ALCOHOL EMBOLIZATION OF CAROTID-CAVERNOUS INDIRECT FISTULAE

Christopher J. Koebbe, M.D.

Department of Neurological Surgery, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Michael Horowitz, M.D.

Departments of Neurological Surgery and Radiology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Charles Jungreis, M.D.

Departments of Neurological Surgery and Radiology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Elad Levy, M.D.

Department of Neurological Surgery, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Misha Pless, M.D.

Departments of Neurological Surgery, Neurology, and Ophthalmology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Reprint requests:
Michael Horowitz, M.D., Department of Neurological Surgery, University of Pittsburgh Medical Center, 200 Lothrop Street, Suite 400-B, Pittsburgh, PA 15213.
Email: horowitzmb@msx.upmc.edu

Received, September 19, 2002.
Accepted, January 8, 2003.

OBJECTIVE: Carotid-cavernous fistulae (CCFs) are abnormal communications between the carotid artery and cavernous sinus that may present with rapid visual deterioration and extraocular paresis as a result of increasing intraocular pressure requiring emergent treatment to preserve vision. We present a technique of balloon-assisted ethanol embolization of the cavernous carotid artery supply to indirect CCFs providing immediate reduction in intraocular pressure with symptomatic improvement.

METHODS: We reviewed clinical and angiographic data and present a retrospective case series illustrating six patients who underwent endovascular embolization because of worsening visual acuity and extraocular motility disorder caused by CCFs. Cerebral angiography revealed significant blood supply from the cavernous carotid artery to these CCFs. We performed ethanol embolization of these branches with distal balloon protection.

RESULTS: Five of the six patients experienced immediate and sustained (mean follow-up, 21 mo) decreases in intraocular pressure, with significant symptom improvement. One patient experienced cavernous sinus thrombosis after conclusion of embolization, which caused a temporary worsening of symptoms that improved gradually over time.

CONCLUSION: Many surgical and endovascular options are available to treat indirect CCFs. Absolute ethanol is a liquid agent that causes immediate vessel sclerosis and occlusion, which makes it a dangerous but potent liquid embolic agent. With distal temporary balloon protection to prevent migration of ethanol, we achieved excellent clinical and angiographic results using absolute ethanol to embolize the cavernous carotid supply to indirect CCFs. This represents a safe and effective method of endovascular management of this complex vascular anomaly.

KEY WORDS: Carotid-cavernous fistula, Endovascular, Ethanol embolization

Neurosurgery 52:1111-1116, 2003 DOI: 10.1227/01.NEU.0000058232.09835.49 www.neurosurgery-online.com

A fistula is a direct connection between an artery and a vein. Carotid-cavernous fistulae (CCFs) form secondary to abnormal communications between the carotid artery and cavernous sinus and may present with pulsatile exophthalmos, bruit, diplopia, chemosis, pain, increased intraocular pressure (IOP), and/or erythema. Barrow et al. (2) classified CCFs as direct (Type A) or dural (indirect; Types B–D). Direct CCFs are high-flow fistulae caused by traumatic or aneurysmal rupture of the cavernous carotid artery into the cavernous sinus. Indirect or dural CCFs are low-flow states caused by anomalous connections between the cavernous sinus and smaller-caliber branches of the cavernous carotid artery, external carotid artery, or both. These lesions present most commonly in perimenopausal women and have been attributed to a number of causes, including congenital abnormalities, spontaneous venous hypertension from thrombosis, atherosclerosis, angiopathy from collagen vascular disorders, syphilitic arteritis, and iatrogenic vessel damage (13). Indirect CCFs occasionally resolve spontaneously or respond to serial manual carotid compression. Treatment is indicated for worsening symptoms, medically refractory increased IOP, or the presence of angiographic features associated with increased risk of hemorrhage, such as pseudoaneurysm, large varices, or cortical venous drainage (6). Man-

Copyright © Congress of Neurological Surgeons. Unauthorized reproduction of this article is prohibited.

agement options include balloon occlusion; arterial embolization of external carotid artery (ECA) and internal carotid artery (ICA) feeders with polyvinyl alcohol, 50% dextrose, or cyanoacrylates; transvenous coil embolization of the cavernous sinus; surgical obliteration; and stereotactic radiosurgery (1, 2, 5, 8, 9, 11, 16). When indirect CCFs cause deterioration in visual function from increased IOP, urgent treatment is necessary. We present a retrospective analysis of six patients treated with absolute ethanol embolization of the cavernous carotid supply to indirect CCFs, resulting in an immediate decrease in IOPs and symptomatic improvement.

