Carotid body tumors managed with preoperative embolization

Report of two cases

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Two patients with large vascular carotid body tumors underwent preoperative intravascular embolization of the major arterial feeders. The tumor vascularity was reduced markedly, and complete surgical extirpation was accomplished without difficulty. The literature on carotid body tumors is briefly reviewed. The role of preoperative embolization in the treatment of these difficult tumors is emphasized.

KEY WORDS • carotid body tumor • paraganglioma • preoperative embolization • supraselective angiography • external carotid artery • ascending pharyngeal artery

Carotid body tumors are uncommon neoplasms that originate at the carotid bifurcation from neural crest paraganglion cells. Their surgical excision can be accompanied by many complications. An early series cites vascular complications in 40% of patients and a 29% mortality rate.16 Despite improvements in anesthetic and operative technique, more recent surgical series continue to indicate occasional mortality and serious morbidity in an average of 11% of patients.1,5,11,14,20,24 This morbidity includes injuries to the ninth through 12th cranial nerves as well as the recurrent laryngeal nerve, the sympathetic nerves, and the mandibular branch of the seventh nerve. In addition, stroke from internal carotid artery (ICA) injury has been reported.3,11,14 Underlying such discouraging statistics is the critical location of these large hypervascular tumors at the carotid bifurcations.

Many authors recommend surgical maneuvers to diminish the likelihood of operative complications, including early identification and control of neural and vascular structures and subadventitial dissection of the tumor from the adherent carotid arteries.1,2,5,9,11,12,14,20,23-26,28 Some authors also recommend early ligation of the external carotid artery (ECA) and its branches to diminish tumor vascularity and facilitate the subadventitial dissection.5,25 Unfortunately, when the tumor is large, the carotid arteries are often engulfed by or posterior to the tumor and are rarely accessible during the initial surgical approach.

Although the ECA branches supplying the tumor are not accessible during the initial surgical approach, they can frequently be catheterized selectively for embolization. This report describes two cases in which preoperative intravascular embolization greatly facilitated the surgical excision of large carotid body tumors. The role of this adjunctive therapy within the overall treatment plan of carotid body tumors is emphasized and the embolization technique described.

Case Reports

Case 1

This 40-year-old man was in excellent health until late 1979, when he noted a non-tender right anterolateral mass on his neck. Surgical exploration at another hospital yielded tissue consistent with reactive lymphoid hyperplasia. Throughout 1981, the mass gradually increased in size. In early 1982, he experienced sudden dizziness without vertigo, associated with mild tachycardia. This resolved spontaneously. During evaluation for gastrointestinal complaints, a large right anterolateral neck mass was noted again by his physicians. Carotid angiography demonstrated the presence of bilateral carotid body tumors, and he was referred to the Massachusetts General Hospital for further evaluation and treatment.

At examination, the patient appeared healthy, with normally functioning cranial nerves. There was a 5-cm J Neurosurg 59:867-870, 1983
Case 1

L. F. Borges, R. C. Heros and G. DeBrun

right anterolateral neck mass and a 1-cm left anterolateral neck mass. Both were non-tender, immobile, and without bruits. Laboratory examination did not reveal evidence of catecholamine hypersecretion.

On April 12, 1982, approximately 80% to 90% of the ECA feeders to the large right-sided tumor were embolized percutaneously (Fig. 1). Over the ensuing few days, the tumor diminished in size by one-third and became tender. On April 16, 1982, the patient underwent an uncomplicated resection of the large right carotid body tumor. Blood loss was negligible. The postoperative course was uneventful and all neural structures functioned normally. Six weeks later, the smaller left-sided tumor was resected without preoperative embolization. Again, the postoperative course was uneventful.

Case 2

This 23-year-old woman was well until she was 16 years of age, when she first noted a left anterolateral neck mass. This enlarged slowly and, when the patient was 22 years old, a normal lymph node was removed surgically from the left side of her neck. Because of continuing enlargement of the neck mass, she was referred to the Massachusetts General Hospital.

She denied pain, hoarseness, hypertension, flushing, or headache. Examination revealed a pulsatile nontender immobile neck mass, 4 cm in size, at the left carotid bifurcation. No bruit was present. The cranial nerves were normal. Laboratory examination did not reveal catecholamine hypersecretion. A carotid body tumor was suspected, and this diagnosis was confirmed by intravenous digital subtraction angiography.

Selective angiography confirmed the diagnosis of a left carotid body tumor, with its main arterial supply from an enlarged branch of the left ascending pharyngeal artery. On November 9, 1982, this feeding artery was embolized percutaneously, with resulting marked reduction in vascularity of the tumor (Fig. 2). On November 10, 1982, the patient underwent complete excision of the left carotid body tumor. The posterior wall of the common carotid artery was entered, but was easily repaired with two 5-0 silk sutures. No blood transfusions were required. Postoperatively, a mild left hypoglossal nerve paresis resolved over several days and a partial left recurrent laryngeal nerve paresis resolved over several weeks. A left Horner's syndrome was still present on follow-up examination 1 month later.

