Endovascular Therapy of Intractable Epistaxis Complicated by Carotid Artery Occlusive Disease

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Summary: Three cases of intractable spontaneous posterior epistaxis refractory to nasal packing and complicated by ipsilateral carotid artery occlusive disease were successfully treated with internal maxillary artery occlusion with microcoils. There were no complications and no recurrent episodes of epistaxis at a mean follow-up of 12 months. The presence of ipsilateral carotid artery disease requires modification of standard distal internal maxillary artery embolization because of the recruitment of external carotid to internal carotid and external carotid to ophthalmic artery collateral pathways, with subsequent risk of particle embolization of these arterial distributions. Proximal internal maxillary artery microcoil embolization eliminates this risk and is equivalent to surgical internal maxillary artery ligation. This procedure also provides additional information about the internal carotid artery collateral circulation.

Index terms: Epistaxis; Arteries, carotid, external; Interventional neuroradiology

Epistaxis is a common otolaryngologic emergency, with 3% of the population seeking medical treatment at some point in their lifetime. The cause of epistaxis is varied and includes trauma, tumor, coagulopathy, and idiopathic spontaneous causes. Although anterior septal epistaxis is easily accessible to direct treatment, 20% of cases arise posteriorly, limiting direct access. Conservative packing may fail in as many as 50% of patients, and these cases of intractable posterior epistaxis may require more aggressive treatment consisting of either traditional surgical arterial ligation or intraarterial embolization (1).

Percutaneous intraarterial embolization for intractable epistaxis has made significant advances since its introduction by Sokoloff et al in 1974, and it has been demonstrated to be an effective and safe primary treatment in recent years (2–6). Selective catheterization and embolization of the distal internal maxillary artery branches with small particles offer a distinct advantage over more proximal surgical arterial ligation in that it lessens the risk of rebleeding attributable to distal collateral circulation and does not block access to the internal maxillary area in the event that retreatment is necessary (7, 8). However, the presence of ipsilateral internal carotid artery (ICA) stenosis or occlusion may preclude the use of small embolic particles for epistaxis therapy because of dangerous external carotid artery–to–ICA anastomoses with the subsequent risk of stroke or retinal artery occlusion. This report demonstrates that successful endovascular therapy of intractable posterior epistaxis still may be performed, despite the presence of internal carotid occlusive disease, by modifying standard embolization techniques with the use of more proximal embolic materials.

Materials and Methods

Three patients (mean age, 70 years; range, 60 to 79 years) of 32 referred for endovascular treatment of intractable epistaxis were found to have severe stenosis or occlusion of the ipsilateral ICA on preembolization angiography. These three patients had between one and four episodes of severe epistaxis that were refractory to posterior nasal packing. There was no history of surgery or embolization.

Angiography of the ipsilateral external carotid and contralateral common carotid arteries was performed to evaluate cerebral perfusion as well as to search for external carotid arterial anastomoses to the ophthalmic artery and the internal carotid system. The degree of safety of endovascular therapy was assessed by evaluation of visible external carotid–to–internal carotid anastomoses, both to avoid cerebral/ophthalmic embolization and to prevent compromise of cerebral perfusion via vital collaterals in the face of ICA occlusive disease. Therapeutic embolization of the internal maxillary artery (IMA) was performed using a coaxial system: 6F introducing catheter in the common carotid artery with a Tracker 18 (Target Therapeutics) microcatheter. Embolization was performed by

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placing platinum microcoils into the pterygopalatine segment of the IMA to complete occlusion of the distal IMA. Postembolization external carotid angiography was performed to evaluate the need for ipsilateral facial artery embolization.

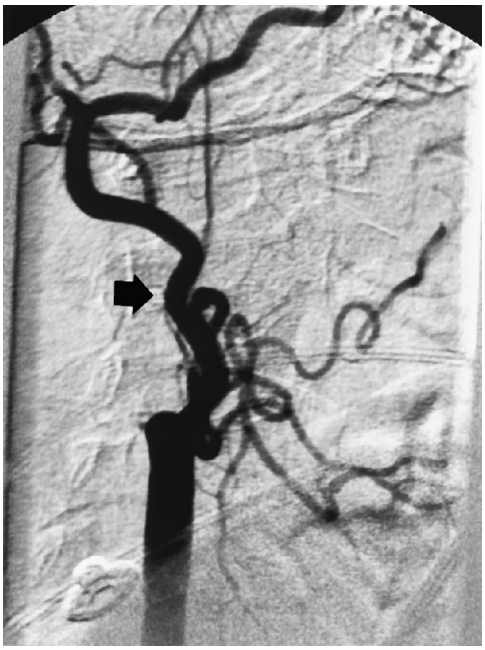

Fig 1. Right internal carotid occlusion is present ipsilateral to epistaxis. *Arrow* indicates right external carotid.