PATIENTS AND METHODS

Between November 1999 and March 2001, six patients were referred from the neuro-ophthalmology service for indirect CCFs with increased IOP and deterioration in visual acuity and/or cranial nerve function. All patients had symptom progression despite maximal medical therapy, including brimonidine (Alphagan; Allergan, Inc., Irvine, CA), timolol (Timoptic; Merck & Co., West Point, PA), and other medications. *Table 1* summarizes patient demographics, severity of visual and cranial nerve impairment, and IOP at the time of embolization and at the most recent follow-up after embolization, as well as the vascular supply of each CCF and the sequence of endovascular procedures performed. Patients 1 to 5 had symptom onset a few weeks before presentation; Patient 6 presented within 24 hours of symptom onset. A photograph of Patient 6 at presentation demonstrates common signs of CCF, including severe conjunctival erythema, chemosis, complete bilateral IIIrd nerve palsies, and disconjugate gaze as a result of bilateral IVth and VIth nerve palsies (*Fig. 1A*).

Each patient underwent angiography of both the ECA and ICA to delineate blood supply to the fistula (*Fig. 2A*). The first patient underwent embolization with conscious sedation only but experienced significant discomfort during alcohol injection, prompting the use of general anesthesia with neurophysiological monitoring for the remainder of the patients. In all patients, a transvenous embolization was first attempted by catheterization of the inferior or superior petrosal sinuses. This could not be achieved in these patients, and a superior ophthalmic surgical exposure or transfacial vein catheterization was forgone in lieu of the following technique. The contribution from the ECA in Type C and D lesions was treated with polyvinyl alcohol particulate embolization of the ECA supply. Type B and D lesions were managed by advancing a nondetachable silicone balloon (Endeavor; Boston Scientific/Medi-tech, Natick, MA) into the horizontal segment of the ICA just distal to the branches supplying the fistula but proximal to the ophthalmic artery. A microcatheter (TurboTracker 18; Boston Scientific/Target, Fremont, CA) was inserted through the 7-French study catheter and alongside the balloon catheter into the posterior genu of the cavernous ICA (proximal to the balloon). The balloon was inflated, and an angiogram through the microcatheter was performed to best visualize the cavernous ICA branches feeding the CCFs with no changes in the

TABLE 1. Summary of six cases of ethanol embolization of the cavernous internal carotid artery supply to indirect carotid-cavernous fistulae[a]

Patient no.	Age (yr)/ sex and follow-up (mo)	VA	IOP (mm Hg)	CN deficits	Blood supply	Embolization procedures	Angiographic outcome	Outcomes at latest follow-up	Complication
1	72/F, 12	Hand motion	29/12	R VI	R Cav ICA	I: R ICA	Complete obliteration	IOP 15, VA 20/15, CN VI improving, Xa only med	Cavernous sinus thrombosis
2	57/M, 15	20/30	24/14	R VI	B Cav ICAs and ECAs, ophthalmic	I: R ECA II: B Cav, ICAs	Residual ophthalmic supply	IOP <20, VA 20/20, CN VI nl, Al dose ↓	None; received radiosurgery after embolization
3	77/F, 15	Hand motion	28/13	R VI	R Cav ICA	I: R ICA	Complete obliteration	IOP 20, VA 20/30, CN VI nl, no meds	None
4	67/F, 17	20/70, 20/100	12/22	Ptosis	B Cav ICAs, L MMA and IMax	I: L ICA II: L IMax	Complete obliteration	IOP 16/18, VA 20/20 B, CNs nl, no meds	None
5	55/M, 30	Count fingers B	37/33	B III, IV, VI	L Cav ICA, L AMA, R IMax	I: L AMA II: L ICA	ICA flow obliterated, slight residual supply from R	IOP 16/13, VA 20/20 B, CNs nl, Xa only med	None; received radiosurgery after embolization
6	77/F, 38	20/100 B	60s both eyes	B III, IV, VI	B Cav ICAs, B MMAs	I: L ICA and L MMA	Minimal residual R ECA supply	IOP 14/14, VA 20/40 and 20/60, R III and VI deficits, on Al/Tr	None; received radiosurgery after embolization

