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Spontaneous closure of a traumatic middle meningeal arterio-venous fistula

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Summary. A case is reported of a traumatic middle meningeal arteriovenous fistula, which was formed on the contralateral side to the head injury without a skull fracture and was no longer demonstrable on the repeated angiogram after 35 days without any surgical intervention. Possible mechanisms of fistula formation and its spontaneous closure are discussed.

Key words: Head injuries – arteriovenous fistula – middle meningeal vessels – spontaneous closure – carotid angiography

Introduction

Traumatic dural arteriovenous fistulas involving the middle meningeal vessels as a complication of head injury were first described by Fincher [1] in 1951. Those that are closed spontaneously however, are rare: to our knowledge only 6 cases [4, 5, 7, 8, 11, 12] have been clearly recorded in the literature. We report a case of a traumatic middle meningeal arteriovenous fistula, which was formed without a fracture and was closed spontaneously 35 days after the head injury, with comments on the mechanisms of fistula formation and its spontaneous closure.

Case report

A 75-year-old woman who fell and struck her head after a syncopal attack, then visited Matsuyama Shimin Hospital complaining of nausea, vomiting and severe left temporalgia on 22 July 1980.

Examinations

On admission, she was alert and blood pressure was 104/62 mmHg, pulse rate 86/min and respiration rate 24/min. There was a contused wound in the right temporal region. Pupils were equal in size and were reactive to light. There was no motor weakness or sensory disturbance in the limbs. No bruit was heard over the head. The neck was supple with full flexion. No abnormalities were found in the laboratory data. Lumbar puncture disclosed an opening pressure of 110 mmH_2O and slightly bloody cerebrospinal fluid. Plain skull roentgenograms showed a linear right temporal fracture, but there was no evidence of a fracture

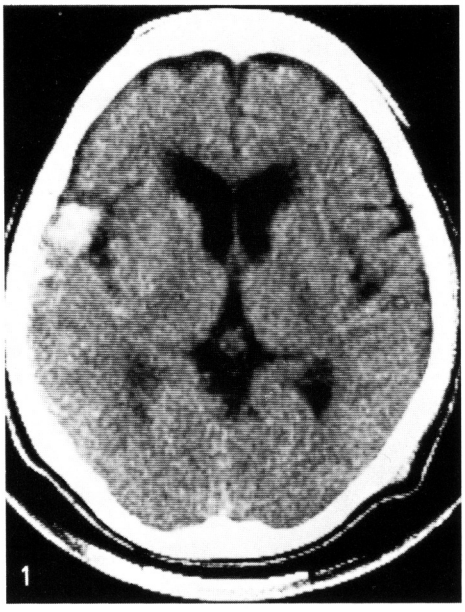
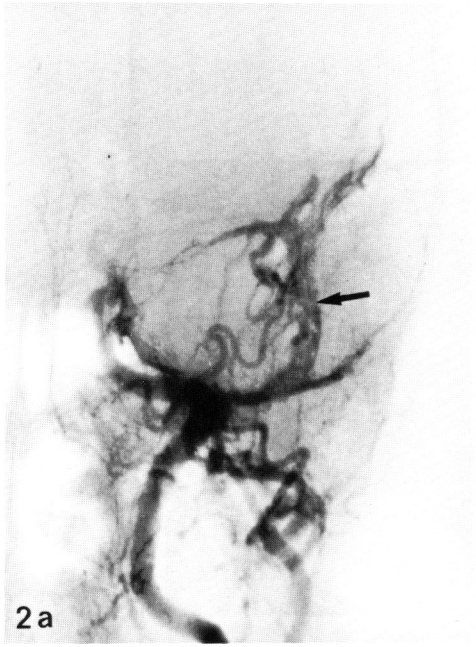
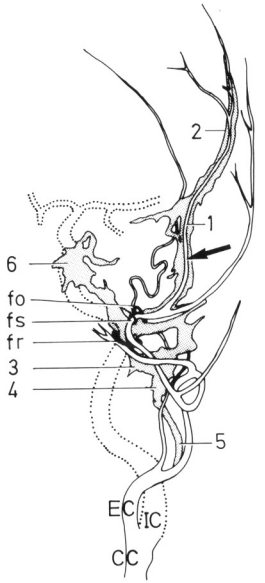


