CONTINUING EDUCATION PROGRAM: FOCUS...

Epistaxis: The role of arterial embolization

A. Reyre\textsuperscript{a,∗}, J. Michel\textsuperscript{b}, L. Santini\textsuperscript{b}, P. Dess\textsuperscript{b}, V. Vidal\textsuperscript{a}, J.-M. Bartoli\textsuperscript{a}, G. Moulin\textsuperscript{a}, A. Varoquaux\textsuperscript{a}

\textsuperscript{a} Service de radiologie adultes, CHU Timone, AP–HM, 264, rue Saint-Pierre, 13385 Marseille cedex 5, France
\textsuperscript{b} Service de chirurgie ORL, CHU Timone, AP–HM, 264, rue Saint-Pierre, 13385 Marseille cedex 5, France

KEYWORDS
Epistaxis; CT angiography; Arteriography; Internal maxillary and sphenopalatine artery; Embolization

Abstract    Epistaxis is defined as flow of blood from the nasal fossae and is a common and benign disorder in the great majority of cases which does not require medical care. It may however become a genuine medical or surgical emergency because of the amount, repeated episodes or patient’s medical vulnerability (such as coronary artery disease patients). Epistaxis may be either primary or a symptom of an underlying disease. Four levels of problems need to be answered faced with epistaxis: recognizing it, and in particular not missing ''epistaxis'' due to swallowed blood or venous hemorrhage, which falls outside of the scope of interventional radiology; establishing the amount and its repercussions, particularly as a decompensating factor in another disease; investigating its cause and in particular never missing a tumor (male adolescents); obtaining hemostasis. Epistaxis varies not only in type and cause but must be considered in its clinical context. Arterial embolization is a treatment of choice for severe refractory epistaxis and some hemorrhages. When carried out by trained operators, it is an effective method with few risks of complications and is increasingly being used in reference centers (Brinjikji et al.). It remains, however, a method which is less widely used than surgery, particularly in the United States where in a series of 69,410 patients treated over the last 10 years for refractory epistaxis, 92.6% underwent surgical ligation, 6.4% embolization and 1% combined treatments (Brinjikji et al.). Epistaxis is occasionally catastrophic and requires extremely urgent management. In each case, close collaboration with the surgeon, the presence of an intensive care anesthetist and at least sedation are all factors which improve management and therefore the results of embolization. All patients and/or their friends/close family should have given ''reliable, clear and appropriate'' information.

© 2015 Éditions françaises de radiologie. Published by Elsevier Masson SAS. All rights reserved.
**General points**

Epistaxis has a prevalence of approximately 60% in the adult population and is the commonest acute disorder seen in ear-nose and throat clinical practice. The natural history of epistaxis is that it recovers untreated in the great majority of cases. According to Small et al. [1], only 6% of cases of epistaxis require medical treatment although some may be fatal without treatment.

The causes can be divided into two subgroups: an idiopathic (or essential) group and a “symptomatic” group in which the epistaxis is the symptom of an underlying disease.

More than 70% of cases are “idiopathic” or “essential” [2]. The majority of cases of essential epistaxis are related to risk factors [3], which are hypertension, hypercholesterolemia, smoking, the NSAID (mostly aspirin) and coagulation disorders. The organic causes of symptomatic epistaxis (Fig. 1) are trauma, surgery particularly trans-sphenoidal [4], ENT tumors, malformations such as hereditary hemorrhagic telangiectasias (Rendu-Osler disease) [5] or occasionally carotid-cavernous sinus fistulae [6].

Regardless of the cause, epistaxis can be serious and life threatening. When it is first seen, it is important to assess the amount and repetition of blood losses and carry out a general laboratory assessment (coagulation, complete blood count and grouping). In severe epistaxis, patients should be managed in a specialist ENT unit.

**Practical radiological anatomy**

A full knowledge of the branches of the external carotid artery is an essential. These should be identified by conventional angiography after selective catheterization of the external carotid artery (Figs. 2–10).

**Normal radiological anatomy**

The sphenopalatine artery is the artery usually responsible for refractory epistaxis. This can be occluded surgically through an endonasal approach or it can be embolized. Possible hazardous anastomoses with branches of the external carotid artery can result in visual or central deficits per- or post-embolization.

The vascular blush is an anastomotic plexus made up of arterial branches (second order) from the external carotid artery, described by Kiesselbach. This is located in the antero-inferior part of the nasal septum and is the site of over 90% of epistaxis. The sphenopalatine artery is its main blood supply (Fig. 3) and this is therefore the artery most often involved in idiopathic epistaxis.

Other branches of the maxillary artery should also be understood.