[a] VA, visual acuity; IOP, intraocular pressure; CN, cranial nerve; R, right; Cav, cavernous; ICA, internal carotid artery; Xa, Xalatan (latanoprost; Pharmacia & Upjohn, Kalamazoo, MI); med, medication; B, bilateral; ECA, external carotid artery; nl, normal; Al, Alphagan (brimonidine; Allergan, Inc., Irvine, CA); L, left; MMA, middle meningeal artery; IMax, internal maxillary artery; AMA, accessory meningeal artery; Tr, Trusopt (dorzolamide; Merck & Co.).

Copyright © Congress of Neurological Surgeons. Unauthorized reproduction of this article is prohibited.

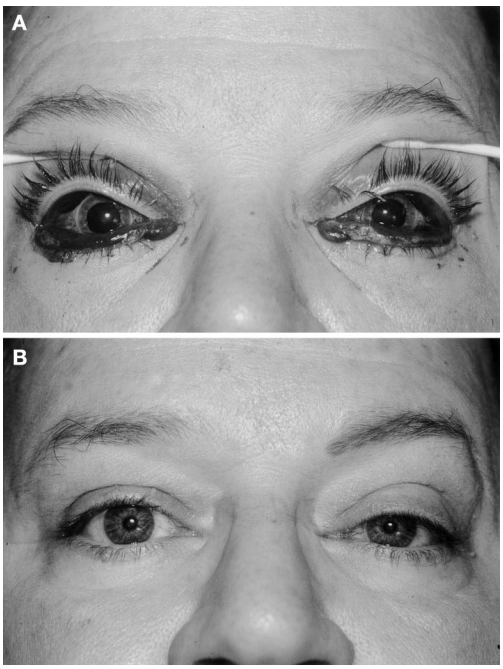


FIGURE 1. A *facial photograph of Patient 2 showing bilateral conjunctival and periorbital edema and hemorrhage before embolization. Note that the pupils are pharmacologically dilated.* B, *photograph of the same patient after embolization.*

size or flow to the cavernous ICA branches (*Fig. 2, B* and *C*). Angiography was repeated with repositioning of the balloon catheter and the endhole catheter several times until the optimal placement was achieved. In an effort to keep the ethanol as concentrated as possible, because dilution diminishes the effect, we did not mix contrast medium with the ethanol. To gauge a safe rate and amount of ethanol to be injected, we used a contrast injection of the same-size bolus of ethanol with the same-size syringe (usually a 1-ml syringe) before ethanol injection. We proceeded only after confirming minimal or no reflux down the ICA or into potentially dangerous anastamoses of the inferolateral trunk and ophthalmic artery. Individual injection volumes usually ranged between 0.3 and 1.0 ml, and total ethanol volume per ICA ranged between 3 and 5 ml. After each ethanol injection and before balloon deflation, vigorous irrigation was undertaken by injection of 20 ml of saline through the microcatheter while aspirating from the sideport of the guiding catheter. Repeat angiography via the microcatheter during balloon occlusion was then used to judge the progress of embolization. A 3- to 5-minute delayed angiogram was typically needed to confirm satisfactory vessel occlusion, because the effect of the alcohol was slightly delayed (*Fig. 2D*). The balloon occlusion time usually ranged from 1 to 5 minutes, with multiple balloon inflations being necessary because any neurophysiological monitoring changes prompted balloon deflation. In Patient 6, electroencephalographic and somatosensory evoked potential monitoring showed no changes during prolonged balloon occlusion,

