Treatment of Residual Facial Arteriovenous Malformations after Embolization with Percutaneous Cryotherapy

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ABSTRACT

This report presents 4 patients (mean age, 22 y; range, 17–26 y) with facial arteriovenous malformations (AVMs) who underwent arterial ethanol and *N*-butyl cyanoacrylate embolization followed by percutaneous cryoablation of residual inaccessible AVMs. After the procedure, minor complications classified as type B according to the Society of Interventional Radiology (SIR) classification system occurred in 75% (3/4) of patients. One patient achieved 90% obliteration of AVM, and 3 patients had complete obliteration of AVM at 1-year follow-up. This reports shows that percutaneous ablation may be a viable treatment option for residual facial AVMs after ethanol and glue embolization.

ABBREVIATIONS

AVM = arteriovenous malformation, NBCA = *N*-butyl cyanoacrylate

An arteriovenous malformation (AVM) is believed to result from an error in vascular development during embryogenesis. The AVM nidus is defined as an abnormal connection between the arterial and venous system without a normal intervening capillary bed (1). AVMs are reported to occur in 0.1% of the general population. The clinical manifestations of AVMs include pain, ulceration, bleeding, destruction and enlargement of tissue, disfigurement, and a high cardiac output state (2). The natural course of an AVM is progressive, invasive, and destructive; thus, early intervention is necessary (2). AVMs are typically treated with embolization of the AVM nidus, followed by surgical excision if needed, and follow-up cosmetic surgery (3,4).

Superselective catheterization or direct puncture of the AVM nidus and the use of ethanol as a permanent embolic agent have shown good clinical and radiologic obliteration rates with an acceptable risk and morbidity (5). Complication rates for ethanol embolization of AVMs are 10%–30%, and the most common complications are local tissue injuries, such as skin blistering or necrosis, and peripheral nerve palsy (6,7). Furthermore, with serial ethanol treatments, the arterial feeders to the nidus and AVM decrease in size and supply the normal surrounding tissues. During this stage of the embolization, injection of ethanol can be dangerous because of the high likelihood of embolization of normal tissue. Given this, we sought other treatment options to obliterate the remaining small residual AVM nidus and prevent damage to normal tissue and potential skin complications.

AVMs of the face can cause cosmetic, functional, and psychological problems and can be life-threatening in some cases. Facial AVMs are often related to ophthalmic or internal maxillary arteriovenous shunts. This can make embolization of these shunts dangerous secondary to the communication of the external carotid artery with the internal carotid through known anastomoses. Furthermore, potential inadvertent embolization of the ophthalmic artery or vasa nervosa of a cranial nerve can cause blindness or a permanent nerve deficit, respectively. Given these potential specific complications to the facial region in the treatment of AVMs, percutaneous ablative technologies may have a role in improving outcome and safety.

Novel techniques to treat venous malformations are described in the literature, including radiofrequency ablation, cryoablation, laser ablation, and magnetic resonance (MR)–guided high-intensity focused

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ultrasound ablation (8). A single case report describes the use of radiofrequency ablation for treatment of a renal AVM (9). A challenge to performing a successful ablation of a vascular lesion is the heat-sink effect from blood flow into the lesion (10). Blood flow can limit cell damage in cold and heat ablative therapies. To limit the heat-sink effect, serial embolizations of the AVM with ethanol were performed until most of the blood flow to the lesion was occluded. The decision to use cold versus heat ablation was based on favorable literature suggesting better visualization of the ablation zone, better cosmetic healing, a predictable ablation zone, and nerve regeneration potential. In contrast, with heat ablation there is a considerable risk of burns to the skin and other facial structures given the superficial location of facial AVMs. Furthermore, with ablative therapies, the endothelial cells of the AVM are destroyed, and angiogenesis factors will not be secreted to stimulate a neovascular response that may allow for a permanent cure of the AVM. This case series highlights a novel therapeutic approach of using percutaneous cryoablation for the treatment of residual inaccessible facial AVMs.

