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Pre-operative embolization of auricular arteriovenous fistula

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Abstract
Treatment of auricular arteriovenous fistula (AVF) is a challenge with surgery being the preferred option until now. We present three cases of auricular AVFs who underwent pre-operative embolization and its outcome on surgery. Three patients were diagnosed to have auricular AVF by angiography. All three patients underwent pre-operative embolization with n-butyl 2-cyanoacrylate after which they underwent surgical resection of the AVF. Pre-operative embolization resulted in significant devascularization of the AVF thus leading to near bloodless and clean surgery. Pre-operative embolization of auricular AVFs is a good treatment option, leading to significantly reduced blood loss during surgical excision.

Key words: Ear, External; Arteriovenous Fistula; Embolisation, Therapeutic

Introduction

An arteriovenous fistula (AVF) is an abnormal communication between an artery and a vein and is an uncommon clinical condition. In particular AVFs involving the auricle are rare, with the World Health Organization listing only a single case during the period between 1966 and 1976. This case involved a 35-year-old male with traumatic AVF of his external ear.[1]

Until now, only one case of AVF of the auricle treated with pre-operative embolization has been reported in the English literature.[2] Good surgical outcome of these cases depends on minimizing blood loss. We present here three cases of congenital AVF of the auricle which underwent pre-operative embolization as a prelude to surgery, resulting in nearly bloodless and clean surgery.

Material and methods

Three patients with congenital AVF malformation of the auricle underwent angiography and embolization. Digital subtraction angiograms of the external and internal carotid arteries on the side of the lesion were carried out first. This was followed by selective angiograms of the posterior auricular, occipital and superficial temporal arteries. Embolization was performed by using a microcatheter in two patients and direct injection of the vascular channel by the percutaneous route in one patient. Once the feeder arteries were identified, a microcatheter (Tracker 18, Target Therapeutics, Fremont, Ca.) was placed in the major arterial feeder of the AVF. A 30 per cent mixture of n-butyl-2-cyanoacrylate with Lipoidol (X-ray contrast agent; Rhone Poulenc, India) was used to embolize the lesion. The microcatheter was then withdrawn immediately after injecting the mixture to prevent glueing of the catheter to the vessel wall. In one patient percutaneous injection of the large venous sacs was carried out after placing a tourniquet fashioned out of the cuff of a surgical glove around the head and below the ear. It effectively blocked the venous outflow. When the veins become prominent a 23G scalp vein was inserted into the venous sac and a 30 per cent mixture of n-butyl 2-cyanoacrylate and Lipoidol was injected through it.

In all patients post-embolization angiograms were performed to investigate the degree of devascularization and the cast of n-butyl 2-cyanoacrylate. All patients underwent surgical excision of the AVF.

Results

In all the three cases selective catheterization of the posterior auricular, superficial temporal and occipital arteries was possible. In all the cases, a major portion of the AVF was supplied by the posterior auricular artery. In two of the cases the superior portion of the AVF was supplied by the superficial temporal artery and in the remaining case, the posterior portion of AVF was supplied by the occipital artery. Two of the AVFs were associated with aneurysmally dilated venous sacs while one was a cirsoid aneurysm with multiple communications between arteries and veins with marked dilation of these vessels. In two cases which underwent endovascular embolization of the segment supplied by the posterior auricular artery, complete blockage of arteriovenous communications was obtained (Figure 1). In the third case which underwent percutaneous embolization, marked reduction of flow into the AVF was obtained (Figure 2). In all the three patients the AVF was excised within one week of embolization. During surgery blood loss was significantly reduced due to previous embolization and also due to clear depictions of regional vascular anatomy by angiography. No post-operative complication was seen in any of the patients. No recurrence of the lesion was seen on subsequent clinical examination.

