26

Therapeutic Embolization of Cranial Tumors

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1. Introduction

The aim of embolization is to devascularize the tumor bed by filling intratumoral vascularization as deep as possible into the precapillary level to initiate tumor necrosis, decrease blood loss during surgery and thus make it easier and possibly, more radical, and in tumor of nasal cavity to stop epistaxis (Berenstein, 2004, Valavanis, 2002, Gupta, 2006, Smith, 2006). The procedure is angiographically guided. Embolization of a tumor is performed by either an intraarterial catheterization approach or direct puncture of the tumor artery. Selection of one of these approaches depends of the location, and number of arterial feeders of the tumor. Result of devascularization is verified by contrast enhanced CT or MR imaging.

Endovascular therapy is not only limited to devascularization procedures for head and neck tumors. Central nervous system lymphomas can be treated with intraarterial chemotherapy infusions after blood brain barrier disruption (Neuwelt, 1991), and squamous cell carcinomas of the skull base and neck are treated with selective infusions of high dose chemotherapy (Simunek, 1993). In this review, we will discuss the endovascular approach to induce tumor ischemia and necrosis of central nervous system and skull base tumors. In spite of the fact that methods of preoperative devascularization of hypervascularized tumors of the head have been established for many years, the number of patients treated endovascularly using this technique is relatively low. This is mainly due to a small proportion of the head and neck tumors suitable for this therapy and controversies in terms of its risk-benefit ratio (Latchaw, 1993).

2. Indications

There are basically three main indications for head and neck vascularized tumor embolization: presurgical embolization, palliative treatment, and to stop epistaxis. Presurgical embolization is believed to reduce blood loss, shorten the operative procedure time and potentially increase the chances of radical surgical resection as well as decrease the incidence of tumor recurrence. Palliative treatment may led to a decrease in tumor mass with improvement of neurological symptoms due to intracranial expansion. In some cases, shrinkage of tumor may alleviate intractable pain.

The most common skull base hypervascular tumors treated with embolization are meningiomas, paragangliomas, hemangiopericytomas, juvenile nasopharyngeal

angiofibromas. The most frequent intraaxially located tumors indicated for embolotherapy are hemangioblastomas, hypervascularized metastases and ependymomas (table 1).

Cranial tumors that are treated with endovascular embolization
1. Meningiomas 2. Paragangliomas 3. Juvenile nasopharyngeal angiofibromas 4. Hemangioblastomas 5. Intracranial and extracranial mestastases 6. Hemangiopericytomas

Table 1**.** The list of the most common hypervascularized tumors in which embolization may be benefitial.

3. Embolization technique

3.1 Functional Angiography

Selective internal, external carotid and vertebral angiograms are followed by selective catheterizations of feeding arteries to analyze arterial supply of the tumor.

Complex vascular anatomy of the skull base requires meticulous digital subtraction angiograms to reveal dangerous anastomoses from external carotid artery branches to internal carotid or vertebral arteries (Russell, 1986, Geibprasert, 2009). These arterial connections should be checked during embolization since they might open up as the occlusion of target artery progresses (Berenstein, 2004, Russell, 1986).

For complex procedures, meticulous angiograms without motion artifacts are required, and simple intravenous sedation may be inadequate. In such procedures general anesthesia will often be appropriate. Arteficial ventilation is stopped during selective injections to avoid any movement artefacts. High-quality biplanar imaging is generally recommended at the skull base and around the eye arterial network studies.

Depending on tumor size and extension, unilateral or bilateral internal and external carotid and vertebral angiograms are performed using 5F catheter system as the first step of embolization procedure. Then selective studies are accomplished via various types and diameters of microcatheters. Their selection depends on diameter of the target artery and also on the operator´s experience. Care should always be exercised to avoid proximal vasospasms which may preclude flow directed embolization. Vasospasms occurrence varies from patient to patient and should be minimized by careful catheterization techniques and administration of vasodilators (Kurata, 1996).

The feeding pedicles should be tested for supply to cranial nerves before infusion of embolic agents. Awake patient can be evaluated for appropriate cranial nerve or visual deficits after selective injection of 1-2 ml of local anesthetic before embolization starts. In the sleeping patient we have to rely on anatomical angiographic studies and thorough knowledge of anatomy.

Ideally the capillary bed of the tumor is slowly saturated with embolic microparticles or liquid agent. This is accomplished under fluoroscopic guidance to assess blood flow velocity in the artery and potential backflow and reflux of embolic agent is observed. During embolization the operator should perform selective angiograms, the quality of which

enables not only to assess progress in occlusion, but to reveal potential filling of collaterals. Some neuroradiologists say: "When you see collaterals, it is always too late."

3.2 Special techniques

For tumors in cavernous region supplied by dural feeders coming off the internal carotid artery the non-detachable balloon is inflated above the feeding arteries origin to protect the ophthalmic artery and all pial networks (Théron, 1986, Gonzales, 1990, Tymianski, 1994, Jungreis, 1991). The microparticles can be safely injected nonselectively into these feeders. Repeated flushing and aspiration should be performed before the balloon is deflated to minimize distal embolization.

Usually, it is most important to embolize surgically inaccessible arterial feeders. Then depending on risk-benefit ratio and compartmental supply of some tumors, second-order size feeders are closed as well. Finally, we prefer to embolize the largest feeders.

The blood flow in the magistral arteries with potentially dangerous collaterals can be modified by temporal balloon occlusion. Distal arterial beds can be protected by microcoil placement to avoid penetration of embolic agent into normal tissue and minimize risk of the skin necrosis or delayed healing of the future surgical wound.

Direct percutaneous puncture of some hypervascular tumors is performed with a system of coaxial needles (Casasco, 1994, Chaloupka, 1999, Abud, 2004, Lonser, 1998, Casasco, 1999). This has to be done under general anesthesia and simultaneous angiographic control. Liquid agents are most used in this technique (Lonser, 1998). Complications from intratumoral anastomoses to surrounding neurovascular structures are observed with this technique, similar to those of transarterial embolization (Casasco, 1999).

3.3 Embolic agents

The operator´s selection of embolic agent is directed by a balance between risk and efficacy. Smaller particles (45-150 μm) (Wakhloo, 1993) and liquid embolic agents (bucrylate, ethanol, ethylenevinylalcohol) penetrate tumor better and achieve a higher degree of necrosis. However, these agents definitely carry a higher risk of collateral tissue ischemia of the vasa nervorum. The critical size of microparticles is generally considered to be above 150 μm (table 2).

The most frequent causes of neurologic deficit after embolization of cranial base tumors
1. selection of inappropriate embolic material
2. reflux of emboli (vasospasm, nonselective injection, too many particles injected too fast)
3. failure to recognize dangerous anastomoses (incomplete angiogram, inadequate analysis)

Table 2. The list of frequent causes of ischemic neurological deficit after embolization in the skull base (Lasjaunias, 1980).

4. Results of embolization

Comparisons between groups of patients with surgical therapy of meningiomas with and without preoperative embolization revealed that embolization reduces blood loss and the number of transfusions needed during operation. This effect is higher in large tumors than

small, and when a high degree of obliteration was achieved. These facts can be generalized to other vascularized tumors of the skull base and brain which are much less frequent than meningeomas. The authors will discusse embolization in specific lesions as meningeomas, paragangliomas, juvenile angiofibromas and some of intraaxial tumors.

4.1 Meningiomas

Meningiomas are the most common extraaxial intracranial tumors in adults. They are derived from meningothelial cells concentrated in the arachnoid villi. The most frequent sites of origin of these tumors are where the arachnoid villi are the most numerous, i.e. the major dural sinuses, veins, and along the root sleeves of the exiting cranial and spinal nerves. However, ectopic meningiomas may arise from ectopic arachnoid cell inclusions (Black, 1993).

The grade of vascularization varies on histological type of meningioma (Toktas, 2010, Ketter, 2008). The World Heath Organization grade II and III, which are labeled as atypical and anaplastic, as well as angioblastic and syncytial types tend to be more vascular then grade I, and fibroblastic type which are considered benign.

For practical reasons three types of this diseases are classified in the literature. The most common is the globular meningioma. It presents with compact rounded mass with invagination of brain. The tumor is flat at base, and originates in at basal or convexity dura, tentorium or falx. The second type is meningioma en plaque, which induces hyperostosis of underlying bone. This type is not indicated for angiography or embolization. The third type is multicentric meningioma. It might be associated with neurofibromatosis type II.

Preoperative angiography is indicated only for purposes of devascularization in potentially highly vascularized tumors evaluated by CT or MR. These are predominantly large in size and located at skull base (Nelson, 1994).

