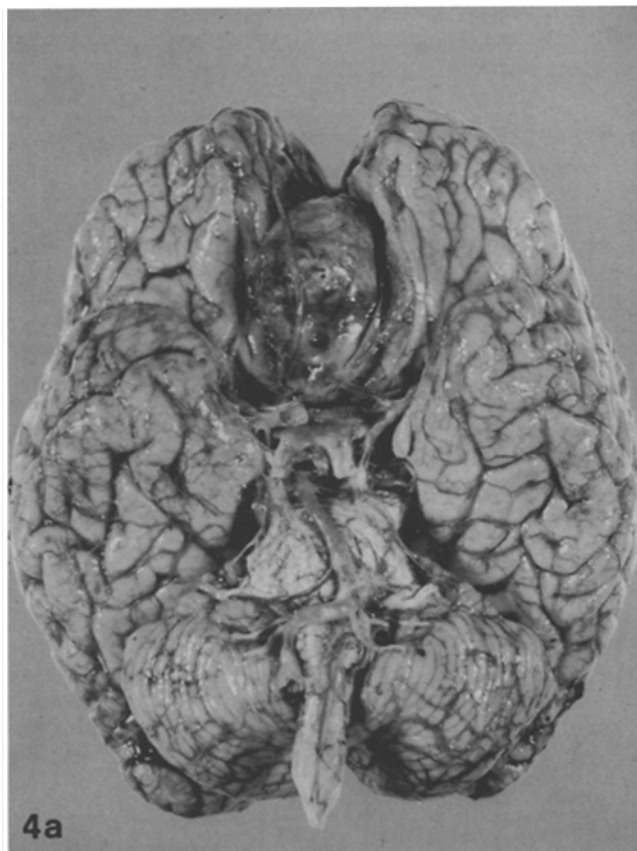
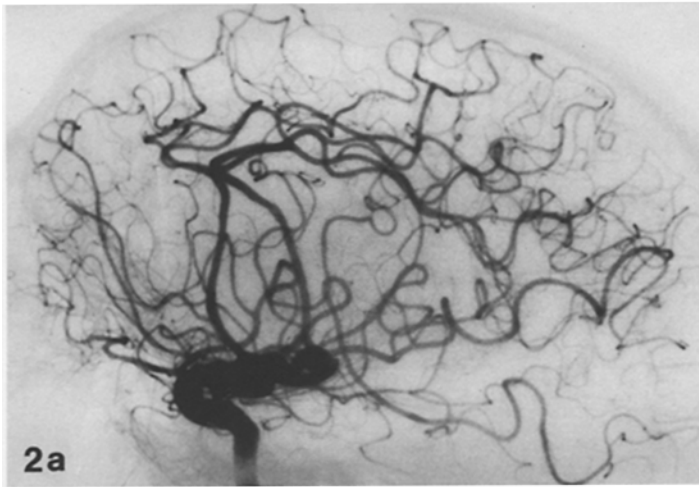
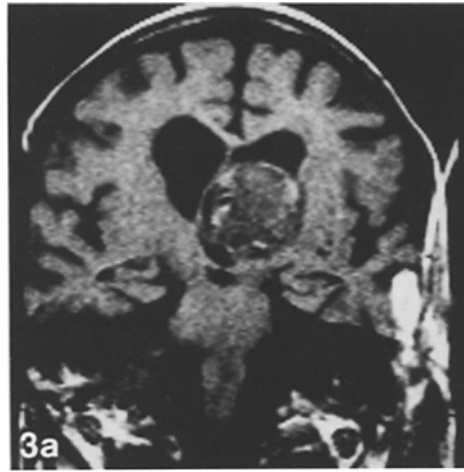
The characteristics of GIA's in MRI scans are multilaminated intensity patterns in the area of the thrombus and a signal void from the residual channel and from the parent vessel of the aneurysm [3]. Signal void is seen on T1- and T2-weighted images in areas of rapidly flowing blood, since the excited protons are removed from a particular section too rapidly to create a spin echo [11]. This phenomenon was also seen in the neck of the GIA in the case described here (Fig. 3). Echo rephasing represents a further MRI feature in the differentiation of vascularized brain lesions [2], but it was not used in the case presented here. These "functional capacities" of MRI in combination with its excellent morphological resolution may add to the higher diagnostic specificity compared with CT.