---

*Figure 1.* Causes of refractory epistaxis by incidence, from Christensen et al. in 2005 [10].
Anastomoses between internal and external carotid artery territories

A variable number of anastomoses exist between branches of the internal and external carotid arteries (aIC-aEC) carrying a risk of neurological accidents with off-target embolization. These “hazardous” anastomoses should always be looked for (Fig. 11). Only the ethmoidal arteries routinely contribute to the vascular supply of the nasal fossae. The anterior and posterior ethmoidal arteries arise from the ophthalmic artery (which itself is the first collateral branch of the internal carotid artery) and supply the upper part of the superior nasal concha and the most superior part of the nasal fossae.

The place of diagnostic imaging

A routine supra-aortic vessel CT angiogram (SAOV) is recommended before any treatment procedure. If the epistaxis is deemed to be severe, treatment should be given without delay.

This must include an image in the arterial phase and an image in the tissue phase. These have two uses:

- etiologic: they can be used to investigate for an underlying disease as the cause of the epistaxis (symptomatic epistaxis):
  - tumor: investigation for a mass in the nasal fossae or cavum or vascular lesions (wall irregularities, pseudoaneurysm),
Figure 4. Middle meningeal artery. Selective arteriography of the external carotid artery (a) and flat panel CT (FPCT) 3D oblique coronal (b) and axial (c) reconstructions. The middle meningeal artery (a, b: white arrow) is generally the second superior branch of the maxillary artery after the superficial temporal artery (a, b: arrowhead). After its origin, it travels vertically in the infratemporal fossa entering the cranium (c: arrows) through the spinous foramen and supplies the dura mater. It is not responsible for epistaxis and does not require embolization in this situation. Its anatomical position generally allows it to be preserved.

Figure 5. Infra-orbital artery. Selective arteriography of the external carotid (a) and flat panel 3D (FPCT) oblique coronal (b) and axial (c) reconstruction. The infra-orbital artery arises from the distal portion of the internal maxillary artery and enters the infra-orbital canal where it travels jointly with the infra-orbital nerve (a branch of V2). Before emerging through the infra-orbital foramen, it divides into an orbital branch supplying the inferior rectus muscle and the lachrymal sac and an anterior branch supplying the superior dental tissues and maxillary sinus mucosa. Some of its branches may anastomose superiorly with the facial artery (through the angular artery) or the ophthalmic artery (through dorsal nasal branches of the ophthalmic artery). It is not therefore directly responsible for posterior epistaxis but because of its anatomical site, it is rarely preserved.
Epistaxis: The role of arterial embolization

Figure 6. Descending palatine artery. Selective arteriography of the external carotid (a) and flat plane 3D (FPCT) oblique coronal (b) and axial (c) reconstruction. Descending palatine artery arises from the distal portion of the internal maxillary artery (a: arrow), crosses the maxillary nerve (V2) next to the pterygopalatine ganglion in the pterygopalatine groove (b: arrowhead) and gives rise to a descending vertical portion (b: arrow) in the greater palatine nerve canal (c: arrow) where it travels together with the nerve carrying the same name. It supplies the pharyngeal mucosa in the soft palate and upper gums. It is therefore not directly responsible for posterior epistaxis but because of its anatomical site, it is rarely preserved.

Figure 7. Inferior alveolar artery. Selective arteriography of the external carotid (a) and flat panel 3D (FPCT) oblique coronal (b) and axial (c) reconstruction. The inferior alveolar artery arises from the initial portion of the internal maxillary artery (a: arrowhead) on its inferior surface. It then runs downwards and anteriorly to join the inferior alveolar canal which it enters at the Spix spine (b, c: arrow). It runs together with the inferior alveolar nerve to the mental foramen where it gives rise to a median incisor branch, a mental branch which anastomoses with branches of the facial artery and a branch supplying the mylohyoid muscle. It is not responsible for epistaxis and does not require embolization in this situation. Because of its anatomical site, it can usually be preserved.
Figure 8. Ascending pharyngeal artery. Selective arteriography of the external carotid (a) and flat panel 3D (FPCT) oblique coronal (b) and axial MIP (c) reconstruction. The ascending pharyngeal artery arises from the initial portion of the external carotid artery immediately above the bifurcation of the common carotid artery (a: arrow). It is the smallest branch of the external carotid artery and runs in an ascending vertical plane in the carotid space. It separates into three vessels. The first is an anterior palatine vessel. The second is an anterior pharyngeal vessel which contributes to the supply to the nasopharyngeal mucosal space and pharyngeal muscles (pharyngeal constrictors and styloid septum muscles; b, c: arrow). Finally, the ascending pharyngeal artery gives rise to a posterior neuromeningeal vessel (a: arrowhead), which enters the base of the cranium at the hypoglossal jugular foramen and which contributes to supplying the mixed nerves (vasa nervorum) (IX, X, XI) and the hypoglossal nerve (XII). The neuromeningeal trunk (posterior) of the ascending pharyngeal artery is not therefore directly responsible for posterior epistaxis and because of its anatomical site, it can usually be preserved. Embolization of this vessel carries risks of neuronal damage.