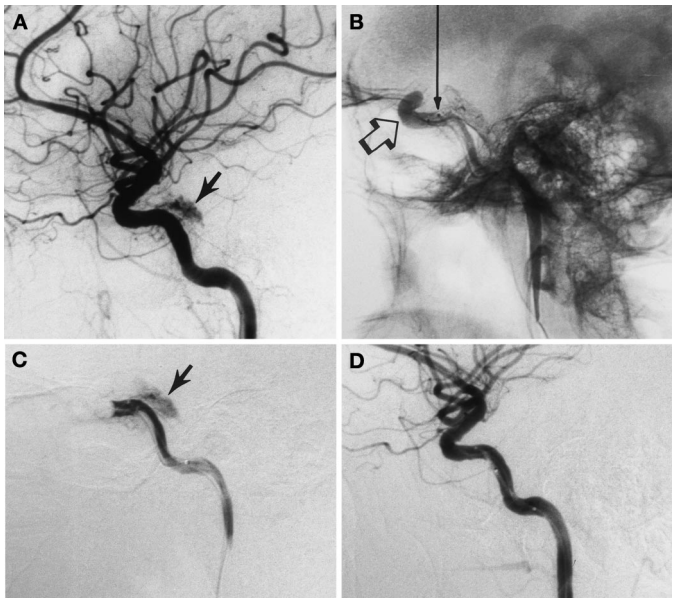


FIGURE 2. A, *lateral projection of a left ICA angiogram in a patient with "indirect" CCF. Many small branches off the cavernous portion of the ICA fill the cavernous sinus (*arrow*).* B, *angiogram showing occlusion balloon (*open arrow*) inflated in the ICA with the tip of the microcatheter (*long arrow*) as close to the fistula as possible.* C, *lateral projection of a left ICA angiogram after contrast injection through the microcatheter. The fistula to the cavernous sinus (*arrow*) is well filled. Ethanol was then injected through the microcatheter.* D, *lateral projection of a left ICA angiogram after embolization and deflation of the balloon. The ICA remains patent, and the fistula is no longer filling.*

prompting us not to deflate the balloon while waiting for the follow-up angiogram, which led to a total occlusion time of nearly 15 minutes with no ischemic sequelae.

RESULTS

All patients showed immediate and sustained decreases in IOP except for one, who normalized in a delayed manner (*Table 1*). Long-term follow-up (mean duration, 21 mo; range, 12–38 mo) showed visual acuity improvement to 20/30 or better in five patients, whereas Patient 6 improved from 20/100 bilaterally to 20/40 in the right eye and 20/60 in the left. IOP remained below 20 mm Hg in all six patients, with termination of medical therapy in two patients and a significant reduction in medical therapies in four patients. Cranial nerve deficits resolved in four patients, whereas two showed improvement in extraocular muscle function. Angiography on completion of the embolization procedures revealed complete obliteration of the indirect CCFs in three patients, whereas there was minimal residual filling in the other three. We did not perform follow-up angiography on these patients, given the continued clinical improvement of all six individuals (*Fig. 1B*). For the three patients with residual flow and incomplete symptom resolution after embolization, we performed stereo-

Copyright © Congress of Neurological Surgeons. Unauthorized reproduction of this article is prohibited.

tactic radiosurgery after embolization, which resulted in further clinical improvement.

DISCUSSION

The first successful treatment of a possible CCF was reported by Travers, a British surgeon, who described a "thrilling tumor in which pulsation ceased upon compression of the vascular structures of the neck" (10). He performed ligation of the common carotid artery, with a good clinical response. Many surgical approaches have since been introduced to treat CCFs, including combined muscle embolization through the cervical carotid with cavernous ICA surgical ligation by Hamby (10), cavernous ICA sacrifice under hypothermic arrest by Parkinson (17), and surgical embolization of the cavernous sinus with hemostatic agents by Mullan (16). Endovascular treatment of direct CCFs has moved to the forefront since the introduction of detachable balloons and is now the definitive treatment of high-flow traumatic or aneurysmal lesions because of its safety and efficacy (4, 5, 19). Indirect CCFs (Types B–D), however, are rarely, if ever, amenable to balloon occlusion or particulate embolization, because they receive arterial supply from multiple ECA and/or cavernous ICA branches too numerous and too small for balloon occlusion. Close clinical observation is indicated for a patient with stable symptoms because of the spontaneous resolution rate of these lesions, ranging from 10 to 74%, and the 30% response rate to serial carotid compression (12). However, indirect CCFs require vigilant observation, because they can cause rapid deterioration in vision and cranial nerve function as a result of retrograde filling of the superior ophthalmic vein and subsequent increasing IOP.

