Available online at www.sciencedirect.com

ScienceDirect

Journal of the Chinese Medical Association 79 (2016) 152−158

www.jcma-online.com

Original Article

Aggressive cavernous sinus dural arteriovenous fistula: Angioarchitecture analysis and embolization by various approaches

Chao-Bao Luo [a,b,c], Feng-Chi Chang [a,b], Michael Mu Huo Teng [b,d,*], Chung-Jung Lin [a,b], An-Guor Wang [b,e], Ta-Wei Ting [c]

[a] *Department of Radiology, Taipei Veterans General Hospital, Taipei, Taiwan, ROC*
[b] *Department of Radiology, School of Medicine, National Yang-Ming University, Taipei, Taiwan, ROC*
[c] *Department of Biomedical Engineering, Yuanpei University of Medical Technology, Hsinchu, Taiwan, ROC*
[d] *Department of Medical Imaging, Cheng Hsin General Hospital, Taipei, Taiwan, ROC*
[e] *Department of Ophthalmology, Taipei Veterans General Hospital, Taipei, Taiwan, ROC*

Received July 7, 2015; accepted September 2, 2015

Abstract

Background: Most cavernous sinus dural arteriovenous fistulas (CSDAVFs) present with benign neuro-ophthalmic symptoms. CSDAVFs manifesting with aggressive neurologic symptoms are rare. The purpose of this study was to analyze the different angioarchitectures of aggressive CSDAVFs and to report our experiences of embolization.

Methods: Over the past 10 years, a total of 118 CSDAVFs were managed by embolization. From the databases containing such patient information, nine patients (7.6%) were found to have aggressive CSDAVFs presenting with neurologic deficits. There were seven women and two men, ranging in age from 51 years to 78 years (mean, 66 years). We retrospectively analyzed the angioarchitectures of aggressive CSDAVFs, further reviewing patient and angiographic as well clinical outcomes after embolization.

Results: The cause of clinically aggressive CSDAVFs was insufficient fistula drainage because of occlusion ($n = 6$) or stenosis ($n = 1$) of the inferior petrous sinus (IPS) or compartment of IPS−cavernous sinus ($n = 2$) with fistula flow reflux to the veins of brainstem ($n = 7$) leading to brainstem ischemia, while two fistula flow reflux to the cortical vein leading to cerebral infarction. Transvenous embolization via IPS to fistula was achieved in one case; six patients underwent transorbital access, while transarterial embolization was performed in two cases. Total fistula occlusion was achieved in eight CSDAVFs. All patients had total ($n = 7$) or partial ($n = 2$) resolution of their symptoms gradually within 6 months. One patient undergoing transarterial embolization had limb weakness because of inadvertent pial artery occlusion. Their overall mean clinical follow-up period was 17 months.

Conclusion: Aggressive CSDAVFs are associated with occlusion/stenosis of the IPS or compartment of IPS−cavernous sinus with leptomeningeal reflux. In this limited case series, aggressive CSDAVFs most presented with brainstem ischemia, followed by nonhemorrhagic/hemorrhagic stroke in the cerebrum. Embolization through various access routes is a feasible method to manage these aggressive CSDAVFs, with an acceptable level of periprocedural risks.

Keywords: cavernous sinus; dural arteriovenous fistula; embolization; stroke

Conflicts of interest: The authors declare that they have no conflicts of interest related to the subject matter or materials discussed in this article.

* Corresponding author. Dr. Michael Mu Huo Teng, Department of Medical Imaging, Cheng Hsin General Hospital, 45, Cheng Hsin Street, Taipei 112, Taiwan, ROC.

E-mail address: mhteng@gmail.com (M.M.H. Teng).

http://dx.doi.org/10.1016/j.jcma.2015.09.001

1. Introduction

Intracranial dural arteriovenous fistulas (DAVFs) are uncommon cerebrovascular lesions appearing in 10−15% of the total intracranial vascular malformations.[1] Intracranial DAVFs have a wide spectrum of clinical presentations,[1−4] although aggressive DAVFs are generally considered as fistulas with insufficient venous drains with leptomeningeal or pial venous reflux.[3] Aggressive DAVFs have a tendency to produce hemorrhagic or nonhemorrhagic stroke or death. Minor neurologic deficit such as cranial nerve palsy was not considered as aggressive DAVFs.[5] Most cavernous sinus DAVFs (CSDAVFs) are low-flow shunts fed by dural branches of the internal and/or external carotid arteries. CSDAVFs commonly present with benign ocular symptoms of orbital venous hypertension, or focal third and/or sixth cranial nerve palsy. CSDAVFs presenting with aggressive behavior with hemorrhagic or nonhemorrhagic neurologic deficit is uncommon.[6−8]

The purpose of this study was to evaluate the angioarchitectures of aggressive CSDAVFs, and to report our experiences of access and embolization to manage aggressive CSDAVFs.