Figure 9. Ascending palatine artery. Selective arteriography of the external carotid (a) and flat panel 3D (FPCT) sagittal MIP (b) and axial MIP (c) reconstruction. The ascending palatine artery (a: arrows) varies in origin and usually arises from the initial portion of the external carotid artery (b: arrow) close to the facial artery [23] (b: arrowhead). Amongst other things, it supplies the palatine tonsils, the roof of the palate and the Eustachian tube (c: arrows). Because of its many anastomoses, it may make a minor contribution to the posterior vascular supply of the floor of the nasal fossae. It is believed to be difficult to catheterize and embolization of this vessel is therefore only rarely an angiographic target to treat refractory epistaxis.
Figure 10. Facial artery. Hyperselective arteriography of the facial artery (a) and selective arteriography of the external carotid artery in coronal MIP (b) and axial MIP (c) 3D flat plane capture CT plan (FPCT) reconstructions. A branch of the external carotid artery, the facial artery, supplies the superficial planes of the face. Through its terminal branches, it supplies the anterior nasal region through the superior labial artery (a: arrow) which gives rise to the infraseptal artery at the internal canthus (b: grey arrow) where it is not unusual for it to have anastomoses with the ophthalmic artery system. It is usually embolized second line and embolization should take account of potentially hazardous anastomoses and possible unsightly discoloration of the lips as a result of colored microbeads. Embolization is usually carried out as a result of persistent epistaxis immediately following embolization of the sphenopalatine arteries.

- vascular malformation: investigation for arteriovenous shunts,
- hereditary telangiectasias: investigation for telangiectasias,
- post-traumatic or postoperative: investigation for an active leak, pseudoaneurysms or wall irregularities;
- preoperative:
  - with a view to endovascular treatment: anatomy of the SAOV, atheroma (risk of CVA),
  - with a view to surgery: identification of the sphenopalatine foramen and ethmoidal arteries.

Figure 11. Example of aIC-aEC anastomoses in a 62-year-old female patient suffering from Rendu Osler disease. Selective angiography of the external carotid artery (a) shows the presence of “blushes” in the nasal fossa mucosa (a: arrow) without any anastomosis being seen with the internal carotid system. Note the absence of opacification of the internal maxillary artery because of prior proximal microcoil embolization. Selective angiography of the internal carotid artery (b) and 3D flat panel image (c: flat panel CT) in sagittal MIP reconstruction shows a large anastomotic network between the internal and external carotid artery systems contributing posterior vascularization to the nasal fossae and nasopharynx. The ethmoidal (b: arrows) arteries are opacified together with multiple arteries at the base of the cranium (a: foramen lacerum artery, foramen rotundum artery and pterygoid foramen artery; c: arrows). Branches of the sphenopalatine artery (a, b: arrowheads) are both opacified by the two arterial systems showing aIC-aEC anastomoses and making possible embolization particularly difficult. Note the aneurysms at the ending of the carotid artery (c: arrowheads).
CT angiography is usually normal in essential epistaxis.

Assessment of severity

Before any treatment is given, it is essential to define whether the epistaxis is mild or severe. Diagrammatically, two clinical pictures can be distinguished.

Mild epistaxis

Blood flow is not particularly heavy and comes in drops from the nostril. It almost always begins unilaterally. ORL examination is straightforward after the patent has wiped their nose and anterior rhinoscopy shows the site of the bleeding, which is generally anterior in Kiesselbach’s plexus. This has no consequences on the patient’s general condition.

Severe epistaxis

The severity of epistaxis depends on different factors, which should be assessed immediately:
- the presence of hemorrhagic shock;
- a fall in hemoglobin: epistaxis is deemed to be severe if the blood hemoglobin is under 8 g;
- the amount: this is not so much assessed by whether the epistaxis is bilateral or anterior/posterior but using objective concepts such as heart rate (pulse), blood pressure, sweating and pallor. Volume is always difficult to estimate and is often over-estimated by the patient or the patient’s friends/family, but can be underestimated if it is swallowed;
- the duration or repetition of epistaxis should be confirmed;
- association with a disease liable to decompensate because of blood loss such as coronary artery disease or carotid artery stenosis;
- the presence of coagulation abnormalities (such as anticoagulation therapy) occasionally makes control of the bleeding more difficult.

The nasal cavities are occasionally difficult to examine because of the amount of the bleed which is often bilateral and anteroposterior.

Treatment of essential epistaxis

This depends on how benign or severe the epistaxis is.

If the epistaxis is symptomatic, treatment is that of the underlying cause, which will be discussed in the next section.

