# 6 The Role of Interventional Radiology

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**Objectives**

(a) Provide an overview of Interventional Radiology in the Maxillofacial & Head and Neck Surgery
(b) Discuss the role of Interventional Radiology in the Management of Head and Neck Vascular Lesions

## 6.1 Outline

### 6.1.1 Section 1: Diagnostic Imaging of the Head and Neck

1. General Imaging [1–14]
   (a) The Head and Neck (H&N) is a complex anatomical region divided into both anatomic and functional sections and spaces.
   (b) The role of imaging in the H&N is to detect, characterize, and delineate lesions, while also determining involved surrounding structures and lymph nodes.
   (c) Imaging is also used for interventional procedure guidance, distant metastasis detection, therapy planning, monitoring response, and surveillance.
   (d) Commonly employed cross-sectional modalities include magnetic resonance imaging (MRI), computed tomography (CT), ultrasound (US), and positron emission tomography computed tomography (PET-CT).
   (e) The primary imaging modality will depend on the main clinical concern, availability, cost, reimbursement, and patient-specific factors such as contrast allergies or implanted metallic devices.
   (f) Most modalities will be complementary, but in any case, the initial study should be one that will both provide all the necessary information for proper management and being safe and cost-effective.
   (g) US is a reasonable first step in the evaluation of any H&N vascular pathology given its rapid deployment, cost-effectiveness, and safety; however, imaging should be tailored to best address the clinical concern.
   (h) MRI, as in other parts of the body, is superior to CT in the evaluation of soft

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tissues, however there are certain caveats when imaging the H&N with MRI. MRI is typically employed to evaluate the suprahyoid neck, where it is less affected by artifact from dental amalgam or motion and is less suitable for the evaluation of the infrahyoid neck, a region that is sensitive to respiratory motion artifact from coughing or breathing.

(i) Common indications for imaging of the H&N include lesion localization, tumor staging, and infection workup. When imaging the head and neck for the purpose of finding a lesion, contrast-enhanced CT (CECT) is generally the preferred initial modality. In some cases, CECT is also generally preferred for staging. For example, CECT is the best first step for staging of Squamous Cell Carcinoma. The same is true for the evaluation of a suspected infection, as CECT is excellent at distinguishing between cellulitis, phlegmon, or abscess.

(j) MRI is the preferred modality in the evaluation of tumor extent, perineural tumor spread, and/or intracranial extension.

(k) It is important to have a general approach to lesions that are found in the H&N. As mentioned before, the H&N is divided into many sections or spaces, and as such, the first step is to determine the space in which the lesion originated. This allows us to formulate a space-specific differential diagnosis.

(l) It is also important to consider whether a lesion could represent a normal structure or variant, or whether the abnormal finding merits no further intervention.

2. Imaging of Vascular Lesions

(a) Radiology plays a crucial role in the diagnosis and management of vascular lesions in the H&N.

(b) Diagnostic imaging can confirm a diagnosis, precisely delineate a lesion, distinguish associated abnormalities from normal variation, and allow for the surveillance and monitoring of a lesion, pre- and post-therapy.

(c) Interventional radiology provides several therapeutic options for vascular anomalies, which will be discussed in greater detail in later sections.

(d) Ultrasound is often used to image vascular anatomy given its ability to demonstrate flow in real time, as well as its ability to quantify flow using Doppler technique.

(e) As mentioned before, ultrasound may be used as the first step in the evaluation of H&N vascular lesions and may help characterize the lesion as having primarily venous or arterial flow.

(f) Although typically not preferred for the evaluation of vascular lesions given the associated ionizing radiation, CT can be employed to assess bony anatomy, as well as to detect calcifications or phleboliths in venous malformations.

(g) Vascular lesions are best assessed when enhanced following the administration of intravenous contrast.

(h) Generally, MRI is the primary modality for the evaluation of vascular anomalies. Its superior soft tissue resolution makes it the preferred imaging tool.

(i) When compared to other modalities, MRI is better able to identify aggressive imaging features that may suggest a more invasive or malignant diagnosis.

(j) A basic MRI protocol of the H&N will at least include precontrast and fat-saturated postcontrast T1-weighted images, as well as T2-weighted images, and will require at least 30 min of scanning time.

(k) MR angiography can be used to evaluate high-flow lesions during arterial and venous phase.

3. Overview of Non-Invasive Imaging Modalities

(a) Ultrasound

(i) General

1. Based on the transmission, reflection, and reception of sound waves of variable frequencies.
2. Higher frequencies (5–15 MHz) are used for the evaluation of superficial structures, such as the

H&N. Lower frequencies (2–4 MHz) are used for the evaluation of deeper structures, such as the abdomen and pelvis.
3. Non-contrast techniques include gray-scale, spectral Doppler, color Doppler, and power Doppler.
4. Contrast-enhanced US (CEUS), recently approved by the FDA, is modality used to evaluate lesion enhancement.

(ii) Techniques
1. Gray-scale
   (a) Used to evaluate anatomy and morphology.
2. Spectral Doppler
   (a) Used to evaluate spectral waveforms, as well as blood flow velocity, direction, and resistance.
3. Color Doppler
   (a) Assigns color to blood velocity and direction (red reflects flow towards the transducer and blue reflects flow away from the transducer).
4. Power Doppler
   (a) Detects slow flow, otherwise not seen on color Doppler. Does not provide information on velocity or direction.
5. CEUS
   (a) Consists of intravenous injection of highly echogenic microbubbles.
   (b) Used to evaluate lesion enhancement, blood flow dynamics, tissue vascularity, and stent grafts.

(iii) Advantages
1. US is widely available, portable, affordable, and entails no ionizing radiation risk.
2. It is excellent at evaluating tumors in real time, including the assessment of adjacent blood vessels and tumor vascularity.
3. CEUS can be used to evaluate lesion enhancement in patients with contraindications to MRI or CT contrast.
4. If the lesion is well visualized, US may be the preferred modality for image-guided interventions.

(iv) Disadvantages
1. Operator dependent.
2. Greatly limited by bone and air.
3. May not be able to fully evaluate larger lesions.

(b) Computed Tomography/Computed Tomography Angiography (CTA)

(i) General
1. CTA is useful in the evaluation of vascular lesions as it can provide information about the vessel wall and lumen, including information on atherosclerotic vascular disease and calcifications.

(ii) Technique
1. Images are acquired and reconstructed in thin sections typically measuring 1–2 mm.
2. For CTA, iodinated contrast is injected intravenously, and imaging is timed in order to obtain maximal arterial enhancement within the area of clinical concern.
3. Maximal arterial enhancement will depend on location and size of target vessel, vascular pathology, and cardiac function.
4. Arterial enhancement can be modified by adjusting contrast agent concentration, flow rate, and volume.

(iii) Advantages
1. Like US, CT is a reasonable first modality as it is also widely available, fast, and relatively affordable.
2. High spatial resolution.
3. Post-processing and 3D reconstruction capabilities.

4. Less sensitive to motion artifact than MRI (including vascular artifact from turbulent or slow flow).
5. Excellent at evaluating bone and calcifications.
6. Generally easier to interpret than MRI.

(iv) Disadvantages
1. Ionizing radiation exposure.
2. Susceptible to streak artifact from metallic material.
3. Risk of contrast-induced nephropathy.
4. Risk of contrast allergies.

(c) Magnetic Resonance Imaging/Magnetic Resonance Angiography (MRA)

(i) General
1. MRI excels at evaluating soft tissues.
2. As mentioned above, it is the preferred modality to evaluate for tumor extent, perivascular/perineural spread, and/or intracranial extension.
3. Imaging of vascular lesions with MRA can be performed using both contrast and non-contrast techniques.
4. Contrast-enhanced MRA (CEMRA) is less sensitive to artifacts and shows higher morphologic detail than non-contrast technique.
5. CEMRA is more technically challenging as it requires good bolus timing and patient cooperation (breath hold, staying still, etc.).
6. MRA without contrast still allows for the evaluation of vessel morphology, as well as blood flow and direction.

(ii) Advantages
1. Multiplanar scanning.
2. Ability to evaluate vasculature without contrast, which is particularly important in patients who are pregnant or have severe renal failure.
3. No ionizing radiation exposure.
4. MRI contrast allergies are rare.
5. Contrast agents are less nephrotoxic.
6. Ability to perform functional imaging (DWI, tissue perfusion, blood flow/volume, etc.)

(iii) Disadvantages
1. Cost.
2. Long acquisition times.
3. Technically more complex accounting for numerous different sequences.
4. More sensitive to artifact, particularly in the H&N region.
5. Less sensitive to small calcifications.
6. Ferromagnetic metal implants, devices, or fragments may prohibit the use of MRI.
7. May require sedation or anesthesia in the pediatric population.

(d) Positron Emission Tomography Computed Tomography

(i) Advantages
1. Whole body imaging.
2. Excellent at detecting distant metastases and nodal involvement.
3. Useful for therapy monitoring.

(ii) Disadvantages
1. Glucose uptake on 18FDG PET-CT can be nonspecific.
2. Cost.
3. Requires patient preparation and cooperation.
4. Radiation exposure.

### 6.1.2 Section 2: General Interventional Radiology [1–18]

1. Interventional radiology originally developed from diagnostic angiography and was officially

born on January 16, 1964 with the first percutaneous angioplasty of a superficial femoral artery (SFA) stenosis by a diagnostic angiographer, Dr. Charles Dotter, whom many consider to be the forefather of Interventional Radiology [1–6, 19]

2. Since that time, the specialty itself and the minimally invasive surgical techniques are increasing in popularity and have been ever-changing
   (a) As Dotter once said: "*The angiographic catheter can be more than a tool for diagnostic observation; used with imagination, it can become an important surgical instrument*."
3. Many interventional radiology procedures have been shown to reduce cost, recovery time, pain, and risk as compared to conventional surgery
4. Interventional radiology procedures, as they relate to the head and neck as well as elsewhere in the body, can be divided into vascular and nonvascular procedures
   (a) Vascular
      (i) Major vascular procedures include diagnostic conventional angiography, angioplasty, atherectomy, thrombolysis, stenting, and embolization
   (b) Nonvascular
      (i) Major nonvascular procedures include percutaneous biopsy/drainage/injection, percutaneous ablation, nonvascular stenting, and pain management
   (c) Interventions within the head and neck are accomplished by a variety of proceduralists including interventional radiologists, neurointerventional radiologists, neurosurgeons, ENT surgeons, etc.
   (d) Specialty selection in regard to "procedural territory" often varies by institution
   (e) A few of the main vascular and nonvascular procedures pertaining to interventional radiologists are discussed below
   (f) Pre/peri-operative workup
      (i) Always thoroughly review history of present illness, labs, medications, and pertinent imaging
         1. If pertinent imaging or labs have not yet been obtained, recommend ordering prior to procedure
         2. Request patient be NPO for at least 6 h (or per hospital policy) if sedation is planned
      (ii) Risk stratify patient via the American Society of Anesthesiologists (ASA) Physical Status Classification System
         1. I: Normal healthy patient
         2. II: Mild to moderate systemic disease
            (a) Obesity, smoker
         3. III: Severe systemic disease that limits normal activity
            (a) ESRD
         4. IV: Severe systemic disease that is constant threat to life
            (a) Sepsis, recurrent variceal bleeds
         5. V: Not expected to survive without intervention
            (a) Ruptured AAA, large hemorrhage
         6. VI: Brain dead
      (iii) Assess bleeding risk [5]
         1. Desired INR < 1.5 and platelets > 50,000/μL for most interventional procedures
         2. Assess patient-specific and procedure-specific bleeding risk
         3. Hold antiplatelets and anticoagulants per procedure/hospital protocol
            (a) Clopidogrel: ideally held for 5 days prior to procedure
            (b) Aspirin: does not need to be withheld
            (c) Warfarin: desired INR for most procedures is <1.5
               (i) FFP or vitamin K for correction if needed
            (d) LMWH (therapeutic dose): held morning of procedure