Fig. 1. Computed tomogram on admission, showing the subarachnoid hematoma localized in the left sylvian fissure





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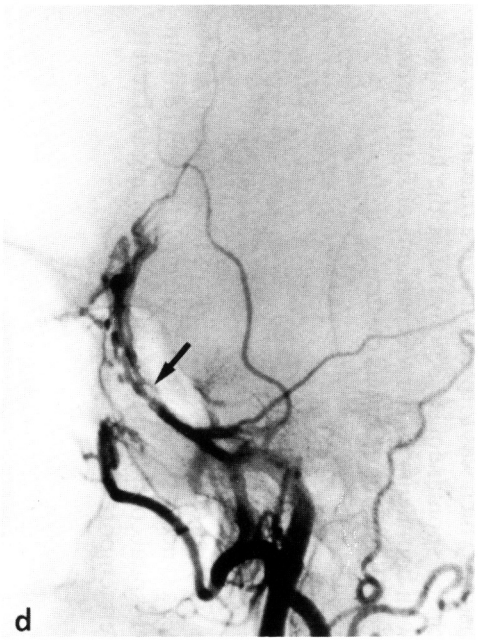

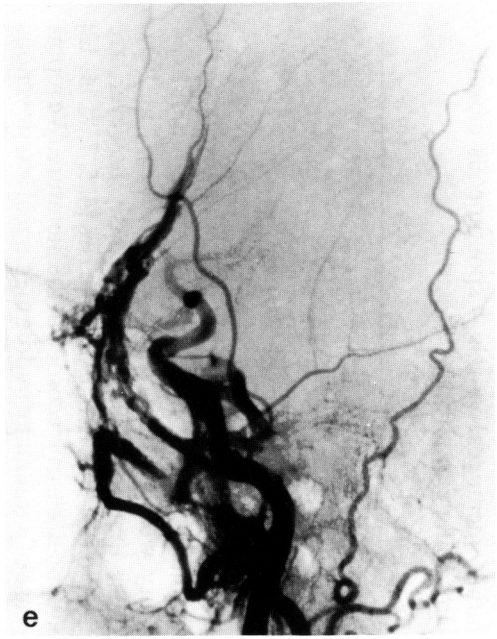

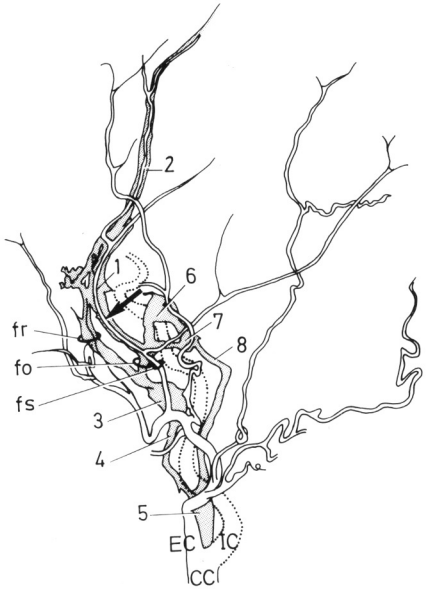

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Table 1. Cases of spontaneous closure of the traumatic middle meningeal arteriovenous fistula

No.	Authors and year	Age (years) sex	Symptoms	Skull fracture on X-P	Concomitant hematoma	Draining venous system	Interval between the head injury and spontaneous closure confirmed on the angiogram
1	Wilson and Cronic (1964)	78 M	lethargy, headache	+	–	superior sagittal sinus	34 days
2	Jackson and du Boulay (1964)	56 F	disorientation, nuchal rigidity	+	–	diploic vein	5 months
3	Ishii et al. (1976)	65 M	awake, aphasia	+	–	diploic vein	1.5 years
4	Taomoto et al. (1976)	55 M	alert, headache, vomiting, nuchal rigidity	+	+	superior sagittal sinus	1 month
5	Kitahara et al. (1977)	27 F	drowsy, headache, blurred vision	+	+	superior sagittal sinus	16 days
6	Odake (1981)	41 F	alert, headache, crural monoparesis	+	+	sphenoparietal sinus	5 months
7	Satoh et al. (present case)	75 F	alert, headache, vomiting	–	+	superior sagittal sinus, pterygoid venous plexus, cavernous sinus	35 days

