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Non-traumatic Arteriovenous Fistulas of the Scalp Treated by a Combination of Embolization and Surgical Removal

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Abstract

Three patients with non-traumatic arteriovenous fistulas (AVFs) of the scalp were treated by embolization using acrylate glue through the transarterial route or by direct puncture of the lesions and then surgical resection. Complete cure was achieved in all three patients. Selective angiography is indispensable in the correct diagnosis of scalp AVFs. Embolization facilitates surgical removal when necessary, and this combination is the treatment of choice for scalp AVFs.

Key words: acrylate glue, arteriovenous fistula, embolization, scalp

Introduction

Non-traumatic arteriovenous fistulas (AVFs) of the scalp are rare.[3,6,12] The clinical manifestation may be symptoms of pulsating mass, headache, local pain, bruits, tinnitus, thrill and, less commonly, hemorrhage and necrosis.[3] Treatment includes ligation of the feeding arteries, surgical removal,[2,3,6] electrothrombosis,[11] embolization, or a combination of these approaches. Embolization has become more common, using transarterial or transvenous methods, or by direct approach to the lesion.[1,4,5,10]

We describe three patients with scalp AVFs treated by a combination of embolization and surgical removal.

Case Presentation

Case 1: A 34-year-old female had noticed the slow enlargement of a pulsating mass at the left temporoparietal region over a 3-year period. Six months prior to admission, she gave birth to a healthy child. Thereafter, the mass further increased in size. She reported no history of scalp trauma. Angiography showed multiple AVFs of the scalp. The feeding arteries were the left superficial temporal artery (STA), the left occipital artery, and the left middle meningeal artery, while the draining veins were the superficial scalp veins (Fig. 1 *left, center*).

Femoral transarterial embolization was performed using a liquid glue; a 1:1 mixture of *N*-butyl cyanoacrylate (NBCA) (Histoacryl blue; B. Braun Melsungen AG, Melsungen, Germany) and lipiodol. This resulted in subtotal occlusion of the fistulas (Fig. 1 *right*). Three weeks later, surgical resection of the AVFs was easily carried out without obvious bleeding. The lesion was located beneath the galea and above the temporal fascia. The small feeding arteries from the left middle meningeal artery had penetrated through the skull bone, so were coagulated and cut. The small holes at the skull bone were occluded with bone wax. The postoperative course was uneventful. There has been no recurrence during the 10 months since surgery.

Case 2: A 22-year-old female had noticed a right frontotemporal pulsating mass at the age of 18 years. The mass had gradually increased in size, especially after she gave birth to a child at the age of 19 years. She reported no history of scalp trauma. Angiography demonstrated a single AVF fed by the bilateral STAs and the right occipital artery, draining to the superfi-

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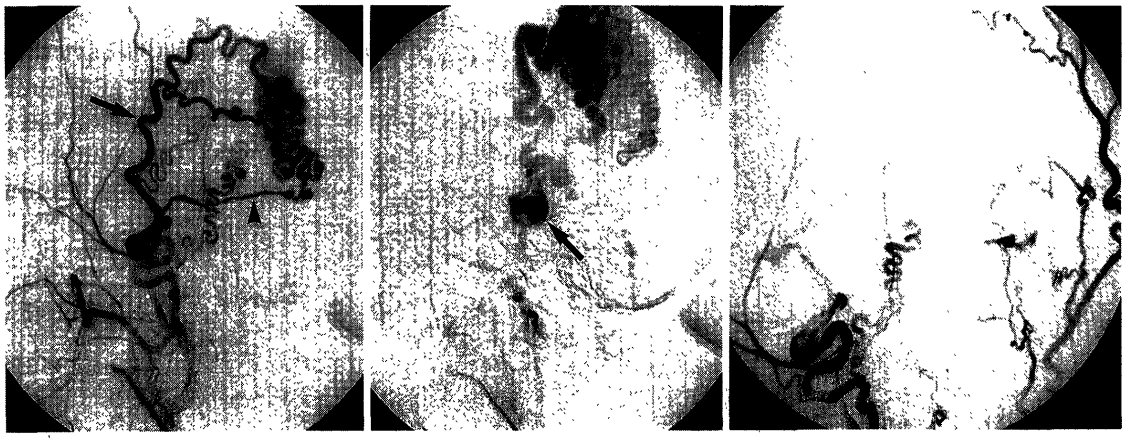

Fig. 1 Case 1. *left*: Left external carotid arteriogram (arterial phase, lateral view) showing the AVFs between the STA (*arrow*), the middle meningeal artery (*arrowhead*), and the superficial temporal veins. *center*: Venous phase arteriogram showing the dilated superficial temporal veins with varicose change (*arrow*). *right*: Arteriogram immediately after embolization showing the shunted flow is markedly reduced.