Angiography demonstrates early opacification of tumoral blush which stays late into the venous phase, so called “mother-in-law” phenomenon. The early draining vein sign is rare, and may be seen in angioblastic meningiomas. Tumorous vascularization has a characteristic “sunburst” pattern. Meningiomas in certain locations have particular arterial feeders. The degree of their pial supply varies.

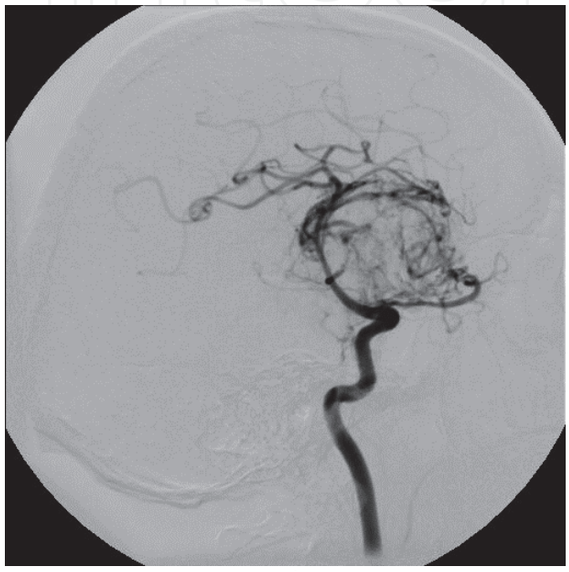
Hypervascularized meningiomas involving the skull base are the most frequently embolized (Macpherson, 1991, Rosen, 2002). Meningiomas originating from the anterior fossa (the olfactory groove, planum sphenoidale) may receive their dural blood supply from the ethmoidal arteries of the ophthalmic artery (Figure 1). Meningiomas of the cavernous sinus and clinoid region are fed by the inferolateral and meningohypophyseal trunks of the internal carotid artery and by the cavernous branch of the middle meningeal artery, recurrent meningeal branch of the ophthalmic artery and the accessory meningeal artery (Valavanis, 2002).

Meningiomas of the middle fossa and sphenoid wing are supplied by the sphenoidal branch of the middle meningeal artery. In general, meningiomas of the lateral third of the sphenoid wing are supplied by external feeders, and tumors of the medial third are supplied off internal carotid artery feeders. Meningiomas located in the tentorium and posterior fossa receive their supply from the internal carotid artery via the marginal tentorial artery, the lateral clival branch, and tentorial branch of the middle meningeal artery. Infratentorial meningeal supply is through the posterior meningeal artery and posterior falx artery which arise from the vertebral artery. Petroclival and cerebellopontine angle meningiomas are

supplied by branches of the internal carotid artery, middle meningeal artery, mastoid artery and ascending pharyngeal artery (Valavanis, 2002, Natarajan, 2007).

Convexity meningiomas receive their dural supply from the middle menigeal artery. There may be bilateral feeders when these lesions are located close to the midline. Meningiomas, invading into the bone may receive transosseous supply from scalp arteries.

Angiography is expected to evaluate relationship of convexity meningiomas to the dural sinuses. If a meningioma has obliterated the sinus, it is easier for the neurosurgeon to remove than if it has compressed the sinus. In this case, preservation of the sinus is necessary (Raza, 2010).



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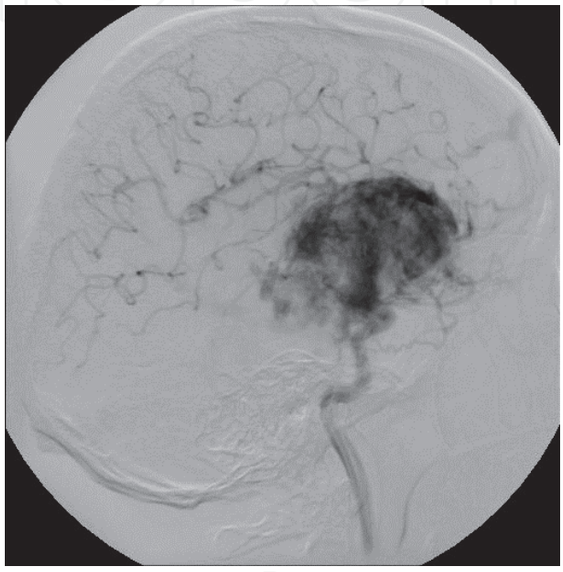

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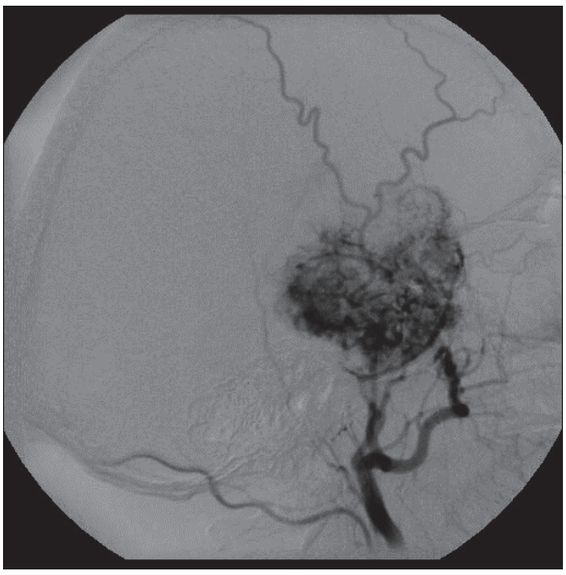

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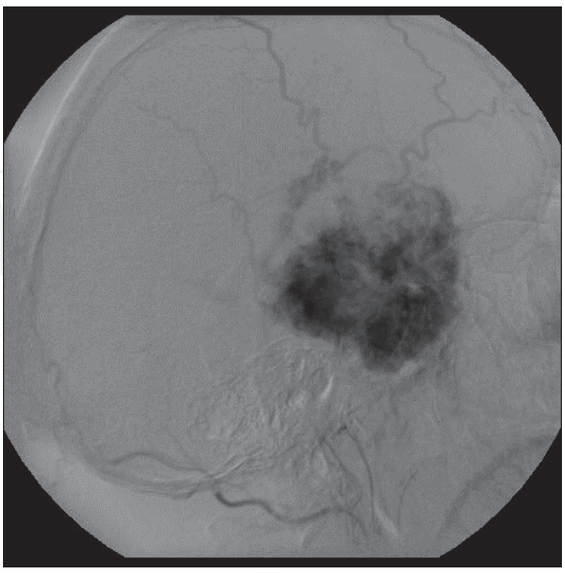

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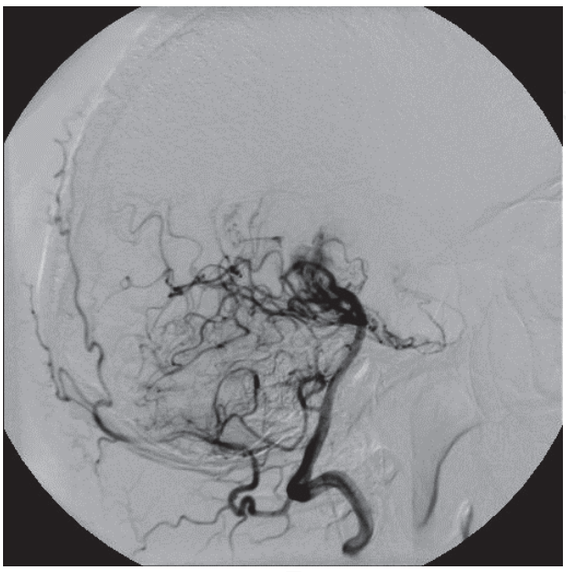

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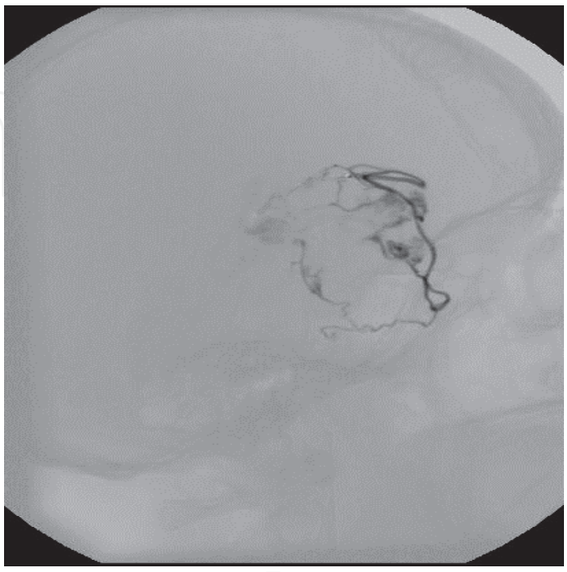