Results

Diagnostic common carotid angiography ipsilateral to the epistaxis demonstrated a high-grade stenosis (greater than 90%) of the ICA in one case (patient A) and ICA occlusion in two cases (patients B and C) (Fig 1). Evaluation of the contralateral common carotid artery demonstrated that the ipsilateral cerebral hemisphere was primarily perfused via the anterior communicating artery in all cases. No antegrade ophthalmic arterial flow was demonstrated in two cases (patients B and C). In one case (patient C), no large arterial anastomoses between the distal IMA and the distal ophthalmic artery were demonstrated, but a choroidal blush was evident with ipsilateral external carotid arteriography, apparently originating from minute external carotid artery collaterals. External carotid angiography revealed a significant external carotid to ICA anastomosis in two cases (patients A and C). Case C demonstrated filling of the ipsilateral carotid terminus, ophthalmic artery, and posterior communicating artery via branches of the middle meningeal artery.

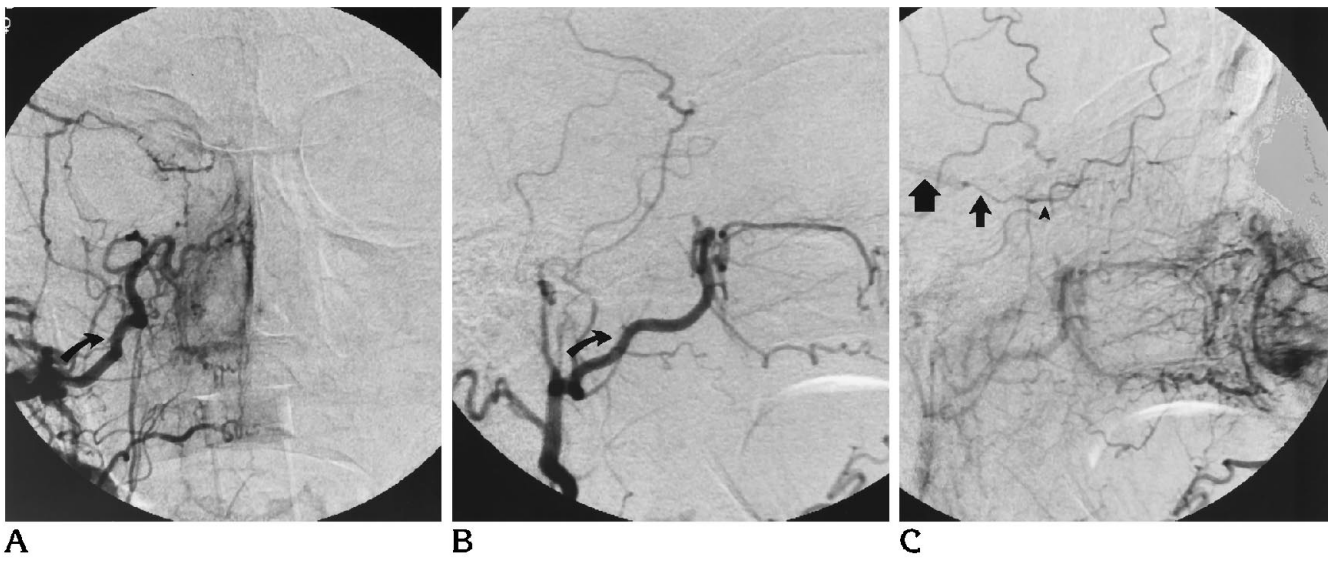

Fig 2. *A–C*, Anteroposterior and lateral views demonstrate carotid occlusion with prominent nasal mucosal hypervascularity. Collateral communication is noted with opacification of the ophthalmic artery (*arrowhead*), posterior communicating artery (*small arrow*) and P2 segment (*large arrow*). *Curved arrow* indicates right IMA.