If the epistaxis is benign

The treatments follow a “crescendo” sequence, increasing in step in the following order:
- Local treatment: “minor measures”:
  - manual bidigital compression for very anterior bleeding;
  - anterior meshing by applying hemostatic meshes (Merocel®);
- endoscopic electrocoagulation with bipolar diathermy forceps under local anesthesia if an anterior lesion is visible;
- if this fails:
  - anteroposterior tamponade: this is usually a treatment for posterior rather than anterior epistaxis. In the latter situation, anterior and posterior tamponade should be applied using a dual balloon tube (Fig. 12);
- if this fails:
  - here again, posterior epistaxis is usually the cause,
  - anteroposterior tamponade may be ineffective in a number of cases (25 to 52% depending on the series), this is defined as failure of two posterior tamponades over 48 h,
  - in this situation, the epistaxis is said to be refractory and is deemed to be severe. This therefore requires surgery or endovascular treatment.

If the epistaxis is deemed to be severe from the outset (or refractory)

In practice, severe epistaxis should be treated initially by anteroposterior tamponade.

If it is life threatening (hemorrhagic shock, fall in hemoglobin to under 8 g), if bleeding persists (refractory epistaxis) or on a background of a very serious underlying cause, it should be treated surgically (electrocoagulation of the sphenopalatine artery) or through an endovascular radiological approach (arterial embolization).

If anterior tamponade fails, the dual balloon tube is introduced as far as the rhinopharynx (Fig. 12). The posterior balloon (arrow) is inflated moderately and blocked in the choana. The anterior balloon (arrowhead) is then inflated in the vestibule of the nostril to isolate the whole nasal fossa. The balloons should be filled with water and deflated every 6 hours to avoid mucosal necrosis.

Surgery

Surgery can be useful for uncontrolled bleeding. Different types of surgery are possible: endonasal electrocoagulation of the sphenopalatine artery (Fig. 13) involves using bipolar diathermy forceps to coagulate the sphenopalatine artery at its emergence (sphenopalatine foramen) into the posterior part of the nasal fossa and is effective in 75% to 85% of cases (or even more in very experienced hands). Its limitations are
mucosal damage making it difficult to view the artery and heavy bleeding due to blood coagulopathies.

The ethmoidal arteries are usually ligated if embolization fails if post-embolization views show revascularization of the nasal fossa by the anterior or posterior ethmoidal artery.

Ligation of the anterior ethmoidal artery is usually performed by a surgical approach through the surgical approach internal canthus rather than endonasally. The posterior ethmoidal artery is ligated in recurrence of bleeding despite ligating the anterior ethmoidal artery.

Ligation of the external carotid artery through cervicotomoy is a technique which has now been abandoned except in very rare cases as it obstructs access to future embolization. Ligating the internal maxillary artery through a vestibular approach is a demanding technique which is poorly suited to emergency use and is rarely carried out.

Endovascular treatment

Anterior essential epistaxes are not treated by embolization unless symptomatic treatment and/or surgery have failed. Posterior epistaxis is a good indication for embolization [8] (Fig. 14) if the epistaxis is severe from the outset (hemorrhagic shock or large fall in hemoglobin with a hemoglobin of under 8 g) or if two posterior tamponades have failed over more than 48 hours (refractory epistaxis).

Embolization technique:

• femoral approach with a valve introducer (6F if possible);
• selective catheterization of the common internal and external carotid artery (if possible using a guide catheter) ipsilateral to the bleeding (and possibly contralateral if the bleeding side is not identified on ENT examination);
• microcatheterization (0.021 inch) of the arteries to be embolized. Flexible "navigating" microcatheters should be used in preference to avoid arterial spasm.

The arterial architecture and bleeding areas should be identified on anteroposterior and lateral views. Anastomoses with the cerebral or ophthalmic arterial territories must be identified, together with those between the sphenopalatine and anterior ethmoidal arteries via the turbinate and infra-orbital arteries. Catheterization of arteries to the neck (subclavian artery and its branches) is generally not required in idiopathic epistaxis although is essential in other cases of ENT hemorrhage, particularly postoperatively in patients who have undergone laryngectomy and are bleeding from their tracheostomy cannula.

Hemorrhage from the anterior ethmoidal artery requires surgical treatment and is not treated endovascularly because of the dangers associated with microcatheterizing the ophthalmic artery.

Embolization of the ipsilateral sphenopalatine artery is sufficient in most cases and may be combined with embolization of the ipsilateral facial artery (Fig. 15), which is very often anastomosed with the sphenopalatine artery through the infra-orbital artery (which significantly reduces the recurrence of hemorrhage) (Fig. 5). In bilateral epistaxis, both sphenopalatine and facial arteries may be embolized.

In some cases, other branches of the external carotid artery may restore flow to the ending of the sphenopalatine artery and the internal maxillary artery through counter-current anastomoses. These only occasionally develop after occlusion of the main trunk and can then be catheterized and occluded.