(iv) Assess post-procedure contrast-induced nephropathy (CIN) risk [4–8]
1. Contrast-induced nephropathy (CIN) is defined by the Acute Kidney Injury Networks as an increase in serum creatinine >25% from baseline or serum creatinine increase of 0.3 mg/dL within 72 h after contrast administration
2. Risk factors for CIN include
   (a) Glomerular filtration rate <60 mL/min (particularly <30 mL/min) or baseline serum creatinine > 1.5 mg/dL
   (b) Age > 75
   (c) Diabetes
   (d) Medical conditions that decrease renal perfusion (i.e. heart failure)
   (e) Use of nephrotoxic medications (i.e. NSAIDS)
   (f) Procedure-related risk factors
      (i) Risk of CIN is dose dependent (procedures with a larger volume of intra-arterial contrast media use pose a greater risk)
3. CIN prevention
   (a) Pre/intra/post-procedural oral and/or IV hydration
      (i) Ideally 1 mL/kg/h of IV hydration 12 h prior to and after elective procedure
   (b) Minimize volume of intra-procedural arterial contrast
      (i) Consider other contrast agents, such as carbon dioxide (CO2) angiography
         1. An absolute contra-indication to $CO^2$ angiography includes cerebral arteriography
      (ii) Note, a dose-toxicity direct relationship has only been shown for intra-arterial use of contrast, not intravenous
   (c) Temporal separation of contrast media procedures
   (d) Hold nephrotoxic medications
      (i) Patients taking metformin who develop CIN are at risk for lactic acidosis
4. Contrast reactions
   (a) Acute reactions to intravenous contrast can be divided into allergic-type and nonallergic-type; both of which can range in severity
   (b) Premedication
      (i) History of severe contrast reaction is considered a relative contraindication to receiving the same class of contrast
      (ii) In a patient with a known contrast allergy, any future administration of that class of contrast is likely to produce a similar reaction
         1. In fact, no evidence that premedication decreases the *incidence* of moderate or severe contrast reactions, it can however decrease the severity
         2. Therefore, if IV contrast is necessary for a patient who has had a previous reaction, a premedication regimen is recommended
      (iii) Premedication regimens vary by institution; those recommended by ACR include

1. Methylprednisolone 32 mg PO 12, 2 h prior ± diphenhydramine (Benadryl) 50 mg PO 1 h prior
2. **Or** prednisone 50 mg PO 13, 7, 1 h prior ± Benadryl 50 mg PO 1 h prior
3. **Or** hydrocortisone 200 mg IV 5 h and 1 h prior and Benadryl 50 mg IV 1 h prior
   (a) Note that IV steroids are only shown to be effective when given *at least > 4 h* prior to administration of contrast (IV steroids given in emergent premedication < 4 h prior to contrast administration has not shown efficacy)

(v) Assess level of sedation needed
1. Local anesthetic
2. Minimal sedation/anxiolysis
3. Moderate sedation
   (a) Most interventional procedures are accomplished with moderate sedation
   (b) Level of sedation where patient can respond to verbal stimuli and protect their airway
   (c) Midazolam (Versed) is a benzodiazepine most commonly used for sedation
      (i) Typical starting dose is 1–2 mg IV with repeat doses 0.5–1 mg IV
      (ii) Onset of action is 1–5 min
      (iii) Lower doses should be considered in elderly patients and patients with liver or kidney failure due to impaired metabolism/clearance/excretion
      (iv) Can have paradoxical reaction whereby patient experiences hyperagitation
   (d) Again, request patient be NPO for at least 6 h (or per hospital policy) if sedation is planned
4. Deep sedation
5. Monitored anesthesia care (MAC)
6. General anesthesia (endotracheal intubation)

(vi) Assess level of intra-/post-operative analgesia needed
1. Fentanyl (Sublimaze) is an opioid commonly used for analgesia in most IR procedures
   (a) Typical starting dose is ~50 μg IV with repeat doses 25–50 μg IV
   (b) Onset of action is within a minute
   (c) Higher doses may be needed for patients with high opiate tolerance
   (d) Hepatic or renal impairment has less effect and dose does not typically need to be lowered

5. Needles, wires, catheters, sheaths, balloons, and stents
   (a) Size
      (i) Needles are measured in gauge
         1. Gauge (g) refers to the outer diameter of the needle
         2. Higher the gauge, smaller the needle
         3. Typical needles encountered in IR include a 21 g needle to do micro puncture access and a 19 g needle to do direct access or transjugular liver biopsies

(ii) Wires are measured in inches
1. Typical wires encountered in IR include an 0.018″ wire used during microaccess and an 0.035″ guidewire
2. 19 g ~ 0.038″

(iii) Catheters and sheaths are measured in French
1. French (Fr) refers to the outer diameter of the catheter or sheath
2. 1 French = 0.33 mm
3. 3 French ~ 1 mm
4. The internal diameter of a catheter or sheath will vary with thickness of the wall of the device
   (a) Thicker wall of device = smaller internal diameter
5. A 5 Fr sheath should accept a 5 Fr catheter

(iv) Balloons and stents are measured in millimeters

(v) It is essential to know the size relationships of these different measuring standards because inserting these devices into the body requires sliding one device through another device

(vi) The "size rule":
1. 19 g ~ 0.038″ ~ 3 Fr ~ 1 mm
   (a) Note, these are all about the same size
   (b) Also note, a 3 Fr catheter will not accept an 0.035″ wire due to catheter wall thickness
      (i) Again, French is the *outer* diameter

6. Needles
   (a) Divided into vascular access needles versus biopsy needles
   (b) Vascular access needles are single-wall needles—have a single beveled (angled) edge with a small notch on the hub that corresponds to the bevel. They are more steerable than trocar needles because a beveled needle will bend toward the distal tip of the bevel
   (c) Will often contain an inner stylet that is pulled out once the needle tip is confirmed, by ultrasound, to be within the vessel lumen
      (i) A 21 g needle accepts a 0.018″ wire (which is what comes in most IR vascular microaccess kits)
         1. Note—a 21 g needle does not accept a 0.035″ wire
   (d) Biopsy needles are made to remove bodily tissue for pathologic analysis
      (i) Biopsy needles come in various designs in regard to the shape of the needle tip
      (ii) Trocar needle: two-part needle system with an outer cannula and a removable inner sharp three-sided trocar needle (*trois carre* = three sided).
         1. Not steerable because of the three-sided tip
      (iii) Chiba needle: two-part high-gauge needle system with a beveled edge that allows steering
7. Wires
   (a) Wires have three main properties
      (i) Size (diameter—typically 0.018″ or 0.035″)
      (ii) Must have at least a 3 French catheter/sheath for an 0.018″ wire
      (iii) Must have at least a 5 French catheter/sheath for an 0.035″ wire
         1. Stiffness (determined by inner core and material constructed from)
         2. Hydrophilic versus non-hydrophilic
   (b) Three basic wire types (based on above properties)
      (i) Microaccess wire: 0.018″ wire that used to get access and then quickly exchanged
      (ii) Maneuver wire: floppy wire that is often hydrophilic ± curved tip to subselect vessels
         1. Standard or stiff Glidewire (Terumo Medical, Somerset, NJ)
         2. Fathom (Boston Scientific, Marlborough, MA)

(iii) Rail wire: stiff wire that provides a stability for catheter exchanges and during angioplasty/stenting
1. Amplatz (Boston Scientific, Marlborough, MA)
2. Rosen (Cook Medical, Bloomington, IN)
3. Lunderquist (Cook Medical, Bloomington, IN)

8. Catheters
   (a) Numerous catheter types available with different nuances
   (b) Divided into nonselective (flush) catheters and selective catheters
   (c) Nonselective flush catheters have multiple side holes which allow for high-flow injections into large arteries or veins and prevent vascular injury at the tip of the catheter during power injections
      (i) Commonly used for large vessel angiogram
   (d) Selective catheters usually have a single end hole and require lower flow rates injections than flush catheters but allow for more precise/selective injection
      (i) Commonly used for embolization and small vessel angiogram
      (ii) Microcatheters are typically 3F or smaller and allow for the subselection of small vessels advanced through a base or "parent" catheter

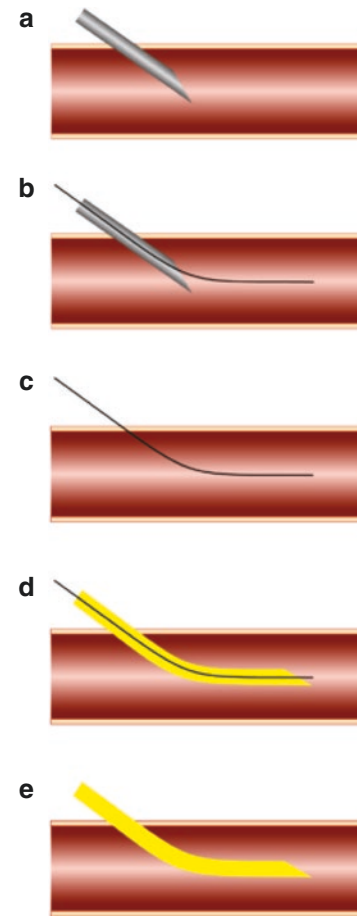


**Fig. 6.1** (**a**) Under ultrasound guidance, a needle and stylet are introduced into the artery; (**b**) stylet removed and microwire introduced through needle; (**c**) needle removed; (**d**) catheter or sheath advanced over microwire; (**e**) microwire removed. (Image: wiki commons)

#### 6.1.2.1 Vascular Access

- Proper vascular access is the foundation for all vascular interventional procedures
- The Seldinger puncture technique (Fig. 6.1a–e)
  - Fundamental interventional radiology maneuver whereby a guidewire/microwire is introduced through a needle puncture which is then exchanged for a sheath/catheter
  - A 21-g needle is needed for the 0.018″ microwire system
  - An 18-g needle is needed for the direct 0.035″ wire system
- Venous access
  - Most commonly accessed veins are the internal jugular and common femoral veins
  - Indications
    Central venous access
    Procedural access (i.e., transjugular intrahepatic portosystemic shunt [TIPS] or IVC filter placement/removal)
    Diagnostic venography
  - Pre-procedural
    Check history and review appropriate imaging
    - Patients with numerous prior venous access procedures or those with longstanding central venous access catheters may be challenging due to stenotic central venous system

Check labs and medications
- Check eGFR and potential nephrotoxic medications, especially if contrast planned
- Desired INR < 1.5 and platelets > 50,000/μL
- Assess patient and procedural risk of bleeding; holding antiplatelets and anticoagulants per procedure/hospital protocol
- Clopidogrel: ideally held for 5 days prior to procedure
- Aspirin: does not need to be withheld
- Warfarin: desired INR for most procedures is <1.5
  - FFP or vitamin K for correction if needed
- LMWH (therapeutic dose): held morning of procedure

NPO for at least 6 h (or per hospital policy) if sedation is planned

– Procedural

Patient supine on table, sterile prepped, ± analgesia/sedation
- Head turned contralaterally to access site in case of internal jugular access

Under ultrasound guidance, perform the Seldinger technique
- Find target vessel and identify important adjacent structures (i.e., carotid artery if accessing internal jugular vein or common femoral artery if accessing common femoral vein)
- Anesthetize skin with lidocaine
- Make a shallow skin nick with #11 scalpel
  - Incision can be made prior to or after 21 g needle insertion
- 21 g needle (with inner stylet) inserted into the vein
  - Needle tip should be always visualized
- 0.018″ microwire advanced through needle and advanced under fluoroscopic observation
- Microaccess sheath (with inner dilator) then advanced over microwire
- Remove inner dilator and microwire
- 0.035″ guidewire advanced through microaccess sheath
- Microaccess sheath removed and desired sheath/catheter advanced over 0.035″ guidewire

• Arterial access

– Most commonly accessed arteries are the common femoral and radial arteries

– Indications

Procedural access
- Angioplasty, atherectomy, thrombolysis, stenting, and embolization

Diagnostic arteriography

– Pre-procedural

Check history and review appropriate imaging

Check labs and medications
- Check eGFR and potential nephrotoxic medications, especially if contrast planned
- Desired INR < 1.5 and platelets > 50,000/μL
- Assess patient and procedural risk of bleeding; hold antiplatelets and anticoagulants per procedure/hospital protocol
- Clopidogrel: ideally held for 5 days prior to procedure
- Aspirin: does not need to be withheld
- Warfarin: desired INR for most procedures is <1.5
  - FFP or vitamin K for correction if needed
- LMWH (therapeutic dose): held morning of procedure

NPO for at least 6 h (or per hospital policy) if sedation is planned

– Procedural

Patient supine on table, sterile prepped, ± analgesia/sedation
- Wrist in hyperextension for radial access

Under ultrasound guidance, perform the Seldinger technique
- Find target vessel and identify important adjacent structures