CASE REPORTS

Institutional review board approval was obtained for this report. The vascular anomalies database was retrospectively searched from January 2005 to July 2015 for peripheral AVMs treated by percutaneous cryoablation. Patient demographic data, clinical charts, indications for treatment, radiographic images, procedure notes, hospital course after the procedure, and follow-up were reviewed. Complications were graded according to the Society of Interventional Radiology (SIR) classification system for complications by outcome.

Two female and 2 male patients with a mean age of 22 years (range, 17–26 y) with facial AVMs underwent percutaneous cryoablation. All patients presented with pain and enlargement of the AVM, and 2 patients presented with bleeding. AVMs were located in the subcutaneous soft tissues of the forehead in 2 cases, in the chin in 1 case, and in the left cheek in 1 case. Schobinger classification was 3 in 2 cases and 2 in 2 cases. Mean size of the initial AVM nidus was 3.5 cm (range, 1.5–6 cm). Arterial feeders to the AVMs were from the ophthalmic artery, superficial temporal artery, and facial artery with variable venous drainage.

TECHNIQUE

Embolization

After standard preparation and induction of general anesthesia, retrograde access of the right common femoral artery was obtained, and a 5-F MPC guide catheter (Depuy Neurovascular, Inc, Raynham, Massachusetts) was advanced into the right and left internal and external carotid arteries. Angiography of the carotid arteries was performed. Arterial feeders to the AVM were identified, and embolization with ethanol and *N*-butyl cyanoacrylate (NBCA) was performed using a microcatheter from an intraarterial perinidal location. In most cases, absolute (99%) ethanol was used, but in some sessions, diluted (50%–70%) ethanol with nonionic contrast medium iopamidol 61% (Isovue-300; Bracco Diagnostics, Inc, Milan, Italy) was used. Flow control was used with placement of a Scepter XC balloon (MicroVention, Inc, Tustin, California) in the arterial inflow or venous outflow Berenstein occlusion balloon (Boston Scientific, Marlborough, Massachusetts) before the injection of ethanol to prolong contact time with the endothelium. In the 8 embolization sessions, an intraarterial approach (6 sessions), occlusion of the venous outflow (4 sessions), and a percutaneous approach (5 sessions) were used. Most of the embolization sessions (7 sessions) included a combination approach. The embolization sessions were staged at 12-week time intervals until the AVM was obliterated or the authors thought the arterial feeders or nidal vessels were too small to treat from an intraarterial or percutaneous approach because of the risk of embolization of normal tissue and potential skin complications. Cryoablation was performed in a salvage attempt to treat residual AVMs given the possible rate of recurrence, often with complex architecture and extensive vascular recruitment.

Cryoablation

The cryoablation procedure was performed under general anesthesia, and the skin area overlying the AVM was prepared and draped in a sterile fashion. The number and size of the probes were determined by the size and shape of the residual AVM. The mean size of the residual AVM nidus was 1.5 cm (range, 1–2 cm); therefore, 2 Endocare PerCryo PERC-17 probes (HealthTronics, Inc, Austin, Texas) were determined to be sufficient to ablate lesions in 2 patients with residual AVMs measuring 2 cm. In the other 2 patients with a smaller residual AVM nidus, 1 cryoprobe was determined to be sufficient to cover the entire lesion. The PERC-17 probe used in the procedures has a $-40^{\circ}C$ isotherm with 1.4 cm diameter and 3.5 cm length. Ultrasound guidance was used to place the probes in the lesion 1.5 cm apart in patients with a 2.0-cm residual AVM nidus so that the entire lesion was covered. A single probe was placed in the center of the lesion in patients with a smaller residual AVM nidus. The probes were placed parallel to the skin surface to avoid formation of the ice ball at the skin surface. Before freezing, the skin area overlying the AVM was protected by injecting a sterile saline solution into the subcutaneous tissue to raise a 1.0- to 1.5-cm margin between the lesion and the skin surface. Furthermore, placing a warm saline solution in sterile gloves on the skin surface