Oclusion materials

The choice of occlusion material depends mostly on the cause of the epistaxis. Microparticles are preferable in most cases of essential epistaxis when non-resorbable microparticles with a diameter of over 500 microns in size are extremely effective. These should however be avoided if anastomoses are present between the sphenopalatine territory and the territory of the anterior ethmoidal artery, particularly if these two territories are contributing to the epistaxis. In order to be used, they require free-flow injection and radioscopy monitoring to avoid any risk of reflux and to identify anastomoses which are unmasked secondarily. These microparticles can cause complications during embolization of the facial artery (skin necrosis and pain).

Insertion of microcoils may be a good alternative: these are either "flushable" or controlled release. They should
be positioned distally in contact with the hemorrhagic area. They have the disadvantage of permanently obstructing the artery and occluding one of the targets for possible re-embolization in the event of a recurrence.

Cyanoacrylate glues (Glubran 2® or Histoacryl®) are difficult to use and require operators trained in their use [7]. They are immediately effective but are reserved for life-threatening cases. Embolization with Onyx® is more straightforward and carries less risk of off-target embolization than the cyanoacrylates. This requires good knowledge of the toxicity and delivery of the substance [8]. When it is used in branches of the internal maxillary artery, the dimethylsulfoxide contained in Onyx® can cause profound bradycardia due to a trigemino-cardiac reflex [9].

**Results: success, recurrence and complications**

The technical success rate of embolization is high, in the region of 80 to 88% [10]. Complications may occur in 8%—13% of cases [10—12]. The most recent studies show better results probably due to advances in techniques, operators, the materials available to them and embolization agents (microcoils, microparticles, etc.).

There is a wide range of potential complications after endovascular treatment of epistaxis. In addition to recurrence of hemorrhage, the literature reports cases of facial neuralgia, septal perforation, sinusitis, and otitis media. Systemic complications may also occur, including inhalation hypoxia, hypovolemia, angina and/or myocardial infarction. The meta-analysis reported by Cullen on 539 patients not only identified the different complications of endovascular treatment but compared their incidence with those of surgery [13].

The most severe complications of arterial embolization are cerebrovascular accident and obstruction of the central artery of the retina. Depending on the series, these occur in between 0 and 2% of cases [14]. In Brinjikji’s retrospective analysis [15] on a population of 64,289 patients treated in the United States between 2003 and 2010, the CVA rate was found to be significantly higher after embolization (0.9%) than after surgery (0.1%) (Fig. 16).

These results raise questions. It is likely that the increasing use of minimally invasive endoscopic techniques to ligate the sphenopalatine artery is improving patient management and secondly, the experience of interventional radiologists, the state of the patient’s arterial system and embolization agents are all factors which influence development of CVA. Only a few authors have produced a critical analysis of the embolization materials and agents used in this situation. Routine use of microcatheterization is contributing towards a reduction in the incidence of CVA [3].

In terms of procedure costs, data from the studies are controversial probably because of the differences in price of the various embolization agents which can be used and differences in cost of the radiology and surgical environments. Embolization appears to be more expensive in surgery [16], although this excess cost is usually compensated by shorter hospitalization stays for embolization [17].

Early rebleeding after embolization is usually treated by further embolization unless blood flow has obviously been restored through the ethmoidal arteries. Collateral vessels are examined angiographically (the anterior ethmoidal and contralateral external carotid arteries). Occlusion of the two facial arteries and two sphenopalatine arteries is a possible option.
Epistaxis: The role of arterial embolization

CVA due to off-target embolization is managed conventionally. 
Post-embolization neuropathy is treated with corticosteroids.

Secondary epistaxis

Embolization of branches of the external carotid artery in ENT oncology

Epistaxis generally involves neoplastic sites in the nasopharynx (Fig. 17). The incidence of this complication is low in cervicofacial surgery (4.3%) [17] although external radiotherapy increases the risk by a factor of 7.6 [18,19].

The bleeding may arise from the tumor by erosion of vascular structures which are invaded, particularly in progressive disease [22], but may occur as a result of tumor necrosis in patients who have responded very well to treatment. The hemorrhage may occur on a background of complete remission as a result of irradiation-induced damage, in which case it is promoted by a local inflammatory factor (osteoradionecrosis, tracheostomy cannula, orostome). Occasionally, hemorrhage in the postoperative period may be due to loosening of a vascular structure (reconstruction flap). Endovascular (hemostatic) treatment is an option with a recognized indication [18]. The general principles of embolizing branches of the external carotid artery are the same as for essential epistaxis (Appendix).

Rendu-Osler disease

Rendu-Osler disease or hereditary hemorrhagic telangiectasiae is an autosomal dominant transmitted angiomatosis belonging to the phacomatoses group. It is managed in a multidisciplinary setting in a local or regional center.

Epistaxis is the most common clinical feature and recurs frequently.