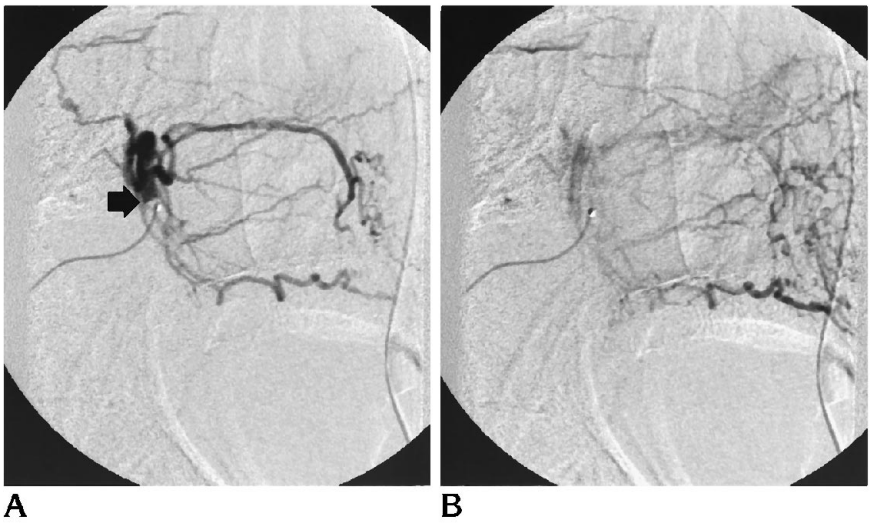

Fig 3. *A* and *B*, Early and late arterial phase with Tracker catheter positioned in distal right IMA (*arrow*).

Evaluation of the external carotid arteriograms revealed an ipsilateral hypervascular nasal mucosal blush in all three cases without evidence of an underlying lesion (Figs 2 and 3).

Successful embolization of the IMA was performed with coils in all three cases. In two cases (patients B and C), multiple 2 × 10-mm helical and 5-mm straight coils were packed into the pterygopalatine segment of the IMA to complete occlusion. In one case (patient A), only a single 2 × 10-mm coil was needed because of spasm of the IMA. Postembolization angiography demonstrated a marked reduction in the nasal mucosa blush in all cases (Fig 4). In one case (patient C), persistent inferior nasal hyperemia was observed, attributable to jugal collaterals to the distal IMA branches from the ipsilateral facial artery (Fig 5). These collaterals were reduced by embolization of the facial artery with polyvinyl alcohol (PVA) particles (500 to 700 μm) (Fig 6). Embolization was considered adequate following IMA occlusion if nasal mucosal blush was absent on posttreatment IMA and external carotid arteriography.

Nasal packing was removed within 6 to 24 hours in all three patients. They were discharged in 2 to 4 days with no evident complications. Follow-up from 5 to 18 months revealed no recurrent epistaxis (Table).

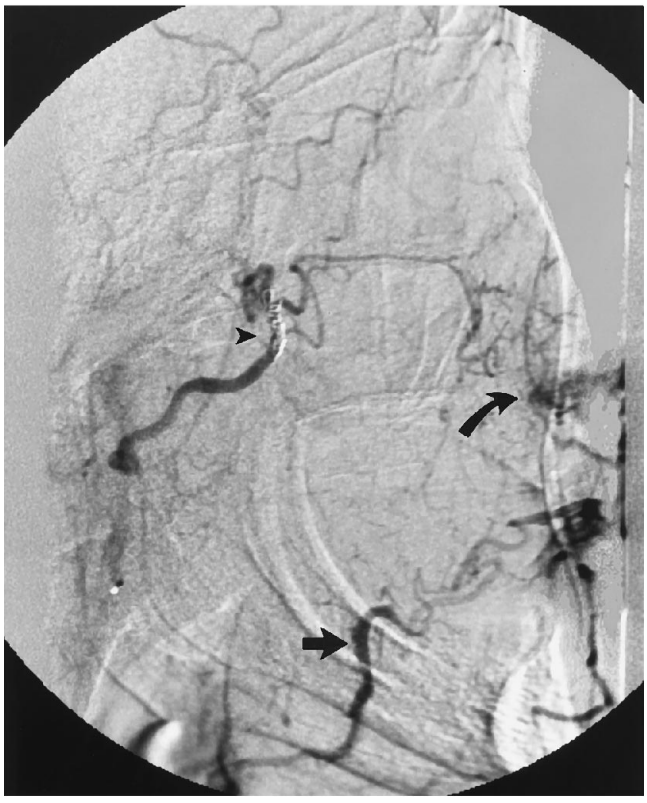

Fig 4. Immediately after microcoil embolization of IMA (*arrowhead*), there is a significant decrease in nasal mucosal vascularity with some anterior-inferior mucosal stain (*curved arrow*) via the facial artery (*arrow*).