Barrow et al. (2) classified indirect CCFs on the basis of arterial supply as Type B (ICA alone), Type C (ECA alone), or Type D (both ICA and ECA). Endovascular management of indirect CCFs involving the cavernous ICA (Types B and D) has historically involved a transvenous approach to the cavernous sinus and/or embolization of ECA branches alone because of inability to catheterize the tiny ICA branches along with fear of stroke from reflux of embolic agents beyond the cavernous ICA. Barrow's initial report of 14 patients included 12 indirect CCFs. Five resolved spontaneously, one patient died of unrelated causes, and one became blind without treatment. The remaining five patients received particulate embolization of the ECA, with four good results and one attempt that failed because of stroke during angiography. Debrun et al. (5) published a larger series involving 32 indirect CCFs and found a 40% treatment failure rate with ECA embolization alone for Type D lesions. Another report indicated that radiation therapy after incomplete embolization of ECA branches was successful in eight of nine patients with Type D CCFs (11). Halbach et al. (8) provided one of the few reports of embolization of cavernous ICA branches involving four indirect CCFs. They used polyvinyl alcohol in two patients, 50% dextrose in one patient, and isobutyl cyanoacrylate in one patient, with complete cure in all four patients during a period of 5 to 48 months of follow-up. Despite the promising results of that series, little support has followed in favor of attempting embolization of ICA branches.

Indirect CCF embolization via a transvenous approach through the superior ophthalmic vein either surgically or endovascularly or via the facial vein or through the inferior petrosal sinus, superior petrosal sinus, or cortical draining vein using an endovascular approach has been successful (3, 9, 14, 15). Halbach et al. (7) report 81% complete cure and 19% partial cure in a series of 54 patients treated with transvenous embolization using balloons, liquid adhesives, and/or coils, with a 4% complication rate. Patients in our series had either no angiographic evidence of an inferior petrosal sinus (unable to catheterize the inferior petrosal sinus despite numerous attempts) or severe angulation of the superior ophthalmic vein, placing it at risk for catheter perforation and retro-orbital hemorrhage. These findings made a transvenous approach impossible. We believed that the presence of a distinct region of feeders from the cavernous carotid would allow our technique to be a safe and effective alternative.

Yakes et al. (20) first reported the use of absolute ethanol in the endovascular management of cerebrovascular malformations in 1997. His group found that absolute ethanol caused immediate endothelial denudation and fractured the vessel wall to the level of the internal elastic lamina without causing angionecrosis or brain necrosis. This resulted in vessel sclerosis without recanalization. Absolute ethanol, however, is a dangerous embolization agent, given the immediate and permanent vessel occlusion it causes if injected into normal brain. The series by Yakes et al. (20) reported complications (only one permanent) in 8 of 17 patients, including hemiparesis, numbness, cognitive dysfunction, and hemianopsia. We chose alcohol for our patients because of its ability, as a liquid, to penetrate the cavernous ICA branches that were not large enough for direct microcatheterization and particulate embolization while minimizing the risk of embolism. However, Phatouros et al. (18) reported a patient with diabetes insipidus who required deamino-8-D-arginine vasopressin therapy for 3 months after injection of 50% dextrose and pure ethanol into an enlarged meningohypophyseal trunk supplying a CCF in which the inferior hypophyseal artery did not opacify during selective preembolization meningohypophyseal injection. In addition to pituitary dysfunction from meningohypophyseal occlusion via the hypophyseal artery, occlusion of the inferolateral trunk can lead to Cranial Nerve III, IV, and/or VI neuropathies or even blindness because of potential connections with the eye via the deep recurrent ophthalmic artery from the inferolateral trunk. A nondetachable balloon was inflated proximal to the ophthalmic artery during embolization of the cavernous ICA feeders to protect the eye and brain from ischemic injury. Venous thrombosis was unlikely, because any ethanol that entered the CCF venous drainage system was diluted and inactivated. We experienced only one procedure-related complication involving cavernous sinus thrombosis causing transient symptom exacerbation that eventually resolved over 4 weeks. This patient (Patient 1) had

Copyright © Congress of Neurological Surgeons. Unauthorized reproduction of this article is prohibited.

possible ethanol-induced occlusion of venous drainage from the cavernous sinus. Once collateral venous outflow developed over a few weeks, the worsened symptoms resolved.