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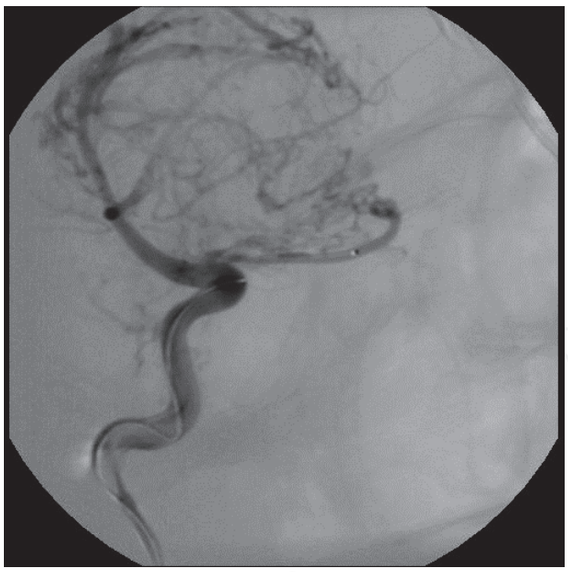

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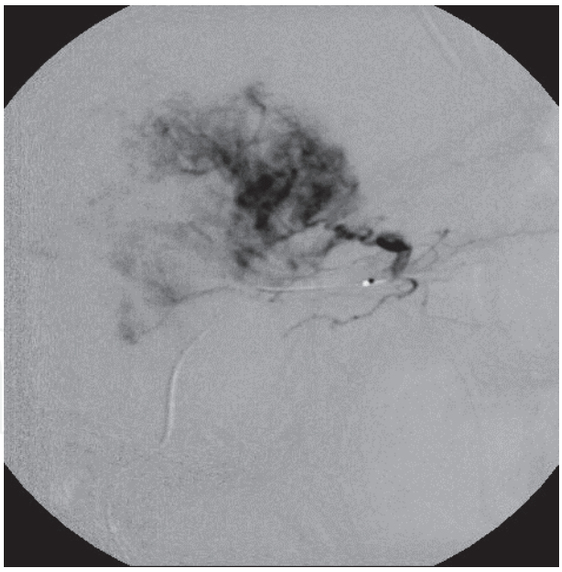

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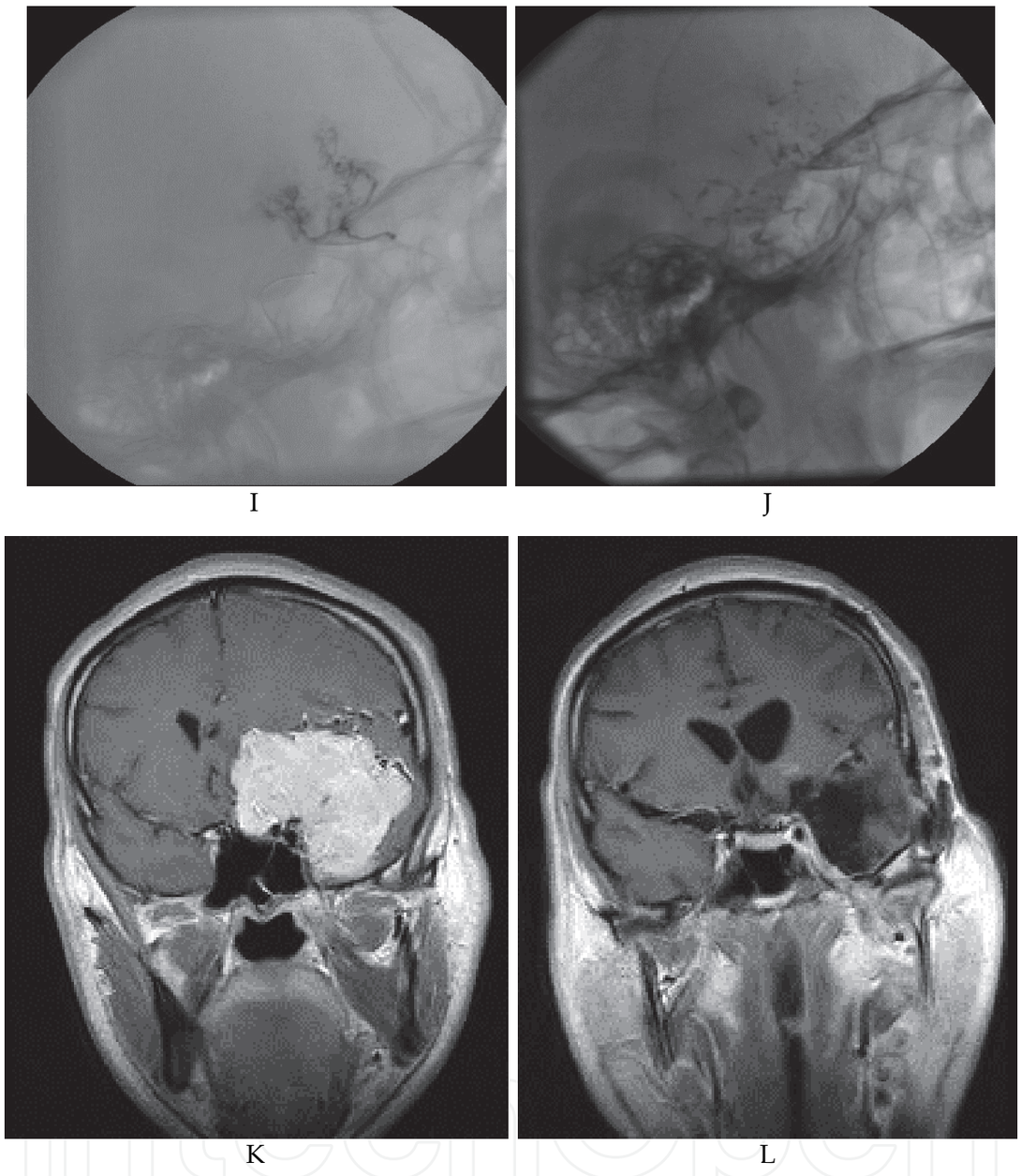


Fig. 1. A 48-year-old male with large meningioma of the sphenoid wing. **A, B.** Selective internal carotid angiogram shows hypervascularized tumor. **C, D.** The external carotid angiogram demonstrates rich vascularization fed from the middle and accessory meningeal arteries. **E.** The posterior part of the tumor is supplied by pial feeders arising from posterior circulation. **F.** Embolization of the tumor was started from hypertrophied pial feeders. **G-I.** After embolization of the external carotid artery branches (not shown), the opththalmic artery feeder was catheterized and embolized. **J.** The lateral view of the skull reveales penetration of radiopaque acrylic glue into the tumor. **K.** Preoperative MR image of the tumor. **L.** Postoperative MR image demonstrated total tumor removal inspite of the fact that preoperative embolization was estimated to be only 50 %. The patient suffered from ipsilateral oculomotor palsy after surgery which improved with time.

4.1.1 Results

In last two decades several comparative studies to prove whether embolization really reduces operative blood loss have been published, including whether it justifies the potential risk.

In 1994 Dean et al. selected 18 matched pairs of meningiomas (size, location, angioblastic versus nonangioblastic) out of 33 embolized and 193 nonembolized meningiomas to compare both groups for blood loss during operation, surgical resection time, cost and complications. Embolization was revealed to be beneficial since the estimated blood loss was significantly lower (533 ml versus 836 ml), number of transfusions was significantly lower (0.39 units versus 1.56 units), surgical resection time was shorter (305.8 minutes versus 337.5 minutes) and length of hospitalization was shorter (10.6 days versus 15.0 days). The mean cost was lower for the embolized group. There were zero major and ten minor complications in embolized group and three major and nine minor complications in the non-embolized group (Dean, 1994).

In 1998 Oka et al. analysed a group of 12 patients with meningiomas larger than 4 cm in diameter which were embolized prior surgery after the year 1990. The control group included 8 patients who were operated without embolization between 1980-1989. The results showed that in tumors smaller than 6 cm, the average blood lost was significantly lower in the embolized group (1.1. units versus 12.8 units).

Differences in the number of blood units required during surgery for patients with meningiomas over 6 cm in size did not reach statistical significance. This paradox was explained by a tendency of larger meningiomas in recruiting blood vessels that were unsuitable for preoperative embolization. The authors did not find a difference in the lenght of the operation between the two groups and the embolized group tended to show a better clinical outcome than the non-embolized group (Oka, 1998).