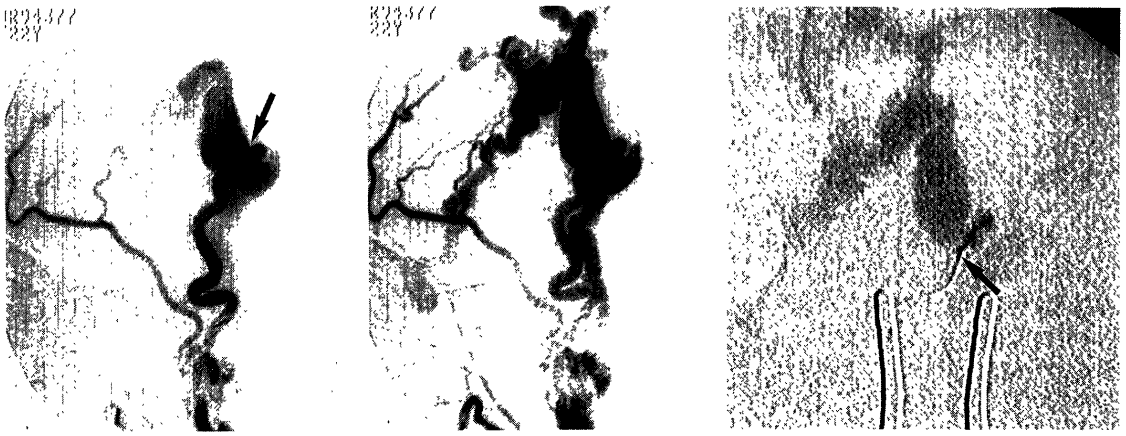

Fig. 2 Case 2. *left*: Right external carotid arteriogram (lateral view) showing the single AVF (*arrow*) between the STA and the scalp veins. *center*: Late arterial phase arteriogram showing the enlarged superficial temporal veins. *right*: Arteriogram immediately before embolization. *Arrow* indicates the tip of the catheter. Liquid glue has subtotally occluded the fistula, but the flow from the opposite (left) STA persists. No post-embolization angiogram was obtained.

cial scalp veins (Fig. 2 *left*, *center*).

Femoral transarterial embolization using a detachable balloon failed because of marked tortuosity of the right STA (Fig. 2 *right*). Glue embolization (NBCA:lipiodol 1:1) of the AVF through a direct puncture of the right STA was subsequently carried out, resulting in nearly total occlusion of the fistula. The fistula was easily resected by surgery 1 week later. The AVF was located above the temporal fascia and beneath the galea. The postoperative course was uneventful. There was no recurrence for 3 years after surgery.

Case 3: A 5-year-old boy had had a gradually increasing mass in the frontal midline region since the age of 2 years. Although he had no history of scalp laceration, minor trauma could not be excluded, since simple head trauma is fairly common in children of this age. The skin above the pulsating mass was slightly red. Angiography showed multiple AVFs, fed by the bilateral STAs, the left middle meningeal artery, and the bilateral ethmoidal arteries, and draining to the bilateral superficial scalp veins and the bilateral superior orbital veins, then to the bilateral cavernous sinuses (Fig. 3 *upper row*). No drainage to the superior sagittal sinus was observed. There was no nidus between the feeding arteries and draining veins,

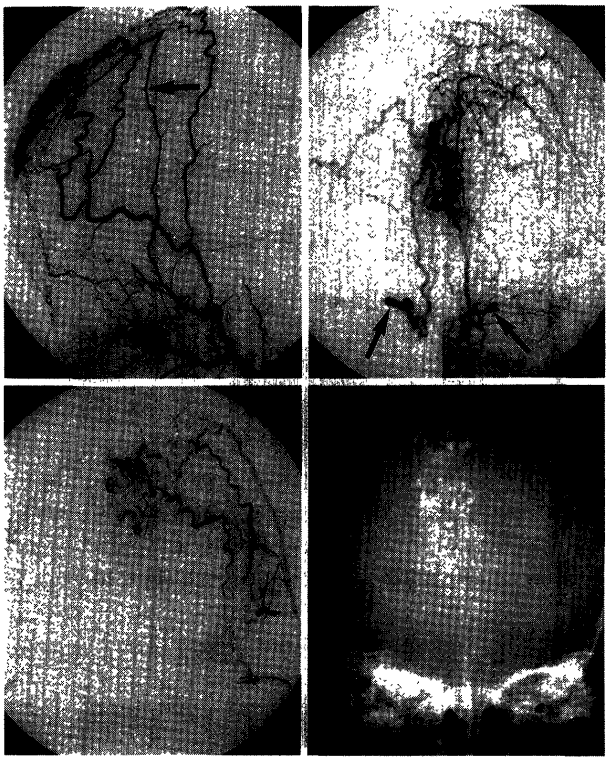

Fig. 3 Case 3. *upper left*: Left external carotid arteriogram (lateral view) showing the scalp AVFs fed by the branches of the left STA and the left middle meningeal artery (*arrow*). *upper right*: Arteriogram (anteroposterior view, late arterial phase) showing the AVFs on the midline draining to the superficial scalp veins and the bilateral superior orbital veins (*arrows*). *lower left*: Post-embolization left superficial temporal arteriogram (anteroposterior view) showing the shunted flow is markedly reduced. *lower right*: Plain skull x-ray film (anteroposterior view) after embolization. The glue is located predominantly in the venous side of the AVFs as well as in the right STA.

but numerous minute connecting fistulas were observed.