Five of the six patients experienced an immediate reduction in IOP and improvement of visual acuity. We performed adjunctive embolization of ECA branches at the time of or within a few days of the cavernous carotid embolization for all Type D lesions to minimize the risk of recurrence by establishment of new collateral branches. For the three patients with residual flow and incomplete symptom resolution after embolization, we performed stereotactic radiosurgery after embolization, with further clinical improvement. We have not obtained follow-up angiography, given no clinical recurrences, although concern for long-term ICA patency after ethanol embolization may prompt us to obtain a computed tomographic angiogram or magnetic resonance angiogram in the future. We conclude that absolute ethanol embolization of cavernous ICA branches supplying indirect CCFs in association with temporary distal balloon protection is safe and effective in providing immediate relief of rapidly declining visual and extraocular motility function and rising IOPs. This technique has become an integral tool in the multimodal management of this complex vascular anomaly.

REFERENCES

1. Barcia-Salorio JL, Soler F, Hernandez G, Barcia JA: Radiosurgical treatment of low flow carotid-cavernous fistulae. **Acta Neurochir Suppl (Wien)** 52: 93–95, 1991.
2. Barrow DL, Spector RH, Braun IF, Landman JA, Tindall SC, Tindall GT: Classification and treatment of spontaneous carotid-cavernous fistulas. **J Neurosurg** 62:248–256, 1985.
3. Bellon RJ, Liu AY, Adler JR Jr, Norbash AM: Percutaneous transfemoral embolization of an indirect carotid-cavernous fistula with cortical venous access to the cavernous sinus. **J Neurosurg** 90:959–963, 1999.
4. Debrun GM: Treatment of traumatic carotid-cavernous fistula using detachable balloon catheters. **AJNR Am J Neuroradiol** 4:355–356, 1983.
5. Debrun GM, Viñuela F, Fox AJ, Davis KR, Ahn HS: Indications for treatment and classification of 132 carotid-cavernous fistulas. **Neurosurgery** 22:285–289, 1988.
6. Halbach VV, Hieshima GB, Higashida RT, Reicher M: Carotid cavernous fistulae: Indications for urgent treatment. **AJNR Am J Neuroradiol** 8:627–633, 1987.
7. Halbach VV, Higashida RT, Hieshima GB, Christopher FD: Endovascular therapy of dural fistulas, in Viñuela F, Halbach VV, Dion JE (eds): *Interventional Neuroradiology: Endovascular Therapy of the Central Nervous System*. New York, Raven Press, 1992, pp 29–50.
8. Halbach VV, Higashida RT, Hieshima GB, Hardin CW: Embolization of branches arising from the cavernous portion of the internal carotid artery. **AJNR Am J Neuroradiol** 10:143–150, 1989.
9. Halbach VV, Higashida RT, Hieshima GB, Hardin CW, Pribani H: Transvenous embolization of dural fistulas involving the cavernous sinus. **AJNR Am J Neuroradiol** 10:377–383, 1989.
10. Hamby WB: *Carotid-Cavernous Fistula*. Springfield, Charles C Thomas, 1966.
11. Hasuo K, Mizushima A, Matsumoto S, Uchino A, Uehara S, Miyoshi M: Type D dural carotid-cavernous fistula: Results of combined treatment with irradiation and particulate embolization. **Acta Radiol** 37:294–298, 1996.
12. Higashida RT, Hieshima GB, Halbach VV, Bentson JR, Goto K: Closure of carotid cavernous sinus fistulae by external compression of the carotid artery and jugular vein. **Acta Radiol Suppl** 369:580–583, 1986.
13. Lewis AI, Tomsick TA, Tew JM Jr: Carotid-cavernous fistulas and intracavernous aneurysms, in Wilkins RH, Rengachary SS (eds): *Neurosurgery*. New York, McGraw-Hill, 1996, vol 3, pp 2529–2540.
14. Miller NR, Monsein LH, Debrun GM, Tamargo RJ, Nauta HJW: Treatment of carotid-cavernous sinus fistulas using a superior ophthalmic vein approach. **J Neurosurg** 83:838–842, 1995.
15. Mounayer C, Poitin M, Spelle L, Moret J: Superior petrosal sinus catheterization for transvenous embolization of a dural carotid cavernous sinus fistula. **AJNR Am J Neuroradiol** 23:1153–1155, 2002.
16. Mullan S: Treatment of carotid-cavernous fistulas by cavernous sinus occlusion. **J Neurosurg** 50:131–144, 1979.
17. Parkinson D: Carotid cavernous fistula: Direct repair with preservation of the carotid artery—Technical note. **J Neurosurg** 38:99–106, 1973.
18. Phatouros CC, Higashida RT, Malek AM, Smith WS, Dowd CF, Halbach VV: Embolization of the meningohypophyseal trunk as a cause of diabetes insipidus. **AJNR Am J Neuroradiol** 20:1115–1118, 1999.
19. Serebinko FA: Balloon catheterization and occlusion of major cerebral vessels. **J Neurosurg** 41:125–145, 1974.
20. Yakes WF, Krauth L, Ecklund J, Swengle R, Dreisbach JN, Siebert CE, Baker R, Miller M, Vanderark G, Fullagar T, Prenger E: Ethanol endovascular management of brain arteriovenous malformations: Initial results. **Neurosurgery** 40:1145–1153, 1997.