  - Common femoral artery access site should be over the medial 1/3rd of the femoral head; high or low puncture could lead to retroperitoneal hematoma or inadvertent superficial femoral artery access, respectively
  - Radial artery access site should be ~2 cm proximal to the radial styloid process
    Some prefer to predilate radial artery with topical nitroglycerine prior to procedure
- Anesthetize skin with lidocaine
  - Skin entry site should be ~2 cm below (for retrograde arterial access) or above (for antegrade arterial access) the arteriotomy site
- Make a shallow skin nick with #11 scalpel
  - Incision can be made prior to or after 21 g needle insertion
  - Skin nick usually not necessary for radial access
- 21 g needle (with inner stylet) inserted into the artery at 45° angle
  - Needle tip should be always visualized
- 0.018″ microwire advanced through needle and advanced under fluoroscopic observation
- Microaccess sheath (with inner dilator) then advanced over microwire
  - Specific radial artery access sheaths are available
- Remove inner dilator and microwire
  - Vasodilatory "cocktail" administered through microaccess sheath
    Nitroglycerin (200 μg) + verapamil (2.5 mg) + heparin (3000 units)
- 0.035″ guidewire advanced through microaccess sheath
- Microaccess sheath removed and desired sheath/catheter advanced over 0.035″ guidewire

#### 6.1.2.2 Embolization [9–18, 20–35]

- Embolization refers to the insertion of an intravascular agent to control hemorrhage or for devascularization purposes
- Indications for embolization
  - Occlude active source or potential source of hemorrhage
    Vascular rupture, aneurysms, pseudoaneurysms, arteriovenous fistulas, and varices
  - Tumor devascularization
  - Flow redistribution to protect tissue or facilitate flow to another tissue
    For example, gastroduodenal artery (GDA) embolization prior to chemo/radioembolization for hepatocellular carcinoma protects the tissues supplied by the GDA from receiving chemotherapy or radiotherapy
  - Treatment of congenital arteriovenous or venous malformations
  - Treatment of venous insufficiency
    For example, saphenous vein occlusion for lower extremity venous insufficiency
- Numerous embolization agents are available and are selected based on the indication for embolization, desired duration of embolization, and specific target(s) for embolization; they can be broadly divided into temporary versus permanent embolic agents
  - For example, a permanent embolic agent would be desired in a patient with an arteriovenous fistula
  - Embolic agents can also be differentiated by size of vessel occlusion (small, medium, or large) and mechanism of action (obstructs flow, thrombosis, or sclerosis)
    In general, smaller embolic agents will embolize more distal vessels (i.e., capillaries) while larger embolic agents will embolize more proximal vessels
    Some embolic agents (i.e., Onyx) can conform to the vessel lumen and therefore, obstruct flow to both small and large vessels

- Temporary embolic agents
  - Gelatin sponge
    Gelfoam (Upjohn Co, Kalamazoo, MI) or Surgifoam (Ethicon, Somerville, NJ)
    - Embolic particles that temporarily obstruct flow
      - Commonly used to temporarily embolize acute arterial hemorrhage
    - Usually completely resorbs with recanalization of the treated vessel with a few weeks
    - Can be prepared as single "torpedo" or "slurry" forms depending on indication for embolization
      - A "slurry" form can be created by cutting a Gelfoam sheet into very tiny squares which can then be mixed with contrast to create a slurry
        Mixed contrast provides fluoroscopic visualization while injecting

    Relatively inexpensive and effective
  - Avitene (Davol Inc, Cranston, RI)
    Embolic particle that temporary obstructs flow
    Made of microfibrillar collagen
    Completely resorbs with recanalization of the treated vessel within 2 months
  - Autologous clot
    Delivery of patient's own thrombosed blood products
    Completely resorbs with recanalization of the treated vessel within hours (very short duration of action)
- Permanent Embolic Agents
  - Coils
    Coils are radiopaque, permanent embolic agents
    Come in various shapes and sizes and primarily work by mechanical obstruction with subsequent recruitment of the patients own clotting cascade and platelet activation for permanent vessel occlusion
    Coated with tiny fibers and/or hydrogel which initiates platelet aggregation
    To consider using coils, the catheter needs to be advanced all the way to the target vessel/embolization area
    Coils should be slightly larger than the diameter of the target vessel lumen to avoid the risk of dislodgement (approximately 10–20% larger than the vessel lumen)
    "Pushable coils": deployed through a microcatheter via a coil-pusher wire with subsequent semi-forceful injection of ~2cc saline; difficult to retrieve after deployment if malpositioned
    "Detachable coils": can be deployed either mechanically or electrically and may be retrieved if malpositioned
    - Useful for high-flow shunts or vital vessels

    Stent-assisted coil embolism
    - Use of a noncovered stent to apply coils
    - Commonly used for pseudoaneurysm or berry aneurysm treatment
      - Noncovered stent placed across pseudoaneurysm neck
      - Microcatheter maneuvered through the stent interstices
      - Coils then introduced through microcatheter
  - Particles
    Particles are essentially tiny solids that induce permanent embolism
    - Gelfoam is technically a particle that only provides temporary embolization

    Usually have good small vessel penetration (better than coils but not as good as liquid embolic agents)
    Nonspherical Embolics—Polyvinyl Alcohol (PVA)
    - PVA particles are nonspherical irregular shavings from PVA blocks that vary in size from 50 to 1200 μm
    - Embolize by clumping together after injection and mechanically occluding the vessel lumen by activating the coagulation cascade (similar to coils)

- Clumping can lead to occlusion in a wide range of vessel calibers—can occlude both small and large vessels; smaller particles (100–300 μm) penetrate more deeply and medium to large particles (300–800 μm) penetrate less deeply
  - Degree of clumping determined by concentration of PVA particles
    A more dilute solution prevents clumping whereas a very concentrated solution will rapidly clump
  - Like glue (discussed below), PVA particles in a very concentrated solution risk occluding the proximal vessel or even the catheter during injection and therefore, should be mixed with dilute contrast prior to administration
    Concentration of PVA to dilute contrast will vary by size of target vessel

Spherical Embolics—"Microspheres"

- Spherical embolic agents, or "microspheres," also vary in size (50–1200 μm) and tend not to clump like nonspherical PVA particles, resulting in a more predictable location of aggregation location and size of vessel occlusion
- Does not usually need to be mixed with dilute contrast prior to administration (just mix with ~3cc of contrast)
- Numerous spherical embolic agents available, including
  - Embospheres (Biosphere Medical, Rockland, MA)
    First microspheres used in humans
  - Contour SE microspheres (Boston Scientific)
  - EmboGold Microspheres (Merit Medical, South Jordan, UT)
  - Embozene microspheres (CeloNova BioSciences, Peachtree City, GA)
- Drug Eluting Particles
  - Promising "newer" drug delivery method whereby drugs our electrically or osmotically bound to particles and gradually elute to provide its therapeutic effect
  - Currently mostly used for chemotherapy/radiation drug delivery however, many non-chemoradiation drugs are currently being researched
    Yttrium-90—beta emitting particle used to treat hepatocellular carcinoma

- Vascular plugs
  Expandable radiopaque nitinol mesh plug that mechanically occludes the target vessel providing permanent embolization
  Allows for single step vessel occlusion with precise positioning
  Should be oversized relative to the target vessel by 25–50%
  Amplatzer Vascular Plug (St. Jude Medical, Saint Paul, MN)
  - Compact vascular plug available in four forms (AVP I, II, III, and IV)
  - Requires a guide catheter or sheath for deployment
  - Newer vascular plugs are being developed that can be deployed through a microcatheter
- Liquids
  Liquid embolic agents usually provide excellent small vessel penetration and occlusion
  - Administration is sometimes difficult to control depending on flow dynamics and embolic agent properties
    - Greatest risk is nontarget embolization due to higher than expected vascular flow

  Ethanol
  - Ethanol (96–98%) is a liquid embolic agent that immediately causes protein denaturing and permanent vessel thrombosis

- Can embolize an entire organ if desired
- Sometimes painful during delivery
- Not fluoroscopically visible unless mixed with Lipiodol—radiopaque substance (~4:1 mix)

Glue (N-butyl cyanoacrylate [n-BCA])

- Non-resorbable, non-radiopaque liquid embolic agent
- Most used to treat vascular malformations, particularly intracranial
- Liquid glue (cyanoacrylate) is a monomer that immediately becomes a solid polymer when contacted with ionic medium (i.e., blood or saline)
  - Glue will solidify within the catheter unless a substance is added that extends the polymerization time
  - Lipiodol (Guerbet, Paris, France)

    Lipiodol (aka ethiodized oil) is a radiopaque poppy seed oil that is typically mixed with cyanoacrylate to extend polymerization time or with ethanol for fluoroscopic visualization
    - Common to also add powdered tantalum during intracranial procedures to increase fluoroscopic visualization during injection

    Typically, ~5:1 lipiodol to cyanoacrylate ratio

    The higher the ratio, the longer the polymerization time
- Preparation and administration can therefore be a tedious and deliberate process
- The final mixture of liquid glue is injected after the catheter has been flushed with D5W
- The major disadvantage/risk of using cyanoacrylate is secondary to rapid polymerization
  - Can result in lack of nidus/small vessel penetration which can even lead to gluing the catheter in place
  - If polymerization time is too long, can pass into the venous circulation resulting in pulmonary emboli

Onyx (ev3 Endovascular Inc, Covidien, Plymouth, MN)

- Onyx is an ethylene vinyl alcohol (EVOH) copolymer mixed with dimethyl sulfoxide (DMSO) and opacified with radiopaque micronized tantalum powder (added for fluoroscopic visualization)
- When Onyx encounters blood, the DMSO rapidly diffuses away resulting in precipitation and solidification of the alcohol polymer—said to have "lava-like" flow as compared to free flowing liquid embolics, such as ethanol
- Forms an elastic "foam" that conforms to the vessel lumen
- Non-adhesive property allows for slower injection and less risk of catheter-to-vessel adhesion contrary to n-BCA glue
  - Precipitation time is mainly determined by the amount of EVOH

    More ethylene vinyl alcohol, quicker precipitation time
  - Two kinds of Onyx preparations

    Onyx 18 = 6% EVOH
    - Precipitates slower and penetrates more deeply and therefore, used for slow-flow vascular malformations/fistulas

    Onyx 34 = 8% EVOH
    - Precipitates quicker and has less penetration therefore, used for high-flow vascular malformations/fistulas

    Final solidification occurs within 5 min for both Onyx preparations

Precipitating hydrophobic embolic liquid (PHIL)

- PHIL is a newer non-adhesive liquid embolic agent made of an iodine-

bound polymer dissolved in dimethyl sulfoxide (DMSO)
  - The bound iodine provides fluoroscopic visualization
- There are four concentrations of PHIL (25%, 30%, 35% and low viscosity [LV])
- Similar to Onyx, the non-adhesive property allows for slower injection and less risk of catheter-to-vessel adhesion
- Unlike Onyx, iodine is covalently bonded to PHIL, rather than mixed with tantalum powder which leads to less glare artifact on follow-up CT scans
- Useful in embolization of neurovascular lesions (AVMs and hypervascular tumors)
- Prepared in a pre-filled sterile 1 mL syringe
- Recent studies have shown that this is a promising liquid embolic agent with very effective small vessel penetration, adequate fluoroscopic visibility, and little reflux

Thrombin
- Off-label use for treatment of arterial access complications such as pseudoaneurysm

– Sclerosants

Sclerotherapy has mostly become the first-line therapy of most venous and lymphatic malformations (low-flow vascular malformations) in the head and neck

Sclerosing agents are detergents that damage the endothelium by inducing an inflammatory reaction with eventual thrombotic vascular occlusion and sclerosis
- Liquid embolic agents are technically considered "sclerosants" however, the term is generally reserved for agents that primarily treat venous/low-flow disease

May be administered intravascularly or percutaneously
- Intravascular transcatheter balloon occlusion and/or coil/gelform embolization may be simultaneously used to prevent nontarget sclerotherapy

Numerous sclerosing agents available with differing effectiveness and side effects

Sodium tetradecyl sulfate (Sotradecol) (AngioDynamics, Latham, NR; Thrombotect, Omega, Montreal, Canada)
- Commonly used for esophageal varices, venous malformations, and varicose veins
- Liquid or foam forms

Ethanolamine oleate
- Mixture of 5% ethanolamine oleate (ethanolamine plus oleic acid) and ethiodol, ratio typically 5:1 or 5:2
- Less penetration than absolute ethanol and associated with less regional side effects (less adjacent tissue damage)
- Main side effect is nephrotoxicity as oleic acid may adhere to serum proteins and travel to the kidneys
  - Prophylactic haptoglobin (serum protein) used to reduce risk of nephrotoxicity
- Indicated for the treatment of esophageal varices that have recently bled, venous malformations, and cyst sclerosis

Bleomycin
- Effective in superficial lymphatic malformations
- Pingyangmycin is a bleomycin derivative commonly used in China

Picibanil (OK-432)
- Lyophilized mixture of group A Streptococcus pyogenes with antineoplastic activity used to treat lymphatic malformations

Doxycycline
- Inexpensive antibiotic sclerosing agent widely used for lymphatic malformations and abscesses

Ethanol
- Liquid sclerosant that immediately denatures proteins upon contact
- Effective but associated with significantly more side effects, particularly in the head and neck, such as adjacent tissue damage (nerve damage) and skin necrosis/ulceration
  - For these reasons, ethanol is not usually recommended in the head and neck

### 6.1.3 Section 3: Nonvascular Procedures of the Head and Neck

Discuss the distinct nonvascular procedures performed by Interventional Radiology.