on the left side of the skull. On the plain computed tomograms (Fig. 1), a subarachnoid hematoma was demonstrated in the left Sylvian fissure. The left carotid angiography (Fig. 2) performed 7 days after the head injury revealed the middle meningeal arteriovenous fistula at the base of the left middle fossa and demonstrated the following interesting venous draining system. Distal to the fistula, draining veins ascended along the sphenoid ridge to the superior sagittal sinus showing the so-called "railway configuration". Another vein running along the greater wing of the sphenoid passed through the foramen rotundum to the pterygoid venous plexus. Proximally, a dilated vein mainly ran out the foramen ovale into the pterygoid venous plexus, the palatine venous plexus and then the internal jugular vein. It was drained partially into the posterior portion of the cavernous sinus, the lateral half of the basilar plexus, the inferior petrosal sinus and from there into the internal jugular vein. A schematic drawing of the middle meningeal arteriovenous fistula is presented in Fig. 3.

Course

The patient was treated only conservatively without any surgical intervention. 34 days after the head injury, her condition had improved remarkably. The left carotid angiogram repeated on the next day (Fig. 4) disclosed complete disappearance of the fistula. Irregularity of the wall of the anterior branch of the middle meningeal artery corresponded to the position of the fistula.

Fig. 2 a–f. Left carotid angiogram 7 days after the head injury (arterial phase). A-P view **(a–c),** Lateral view **(d–f).** *Arrow* indicates the point of fistula, determined retrospectively. Dilated middle meningeal veins draining to the superior sagittal sinus show the so-called "railway configuration" or "tram track sign". Early filling of the pterygoid venous plexus, the cavernous sinus, the basilar plexus, and the inferior petrosal sinus are also demonstrated

Fig. 3 a and b. Schematic drawing of the middle meningeal arteriovenous fistula. A-P view **(a),** Lateral view **(b).** Moderately dilated anterior branch of the middle meningeal artery *(1)* forms the fistula *(arrow)* to the accompanying middle meningeal veins *(2)*, which ascend along the sphenoid ridge to the superior sagittal sinus showing the so-called "railway configuration". From the most anterior part of the internal surface of the greater wing of the sphenoid a dilated vein passes through the foramen rotundum *(fr)* to the pterygoid venous plexus *(3)*. Proximal to the fistula another vein runs out of the foramen ovale *(fo)* to the same plexus *(3)*. It drains mainly to the palatine venous plexus *(4)*, then to the internal jugular vein *(5)*, but also partially to the posterior portion of the cavernous sinus *(6)*, the lateral half of the basilar plexus *(7)*, the inferior petrosal sinus *(8)*, and then into the internal jugular vein *(5)*. *fs:* foramen spinosum, *CC:* common carotid artery, *IC:* internal carotid artery, *EC:* external carotid artery