ENT embolization is only considered in patients with frequent episodes of bleeding who cannot live a satisfactory everyday life. Embolization is performed by microparticles (between 500 and 700 μm in size) (Fig. 18) as distally as possible, preserving the arterial pedicles vascularizing the nasal fossae to allow repeated embolizations. Microcoils are therefore not recommended or even contra-indicated in order to avoid occluding the approach route.

Embolization of benign tumors

Most cases involve preoperative embolization and the tumors involved are mostly nasopharyngeal (angio-)fibromas (Figs. 19–21) and occasionally paragangliomas which may be embolized preoperatively to reduce the risk of surgical bleeding [20,21].

Angiofibromas occasionally have a significant vascular component from the internal carotid artery. Embolization of the internal carotid artery territory carries neurological risks and should be reserved for extremely specialized centers.

Embolization of the sphenopalatine artery is generally insufficient and devascularization requires the facial artery and ascending pharyngeal artery to be embolized, occasionally bilaterally. Embolization of the ascending pharyngeal artery can lead to paralysis of mixed nerves and should be carried out with caution with preoperative preference given to microcoils. Anastomoses may exist with the vertebobasilar territory which should be looked for carefully during diagnostic arteriography.
Figure 17. Epistaxis from a round, median nasopharyngeal tissue lesion vascularized on CT (a: arrowheads) seen as an isointense appearance on T2-weighted imaging (b: arrowheads), with reduced diffusion (ADC = $0.5 \times 10^{-3}$ mm$^2$/sec, c: arrowheads) and vivid enhancement after IV of gadolinium chelate on T1-weighted image with fat saturation (d: arrowheads). Hyperselective catheterization of the anterior trunk of the ascending pharyngeal artery (e: arrows) shows a tumor blush (e: arrowheads). Devascularization was achieved by inserting controlled release microcoils (f: arrow) showing devascularization of the tumor process in a later view (f: arrowheads). After embolization, biopsy showed a plasmacytoma allowing this to be managed specifically.

Figure 18. Embolization in Rendu-Osler disease. External carotid artery arteriography before embolization (a) showing distal hypervascularization of the nasal fossa by branches of the sphenopalatine artery (a: black arrows) and by branches of the facial artery (a: white arrows). A repeat view after embolization (b) carried out using 700 µm microparticles shows satisfactory occlusion of the sphenopalatine and facial arteries. Note a symptomatic refeeding of the nasal fossa vascularization through the anterior ethmoidal artery (b: white arrow). Telangiectasiae are also present in the lingual artery (b: black arrow).
Figure 19. Juvenile angiofibroma. T2-weighted axial (a), T1-weighted axial (b), T1-weighted after IV of gadolinium chelate and fat saturation (c), ADC mapping (d), T2-weighted coronal (e) and T1-weighted 3D with gadolinium enhancement and fat saturation (f). A mass is present in the right nasal fossa (a–d: arrows) exhibiting a few "flow voids" on T2-weighted imaging (a: arrowheads) and vivid enhancement after IV of gadolinium chelate (b, c), with no reduction in ADC values ($1.4 \times 10^{-3} \text{ mm}^2/\text{sec}$, e: arrows) entering the sphenopalatine foramen (e, f: arrows).

Figure 20. Juvenile angiofibroma. 3D dynamic gadolinium-chelate enhanced MR angiography every 4 seconds in a sagittal plane (a–f) showing the hypervascular nature of the lesion (a–f: arrowheads), uptake in which is almost synchronous with the basilar trunk and cerebral arteries (b: arrows).
Figure 21. Preoperative embolization of a nasopharyngeal fibroma. Nineteen-year-old male patient with a Radkowski classification type 1B nasopharyngeal fibroma. Diagnostic angiography of the external carotid artery (reflux into the internal, a) shows a tumor ‘blush’ (a: arrowheads) from the sphenopalatine artery (a: arrows). Devascularization was achieved by hyperselective catheterization and embolization of the arterial trunk with Onyx® 34 (ethylene vinyl alcohol copolymer, EV3 Micro Therapeutics Inc., Irvine, CA) (b: arrowheads), with no episodes of bradycardia. A repeat view shows complete devascularization of the mass (c: arrowheads).

Conclusion

Embolization now currently offers huge benefit in the management of epistaxis. Its results are improved by multidisciplinary collaboration (surgeons and intensive care physicians), by strictly selecting its indications, knowledge of the vascular anatomy and mastering the process of microcatheterization and the embolization agents.