1. Image-Guided Biopsies
   (a) Superficial masses may be amenable to non-image guided biopsy in the clinician's office. Deeper head and neck lesions may be percutaneously biopsied under image guidance, with CT and US being the most commonly employed imaging modalities.
   (b) After adequate head positioning, virtually all major spaces of the head and neck are amenable to image- guided biopsy. Intravenous contrast may be used to better delineate surrounding vascular structures and most procedures can be performed with local anesthetics and moderate sedation.
   (c) Biopsies of the upper cervical spine and upper aerodigestive tract may require general anesthesia.
   (d) Typically, a coaxial needle technique is used. An 18–19-gauge guiding needle is advanced near the target lesion, followed by advancement of the biopsy needle into the lesion. 20–22-gauge needles, such as a Chiba needle, are used to obtain aspirates, while a 20-gauge cutting needle can be used for acquiring core samples.
   (e) Approximately 90% of biopsies yield diagnostic samples.
   (f) Complications include pain, vasovagal reactions, bleeding, and infection. Significant blood vessel or nerve injury is rare.
2. Thermal Ablation Methods
   (a) General
      (i) Apply direct thermal therapy to a lesion with the purpose of producing significant or complete tumor ablation.
      (ii) Techniques include cryoablation, microwave ablation, and radiofrequency ablation.
      (iii) Can be used to treat benign and malignant tumors, though most commonly used for palliative treatment of unresectable malignancies or treatment of potentially resectable lesions in patients who are poor surgical candidates.
      (iv) Advantages include direct visualization of tumor response, reduced recovery time, and absence of a surgical scar.
   (b) Radiofrequency Ablation
      (i) Radiofrequency ablation is a relatively low risk procedure that utilizes electromagnetic radiation to create frictional heat and tissue necrosis. The goal is to produce a temperature between 55 and 100 °C for approximately 4–6 min. To ensure adequate tumor ablation, 0.5–1.0 cm of normal tissue surrounding the tumor must also be ablated.
      (ii) Nearby vasculature may dissipate heat and make tumor ablation harder, a concept known as heat sink. Additionally, one must be cautious of ablating tumors that are near the carotid artery, as this conveys an increased risk of stroke and carotid blowout.
      (iii) Major disadvantages include limited use with larger masses, the risk of vaporization, charring, and carbon-

ization at temperatures above 100 °C which in turn cause an insulating effect that may prevent tumor ablation, and the time to necrosis.

(iv) To treat larger masses, up to three radiofrequency probes can be placed within no more than 1 cm apart to generate a larger ablation zone.

(c) Cryoablation

(i) Cryoablation is a procedure in which there is rapid freezing of intratumoral tissue resulting in intracellular and/or extracellular ice crystal formation causing direct damage to cell structures or cell desiccation, respectively. The procedure involves several freeze and thaw cycles which can take up to 25–30 min. The goal is to produce a temperature between −35 and −20°C. Like radiofrequency ablation, a 0.5–1.0 cm margin of normal tissue ablation is recommended to ensure adequate success.

(ii) Major disadvantages include longer treatment time and the possibility of cryoreaction or cryoshock. Cryoreaction presents with tachycardia, tachypnea, fever, chills, and elevated creatinine levels. Cryoshock presents with multi-organ failure, severe coagulopathy, disseminated intravascular coagulation, and acute respiratory distress syndrome. These are thought to be the result of released cytokines from cellular debris after tumor reperfusion.

(d) Microwave Ablation

(i) Microwave ablation is a procedure that utilizes electromagnetic energy, water molecule agitation, and heat to cause coagulation necrosis. It occurs between frequencies of 900–2450 MHz, with the largest ablation zones seen at a frequency of 915 MHz.

(ii) Microwave ablation has similar results as radiofrequency ablation with the added benefits of increased speed, higher temperatures, and larger ablation zones. It is also less susceptible to electrical impedance and heat sinks.

(e) Percutaneous Sclerotherapy

(i) General

1. Involves the use of sclerosing agents administered via percutaneous catheterization to treat abnormalities of the head and neck such as low-flow vascular malformations, lymphatic malformations, plunging ranulas, sialoceles, and cysts.
2. Usually performed in the interventional suite with moderate sedation or under general anesthesia.
3. Typically, should be avoided in cystic or necrotic tumors, given the risk of producing a communication with surrounding vital structures and acutely infected lesions.

(ii) Low-flow Vascular Malformations

1. Sclerotherapy of the head and neck is most commonly performed for venous and/or lymphatic malformations. These lesions can cause symptoms ranging from pain and swelling to significant functional impairment including hemodynamic effects and airway compromise from mass effect. MRI is particularly useful for the evaluation of these lesions. The presence of phleboliths and enhancement in venous malformations helps distinguish them from lymphatic malformations.
2. The most commonly used sclerosing agents for venous malformations include ethanol, sodium tetradecyl sulfate, and polidocanol.
3. The most commonly used sclerosing agents for lymphatic malfor-

mations include picibanil (OK-432) and doxycycline.

4. Multiple sclerotherapy sessions may be needed for larger or diffuse lesions.
5. Percutaneous Sclerotherapy of Venous Malformations (Direct Stick Embolization):
   (a) A 21-gauge butterfly needle is advanced into the venous malformation under ultrasound guidance.
   (b) Venous blood is freely aspirated from the lesion, confirming the vascular nature and flow pattern of filling of the lesion.
   (c) Angiography is performed with injection of 50% contrast to evaluate the size and venous drainage pattern of the lesion, as well as to exclude arterial cannulation. Volume of the sclerosant can be estimated at this time. The lesion is then serially injected under fluoroscopic guidance with boluses of 1:1 3% sodium tetradecyl sulfate (sotradecol):ethiodized oil (ethiodol) and the amount of sclerosing agent deployed is mindfully noted. Care is taken not to allow the sclerosing agent to enter the draining veins.
   (d) The procedure is then repeated for any other venous malformation in the adjacent tissues with similar 1:1 sotradecol:ethiodol formulation, in an incremental fashion. The total sclerosing agent deployed is similar for lesions in the oral mucosa, tongue, lip, and remaining upper aerodigestive tract.
   (e) The needle is then withdrawn from the venous malformation after 5 min following completion of the injections and another 5 min of very gentle pressure are applied to the puncture site following removal of the needle. At the completion of the procedure the face, neck, and upper chest are re-cleansed with sterile saline solution. The skin of the neck and upper chest was no longer erythematous and without an urticarial reaction.
   (f) (Different case?)
   (g) A butterfly needle was then used to access the posterior pouch of the venolymphatic malformation with biplanar fluoroscopy.
   (h) Free flowing blood aspiration out of the venolymphatic pouch and a pouchogram is performed to ensure access into the venolymphatic pouch proper.
   (i) The needle is then flushed and, under biplane fluoroscopy, an injection mixture of 1:1:1 of 3% Sotradecol, Ethiodol and air (for foam) is instilled to cause sclerosis of any vascular island or pouch. Additional pools or pouches in different planes and deeper are again evaluated by pouchogram to ensure access is within the pouch and sclerosing agent is instilled in a similar fashion.
   (j) All needles are then removed and light pressure is applied for hemostasis.
   (k) Notably, intraoperative ultrasound and biplane fluoroscopy are essential in finding additional pools or pouches.

### 6.1.4 Section 4: Vascular Procedures of the Head and Neck

#### 6.1.4.1 Management of Acute Hemorrhage

- Epistaxis
  - Causes
    Idiopathic
    Trauma with vascular injury
    - Post nasotracheal intubation

    Tumor
    - Primary or metastatic tumors involving the nasal mucosa may present with intractable epistaxis
    - Primary tumor: classically, juvenile nasopharyngeal angiofibroma (JNA)

    Hereditary hemorrhagic telangiectasia (HHT)
    - Osler-Weber-Rendu syndrome
    - Autosomal dominant disease that affects the nasal mucosa, abdominal viscera, and central nervous system characterized by numerous telangiectasias and AVMs
  - Treatment
    Varies by cause
    Majority are treated by nasal packing or cutaneous electrocauterization
    Refractory epistaxis not responsive to anterior and posterior nasal packs associated with significant blood loss may be amenable for arterial embolization
  - Pre-procedural considerations
    Pre-procedural imaging should be reviewed and should include a CT/CTA to exclude possible mass/tumor causing hemorrhage
    Gather relevant past medical history and labs
    - Correct the coagulopathy if present (desired INR < 1.5)
    - Hold anticoagulation if still taking
    - CBC (desired platelets > 50,000)
  - Procedural
    General anesthesia is typically recommended for airway protection as patient may hemorrhage while in supine position
    Obtain arterial access
    - Most commonly right common femoral artery approach

    Angiographic protocol for the evaluation of epistaxis includes assessment of bilateral internal/external carotids, internal maxillary (particularly sphenopalatine branches), and facial arteries
    Vascular supply to nasal mucosa
    - Supplied by the distal internal maxillary and facial arteries (sphenopalatine branches)
    - The ophthalmic artery may supply nasal mucosa via ethmoidal branches

    Epistaxis usually successfully treated with embolization of bilateral sphenopalatine arteries ± ipsilateral distal facial branch of internal maxillary artery
    - It is recommended that the contralateral sphenopalatine artery be angiographically evaluated even if there is unilateral bleeding as there may be contralateral collateralization

    If epistaxis persists, then hemorrhage may be from ethmoidal branches of the ophthalmic artery which are typically not embolized due to risk of vision loss
    Polyvinyl alcohol (PVA) or embozene particles are suitable embolic agents
  - Complications
    Major complications include routine risks of cerebral angiography as well as ischemic stroke by inadvertent embolization of intracranial collateral vessels and skin/mucosal necrosis most commonly due to too small particle size

### 6.1.5 Case: Epistaxis from Traumatic Nasotracheal Intubation Requiring Embolization

(Fig. 6.2)

#### 6.1.5.1 Case: Refractory Epistaxis of Unknown Etiology

Access to the right common femoral artery was obtained with Seldinger technique. A shuttle sheath was advanced into the thoracic aorta over

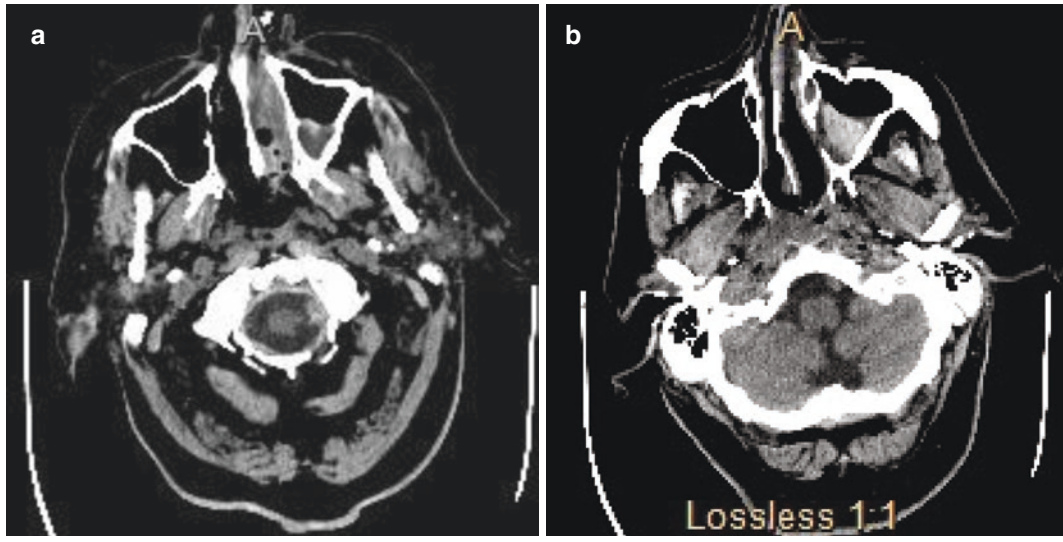


**Fig. 6.2** (**a**, **b**) Non-contrast CT head demonstrating left nasal intubation with associated hemorrhage and blood products in the left maxillary antrum. Embolization was requested and performed. Patient's right groin was prepped and draped in usual sterile fashion. Right common femoral artery found to be patent by ultrasound (**c**). Ultrasound image archived. (**c**) Access was then obtained by Seldinger technique. A 5-French Berenstein catheter was advanced through the sheath into the aortic arch and the left common carotid artery was selectively catheterized. Digital subtraction arteriograms (DSA) were then obtained. (**d**) DSA of the left common carotid artery arteriogram. A microcatheter was then advanced into the left external carotid artery and eventually into the left internal maxillary artery. (**e**) DSA image demonstrating bifurcation into the left superficial temporal artery and proximal aspect of the left internal maxillary artery. (**f** and **g**) sagittal and coronal section—Microcatheter was advanced into the distal left internal maxillary artery near the bifurcation of the internal maxillary artery into the sphenopalatine artery and greater palatine artery. Digital subtraction arteriograms demonstrated hyperemia over the nasal mucosa and posterior nasal pharynx. Microcatheter could not be advanced further and the left greater palatine artery was chosen to be sacrificed. Multiple DSAs were obtained. No collateral flow to the internal carotid circulation was identified. Ophthalmic artery was not visualized. Again hyperemic flow was identified within the nasal cavity and nasal pharynx. The sphenopalatine and greater palatine arteries were then embolized with 450 μm embozene particles. A follow-up lateral DSA demonstrated obstruction of flow at the origin of the left sphenopalatine and midway through the left greater palatine artery. (**h**) Post embolization angiogram of the demonstrating lack of hyperemia or significant flow to the nasopharynx. Microcatheter was then pulled back and angiograms of bilateral maxillary, facial, and right sphenopalatine arteries were obtained which did not demonstrate hyperemia or significant flow to the nasopharynx