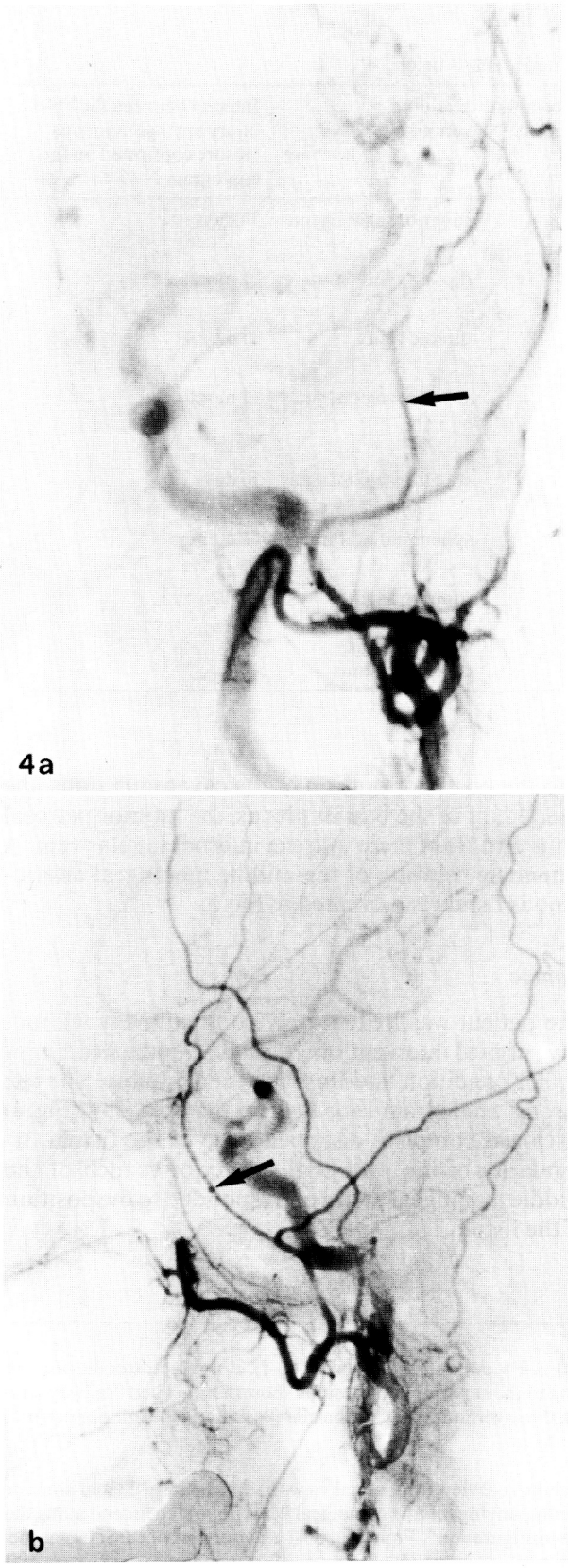

Fig. 4a and b. Repeated left carotid angiograms 35 days after the head injury (arterial phase). A-P view **(a),** Lateral view **(b).** Note disappearance of the fistula and the irregular wall of the anterior branch of the middle meningeal artery *(arrow),* which corresponds to the point of the fistula

Discussion

Traumatic middle meningeal arteriovenous fistula occurs as a complication of the tearing of middle meningeal vessels [10] associated with a skull fracture crossing them. In our case, the fistula was formed on the contralateral side to the head injury without a skull fracture. In such a condition, middle meningeal vessels might be injured by the dura mater/bone separation without a skull fracture [2]. There are certain identifyable influences in the development of such fistulas. The middle meningeal artery runs along the outer surface of the dura mater accompanied by the paired veins, which sometimes form the spheno-bregmatic sinuses. The dura mater is firmly adherent to the bone at the bottom of the vascular groove [6]. Histologically, medial defects in the meningeal arteries are commonly observed at points of branching as they also are in the other intracranial arteries [3]. Moreover, pathological changes such as arteriosclerosis play a role in relation to decrease of the elasticity of the meningeal arteries. The fistula therefore, seems to be formed by interaction between the influences of the head injury [regardless of the existence of a skull fracture], and of the anatomical, histological and pathological condition of the meningeal vessels themselves. The precise relationship of the fistula to the intrasylvian subarachnoid hemorrhage shown by CT remains unknown.

Spontaneous closure of the fistula is rare. There are only seven cases, including ours, confirmed by the angiography (Table 1). Analysis of these cases prompts the following comments: spontaneous closure tends to occur in the rather aged patient who has suffered a relatively minor head injury without, or with only a small, concomitant epidural hematoma. Symptoms are slight and the clinical course is good. The damage to the meningeal vessels in this condition is considered to be of little significance. In our case, the aged patient suffered a minor head injury without any neurological deficit. Treated conservatively without surgical intervention, the patient suddenly lost her severe left temporalgia on the 34th day after the head injury. The arteriovenous fistula was no longer demonstrable by repeated angiography on the next day. Though the precise mechanisms of spontaneous closure are not known, it may have resulted from thrombosis at the point of trauma to the vessels [9, 12]. In our case, it is thought to have been caused by a thrombus at the point of the fistula. Irregularity of the wall of the anterior branch of the middle meningeal artery on the repeated angiograms supports this view.

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