In 2000 Bendszus et al. compared 30 patients who were preoperatively embolized for meningiomas at one center with another 30 patients at a second center who were not. Both protocols were considered to be routine at each neurosurgical center. The mean tumor sizes were 29.6 cm^3 in embolized group versus 29.9 cm^3 in non-embolized group. There were no effect on blood loss (636 ml in embolized group versus 646 ml in non-embolized group) except when there was more than 90 % tumor devascularization as assessed by postembolization MR imaging. The observations of the neurosurgeons regarding hemostasis, tumor consistency, and intratumoral necrosis did not differ significantly. There was one permanent neurological deficit (3%) caused by embolization. The rates of surgical morbidity with permanent neurological worsening were 16 % in a center where embolization was used, and 20 % in a center without preoperative embolization (Bendszus, 2000).

The efficacy of preoperative meningiomas embolization was studied retrospectively in numerous monocentric studies. Investigators analysed embolic agent, methods of tumor necrosis verification, proportion of the feeders from the external and internal carotid artery, timing of surgery after embolization and complications.

4.1.2 Embolic agents

In 1993 Wakhloo et al. compared group of 14 patients whose meningiomas were embolized with polyvinylalcohol particles of 150 to 300 μm in size, with a group of 20 patients where smaller (50 to 150 μm) particles were used. The patients were evaluated by CT, MR, MR

spectroscopy and MR volumetry of their tumors. Angiography after embolization demonstrated the total elimination of tumor blush in all patients. Contrast-enhanced MR after larger particles embolization proved a reduction of tumor enhancement in only 14 %. While after smaller particles necrosis ranged from 30 to 95 % was proved in 60 % with some volume reduction in 20 %. The appearance of the tumor at surgery, and histopathologic examination of different parts of the tumor confirmed MR finding of necrosis. The authors concluded that extended embolization with 50 to 150 μm microparticles improved the surgical treatment of meningiomas, as compared with larger particles embolization. They suggested to use this technique in palliative embolization in patients who are not candidates for surgery (Wakhloo, 1993).

Small particles penetrated deep into the tumorous vasculature, while larger particles produced proximal occlusion of feeding arteries. This proximal occlusion results in clot formation which, over time, can be recanalized and such embolization is less efficient. On the other hand, the operator has to balance the use of the aggressive embolization techniques in a particular vascular territory against possible complications. Another study which compared a historically new embolic agent with polyvinylalcohol microparticles was published in 2000 (Bendszus, 2000). The new embolic microparticles were much more sophisticated. They are trisacryl gelatin microspheres non absorbable collagen-coated, precisely calibrated microspheres (Embospheres, Guerbet). The study included 60 patients and revealed that use of the trisacrylmicrospheres in size 150 to 300 μm were significantly more efficient than embolization with polyvinylalcohol microparticles of both sizes of 45 to 150 μm or 150 to 300 μm regarding blood loss at surgery. The result was explained by deeper arterial penetration of the trisacryl microspheres (Wakhloo, 1993).

In some anatomical territories or if the tip of microcatheter is positioned inside tumoral vasculature, successful embolization using liquid agents have been reported. The use of n-butyl-2-cyanoacrylate (Histoacryl, B. Braun) is recommended in presence of intratumoral arteriovenous shunts. This liquid agent is diluted by oily contrast medium which delays rapidity of its solidification.

Use of highly concentrated ethanol for skull base meningiomas was reported in a series of seven patients at cavernous region. The cavernous part of the internal carotid artery was occluded with a non detachable balloon and from 1 to 12 ml of ethanol was injected during 5-40 minutes. In 2 patients (30 %) permanent cranial nerve palsies developed (Jungreis, 1991).

Another liquid embolic agent is composed of ethylenevinylalcohol copolymer suspended in dimethylsulfoxide with tantalum added for radioopacity (Onyx, ev3). This agent is increasingly used either for transcatheter of direct needle puncture embolization of tumors. The technique of its injection is quite different than on previous agents. The injection is slow and penetration of the agent can be clearly seen on fluoroscopy (Gemmete. 2010) (Figure 2).

4.1.3 Tumor necrosis verification

Angiography performed at the end of embolization is considered not to be valid proof of effective embolization even if the tumor blush disappears completely (Valavanis, 2002, Benszus, 2000). This could be either due to arterial spasms or proximal extratumoral occlusion of feeders. Gadolinium enhanced MR provides more valuable imaging of tumor necrosis. MR spectroscopy has been studied, but is not used routinely (Bendszus, 2000). In

our practice – contrast enhanced CT scanning is most likely equal to MR for evaluating those meningiomas that have not been operated and for their remmants (Figure 3).

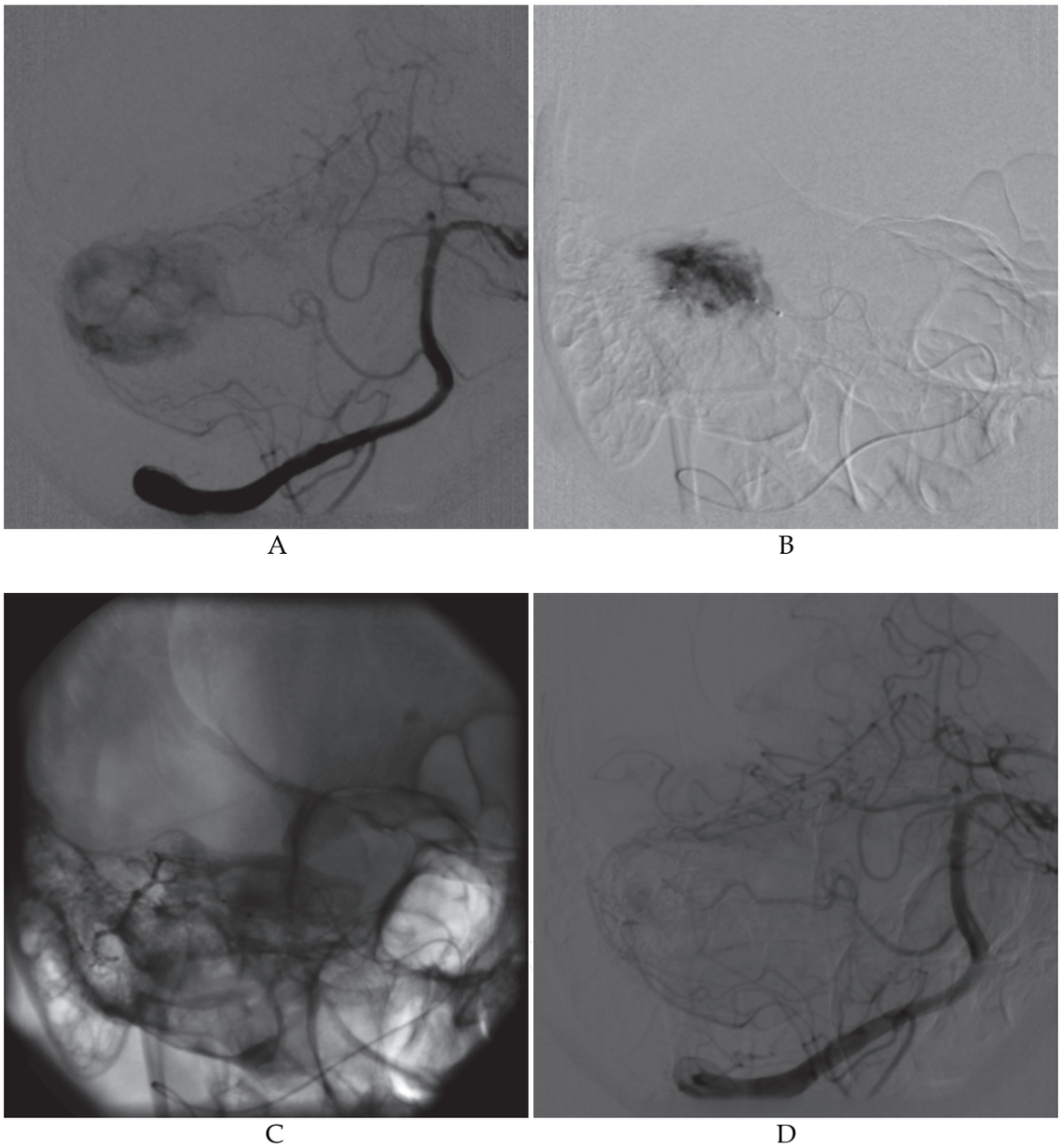
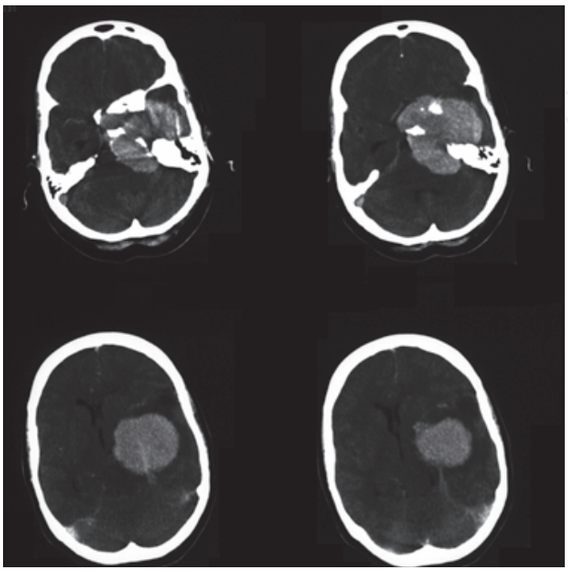