Take-home messages

- there are two types of epistaxis:
  - essential,
  - symptomatic, secondary to underlying disease;
- arterial embolization is a treatment of choice for severe refractory and symptomatic epistaxis;
- full knowledge of the anatomy of the branches of the external carotid artery is an essential requirement for embolization. Its main indications are:
  - essential epistaxis which is serious from the outset and life threatening or refractory after 48 hours of correct medical treatment,
  - severe epistaxis in ORL oncology either through invasion or postoperatively,
  - preoperative devascularization of hypervascular tumors (paragangliomas and nasopharyngeal fibromas),
  - frequent episodes of epistaxis in Rendu-Osler disease;
- the treatment of benign essential epistaxis is initially local and then ‘crescendo’ escalation;
- hemorrhages from the anterior ethmoidal artery require surgical treatment;
- the occlusion materials vary depending on the type of epistaxis:
  - microparticles (from 500 to 700 microns) for essential epistaxis or Rendu-Osler disease,
  - microcoils or glue, particularly in secondary epistaxis or with hazardous anastomoses,
  - microcoils are not recommended in Rendu-Osler disease;
- microparticles can only be used when no hazardous anastomoses with the internal carotid artery or ophthalmic artery territory are present and require free flow injection under radioscopy guidance;
- positioning microcoils may be a good alternative. These have the disadvantage of permanently obstructing the artery (re-embolization is impossible in the event of recurrence);
- amongst the complications of arterial embolization, cerebrovascular accident and central retinal artery obstruction are the most severe;
- the complication rate in a trained operator’s hands is no greater than that of surgery.

Clinical case

A 70-year-old man presents with recurrent low to moderate volume epistaxis. He has a past history of T2N0M0 conventional cell renal carcinoma, and a coronary stent in his left anterior descending artery. Clinical examination reveals a mass in the right nasal fossa.

An MRI is performed including: T2-weighted images (A), Diffusion-weighted MR imaging (B), T1-weighted images (C), dynamic MR angiography (D), a time-intensity (infusion) curve (E) and T1-weighted gadolinium-chelate enhanced image (F) (Fig. 22).

Questions

1. Describe the abnormalities present on MR images.
2. What are the correct answers?
   A. The epistaxis is secondary to a right nasal fossa tissue lesion.
   B. The hypervascular appearances with lavage (E) and the high apparent diffusion coefficient of the lesion (1.8 × 10⁻³ mm²/s; B) are both in favor of a hematogenous metastasis from a renal cancer.
   C. The patient’s age and lack of invasion of the pterygopalatine groove do not support an angiofibroma.
Epistaxis: The role of arterial embolization

Figure 22. Sinus MRI. a: T2-weighted axial MRI showing a well-delineated lesion in the right posterior nasal fossa with clear hyperintensity on T2-weighted imaging; b: diffusion-weighted MRI (apparent diffusion coefficient): the lesion has high ADC values; c: axial T1-weighted imaging: right posterior nasal fossa lesion, isointense on T1-weighted imaging; d: dynamic MR angiography after IV administration of gadolinium chelate: early enhancement of the lesion in the arterial phase; e: T1-weighted DCE perfusion MRI showing the hypervascular nature of the lesion with a washout phase; f: T1-weighted MRI after IV of gadolinium-chelate shows: extensive homogeneous enhancement of the lesion.

D. A biopsy under a local anesthetic is justified before excision of the lesion.
E. Receipt of anticoagulants or platelet antiaggregants is a contra-indication to possible embolization.

3. An arteriography is performed with a view to embolization (Fig. 23). What are the correct answers?

A. Fig. 23a is a selective arteriography view of the external carotid artery showing partial opacification of the internal carotid artery probably due to reflux.

B. Fig. 23b is a hyperselective arteriography view of the facial artery showing a tumor blush identical in size to series (A).
C. Onyx® microcoils and calibrated spherical particles ≥ 700 μm are both embolization agents which can be used.
D. Fig. 23c is a hyperselective arteriography view of the ending of the internal maxillary artery post-embolization showing complete devascularization of the blush and preservation of the infra-orbital and descending palatine arteries.

Figure 23. Arteriography. a: selective arteriography of the right external carotid artery showing a lesion blush within the l a nasal fossa which has multiple arterial supplies including several branches of the internal maxillary artery: sphenopalatine artery, descending palatine artery and superior alveolar artery; b: hyperselective arteriography after microcatheterization of the internal maxillary artery: a lesion blush is seen with the different arterial supplies; c: repeat hyperselective arteriography after embolization with 700 μm spherical microparticles. Complete devascularization of the lesion. Note that the infra-orbital artery is preserved.
E. At this stage in the procedure (C), the lesion may carry a risk of revascularization from the contralateral or intracranial arterial system.

Answers
1. A: MRI T2-weighted axial section: well-circumscribed lesion in the right posterior fossa with hypersignal.
   B: MRI diffusion-weighted sequence (apparent diffusion coefficient): this lesion shows high ADC values.
   C: MRI T1-weighted axial section: isointense lesion of the right posterior fossa.
   D: dynamic MR angiography after IV of gadolinium-chelate: early lesion enhancement in the arterial phase.
   E: MRI perfusion sequence T1 DCE: hypervascular nature of the lesion with washing stage.
   F: T1-weighted MRI after IV of gadolinium-chelate: large and homogeneous enhancement of the lesion.