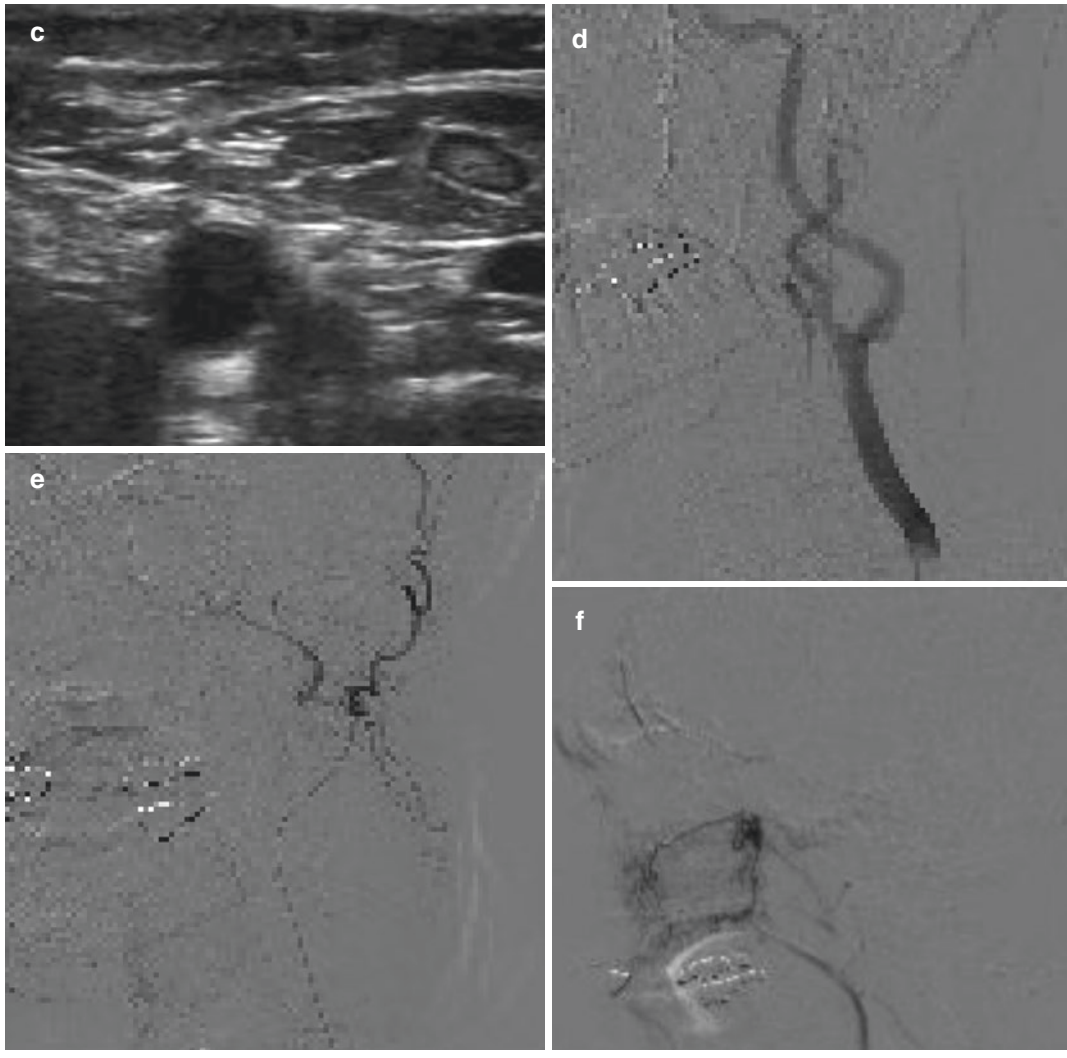


**Fig. 6.2** (continued)

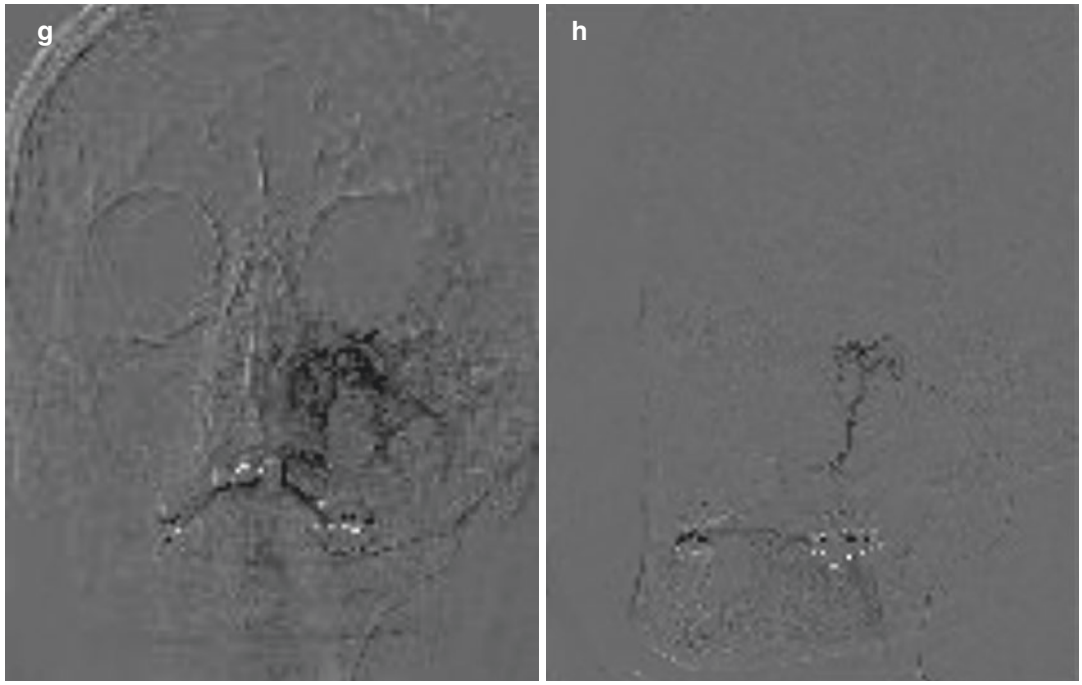


**Fig. 6.2** (continued)

a Bentson guidewire. A 5-French vertebral catheter was used to navigate the great vessels of the aortic arch, and the common carotid artery was selected. Right common carotid arteriogram was performed demonstrating appropriate placement (Fig. 6.3).

#### 6.1.5.2 Case: Large Left Glomus Vagale Paraganglioma Treated with Embolization (Fig. 6.4)

- Traumatic Vascular Injuries
  - General

    Traumatic vascular injuries of the head and neck can occur from blunt or penetrating trauma. If osseous fractures are seen adjacent to the expected location of vascular structures on CT, CTA may be necessary to further evaluate for vascular injury. In cases of penetrating injury, the injury tract can also be evaluated at time of CT. Alternatively, angiography can be performed and would also be effective at identifying vascular lacerations, occlusions, pseudoaneurysms, and/or vasospasms.

    Angiography is performed after standard heparin bolus with evaluation of the aortic arch, carotid arteries and major branches, and vertebral arteries, but the protocol may vary based on the nature of vascular injury. Routine risks of cerebral arteriography apply, including additional risk of vascular injury and stroke.

    Continuous ICU neurologic and hemodynamic monitoring may be required, particularly if there is vertebral or internal carotid artery injury or if treatment involved major vessel occlusion.
  - Oronasal bleeding from facial fractures

    Usually requires the embolization of injured ECA branches with temporary embolic agents such as PVA foam particles or gelfoam pledgets. NBCA glue or Onyx can be used for the treatment of fistulas or pseudoaneurysms.

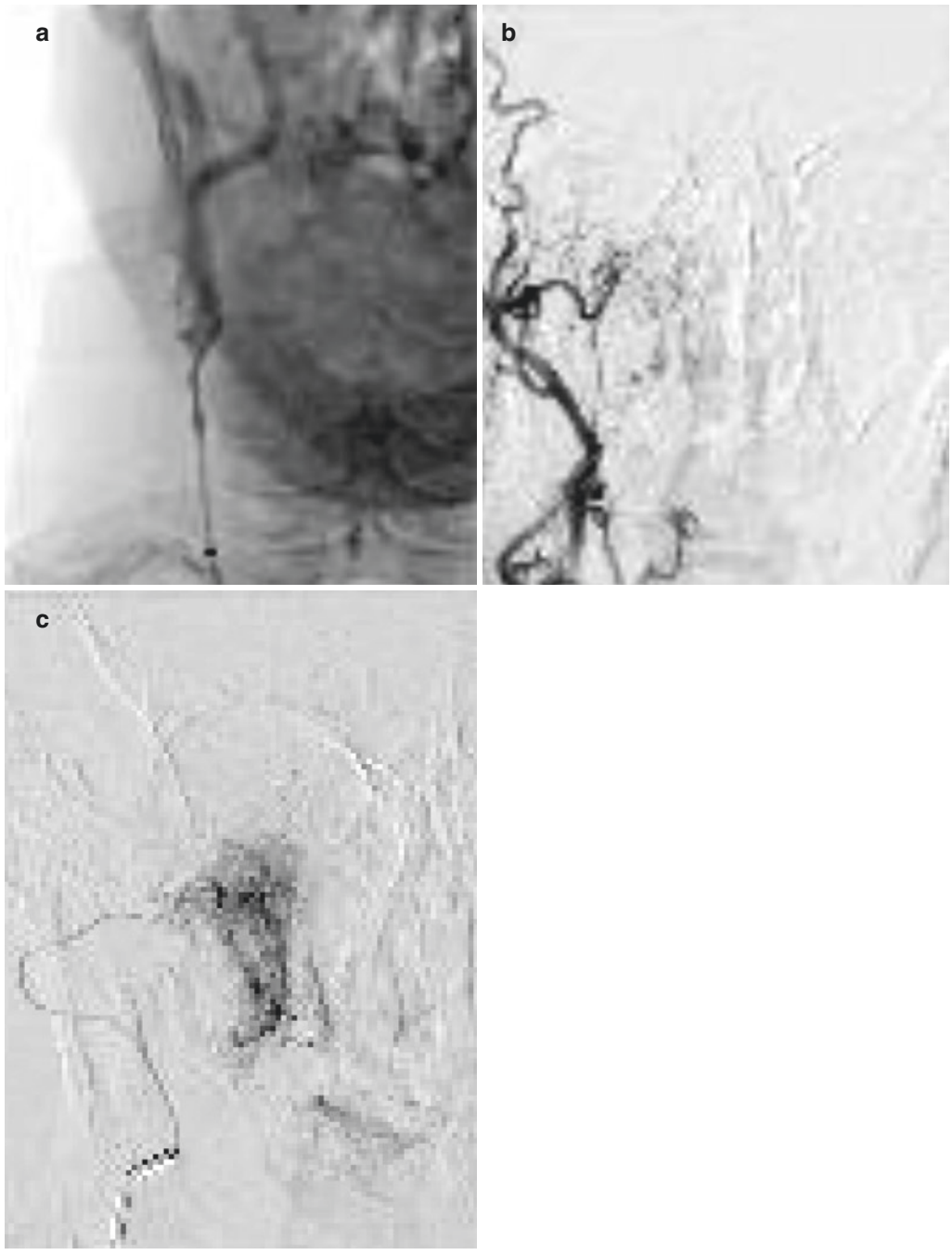


**Fig. 6.3** (**a**) A right carotid arteriogram with access through the aortic arch. (**b**) Using a stiff Glidewire, the vertebral catheter was used to select the right external carotid artery. Right external carotid PA DSA was performed documenting appropriate placement of the vertebral catheter. Mild hyperemia can be seen within the sphenopalatine branch of the internal maxillary artery. (**c**) The microcatheter/microwire system was then subselectively advanced into the sphenopalatine branch of the right internal maxillary artery. Subselective DSA was performed confirming appropriate placement and demonstrating no branches supplying the orbits or other vital structures. The hyperemia is again noted. At this point, 400 μm Embozene particle embolization was performed under fluoroscopic guidance, with 1/5 of a viral used until near stasis was noted