Fig. 2**. A.** A 42-year-old man with intraaxial hypervascularized hemangioblastoma supplyied mainly by the anterior inferior cerebellar artery. **B.** Selective injection through the Sonic 1,5F microcatheter (Balt). **C.** The tumor was embolized with 0.4 ml of ethylenevinylalcohol (Onyx 18, ev3). Filling of the tumorous arteries with radiopaque liquid can be seen. **D.** Postembolization arteriogram depicting the lack of tumor blush with preservation of the anterior inferior cerebellar artery. The patient was operated.



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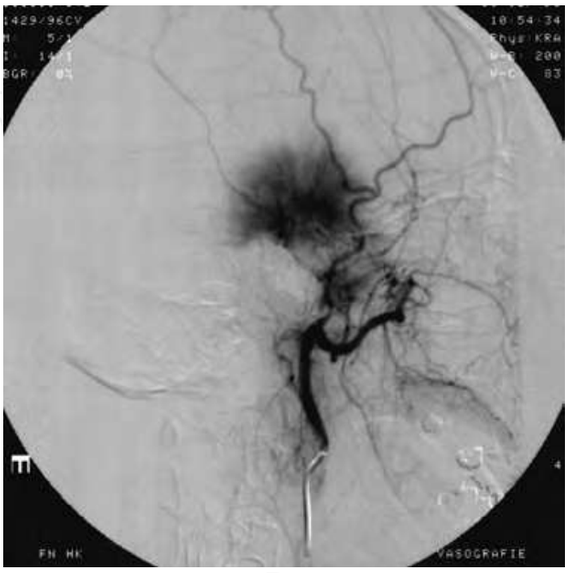

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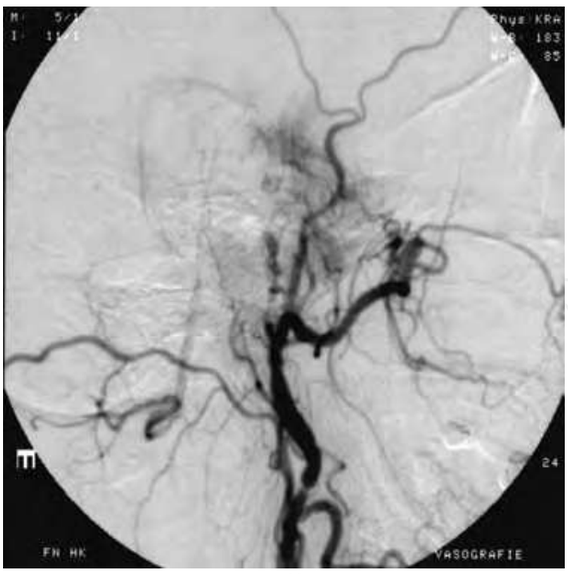

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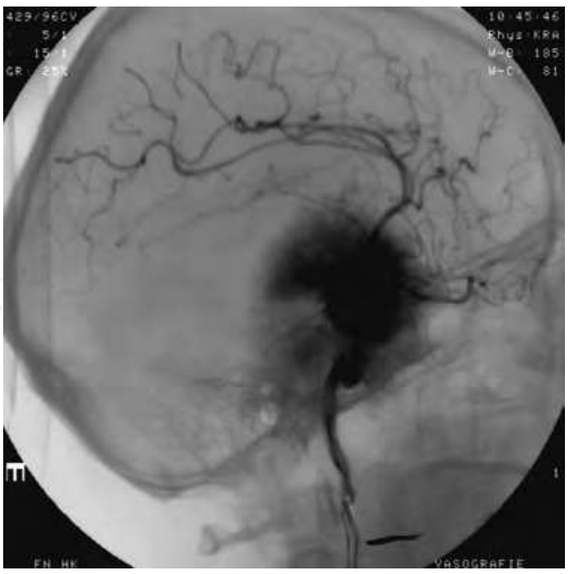

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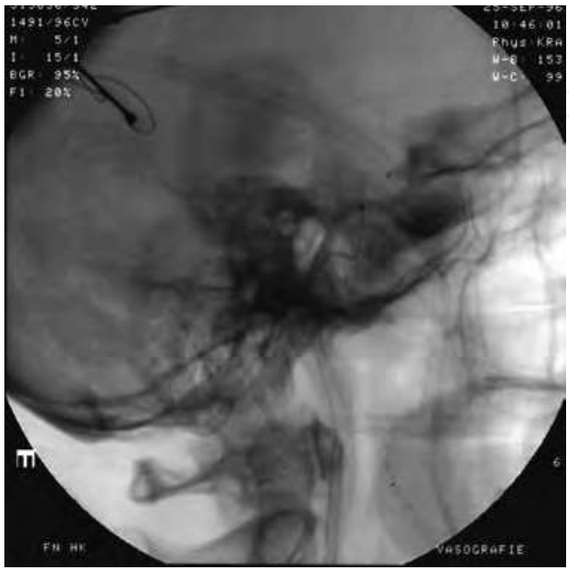
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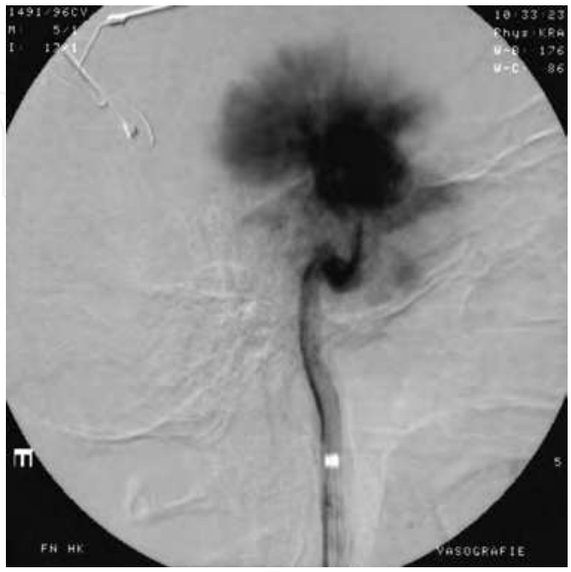
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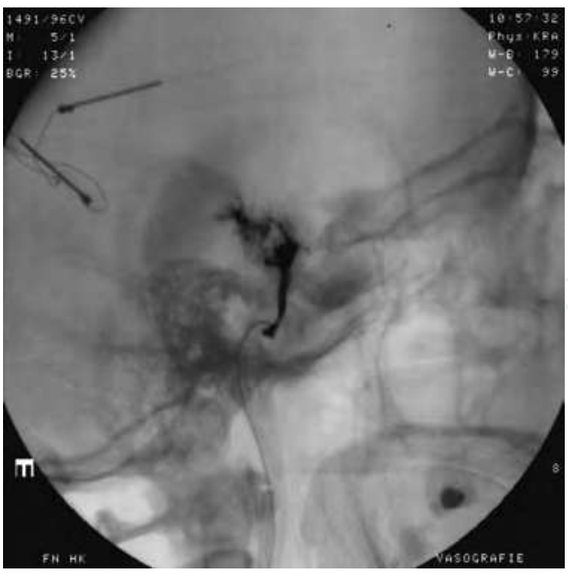
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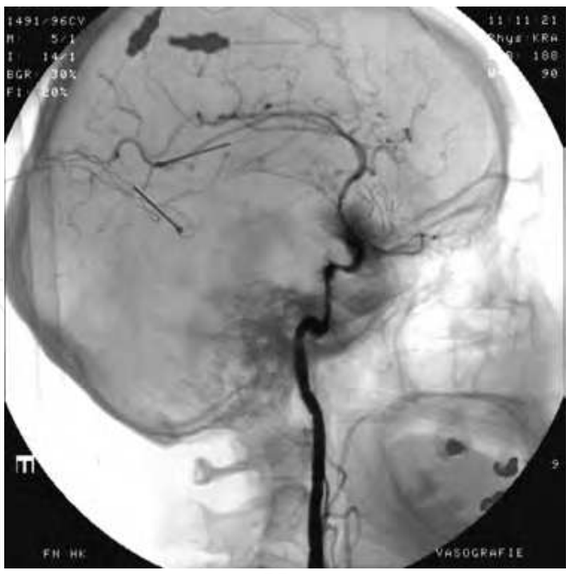
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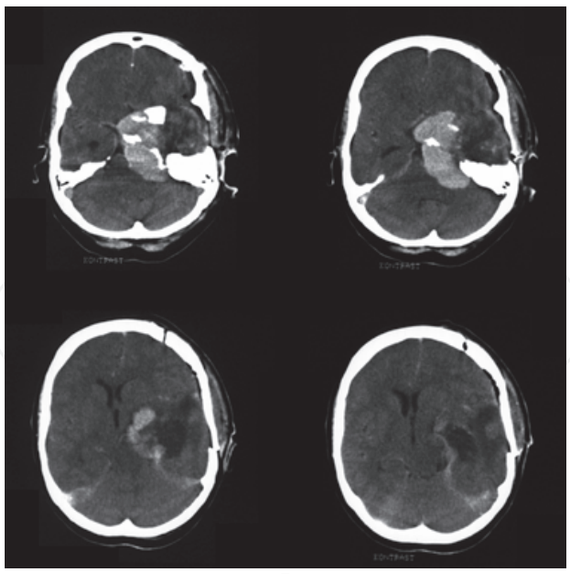