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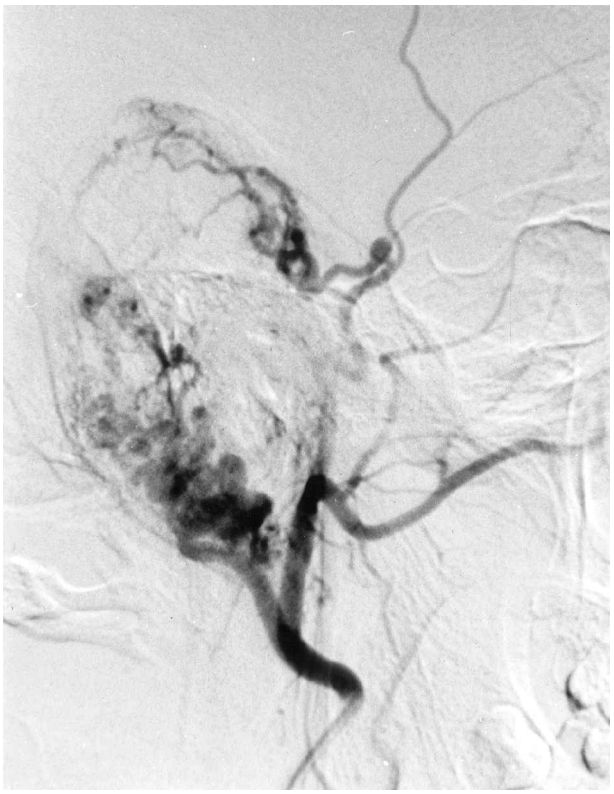

(a)

(b)

(c)

FIG. 1

(a) Selective posterior auricular angiogram lateral view showing the auricular anteriovenous fistula with early shunting of contrast into the external jugular vein. (b) Post-embolization external carotid angiogram PA view showing non-filling of the fistula and presence of supply to the auricle from the superficial temporal artery, (c) Cast of the n-butyl 2-cyanoacrylate as seen in the digital X-ray PA view of the auricle.

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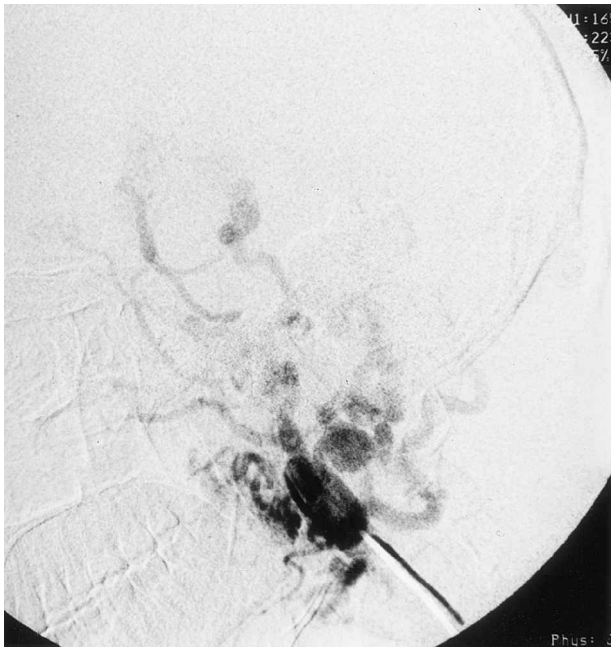
(a)



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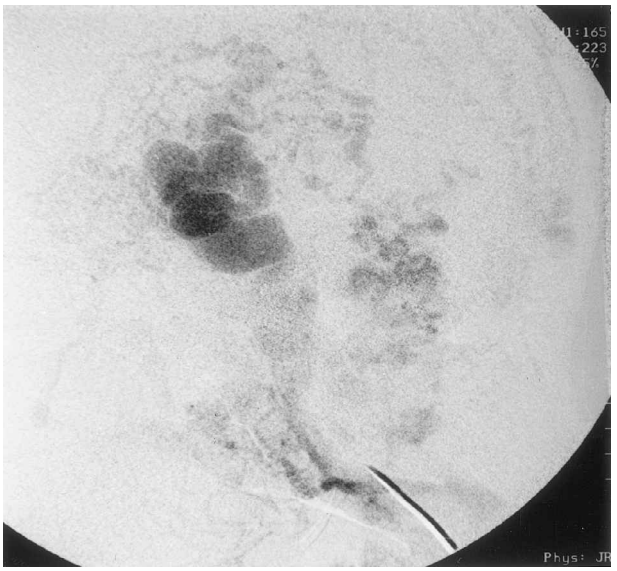

(c)

(d)

FIG. 2

(a) Axial contrast-enhanced CT showing enlarged pinna with enhancing and enlarged vascular channels, (b & c) percutaneous injection of contrast into the AVF showing rapid filling of the dilated and tortuous venous channels of the arterio-venous fistula, (d) Post-embolization digital X-ray PA view showing the cast of the n-butyl 2-cyanoacrylate in the AVF.