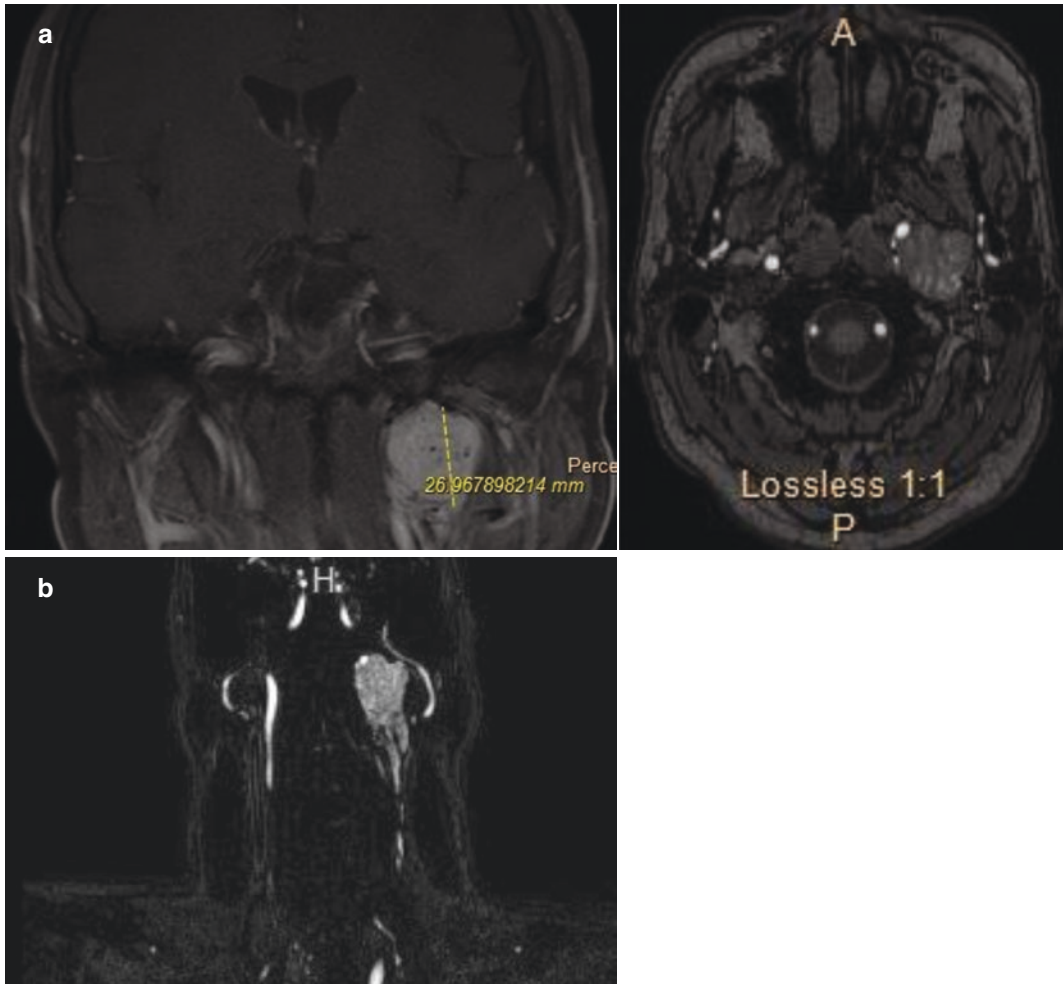


**Fig. 6.4** (**a**, **b**) Post contrast coronal and axial T1, coronal time-of-flight MRA. (**c**) coronal MIP demonstrating large hypervascular mass adjacent to the left ICA near the base of the skull, characteristic of glomus vagale. Post contrast coronal T1, coronal time-of-flight MRA, and coronal MIP demonstrating large hypervascular mass adjacent to the left ICA near the base of the skull, characteristic of glomus vagale. (**c**) DSA demonstrating large hypervascular mass adjacent to the left ICA near the base of the skull, characteristic of glomus vagale. DSA demonstrating large hypervascular mass adjacent to the left ICA near the base of the skull, characteristic of glomus vagale. (**d**) DSA demonstrating feeder vessel to the glomus vagale. DSA demonstrating feeder vessel. (**e**) Subselective DSA image of hypervascular mass. (**f**–**h**) Subselective DSA image of hypervascular mass and Pre and Postembolization DSA demonstrating stasis of flow to the previously seen hypervascular mass. Pre and Postembolization DSA demonstrating stasis of flow to the previously seen hypervascular mass

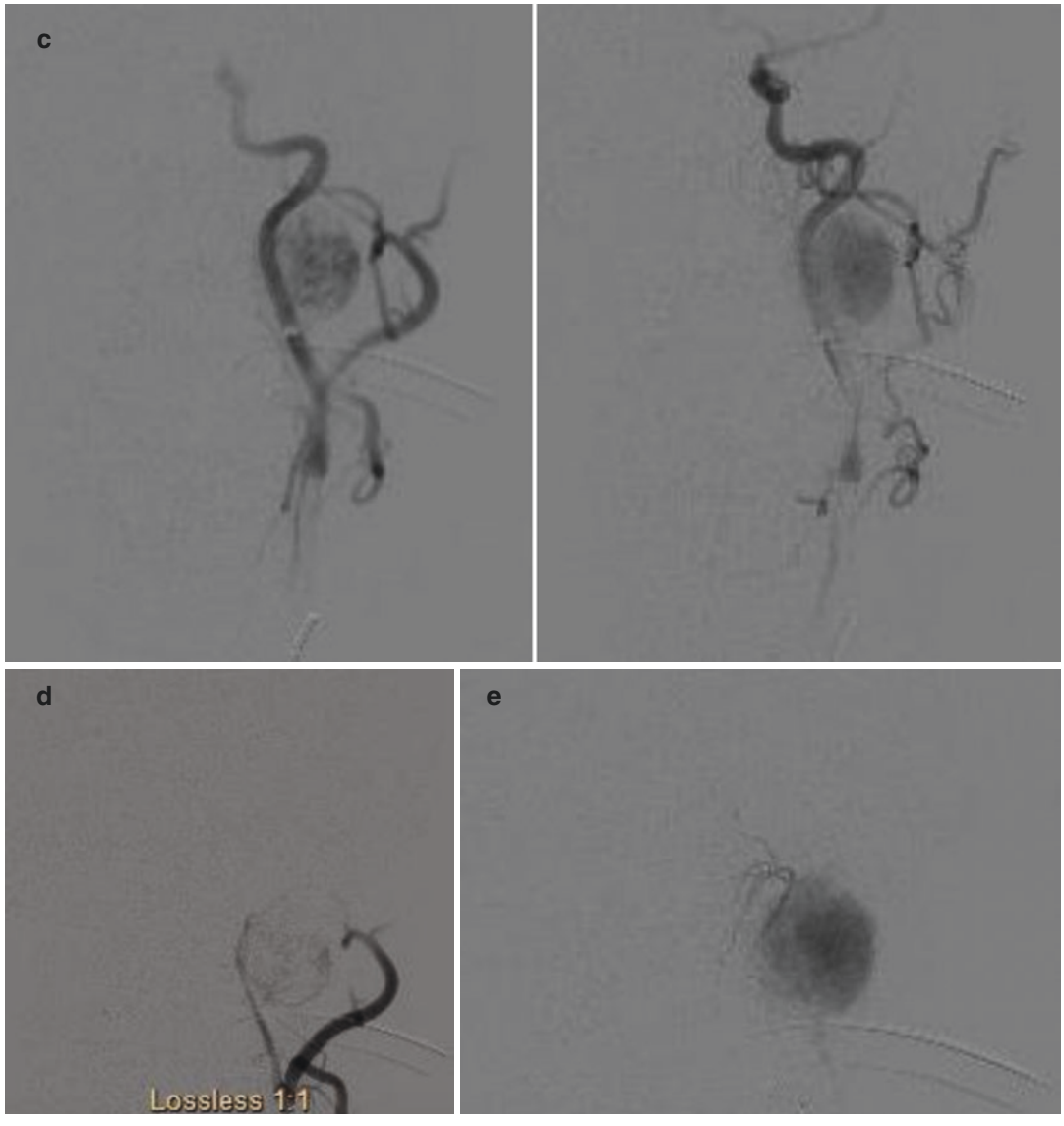


**Fig. 6.4** (continued)

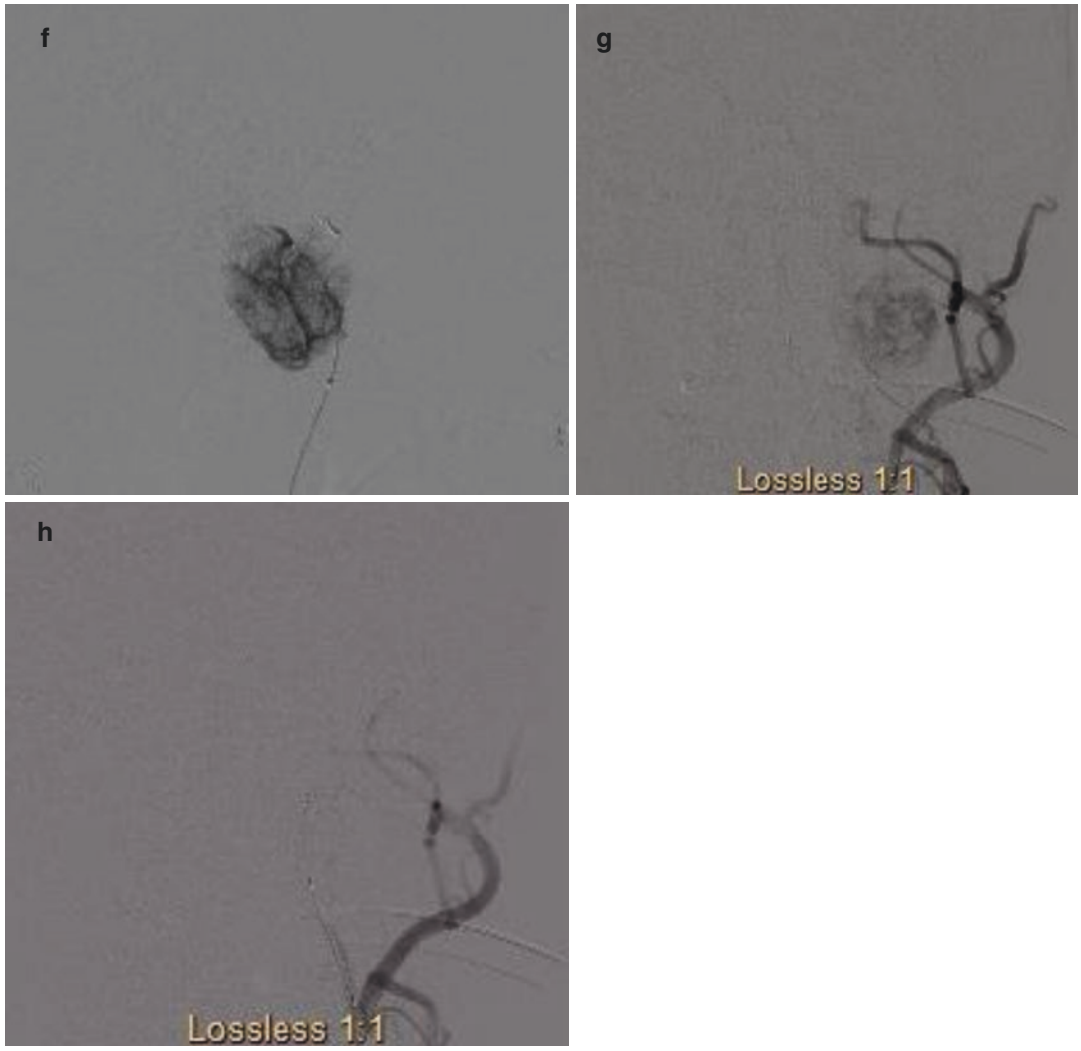


**Fig. 6.4** (continued)

– Carotid/Vertebral Artery Injury

Injury in the form of laceration or rupture can occur anywhere along the course of the carotid artery and branches and special attention on imaging should be made to fixed points, such as where the internal carotid artery enters the dura or within the cavernous sinus. Ultimately, injury can lead to occlusion, pseudoaneurysm or fistula formation. Expectant management is usually employed in the event of traumatic carotid occlusion as surgical bypass is usually not possible. Pseudoaneurysms, on the other hand, may be amenable to stents ± coils deployed into the excluded pseudoaneurysm. Alternatively, surgical repair or, if adequate collateral flow is demonstrated, parent artery occlusion may be performed.