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Fig. 3. **A.** A 45-year-old woman with large hypervascular meningioma which occupies the middle fossa. **B.** The external carotid angiogram reveals typical sunburst pattern of meningioma. **C.** There is minimal residual blush after embolization of the middle and accessory meningeal arteries. **D.** The majority of the tumor is fed from the internal carotid artery branches. **E.** The guiding catheter was placed into the internal carotid artery and nondetachable balloon was inflated below the level of the ophthalmic artery origin. The second parallel microcatheter was placed below the balloon for injection of microparticles (polyvinylalcohol 150-300 μm). **F.** The internal carotid artery angiogram with balloon inflated above the tumor feeders. **G.** The internal carotid artery angiogram after embolization demonstrates some filling of intratumoral arteries. The blood from the internal carotid artery was aspirated before the balloon was deflated. **H.** The internal carotid artery angiogram revealed significant tumorous blush reduction. **I.** The contrast enhanced CT performed after surgery revealed persistent midline portion of the tumor. The tumor remnant was treated by stereotactic radiosurgery.

4.1.4 Embolization of the internal carotid artery feeders

This point is of importance for skull base meningiomas. Some of the internal carotid branches are not accessible at the beginning of surgery, but their embolization presents a much higher risk for the patient than standard embolization of the external carotid artery branches. But if we look closer at the distribution of permanent neurological deficit after embolization, the artery with the highest risk is the ascending pharyngeal artery followed by the meningohypophyseal trunk, accessory meningeal artery, middle meningeal artery and ophthalmic artery. In this study, which included 167 meningiomas with 280 feeders the most frequently embolized artery was the middle meningeal artery followed by meningohypophyseal trunk and ascending pharyngeal artery. The rate of permanent deficit was 9 % (Rosen, 2002) (table 3).

In the other study, which included, 119 meningiomas with embolization of 18 % of the internal carotid artery feeders, permanent complication rate was 2.5 %. None of them was not in relation to the internal carotid artery feeder embolization (Waldron, 2011).

Most frequent permanent neurological deficit after cranial base meningiomas embolization. Distribution according embolized arteries
1. ascending pharyngeal artery 2. meningohypophyseal trunk 3. accessory meningeal artery 4. middle meningeal artery 5. ophthalmic artery

Table 3. The list of the arteries with the most frequent permanent neurological deficit after cranial base meningiomas embolization (Rosen, 2002).

4.1.5 Timing of surgery after embolization

It is general practice to schedule meningioma surgery within 24 hours after embolization, especially to prevent revascularization and avoid problems due to tumor swelling in some cases.

Two interesting studies have been performed. The first proved no difference in operative blood loss in delayed surgery and the greatest degree of tumor softening at surgery was found between 7th and 9th day after embolization (Kai, 2002).

The second study compared two groups of patients. In the first group of 28 patients surgery was done within 24 hours, while in the second group the meningioma was resected later (2-7 days). There was found to be significantly decreased in operative blood loss in the delayed surgery group (Chun, 2002).

4.1.6 Neurologic complications

Causes of neurologic complications after meningiomas embolization include brain or cranial nerve ischemia, hemorrhage, and tumoral swelling (Yu, 2004).

Depending on aggressiveness of the operator the rate of ischemic complications is reported from 0 % to 9 %. Bendzus et al reported on 185 consecutive cases undergoing microspheres embolization of meningiomas, 12 (7 %) complications related to the procedure. Six of them were ischemic (hemiparesis and amaurosis) and another six hemorrhagic. The authors concluded that microspheres embolization of meningiomas is associated with substantial risk of ischemic and hemorrhage events. The individual risk-to-benefit ratio of embolization should be thoroughly considered (Bendszus, 2005).

Kallmes et al. reviewed literature in 1997 and found only seven cases of hemorrhage of a meningioma after embolotherapy at that time. All those meningiomas were large (> 6 cm) and highly vascular (Kallmes, 1997). In the report of Bendzus et al. (Bendszus, 2005) were observed 6 cases of postembolization meningioma hemorrhage occurred in a series of 185 cases. One of them was fatal and the others necessitated urgent surgical tumor removal, but resulted in no persistent neurological deficit. The atypical meningiomas, and tumors with previous hemorrhage, with cystic changes within tumor were positive predictors of hemorrhage. Worsening of mass effects after embolization of meningiomas with significant edema should be prevented by pretreatment with steroids (Gilad, 2009).

4.2 Paragangliomas

Paragangliomas are derived from paragangliomic chemoreceptor cells of neural crest origin. These neoplasms are locally aggressive with a low metastatic potential (Gerosa, 2006). The

most common location in the head and neck region for these tumors is along the temporal bone (almost 50 %) involving the tympanic plexus or jugular fossa, followed by the carotid bifurcation and vagal nerve. Detailed description of last two entities is beyond the scope of this chapter. Multicentricity is observed in 22 %, especially in patients with a positive family history. Secretory catecholamines activity occurs in 1 % (Berenstein, 2004, Valavanis, 2002).
Definitive treatment for these tumor is surgical excision. Preoperative arterial embolization has been accepted for selected cases of head and neck paragangliomas. A ten year survival of only in 29 % in untreated temporal paragangliomas justifies aggressive treatment of these patients (Valavanis, 2002, Valavanis, 1986, Tikkakoski, 1997, Persky, 2002).

4.2.1 Glomus tympanicum tumor

Glomus tympanicum tumor is the most common tumor in the middle ear, which presents with hearing loss and pulsatile tinnitus. It originates from the tympanic plexus on cochlear promontory of the middle ear. The extent of bone involvement can be demonstrated on CT using bone algorithm protocol. Local spread may cause invasion of the carotid canal, jugular foramen, facial and hypoglossal nerves canals, cerebellopontine angle and mastoid process. MRI provides typical "salt and pepper" appearance of hypervascular soft tissue tumor and relationship to the carotid artery (Valavanis, 2002, Van den Berg, 2002).