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Discussion

AVFs can occur in the following forms:[3]

(1) a direct fistulous communication between an artery and a vein;
(2) AVF associated with interposed aneurysmally dilated sacs;
(3) a cirsoid aneurysm in which there are usually multiple communications between arteries and veins as well as invariably marked dilatation of these blood vessels in the area of the lesion;
(4) AVF associated with hypervascular tumours.

AVFs can also be divided as congenital or acquired (traumatic).[3] In congenital AVF the arterial and venous components of the embryonic vascular system fail to separate from the common vessel of origin. This results in multiple communications between the otherwise normal arteries and veins. The acquired (traumatic) AVFs are caused when blood vessels involved in the injury are torn open and blood from the artery finds its way into the vein. When the abnormal arteriovenous communication becomes established the affected vein grows large and tortuous due to direct transmission of arterial blood pressure. This can lead to complications such as ulcerations, haemorrhage, haemodynamic embarrassment, coagulopathies and disfigurement.[4–6] Arterial supply of the auricle comes from the posterior auricular and superficial temporal arteries, which are branches of the external carotid artery and thus these arteries are enlarged in cases of AVF of the auricle. The internal carotid artery which supplies the external auditory canal and tympanic membrane, communicates with the external carotid artery along the walls of the auditory canal. These communications are of clinical importance in that they continue to provide an abundant arterial supply to the auricle in patients who have ligation or embolization of branches of external carotid artery. Blood from the auricle drains into the posterior fascial and posterior auricular veins. It has also been suggested that branches of the external carotid artery not supplying the AVF may atrophy as the major blood supply is diverted to the superficial temporal and posterior auricular arteries.[7]

The main aim of treatment is to obliterate all arteriovenous communications and this depends on the nature of the fistulous communications. In cases of traumatic AVFs with few artery to vein communications, a simple ligation of the abnormal communicating vessels may be sufficient to effect a cure.[3] In congenital AVFs abnormally communicating vessels are so numerous that the whole auricle is often turned into a deformed mass of vascular lesions, making simple ligations ineffective.[3] Moreover, although arterial ligation initially causes a temporary redution in the size of the lesion, the lesion then re-expands as collateral vessels dilate.[8] Along with arterial ligation, excision of whole AVF or amputation of the auricle has been recommended.[9]

Embolization of AVF's has been used pre-operatively to decrease operative blood loss and therefore permit an easier, more precise resection.[8,10] Moreover, embolization can be performed with very little risk and significantly less morbidity and hospitalization than with any other form of treatment.

For embolization a permanent agent should be used. In general particulate agents are usually not suitable since they tend to pass through the large arteriovenous connections of these lesions. N-butyl 2-cyanoacrylate is the preferred embolization agent because despite the presence of high flow fistulae, this agent polymerizes rapidly and forms a cast which instantly reduces flow through the fistula.[11] Moreover, due to its low viscosity n-butyl 2-cyanoacrylate can be easily injected through a microcatheter.[11] Polymerization will occur when the material gets in contact with any ionic solution such as contrast media, blood vessel intima or a basic pH solution. N-butyl 2-cyanoacrlate is mixed with Lipoidol which renders the agent radiopaque and effectively alters the time for polymerization depending on the concentration. Embolization with n-butyl 2-cyanoacrylate can be carried out by placing a microcatheter in the feeding artery. Microcatheters are usually flow directed and tend to easily navigate tortuous vessels. The advantage of embolization by microcatheter is that they can be precisely located at the preferred site for injection of n-butyl 2-cyanoacrylate. Percutaneous injection of n-butyl 2 cyanoacrylate can be carried out in larger vessels which can be identified on palpation.[12] Injection of n-butyl 2-cyanoacrylate into vessels is performed under fluoroscopic control. Movement of n-butyl 2-cyanoacrylate can be observed and injection terminated when there is retrograde flow into adjacent non-involved vessels. No definite post-embolization care is usually necessary except for a precautionary period of rest. Complications such as glueing of the catheter to the vessel wall, ischaemia and necrosis of adjacent structures are uncommon[2] and can be minimized with good techniques.[11]

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