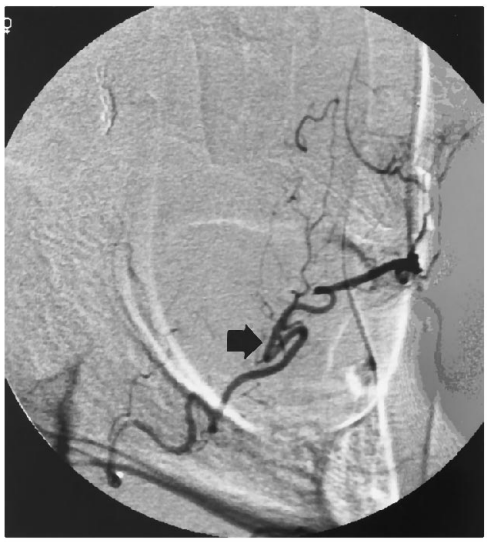

Fig 5. Selective catheterization of the facial artery (*arrow*) followed by embolization with PVA (500 to 700 μm).

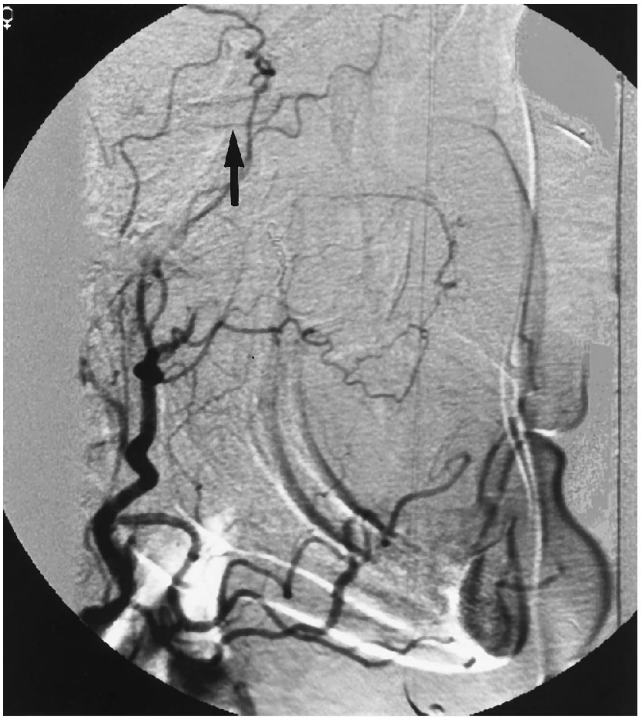

Fig 6. After IMA and facial embolization, right carotid arteriogram demonstrates successful reduction in nasal mucosal vascularity. The ophthalmic artery still opacifies (*arrow*).

Discussion

Intractable posterior epistaxis is epistaxis that is refractory to trials of anterior and posterior nasal packing and may be life threatening. These cases often require surgical or endovascular therapy to avoid the morbidity of prolonged nasal packing (1, 9). Early reports demonstrated the efficacy of intraarterial embolization treatment for epistaxis. However, other reports have cited complication rates higher than those seen with surgical arterial ligation (2, 10, 11).

With the development of coaxial microcatheter systems and the heightened awareness of functional facial vascular anatomy, success rates of 91% to 97% with complication rates of 0% to 3% have been reported within the past 5 years (3, 5, 6). Meticulous angiography of the IMA is important to exclude small but dangerous external to ICA or ophthalmic artery anastomoses as well as anatomic variants such as an ophthalmic artery origin from the middle meningeal artery. Also, the appropriate embolic particle size is important to achieve distal nasal artery occlusion without causing skin or cranial nerve ischemia/necrosis. These considerations significantly decrease the previously reported major complications including stroke, skin necrosis and facial nerve paralysis (11–16). We have found that PVA particles (350 to 500 μm) provide adequate distal embolization while minimizing these risks in the absence of ipsilateral internal carotid occlusive disease.