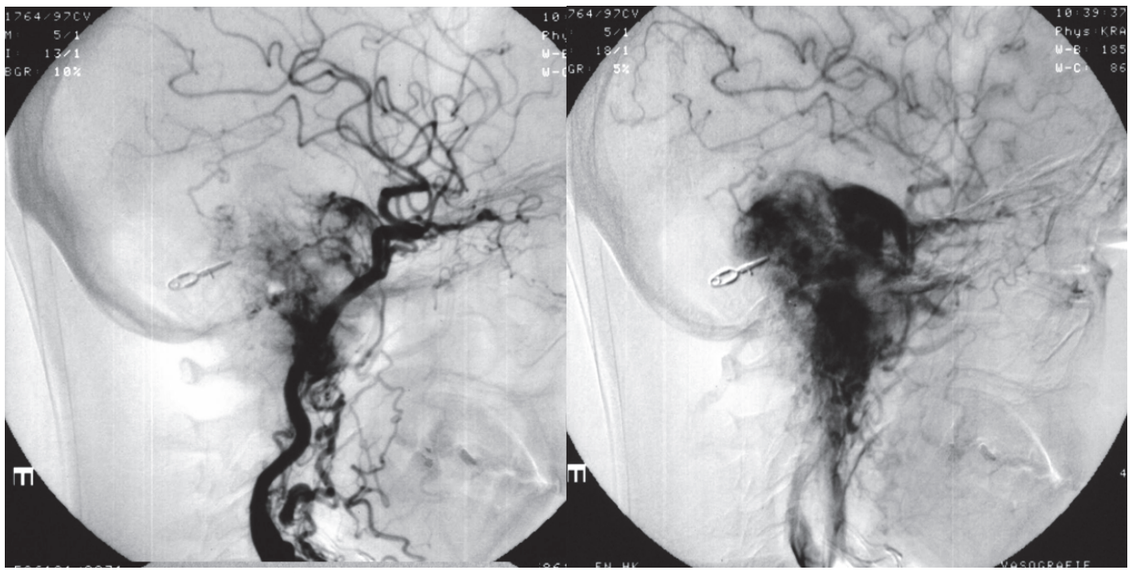
4.2.2 Glomus jugulare tumor

This is the most common tumor in the jugular fossa with intracranial extension. It arises from the adventitia of the jugular vein and shows similarity to tympanicum tumor with hearing loss and tinnitus. CT may reveal destruction of the posteroinferior petrous pyramid (Ramina, 2005).

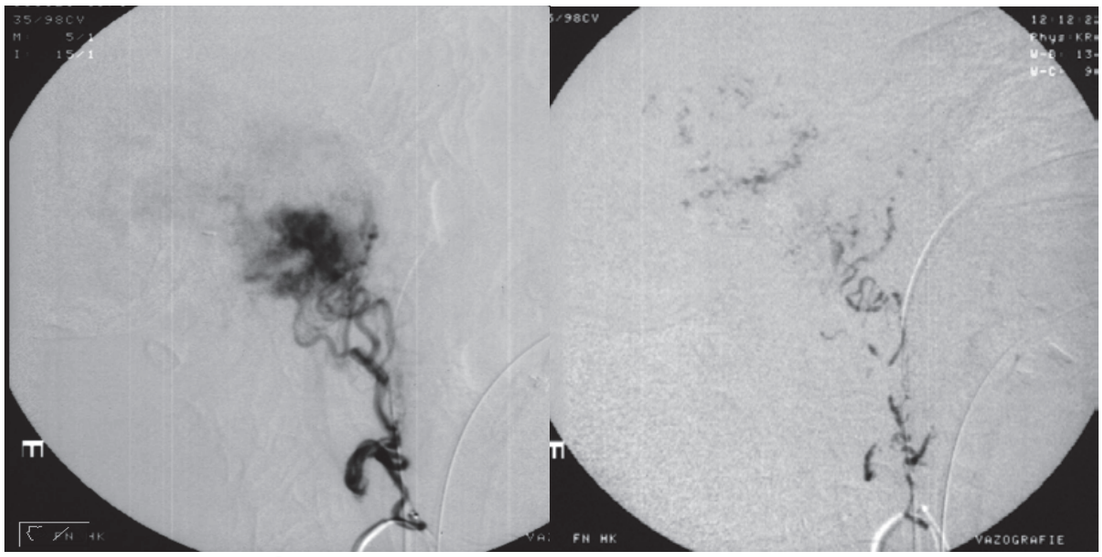
4.2.3 Preoperative and preembolization angiography

Paragangliomas are hypervascularized with enlarged arterial feeders. Catheterization angiography should include separate internal and external carotid artery studies with venous phase imaging.
The ascending pharyngeal artery is the most important feeder of paragangliomas in all four regions and therefore has been called "the artery of the paraganglioma" (Lasjaunias, 1976, Lasjaunias, 1978, Cavalcanti, 2009). While the tympanic paraganglioma is sometimes difficult to visualize because of its small size, the jugular paraganglioma is usually seen as a hypervascular mass with persistent homogeneous reticular stain. On the basis of the compartmental theory, temporal paragangliomas supplied by both neuromeningeal and tympanic branches of the ascending pharyngeal artery may represent two separate tumors appearing as one mass. The compartmental arrangement of paragangliomas has been described in which one artery supplies a single area with a specific venous drainage. More than one compartment occurs in 85 % cases. This type of tumor compartmentalization is explained by its encapsulation, which restricts recruitment of feeders from adjacent territories (Moret, 1982).
The presence of other arterial supply can be a sign of intracranial extension of skull base paraganglioma. These feeders are recruited from meningeal arteries of both external and internal carotid arteries. True intradural tumor ingrowth is proved if there is supply from the anterior inferior and posterior inferior cerebellar arteries.

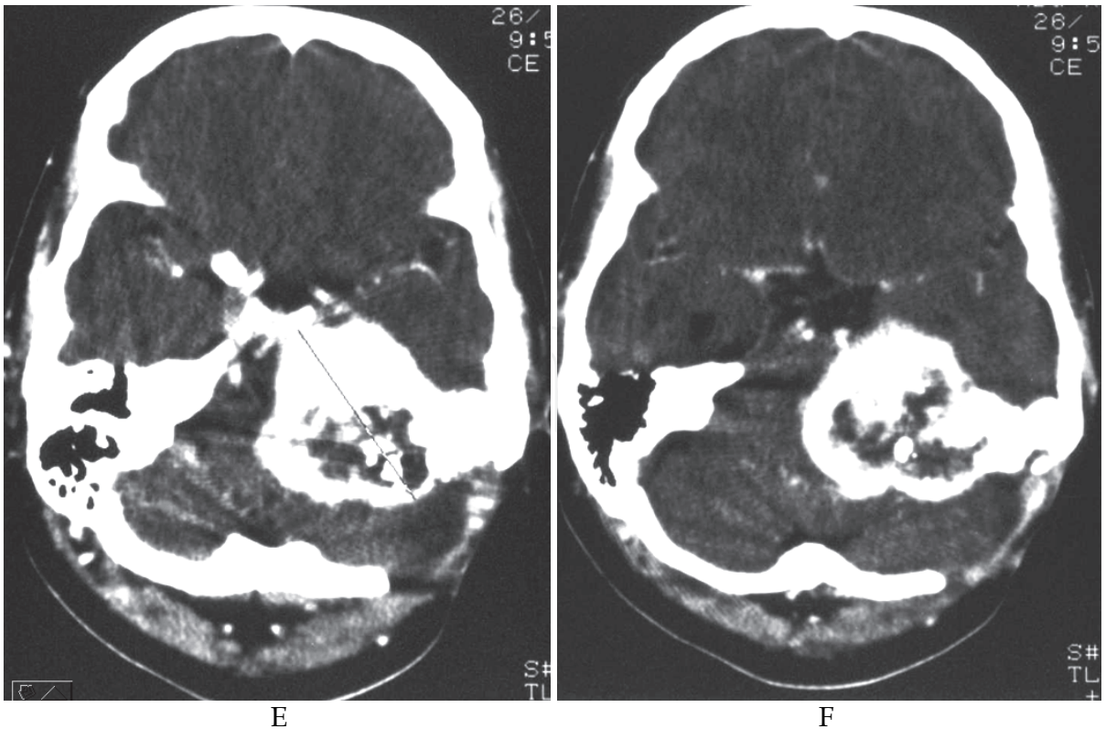
Patency of the internal jugular vein may be impossible to prove due to washout from arteriovenous shunting of the tumor. It is recommended to evaluate jugular vein on postembolization angiography (Valavanis, 2002).
Preoperative or intraoperative occlusion of the internal carotid artery should be considered when CT shows extensive destruction of the horizontal portion of the carotid canal or the tumor spreads to foramen lacerum and cavernous sinus. In such instances, the balloon test occlusion is useful as a part of angiographic work up.
Similarly to meningiomas, there is controversy as to whether embolization is useful before surgical resection. Tikkakoski et al. compared blood loss between paragangliomas embolized before surgery with that of non embolized paragangliomas and revealed a significantly
($p < 0.04$) lower amount of blood loss in the embolized group (588 ml versus 1374 ml) (Tikkakoski, 1997).
The diameter of arteries supplying paragangliomas is about 90 μm proximally and enlarges deep within the tumor. In the central part of the tumor arteries may reach a diameter of 300-600 μm. The most frequently used embolic agent are microparticles and in order to reach central part of the tumor, their diameter should be less than 90 μm then. The presence of arteriovenous shunting in some paragangliomas warrants the use of n-butyl-2-cyanoacrylate (Figure 4) or ethylenevinylalcohol (Valavanis, 2002).