Embolization Technique

Prior to embolization, careful selective mapping of the feeders of the tumor is mandatory. The carotid bifurcation shows the typical crab-like appearance with both the ICA and ECA pushed apart by the tumor. There are almost always a few tiny collateral vessels from the origin of both the ICA and ECA. Of course, these branches which are small in caliber and close to the bifurcation should not be embolized because of the proximity of the ICA, with a high risk of reflux of emboli into that vessel. Global angiography of the trunk of the ECA will give an idea of the size of the tumoral blush and the precocity of the venous drainage of the
Preoperative embolization of carotid body tumors

FIG. 2. Case 2. Pre-embolization (left) and postembolization (right) selective angiograms showing the external carotid artery supply to the left carotid body tumor.

The carotid bodies, extending approximately 4 mm in length between each ICA and ECA, are among the smallest organs within the body. Yet, their blood flow is many times higher than that in the brain, which facilitates their function of maintaining arterial pO2, pCO2, and pH within normal limits.6 Hyperplasia13 and frank neoplasms22 can develop when these sensors are stressed by chronic hypoxia, but neoplastic transformation most commonly occurs without known antecedent provocation. Carotid body tumors usually occur sporadically; however, they may be familial in occurrence.4,10,18,21,27 Familial carotid body tumors appear to have an autosomal dominant mode of genetic transmission10 and are characterized by an increase frequency of bilateral tumors.18,21 These tumors are infrequently malignant,11,15,17,18 but metastases may develop years after extirpation of the primary lesion.19 Since these tumors compress important neurovascular structures as they enlarge, and have malignant potential, most authors have recommended that they be surgically excised.

Although neoplastic transformation of the carotid body involves primarily the epithelioid cells, the vascular sinusoids also replicate, which results in very vascular tumors.11 The hypervascularity of these tumors produces a characteristic “tumor blush” that can be seen angiographically, and can also cause life-threatening hemorrhage during surgical extirpation.

Although the carotid body is nestled within the carotid bifurcation, its normal blood supply is derived from the ECA, usually from small branches of the occipital and ascending pharyngeal arteries which descend from superomedially to the carotid bifurcation.6 During neoplastic transformation, these vessels enlarge and form the major blood supply to the tumor (Figs. 1 and 2).3 As the tumor expands lateral to these feeding vessels, it intertwines itself around the major segments of the carotid bifurcation and obscures the bifurcation as well as the feeding vessels to the tumor. A second, and lesser, vascular supply then develops when the arterioles within the normal adventitia of the carotid arteries become intimately attached to the tumor capsule and enlarge.

Removal of carotid body tumors requires that the surgeon deal with both vascular components of these tumors. Surgical methods of controlling the adventitial arterial supply have been well described in the literature and center around subadventitial dissection of the tumor.1 The bipolar coagulating forceps are particularly useful for this maneuver. Unfortunately, the major arterial supply to these tumors—from ECA branches—is not accessible surgically until the tumor has been dissected from the major neurovascular structures. Although the tumor hides these vessels from the surgeon, it also causes them to dilate so that they can be reached by percutaneously directed intravascular catheters and thus become acces-

* Polyvinyl alcohol (Ivalon) manufactured by Unipoint Industries, High Point, North Carolina.
sible to the interventional radiologist. Since these ECA branches do not supply intracranial structures, they may be injected selectively with emboli without major risk of stroke. As the two present cases illustrate, embolic occlusion of the ECA supply to the tumor causes marked reduction of the tumor’s vascularity (Figs. 1 and 2). In Case 1, the tumor subsequently decreased in size by one-third, and in both cases the reduced vascularity facilitated the surgical dissection. Therefore, this technique can be expected to reduce the likelihood of serious operative morbidity with large tumors.

Preoperative embolization would not be recommended for all carotid body tumors, and in fact was not used in treating the contralateral tumor in Case 1. This lesion was small enough that its supply from the ECA could be controlled surgically prior to dissecting the tumor from the carotid bifurcation. In tumors that do not have distinct large feeding arteries from the ECA, embolization is not indicated. In these cases, the blood supply comes from direct short vessels from the bifurcation, and attempts at embolization may result in an inadvertent embolization of the ICA, with stroke resulting. Thus, to determine whether or not preoperative embolization would be useful and safe, detailed superselective angiography of ECA branches and also of branches of the thyrocervical trunk is necessary. In our view, preoperative embolization is indicated in large tumors as an adjunct to surgical extirpation when detailed angiography reveals that the tumor mass is obscuring the major tumor vessels from early direct surgical attack and when these tumor vessels can be safely catheterized at a distance from the bifurcation without danger of intracranial embolization.

The emboli used in these two cases were 150-μ particles of polyvinyl alcohol. This was found to be more effective than Gelfoam in obliterating tumor beds. However, recanalization of the nidus is likely to occur, and is one reason why surgical removal of the tumor is recommended not more than 2 weeks after embolization. Another reason for extirpation is that it is difficult and rare to totally obliterate the abnormal vessels of the tumor with emboli, particularly those vessels that originate close to the carotid bifurcation. Therefore, embolic occlusion of the ECA vessels supplying the tumor would not be expected to result in cure without other procedures. Although it would have been possible to permanently occlude these major tumor vessels with a cyanoacrylate glue (Bucrylate), the tumor would have become rock hard and difficult to remove surgically.

References