2. Correct answers: A, C, Comments:
   B: the high ADC suggests a benign lesion.
   D: a biopsy is contra-indicated in hypervascular nasal sinus lesions.

3. Correct answers: A, C, D, E. Comments:
   B: internal maxillary artery (ending). This patient underwent distal embolization with calibrated spherical microparticles ≥ 700 µm in size (Embozen® Mircosphere 700 µm CelonoVa®, C) followed by occlusion of the trunk of the internal maxillary artery with microcoils. No significant bleeding occurred on endonasal excision. The final diagnosis was a benign angiomatosus nasal fossa polyp.

Indication
- Posterior epistaxis, refractory after 48 hours of correct medical treatment
- Serious epistaxis, life threatening from the outset.

Assessment of patient status
In all cases
Laboratory assessment: PR APTT INR, CBC, platelets and blood group.

Controlled hemorrhage
Assessment of cause and feasibility assessment: Supra-aortic vessel CT angiography from the aortic arch to the circle of Willis and facial bone CT.

Hemodynamic stability not controlled by intensive care
Either endovascular treatment with embolization or surgery should be discussed with the ENT surgeons and intensive care physicians.

In the operating suite
Patient placed lying down with a head rest, sedated, radioscopy in position, infusion, informed and ‘reassured’.
   One operator, one operator’s assistant and an operating suite technician.
   Anesthetist/Intensive care physician for station and general anesthesia if required, monitoring and correcting hemodynamic indices.

On the angiography table
6F valve introducer. Consider 35 cm long introducer depending on morphology of the iliofemoral arterial system. 6F guide catheter (Envoy®, 100 cm) on 0.035” hydrophilic guide (Terumo®) and antireflux valve.

Microcatheter (at least 135 cm long) suitable for the embolization agents (compatible with Onyx® if necessary) with a straight or pre-shaped (45° or 90°) tip and two radio-opaque markers if controlled release coils are used. 45° and 90° curved hydrophilic microguides. The introducers, catheters and microcatheters should be rinsed and infused by bags and blood pressure.

In the angiography suite trolley
- Microcoils suitable for the microcatheter.
- Calibrated microparticles (Luer-lock syringe prefilled to 500–700 µm).
- Cyanoacrylates, (Histoacryle® or Glubran2®).
- Ethylene vinyl alcohol copolymer (Onyx®), rarely used in emergencies.

‘‘Conventional’’ procedure
Femoral artery approach, valve introducer rinsed and perfused, guide catheter rinsed and perfused.

Acknowledgements
Thanks to Alexis Jacquier (University Professor and Hospital Consultant), Hervé Brunel (Hospital Consultant), Jérôme Soussan (Hospital Consultant), Aurélie Dehaene (Hospital Consultant), Antonin Flavian (Senior Clinical Registrar), Jean-Marie Caporossi (Senior Clinical Registrar), Cléo Gaudon (Senior Clinical Registrar), and to Jean Izaaryene (Senior Clinical Registrar) for their clinical and imaging contributions.

Disclosure of interest
The authors declare that they have no conflicts of interest concerning this article.
• Injection into the common carotid artery after selective catheterization of the bleeding side with long series to study jugular venous return.
• Selective injection of the internal carotid artery with anteroposterior and lateral views (to study the ophthalmic artery and anterior ethmoidal artery).
• Selective injection of the external carotid artery, posterolateral and lateral views including the nasal fossae in the field.
• Investigation for the target and identifying hazardous anastomoses: foramen lacerum artery, clival branches, pterygoid artery, foramen ovale artery, foramen rotundum artery and in the ophthalmic artery.
• Use of a microguide and microcatheter couple from the start leaving a guide catheter at the origin of the external carotid artery. Hyperselective microcatheterization of the target (failing this the sphenopalatine artery).
• Use of arterial tracing. Do not catheterize the branches of the external carotid artery with the 0.035 guide to avoid spasm.

If hazardous anastomoses are present, place the microcatheter beyond the anastomosis but always consider the risk of reflux during embolization. If necessary, carry out proximal embolization (of the trunk) of the hazardous anastomosis using microcoils before the microparticles.

In the event of arterial spasm, to enable free flow embolization, use an in situ intraluminal perfusion of nitrates (1 mg of Risordan®) with the agreement of the anesthetist-intensive care physician.

Embolization always free flow as close as possible to the target and a radioscopy control.

Angiography images are taken routinely at the end of the procedure to investigate for collateral circulation and revascularization of the target. If the sphenopalatine artery is revascularized from the facial artery, the facial artery should be embolized with very distal microspheres or microcoils.

The final check investigates for revascularization of the nasal fossa particularly by the ethmoidal arteries.

Leave the femoral introducer in situ until hemodynamic and neurological control has been confirmed clinically by the ORL team in the angiography suite.

References