A B



C D



E F

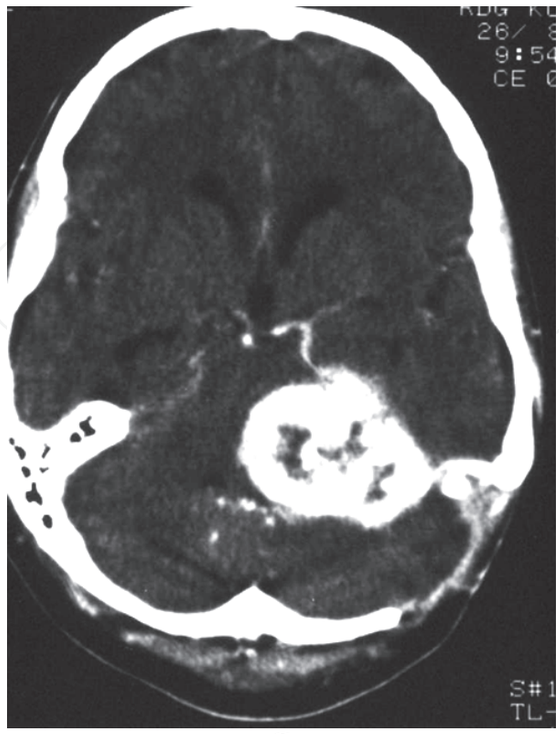


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Fig. 4. A 48-year-old woman with large paraganglioma. **A.** The left common carotid angiogram reveals ligation of the external carotid artery which was done during previous surgery. **B.** The late arterial phase shows opacification of hypervascularized tumor. **C.** The external carotid was reached via collateral branch from the superior thyreoidal artery. **D.** The substracted image demonstrates injection and penetration of the acrylic glue into the vascular bed of the tumor. **E-G.** Follow-up contrast enhanced CT scans revealed central necrosis with radiopaque glue deposit inside it.

The use of percutaneous, direct puncture therapeutic embolization is relatively new technique. Successful devascularization with direct percutaneous injection of n-butyl-2-cyanoacrylate ethanol or ethylenevinylalcohol has been reported (Casasco, 1994, Gemmete, 2010).

4.3 Juvenile nasopharyngeal angiofibroma

Juvenile nasopharyngeal angiofibroma is a benign hypervascular tumor. Although histologically benign, they behave in a locally invasive manner having a propensity to thin and erode bone and displace adjacent structures. The lesion originates from the postero-lateral wall of the nasal cavity in close proximity to the superior aspect of the middle sphenopalatine foramen where the posterior part of the middle turbinate attaches. The tumor later occupies the nasopharynx and posterior nasal cavity, sphenoid sinus, middle cranial fossa, maxillary sinus, oropharynx infratemporal fossa, orbit, and parapharyngeal space (Berenstein, 2004).

Symptoms are related to size and direction of spread. They may include repeat epistaxis (in 59 %), which may be life threatening, obstruction of paranasal sinuses and auditory canal, anosmia, decreased hearing and typical nasal speech due to nasal obstruction. It may be

difficult to differentiate paranasal tumor invasion from purulent sinusitis. Sarcomatous transformation has been reported after radiation therapy. Juvenile nasopharyngeal angiofibroma occurs in young males between 8 and 23 years of age. Tumors decrease in size with exogenous estrogen administration, and increase in size with testosterone (Johns, 1980).

CT and MR can preoperatively reveal bone involvement and extent of the tumor (Chagnaud, 1998, Kania, 2005). Since the introduction of preoperative embolization, both the blood loss and operative time have decreased (Garcia-Cervigon, 1988, Moulin, 1995). More recent transnasal endoscopic surgical techniques may be applied to many of these tumors and tend to rely on preoperative devascularization to decrease blood loss, enhancing the chances for complete tumor resection (Scholtz, 2001, Douglas, 2006). Polyvinylalcohol microparticles are used by most of operators.

A systematic angiographic protocol of juvenile nasopharyngeal angiofibromas begins with injection of the contralateral common carotid artery followed by the ipsilateral common and internal carotid arteries injections. Then the ipsilateral external carotid angiography for evaluation of the maxillary and ascending pharyngeal arteries is performed. The maxillary, accessory meningeal, ascending pharyngeal, and ascending palatine arteries are the most often embolized. If the tumor crosses the midline embolization of appropriate contralateral feeders is done.

In cases with extensive tumorous spread, the supply from the internal carotid artery branches may be embolized if possible, especially if the internal carotid artery needs to be sacrificed prior to surgery. Neurologic deficit is the most feared complication of preoperative embolization. Particularly blindness is of major concern as a result of ethmoidal supply (Lasjaunias, 1980).

Since the introduction of embolization of juvenile nasopharyngeal angiofibroma by Roberson in 1972 (Roberson, 1972) there have been several studies proving benefit of preoperative embolization by decreasing blood loss and operative times. One of the most recent proved, that additional supply from the internal carotid artery in high-grade tumors correlated with recurrences and if only the external carotid artery feeders were embolized, such preoperative embolization did not significantly decrease intraoperative blood loss (Liu,2000, Petruson, 2002).

The majority of studies are 10 to 20 years old and many reports do not even describe embolization techniques at the time (McCombe, 1990). Tumor recurrence after surgery may correlate with preoperative embolization. This fact could be due to tumor postsurgical remnants which are more probable after previous embolization (Petruson, 2002).

4.4 Hemangioblastomas

Hemangioblastomas are highly vascular benign tumors which occur predominantly in the cerebellum and spinal cord. Although 10 % to 20 % are associated with von Hippel-Lindau disease, most occur sporadically. Von Hippel-Lindau disease is a autosomal dominantly inherited disorder with incomplete penetrance and expression (Tampieri, 1993, Gläsker, 2005).

Hemangioblastomas become symptomatic by progressive growth either of the hemangioblastic nodule or an associated cyst or, more acutely, by spontaneous hemorrhage.

The role of angiography is to demonstrate vascular supply. Cerebellar hemangioblastomas are opacified from cerebellar arteries, but additional feeders from meningeal tentorial and vertebral feeders have been reported. However, angiography and preoperative embolization

are done only if the tumor is larger than 1,5 cm since such small hemangiomas are not associated with significant risk of pre-, intra- or postoperative hemorrhage (Gläsker, 2005, Gerlach, 2002).
Preoperative embolization of larger tumors may be performed without additional neurologic deficit even if the tumor is fed by more than one pedicle (Tampieri, 1993, Eskridge, 1996).
However, there have been reports of worsening of preexisting hydrocephalus within 12 hours of embolization (Cornelius, 2007), and intratumoral hemorrhages after small microparticles embolization. The authors hypothesized that 45-150 μm could initiated processes which led to fatal hemorrhage. The authors conclusion is, that if preoperative embolization of cerebellar hemangioblastomas appears absolutely necessary, proximal glue embolization or larger microparticles could be safer (Limaye, 2000, Cornelius, 2007).

4.5 Other hypervascularized tumors

Among other hypervascularized cranial tumors, which have been reported to be embolized, are hemangiopericytomas (Craven, 1992), chorioid plexus papillomas (Oten, 2006), metastases, schwannomas, hemangiomas, neurinomas, esthesioneuroblastomas (Berenstein, 2004). The embolization techniques follows that of meningiomas which are by far the most common tumors in this area.

5. Conclusion

We have rewieved embolization therapy of the cranial tumors. Pre-surgical embolotherapy for hypervascularized tumors is a well-established method. First of all, familiarity with the arterial anatomy of this region is fundamental for safe and successful treatment. Appropriate imaging work up and periprocedural detailed angiographic studies as well as careful selection of embolic agents help to decrease the ischemic complications.

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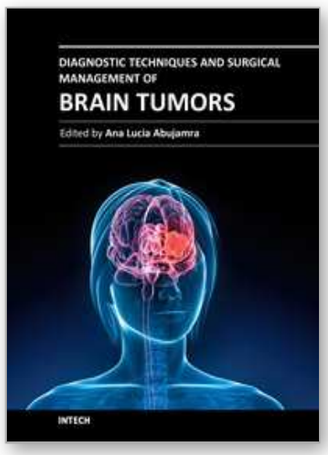

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