Epistaxis ipsilateral to carotid occlusion/severe stenosis is probably related to an attempt to collateralize the distal ICA via branches from the IMA with resultant increased perfusion pressure. In the face of ipsilateral ICA stenosis or occlusion, the treatment of intractable epistaxis is more complex. The surgical risks are higher because of the presence of significant carotid artery disease as well as frequently coexistent cardiovasculopathy. Endovascular therapeutic risks also increase because of the presence of external carotid artery–to–ICA (middle meningeal, accessory meningeal, internal maxillary arteries) and external carotid artery–to–ophthalmic artery (IMA and facial artery) anastomoses (7, 13, 15). Because of the prevalence of these collaterals in internal carotid occlusive disease, standard embolization with small particles ($<$ 500 μm) theoretically could cause blindness from retinal artery

Intractable spontaneous posterior epistaxis treated with internal maxillary artery occlusion

Patient	Age, y	Epistaxis	Ipsilateral ICA	Treatment	Complication	Follow-up
A	70	right	95% stenosis	2 × 10-mm coil (n = 1), IMA	None	No rebleed at 12 mo
B	79	right	occluded	2 × 10-mm coil (n = 2), 5-mm straight coil (n = 3), IMA	None	No rebleed at 18 mo
C	60	right	occluded	2 × 10-mm coil (n = 3), 5-mm straight coil (n = 5), PVA (500 to 700 μm particles), facial artery	None	No rebleed at 5 mo

occlusion or stroke attributable to cerebral emboli. However, we have found that endovascular therapy still may be safely and effectively performed with the use of larger, more proximal occlusive agents (endovascular coils) in the pterygopalatine segment of the IMA.

Although this approach negates the theoretic advantage of intraarterial embolization in the IMA distal to the collateral arterial supply (via anterior and posterior ethmoidal arteries and jugal branches of the facial artery), this method is theoretically similar to the transantral IMA surgical ligation, which has an overall success rate of 80% to 90% (17, 18). As Parns et al suggested, the basic principle in the treatment of idiopathic intractable epistaxis appears to be reducing the perfusion pressure at the bleeding site (4). Because our three cases of epistaxis occurred ipsilateral to ICA occlusive disease, the role of perfusion pressure reduction would seem to be an important factor.

Our standard protocol for intraarterial embolization of intractable spontaneous epistaxis consists of the use of PVA particles (350 to 500 μm), delivered by a coaxial microcatheter system with the Tracker catheter placed in the pterygopalatine segment of both IMAs and, if a significant contributor, the ipsilateral facial artery. We recommend the following modifications in cases of epistaxis with ipsilateral ICA occlusive disease.

After assessment of cerebral perfusion as well as external carotid artery–to–ICA anastomoses, a determination of the relative safety of embolization is made. If the IMA is the predominant source of perfusion to the supraclinoid ICA, coil embolization of the IMA is relatively contraindicated, although each case must be considered individually. If ipsilateral cerebral perfusion is predominantly derived from other sources, IMA coil embolization to complete occlusion may be performed and has proved effective and safe in our limited experience. If nasal mucosal blush persists, additional embolization of the ipsilateral facial artery may be performed (7, 13, 18) with larger PVA particles (500 to 700 μm).

We chose not to embolize the contralateral IMA, which could be a source for persistent or recurrent epistaxis, for two reasons. First, these patients already have significant vascular disease, and the benefit of catheterization and embolization in the contralateral carotid artery probably does not outweigh the risk of vascular injury/stroke. Second, the underlying cause of the epistaxis ipsilateral to internal carotid occlusive disease is increased perfusion pressure. Once the perfusion pressure is diminished by proximal embolization, the risk of recurrent epistaxis via small collaterals is likely to be minimal, as seen in our series.

In summary, endovascular coil embolization of the pterygopalatine segment of the IMA has been a safe and effective treatment for intractable epistaxis associated with ipsilateral ICA occlusive disease in our experience. Although embolization with PVA larger than functional anastomoses also may be safe, we did not test this hypothesis. Although our experience is limited, we anticipate long-term success rates at least equal to those of surgical transantral IMA ligation.

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