COMMENTS

This article describes the usefulness of ethanol infusion into the cavernous internal carotid artery (ICA) with distal temporary balloon occlusion for treatment of indirect carotid-cavernous fistulae (CCFs). Transvenous coil embolization of the cavernous sinus is a safe and effective method of treatment for these lesions, but lack of venous access can preclude this approach. In such cases or when residual fistula is apparent, one can be left with the prospect of selectively catheterizing small and tortuous cavernous ICA branches, which may be the dominant supply to these fistulae. The authors describe a novel solution to this problem, with marked improvement in symptomatology and intraocular pressure (IOP) measurements in all six of their patients. Despite their encouraging results, the number of patients is small, and the potential for injury to neural structures as a result of ethanol infusion into the cavernous ICA exists. We would advocate conservative measures, such as carotid compression and/or palliative embolization of external carotid artery feeders, and leave the described procedure as a last resort until the results can be substantiated by a larger patient experience.

Perry P. Ng
Randall T. Higashida
Interventional Neuroradiologists
San Francisco, California

Koebbe et al. report a series of six patients with indirect CCFs who underwent endovascular treatment with ethanol embolization of ICA feeding arteries. Ethanol injection is potentially dangerous, but it may be an attractive alternative to the transvenous approach. In this small series, the authors have shown that ethanol embolization can be done safely. However, some questions about this technique remain. First, only three of the six patients had fistula obliteration and symptom resolution with embolization. The other three patients had incomplete resolution of the CCFs, demonstrated by residual flow on angiography after embolization and continued symptoms. These patients went on to have further treat-

Copyright © Congress of Neurological Surgeons. Unauthorized reproduction of this article is prohibited.

ment with stereotactic radiosurgery. These results raise questions about the efficacy of ethanol embolization for indirect CCFs. Secondly, the mean follow-up period of 21 months does not assure us that this treatment is truly durable. Larger case series and longer clinical follow-up in the future should shed light on these issues.

Mark R. Harrigan
L. Nelson Hopkins
Buffalo, New York

Koebbe et al. describe their experience in the treatment of six patients with worsening visual acuity treated with embolization of their CCFs with absolute ethanol and balloon occlusion. Five of the six patients in their group experienced immediate, sustained relief and reduction of IOP. One patient, however, did develop cavernous sinus thrombosis, which caused a worsening of the symptoms, albeit gradual, and these improved gradually over time. There is no question that absolute ethanol is a very effective embolic agent, as has been shown in peripheral interventional radiology.

The risk of using this agent intracranially is not insignificant, and I think that the complications that Yakes et al. (1) specified were quite high in terms of neurological events. There are other methods that carry less risk than using balloon occlusion in ethanol infusion and have very good results and perhaps a lower incidence of cavernous sinus thrombosis. In addition, the effects of the alcohol on the carotid artery in the long term are as yet unknown.