Similarly, vertebral artery injury can occur anywhere along its course and special attention on imaging should be made to fixed points, such as the entry sites into the vertebral foramen and at the craniocervical junction. As mentioned above, osseous irregularity on CT, particularly at the vertebral fora-

men, may prompt further evaluation with CTA to exclude underlying injury. After determination of collateral flow, vertebral artery injury may require embolization or sacrifice, which can be performed with platinum micro-coils.

– Traumatic Carotid-Cavernous/Vertebrojugular Fistulas

Carotid-Cavernous and vetebrojugular fistulas are typically seen after penetrating trauma, however, can be seen in the setting of any severe head and neck injury, as well as iatrogenic injury. Carotid-Cavernous fistulas classically present with pulsatile exophthalmos, though symptoms largely depend on the pattern of venous drainage. Additional symptoms include chemosis, proptosis, progressive visual loss, pulsatile tinnitus, and intracranial hemorrhage, including intraparenchymal hemorrhage in the setting of cortical venous drainage. Vertebrojugular fistulas present with cerebral ischemic symptoms relating to "stolen" arterial blood from the posterior circulation or hemodynamic instability from arteriovenous shunting. Emergent treatment is usually not required unless there is risk of vision loss in the case of carotid-cavernous fistulas. For both types of fistulas, treatment usually involves the closure of the fistula using an arterial approach, but venous approach may also be employed based on fistula location or venous drainage pattern. In severe cases, parent artery occlusion or sacrifice may be necessary.

- Malignancy
  – Tumoral Hemorrhage and Carotid Blowout Syndrome [16, 22, 32]

When assessing upper aerodigestive tract bleeding from malignant disease, it is important to also assess the extent of tumor spread in order to appropriately guide endovascular treatment. This can be done by reviewing prior imaging or performing emergent CT or MRI if the patient's condition allows.

Bleeding can occur from increased tumor neovascularity or direct injury of adjacent vasculature. Treatment usually involves direct embolization of injured vasculature or pseudoaneurysms but may also require the occlusion or "sacrifice" of arterial branches supplying the tumor, arising either from the external, internal, or common carotid arteries. Prior to occlusion of the internal carotid artery, a Balloon Occlusion Tolerance (BOT) test can be performed to assess tolerance of carotid occlusion. It usually involves diagnostic arteriography to evaluate for collateral flow, clinical testing after temporary balloon occlusion, and HMPAO SPECT to evaluate cerebral blood flow after temporary occlusion. Endovascular occlusion is usually performed by employing detachable platinum coils with or without the use of a closure device.

Carotid Blowout Syndrome usually refers to acute, though sometimes chronic, bleeding of the upper aerodigestive tract from direct tumor erosion of the major head and neck vasculature. It is usually treated with carotid sacrifice after performing temporary BOT testing. In some life-threatening instances, carotid sacrifice must be performed without provocative testing and the risk of stroke is significantly higher.

Tumoral hemorrhage refers to upper aerodigestive bleeding from increased tumor vascularity or tumor injury of smaller surrounding vessels. It is usually treated with direct embolization, however, may require carotid sacrifice in more severe cases.

Postprocedural management of carotid occlusion or sacrifice entails close neuro-intensive ICU monitoring and hemodynamic therapy (Fig. 6.5).

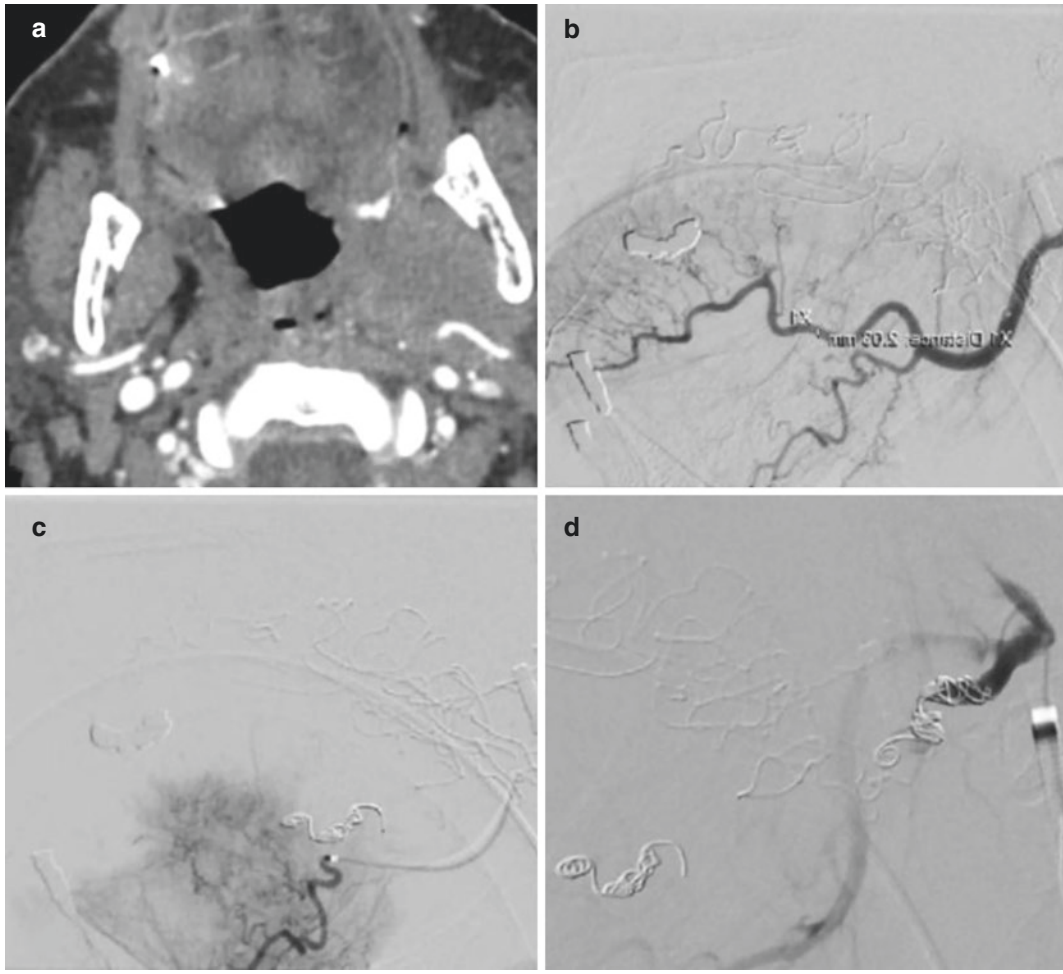


**Fig. 6.5** (**a**–**d**) Case of a 73-year-old male with history of poorly-differentiated squamous cell carcinoma within the left oropharyngeal wall presenting with persistent oropharyngeal bleeding which was treated coil embolization of the left lingual artery. (**a**) Axial CTA image through the oropharynx demonstrates masslike fullness of the left oropharyngeal wall tonsillar pillar region with a small focus of contrast within the left glossotonsillar sulcus suspicious for pseudoaneurysm and hemorrhage source. (**b**) Lateral DSA image shows no clear contrast extravasation or pseudoaneurysm arising from the left lingual artery and branches. (**c**, **d**) Based on the location of the SCC and continued oropharyngeal bleeding, the left lingual artery was embolized. Embolization microcoils were deployed distal to the origin of the sub-lingual artery and dorsal lingual arteries to prevent backfeeding from the contralateral lingual artery. Gelfom pledgets were then injected into the sublingual artery, followed by the injection of 200 μm PVA particles at the level of the lingual artery. Finally, microcoils were deployed in the proximal portion of the lingual artery to prevent recanalization

– Management of Vascular Lesions [1, 3, 8–14]

  Intra-arterial Chemotherapy

  • Intra-arterial chemotherapy has been used to treat both intracranial and extracranial head and neck tumors. Notably in the extracranial head and neck, intra-arterial chemotherapy has been used in the treatment of carcinomas, but use has also been reported in the treatment of metastases, malignant melanoma, and neuroblastomas.
  • The radiation and platinum (RADPLAT) protocol involves the

direct tumor administration of extremely high doses of cisplatin trans-arterially, while circulating the antagonist sodium thiosulfate as to minimize systemic toxicity. The increased dose of cisplatin increases tumor cytotoxicity and increases the effectiveness of subsequent radiation therapy.

- Transfemoral carotid arteriography is performed with selective catheterization of the ECA branch supplying the tumor. Infusion of 150 mg/m$^2$ of cisplatin over a period of 3–5 min is performed. During this time, concomitant administration of an intravenous infusion of 9 g/m$^2$ of sodium thiosulfate over a period of 3–5 min is performed, followed by an administration of 12 g/m$^2$ over a period of 6 h. In disease extending across midline, bilateral infusions may be performed. Treatment consists of four cycles of intra-arterial chemotherapy (days 1, 8, 15, and 22) and once daily radiation therapy 5 days a week.
- Results and techniques are variable based on practitioner and institution protocol. More recent studies have shown that RADPLAT is not superior to intravenous chemoradiotherapy in the treatment of advanced head and neck cancer, however it has been suggested that further investigation is warranted.

Tumor Embolization [16–18, 20–24]

- Cerebral angiography is first performed with selective injections of the internal and external carotid arteries. The artery supplying the tumor is accessed with a microcatheter and angiography is performed with special attention to anastomoses between the carotid and vertebral arteries. Any potentially dangerous anastomoses are coil embolized prior to particulate embolization.
- The aim of tumor embolization in the head and neck is the devascularization of lesions prior to surgical excision or to stop tumoral bleeding. It is frequently performed by transarterial route and the goal is to selectively occlude the external carotid artery feeders that ideally supply the arteriolar capillary beds in the tumor parenchyma with embolic material, as to avoid collateralization. This minimizes blood loss during surgery, provides a clean field for the surgical resection, improves resectability, makes the lesion amenable to different surgical procedures not previously feasible, reduces morbidity, shortens hospital stay, and reduces the rate of tumor recurrence.
- Embolization is usually done 24–72 h prior to surgical resection to allow maximal thrombosis and prevent recanalization and formation of collaterals.
- Skull base tumors that are supplied by branches of the ICA can be difficult to cannulate and convey an increased risk of embolic agent reflux and nontarget embolization. In these cases, direct percutaneous embolization of the tumor can be performed with liquid embolizing agents such as NBCA glue or Onyx.
- Benign vascular lesions may be treated with embolization without the need of subsequent surgery, as is the case of some congenital hemangiomas.
- Commonly embolized tumors of the head and neck include glomus tumors, angiofibromas, and meningiomas. The most common use of embolization is seen in the management of juvenile nasal angiofibromas—highly vascular tumors originating in the pterygo-palatine fossa which can expand aggressively through the sphenopalatine foramen

to involve other surrounding regions. Other tumors amenable to preoperative tumor embolization include esthesioneuroblastomas, schwannomas, rhabdomyosarcomas, plasmacytomas, chordomas, hemangiopericytomas, diffuse neurofibromas, and metastases.

- Commonly used embolizing agents include polyvinyl alcohol (PVA) particles and embospheres, usually in the size range of 100–300 microns, as well as liquid embolic agents, Onyx, gelatin sponge, and coils. Embolization coils are typically not used if open surgical resection is to be performed, however they may be useful if endoscopic resection is planned as proximal coil embolization of the feeding artery after particulate embolization of the tumor may provide added protection from vessel injury and intraoperative bleeding.
- As mentioned previously, Onyx can be used to devascularize tumor directly with relatively low risk. It has inherent properties that allow for a more controlled injection with superior penetration than NBCA glue. It is mechanically occlusive, but does not adhere to vessel walls, allowing it to be used as a single injection administered slowly over a longer period. If unwanted filling of normal vasculature occurs, the treatment can be stopped and resumed after a short period of approximately 2 min. Solidification will continue to occur in the region of previously embolized tumor and once the injection is restarted the agent will follow the route of least resistance and continue to embolize the remaining portions of the tumor.
- Overall, tumor embolization is a safe procedure with a serious complication rate of <2%. Complications include facial numbness, mucosal necrosis, blindness, or cerebrovascular accidents which are usually related to particle reflux, poor technique, or unidentified anastomoses.