2. Methods

2.1. Patients

From May 2004 to April 2014, a total of 102 patients with 118 CSDAVFs were referred to our institute to assess the feasibility of embolization because of intolerable neuro-ophthalmic symptoms or hemorrhagic/nonhemorrhagic stroke. In the databases, there were nine cases of CSDAVFs (7.6%) presenting with clinically aggressive behavior with ischemic stroke ($n = 9$; Figs. 1 and 2) and being associated with cerebral hemorrhagic transformation ($n = 1$). These patients consisted of two men and seven women, and their ages ranged from 51 years to 78 years (mean, 66 years). Prior to conducting this study, informed consent was obtained from all nine patients; their clinical data are summarized in Table 1.

2.2. Techniques of embolization

Embolization was performed on all patients under general anesthesia using bilateral femoral approaches, with the placement of 5-F and 6-F femoral sheaths in the left femoral artery and right femoral vein, respectively. Activated clotting time was monitored and maintained at a value twice the baseline value by intravenous administration of heparin. In all patients, transvenous access to CSDAVFs via the inferior petrous sinus (IPS) and coiling of the fistula was attempted first. This procedure was performed by placing a 4-F diagnostic catheter into the feeding carotid artery as a guide for subsequent transvenous catheterization; then a 6-F guiding catheter (Envoy; Codman & Shurtleff, Rayhnam, MA, USA) was placed into the internal jugular vein, followed by retrograde catheterization of the IPS and then the fistula site of the cavernous sinus (CS) for coiling. However, this route of access was successful only in one patient (Fig. 2). Access to the other eight aggressive CSDAVFs failed after many attempts because of total occlusion ($n = 6$; Fig. 1) or stenosis of the IPS. In these six patients with brainstem ischemia and edema, we decided to undertake a more invasive technique using a transorbital access to the fistula site by direct needle puncture of the proximal ophthalmic vein (OV; $n = 4$; Fig. 1) or CS ($n = 2$) under fluoroscopic and roadmapping guidance. An 18-gauge puncture needle (Terumo Corporation, Tokyo, Japan) was utilized. To avoid injury, the eyeball was pushed to its superior aspect, and the puncture needle was carefully advanced via the inferior orbital rim. During the advancement of the puncture needle, it was adjusted and checked intermittently to target the proximal OV or anterior CS depending upon the inclination of the inferior orbital wall. Once the tip of the puncture needle reached the OV or CS, the arterialized blood flowed from the fistula to the puncture needle, which was confirmed by hand-injection angiography. The inner metallic puncture needle was removed for subsequent microcatheter navigation into the fistula. Coiling of the fistula was initiated and continued until total fistula occlusion or recoil of the microcatheter back to the OV. In one patient, a microcatheter recoiled back to the anterior CS during coiling with residual fistula, to enhance the effect of fistula occlusion; Onyx-18 (ev3, Inc., Irvine, CA, USA) was slowly infused into the fistula site.

Two patients underwent transarterial embolization because they refused to undertake the risk of transorbital approach. Transarterial embolization was performed by navigation of a microcatheter into the accessory meningeal arteries by delivery of a 25% Lipiodol/liquid adhesive mixture ($n = 1$) or particle ($n = 1$) until the embolizer refluxed to the tip of the microcatheter or the fistula flow was sluggish.

A postembolization angiogram was performed immediately after the completion of the procedure to check for fistula occlusion. Seven cases had neuroimaging follow-up; conventional digital subtraction angiography (DSA) was obtained in two cases, while seven cases underwent magnetic resonance angiography (MRA) to evaluate the neuroimaging outcome of embolized aggressive CSDAVFs (mean, 8 months). All patients have been followed up clinically for an average of 17 months (range, 8−25 months).

3. Results

The demography and treatment outcomes of nine patients with aggressive CSDAVFs are summarized in Table 1. The clinical manifestations of these patients were chemosis ($n = 7$), limb weakness ($n = 8$), proptosis ($n = 2$), diplopia ($n = 2$), aphasia ($n = 1$), bruit ($n = 1$), unstable gait ($n = 1$), and respiratory failure ($n = 1$). Prior to performing embolization, all patients underwent brain computed tomography ($n = 3$) and/or MRA ($n = 8$). Brainstem ischemia and edema were detected in eight patients (Figs. 1 and 2); the other patient presented with ischemia with hemorrhagic transformation in the left temporal lobe. Pre-embolization carotid and vertebral DSAs showed all cases of aggressive CSDAVFs were fed by dural branches of internal and external carotid arteries