This is an extremely dangerous procedure; certainly, if no other options are available, this is a good alternative. However, I think catheterization via either the inferior petrosal sinus or transvenous approach, which carry much less risk, should be undertaken before quite so radical a procedure is performed, as the authors have recommended. The effects of alcohol on the central nervous system and peripheral nerves are well known to the neurological and neurosurgical community and cannot be taken lightly.

Robert H. Rosenwasser
Philadelphia, Pennsylvania

---

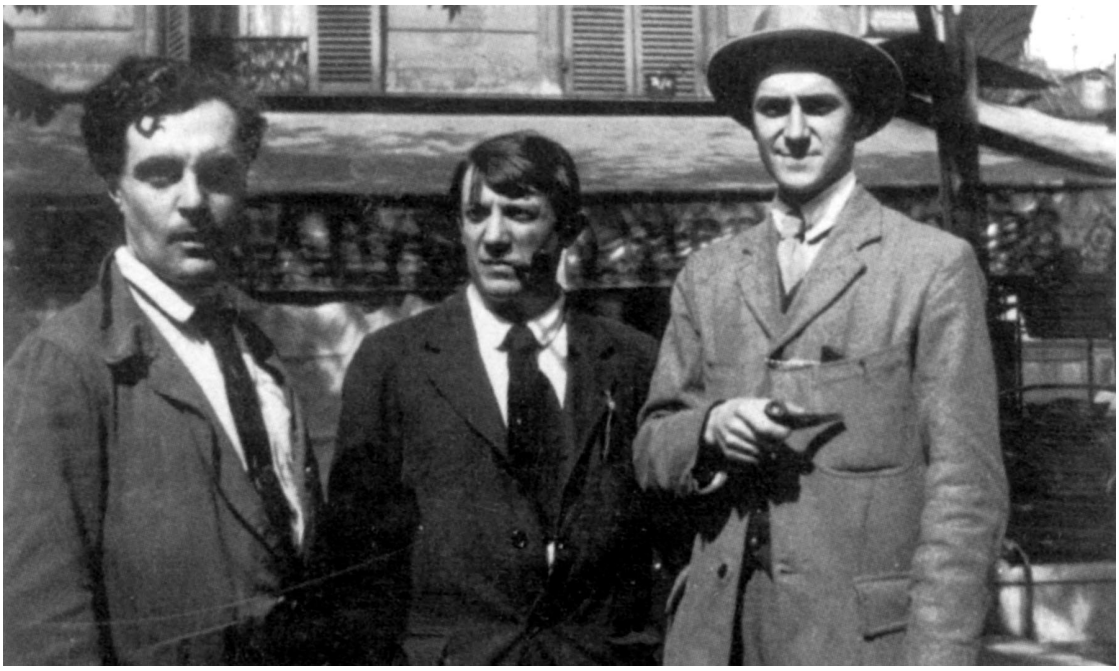
1. Yakes WF, Krauth L, Ecklund J, Swengle R, Dreisbach JN, Siebert CE, Baker R, Miller M, Vanderark G, Fullagar T, Prenger E: Ethanol endovascular management of brain arteriovenous malformations: Initial results. **Neurosurgery** 40:1145–1153, 1997.

The authors report six patients with indirect CCFs who underwent embolization of small feeding branches from the ICA by use of absolute ethanol. Although this agent has been used previously to treat arteriovenous malformations and venous angiomas, its use for CCFs is novel. An occlusion balloon catheter prevented embolization of alcohol into distal branches of the ICA, including the ophthalmic artery.

The clinical outcomes in these patients are well documented, but it will also be necessary to obtain long-term angiographic follow-up to support the safety and efficacy of this technique. Delayed angiography not only will confirm the durability of the method but also can refute concerns about possible sclerosis of the ICA leading to stenosis or occlusion. Because the patients tolerated temporary balloon occlusion of up to 15 minutes during the procedure, it is possible that subacute or chronic occlusion might remain clinically silent.

As the authors state, multiple options exist for the endovascular treatment of indirect CCFs. The technique reported in this article adds to that array and will most likely assume an increasing role in the management of these vascular anomalies.

Arun Paul Amar
Los Angeles, California



Photograph (left to right) *of Modigliani, Pablo Picasso, and André Salmon.*

Copyright © Congress of Neurological Surgeons. Unauthorized reproduction of this article is prohibited.