Fig. 1. Completely thrombosed ACoA aneurysm. Non-contrast axial CT scan demonstrating a hyperdense suprasellar midline tumour of the anterior fossa extending into left and right anterior horns of the lateral ventricles. The corresponding contrast scan revealed no enhancement of the aneurysm (picture not shown)

Fig. 2a, b. Intraarterial internal carotid angiogram (subtraction technique) of a thrombosed giant anterior communicating artery (ACoA) aneurysm. In the anteroposterior view the aneurysm is located between the two anterior cerebral arteries, which are laterally displaced to either side. Apart from a small deposit at the base there is no contrast enhancement inside the aneurysm

Fig. 3. Giant ACoA aneurysm. T1-weighted (TR = 600 ms/TE = 32 ms) coronal (a) and T2-weighted (TR = 2000 ms/TE = 90 ms) axial (b) MR images. Medium signal intensity persisting on all T1-weighted images represents thrombus filling the whole aneurysm. The hypointense area adjacent to the aneurysm represents signal void from blood flow in the former neck of the aneurysm on the ACoA. On the T2-weighted image (b) the thrombosed area appears hypointense, since the thrombotic material contains large deposits of haemosiderin and partially calcified areas. The pictures demonstrate the extension of the aneurysm into the third ventricle and the anterior corpus callosum, thereby pushing the septum pellucidum to the left. Only minimal perianeurysmal brain oedema is seen

Fig. 4. a The basal view of the brain demonstrates the giant aneurysm in situ. It is located between the frontal lobes and compression of the surrounding brain parenchyma, which contains multiple minute perianeurysmal haemorrhages, can be seen. **b** The circle of Willis, displaying the large size of the giant aneurysm, which covers the vessels between both middle cerebral arteries. The aneurysm is completely filled with multiple layers of calcified thrombotic material



This case describes the unusual occurrence of a giant ACoA aneurysm presenting with an organic brain syndrome without focal neurological defects.

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References

1. Adams RD, Victor M (1985) Neurologic disorders caused by lesions in particular parts of the cerebrum. In: Principles of neurology, 3rd edn. McGraw-Hill, New York, pp 329–334
2. Alvarez O, Hyman RA (1986) Even echo MR rephasing in the diagnosis of giant intracranial aneurysm. *J Comput Assist Tomogr* 10: 6999–7001
3. Atlas SW, Grossman RI, Goldberg HI, Hackney DB, Bilaniuk LT, Zimmerman RA (1987) Partially thrombosed giant intracranial aneurysms: correlation of MR and pathologic findings. *Radiology* 162: 111–114
4. Bull JWD (1969) Massive aneurysms at the base of the brain. *Brain* 92: 535–570
5. Olsen WL, Brant-Zawadzki M, Hodes J, Norman D, Newton TH (1987) Giant intracranial aneurysms: MR imaging. *Radiology* 163: 431–435
6. Poeck K (ed) (1982) *Klinische Neuropsychologie*, 1st edn. Thieme, Stuttgart, pp 107–112, 167–177
7. Russegger L, Grunert V (1987) A thrombosed giant MCA aneurysm in a ten-week-old infant. *Neurochirurgia* 30: 186–189
8. Schubiger O, Valavanis A, Wichmann W (1987) Growth-mechanism of giant intracranial aneurysms; demonstration by CT and MR imaging. *Neuroradiology* 29: 266–271
9. Stockland B (1986) Giant intracranial aneurysm. A case report. *S Afr Med J* 70: 693–694
10. Sundt TM, Piepgras DG (1979) Surgical approach to giant intracranial aneurysms. Operative experience with 80 cases. *J Neurosurg* 51: 731–742
11. Von Schulthess GK, Higgins CB (1985) Blood flow imaging with MR: spin phase phenomena. *Radiology* 157: 687–695
12. Whittle IR, Williams DB, Halmagyi GM, Besser M (1982) Spontaneous thrombosis of a giant intracranial aneurysm and ipsilateral internal carotid artery. *J Neurosurg* 56: 287–289
13. Whittle IR, Dorsch NW, Besser M (1984) Giant intracranial aneurysms: diagnosis, management and outcome. *Surg Neurol* 21: 218–230
14. Zhou LF, Jiang DJ (1987) Large and giant intracranial aneurysms. Diagnosis and surgical treatment. *Chin Med J* 100: 392–397

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