Direct puncture of the lesion (venous side) was carried out at three different sites using 22-gauge elastic needles. While manual compression was applied to the area around the lesion, liquid glue (NBCA:lipiodol 1:1) was injected into the lesion. Glue was also injected through the right STA. These procedures resulted in marked reduction of the shunted flow (Fig. 3 *lower row*). We were concerned about the possible development of AVFs in the overlying red skin, so the AVFs with the overlying skin were totally resected 2 days later. The lesion was located above the periosteum and beneath the galea. A defect of the periosteum was observed at the frontal pole of the lesion. The feeding arteries from the middle meningeal artery had penetrated through the sagittal suture. Minute bony holes at the sagittal suture were occluded with bone wax. The skin defect was approximated with primary closure. Cosmetically, no obvious deformity was noted. There was no recurrence for 6 months after surgery.

Discussion

The etiology of scalp AVFs may be spontaneous or traumatic.[3,6,12] Spontaneous AVF of the scalp may present at birth, but in the most patients is asymptomatic until puberty. Trauma, pregnancy, or hormonal change causes deterioration of the symptoms, as occurred in our series. Traumatic AVF of the scalp develops months or years after scalp trauma.

The terminology referring to the scalp AVFs is inconsistent and confused because a variety of terms, such as recemose aneurysm, cirsoid aneurysm, varicose aneurysm, arteriovenous aneurysm, aneurysmal varix, plexiform angioma, hemangioma, and arteriovenous malformation, are commonly used to describe AVFs.[4,6,13] This can be attributed to the confusion in the classification of vascular lesions in the head and neck regions, and to the lack of angiographic characterization of such lesions in diagnosis and treatment.[7]

Differential diagnoses include aneurysm of the STA, arteriovenous malformation of the scalp, sinus pericranii, venous malformation, and cavernous hemangioma. Selective angiography is indispensable for diagnosis. Magnetic resonance (MR) imaging provides additional information. All the cases presented here were first diagnosed as "hemangioma" in other hospitals. Venous malformation and cavernous hemangioma have no arteriovenous shunts as shown by angiography, and are well-demarcated lesions with no flow void signs on MR images. AVFs and arteriovenous malformations show flow void signs on MR images due to the rapid flow in the lesions. Furthermore, dural AVFs, which are thought to be acquired lesions involving the dural sinuses, are sometimes included among scalp AVFs.

Until recently, scalp AVFs have been treated by feeder ligation and surgical resection.[2,3,6] The former improves clinical symptoms only temporarily, as AVFs inevitably recur after recruitment of the collaterals, and results in more difficult access to the lesions in future endovascular treatment.[6] The latter often involves extensive treatment because all the fistulas must be removed. Profuse hemorrhage during surgery is a common complication. Fisher-Jeffes *et al.*[3] reported total surgical excision of scalp AVFs

in 24 patients, with surgical complications of scalp necrosis, bleeding, or wound sepsis in six patients (25%) and recurrence in four (17%). Electrothrombosis is another alternative, but the effect is palliative.[11]

Advances in modern interventional neuroradiology, and diagnostic and therapeutic angiography have changed the management strategy for vascular lesions in the head and neck regions.[7,9] The diagnosis of AVFs should be based on the feeding arteries, draining veins, sites and number of the fistulas, their relationship to the adjacent normal vessels, and the amount of shunted flow. Furthermore, the accessibility through the arterial and venous routes should be evaluated.

Endovascular treatment is becoming more accepted as the definitive or presurgical treatment for scalp AVFs. Transarterial embolization and, more recently, transvenous and direct embolization of AVFs have been reported.[1,4,5,10] Tortuosity of the feeding vessels and draining veins often prevent access to the lesion through the transarterial or transvenous routes. In this situation, direct puncture of the lesion is an alternative.[1,13] The materials used for endovascular occlusion of AVFs include liquid glue, coils, balloons, silk sutures, polyvinyl alcohol particulates, and pure alcohol. These materials are deposited either directly into the fistula or predominantly at the venous or arterial side.

It is often difficult to cure scalp AVFs solely with endovascular techniques. Surgical resection is also usually required,[1,8,10] and is facilitated by subtotal occlusion of the AVFs by embolization. AVFs are located on the periosteum or the temporal fascia and beneath the galea. The minute feeding arteries may arise from the middle meningeal artery through the skull bone. These vessels are difficult to occlude with endovascular techniques alone and surgical excision may be necessary to avoid recurrence from the residual meningeal contribution.[3] However, we believe that a craniotomy to expose the dural feeders is unnecessary, since the fistula is outside the skull bone. Even when endovascular occlusion of the fistulas is complete and successful, the embolic materials, such as glue or coils, may cause a disfiguring mass. Therefore, resection will achieve a better cosmetic outcome. If the resected area is large, reconstruction using a skin graft, local flap, free-tissue transfer, or tissue expanders for the adjacent scalp is necessary.[8] In such cases, a multidisciplinary approach in collaboration with plastic surgeons is indispensable for the treatment of scalp AVFs.[8]

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