- Vascular Malformations
  - Arteriovenous Malformations/Fistulas of the Head and Neck [1–39]

    See **Percutaneous Sclerotherapy** for more information on treatment of Low-flow Vascular Malformations.

    High-flow Vascular Malformations (HFVMs) are **rare** and can involve the soft tissues and bones of the head and neck. They commonly present as cosmetic deformities, however, can also cause pain, bleeding, and high-output heart failure from arteriovenous shunting.

    HFVMs treatment can be challenging depending on the lesion characteristics and are known to **frequently recur**. **Small lesions can be treated with endovascular embolization alone, however larger lesions may require surgical resection.**

    Distinction between a **vascular nidus versus an arteriovenous fistula** proper may also be challenging. Arteriovenous fistulas are less likely to recur after treatment.

    Commonly used embolic agents include ethanol, NBCA glue, Onyx, and coils. Trans-arterial NBCA glue has been shown to be more effective and permanent when compared to other embolization particles but may require staged procedures.

    As in low-flow vascular malformations, **direct puncture embolization** or percutaneous sclerotherapy can be performed to embolize the nidus and/or draining vein with NBCA glue or Onyx. Manual digital compression of the draining vein or the application of a compression device in cases with mul-

tiple draining venous channels can be used to alter flow and facilitate complete filling of the nidus. When compared to trans-arterial embolization, direct puncture embolization is associated with decreased risk of ischemic complications and reduced procedure times.

Given the rarity of these lesions, no standard of therapy has been determined (Fig. 6.6).

### 6.1.6 Section 5: Complications and Post-procedure Management

Provides an overview of common complications and post-procedural management

1. Pain and Fever
   (a) Perform a brief history evaluation. The pain description will usually coincide

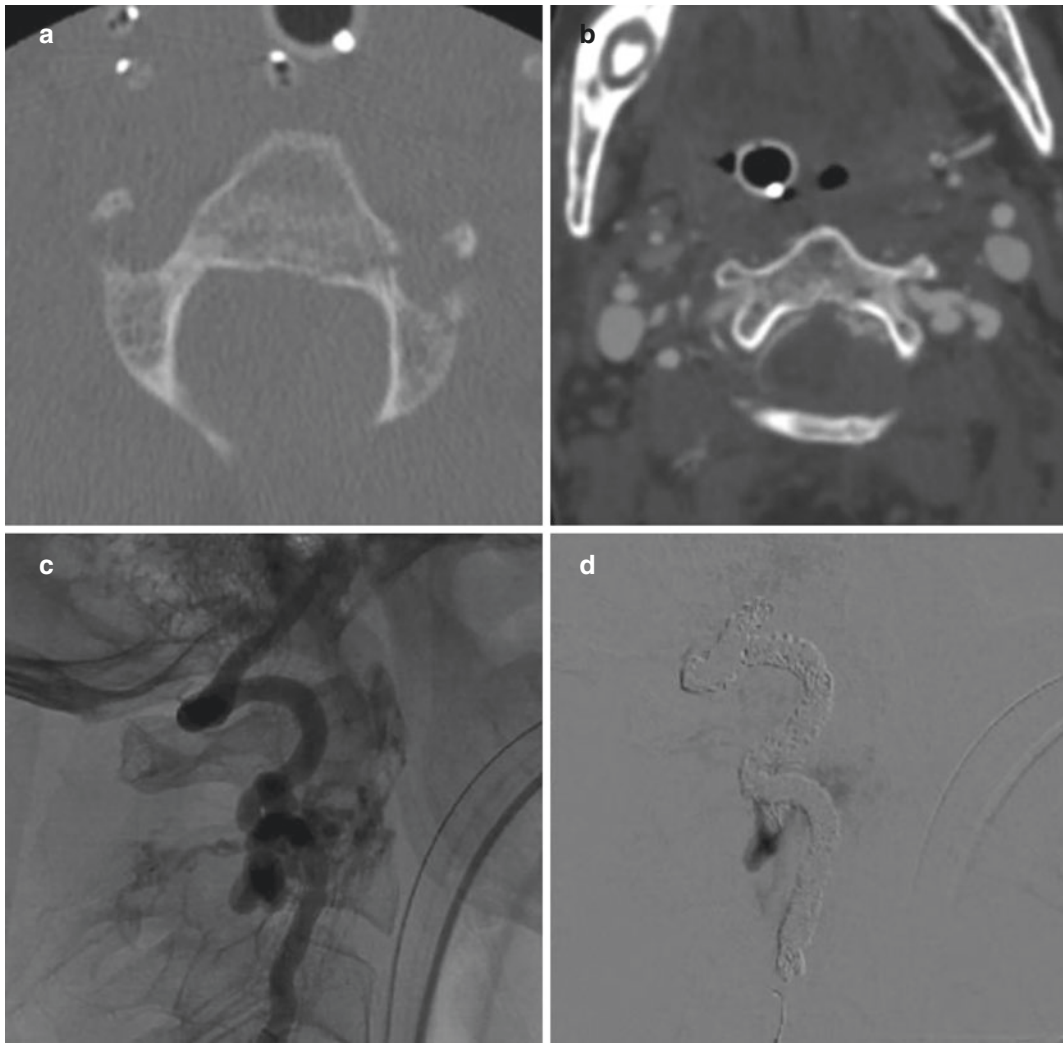


**Fig. 6.6** (**a**–**d**) Case of a 72-year-old female presenting for MVC with Traumatic Vertebral Artery Pseudoaneurysm and Vertebral-Venous Fistula S/P Coil Embolization. (**a**) Axial NECT image of the cervical spine demonstrates a fracture of the dorsal C2 body with extension into the left foramen transversarium. (**b**) Axial CTA image through the level of the fracture shows a multilobulated pseudoaneurysm with suggestion of AV fistula formation. (**c**) Lateral fluoroscopic spot image during left vertebral artery angiogram confirms a large pseudoaneurysm with vertebral-venous fistula formation. (**d**) Lateral DSA image status post coil embolization

with the type of procedure performed. If the pain is greater than expected or refractory, this may prompt an investigation for an alternative cause of pain or a potential complication from the procedure.

(b) Many IR procedures will usually require minimal analgesia, if any. This can be accomplished with Acetaminophen (500–1000 mg q4 h for a maximum dose of 4 g in 24 h) or Ibuprofen (400–600 mg q4–6 h for maximum dose of 3.2 g in 24 h).

(c) Other more invasive or inherently painful procedures will require a higher level of analgesia. This can be accomplished with opioids, preferably administered PO, such as Oxycodone (PO 5–10 mg q4–6h) or Hydromorphone (PO 2–6 mg q4–6 h or IV 0.2–0.5 mg q4–6 h).

(d) Like with any other procedure, the inflammatory response may elicit a fever in the early post-operative period; however, this should prompt a brief history and physical evaluation to exclude any other underlying cause. It is important to note the type of procedure, the time since the procedure was performed, and any intraoperative complications that may have occurred. Any line, tube, or incision should be examined for signs of infection.

(e) Postembolization syndrome is a known potential complication of embolization of usually malignant soft tissue masses and is characterized by noninfectious fevers in the first 2–3 days after the procedure.

(f) Once the etiology has been identified, fever can be treated with Acetaminophen and/or Ibuprofen as clinically indicated.

(g) Deep venous thrombosis, pulmonary emboli, or drug reactions should be kept in the back of the mind for causes of fevers in the post-operative state.

2. Bleeding

(a) Bleeding after an IR procedure can occur at the level of the puncture site, including site of vascular or percutaneous access. It is important to determine what type of procedure was performed and what kind of closure device may have been implemented.

(b) A review of the patient's chart for any anticoagulation medication, bleeding disorders, or chronic medical condition that may lead to increased bleeding risk should be performed.

(c) Treatment will be dictated by the patient's hemodynamic status and the characteristics of the bleed (venous vs. arterial, deep vs. superficial).

(d) Venous or superficial bleeds may subside from holding pressure at the site of bleeding. If the patient is not on anticoagulation medication, an absorbable suture may be employed.

(e) Arterial bleeds may also subside from holding pressure at the bleeding site, however one must be careful not to occlude the artery. A vascular surgery consult may be required if bleeding persists.

(f) Additionally, suspicion for puncture site pseudoaneurysms can be further evaluated with US.

3. Retroperitoneal Hematoma

(a) Arterial puncture above the inguinal ligament may lead to uncontrollable puncture site bleeding or retroperitoneal hemorrhage.

(b) Retroperitoneal hemorrhage in turn can lead to an insidious and devastating decline in the patient's clinical condition and may be life-threatening. Hypotension and tachycardia may be the only clinical signs of ongoing retroperitoneal hemorrhage.

(c) Depending on the patient's hemodynamic status an urgent non-contrast CT or CTA of the abdomen and pelvis may be performed as the first step in diagnostic management; however, hemorrhagic shock should prompt immediate surgical intervention.

(d) The standard therapeutic management is immediate surgical repair, however, if surgery is unavailable or contraindicated,

a covered stent placed across the vascular injury can be employed.

4. Neurological Complications
   (a) In order to adequately assess for neurological complications during or after an IR procedure, it is important to perform a neurological exam prior to the procedure and have an idea of the patient's baseline neurological function, as subtle changes in function may indicate a possible complication.
   (b) Intracranial hemorrhage, specifically subarachnoid hemorrhage, is an uncommon complication that may result from intracranial vessel perforation when manipulating microwires or catheters in the intracranial vasculature. When small and distal branches are being canalized, fast-acting embolic agents can be immediately employed to seal the perforation site and reduce the size of hemorrhage. When perforation has occurred, the microwire or catheter may be seen outside of the vessel on imaging. Alternatively, hypotension, bradycardia, or signs of increased intracranial pressure may suggest vascular injury. Immediate neurosurgical consult may be needed if there are any signs to suggest herniation.
   (c) Small and asymptomatic ischemic infarcts can occur during head and neck IR procedures. They usually result from tiny emboli which can arise from stagnant blood in catheters to tiny air bubble from poor technique. Large or symptomatic ischemic infarcts are rare and can often be identified and treated interprocedurally with thrombectomy or thrombolysis.
   (d) Catheter-associated or mechanically induced vasospasm of the head and neck vasculature may occur when manipulating intra-arterial catheters. Not only may this prevent advancement of the catheter, but the consequent cerebral hypoperfusion may lead to thrombotic and ischemic complications. Most cases are asymptomatic and self-limiting. Patients with evidence of vasospasm during diagnostic angiography, with high levels of anxiety, or tortuous vessels are more prone to catheter-induced vasospasm. It has been suggested that a patient may be administered a tranquilizer (etizolam or brotizolam), warm compresses, and/or an intra-arterial injection of lidocaine/nicardipine either prophylactically or as a means of treatment.
   (e) Continued advancements in microcatheter design have decreased the risk of immediate and delayed complications by minimizing trauma and interruption of vascular flow. Given the continued decrease in size of microcatheters, smaller vessels can be canalized for more selective treatment, thus reducing the risk of potential complications.
   (f) https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4533334/

#### 6.1.6.1 Additional Case of Direct Stick embolization

Left hemifacial, oral and pharyngeal low-flow vascular malformation.

The case below Fig. 6.7a–d was referred to clinic for obstructive sleep apnea and vascular lesion impeding his denture wearing. He had extensive cutaneous, oromucosal lesion causing palatal, pharyngeal and nasal area petulance superiorly (Figs. 6.8 and 6.9).

21-gauge butterfly needles are easily advanced into the venous malformation of the facial areas or left cheek as illustrated (Fig. 6.7) under ultrasound guidance.

Venous blood is freely aspirated from the lesion, confirming the vascular nature and flow pattern of filling of the lesion.

Angiography is performed with injection of 50% contrast to evaluate the size and venous drainage pattern of the lesion. The lesion is then serially injected under fluoroscopic guidance with boluses of 1:1 3% sodium tetradecyl sulfate (sotradecol):ethiodized oil (ethiodol) and the amount in millileters of sclerosing agent is deployed mindfully. Care is taken not to allow the sclerosing agent to enter into the draining veins.

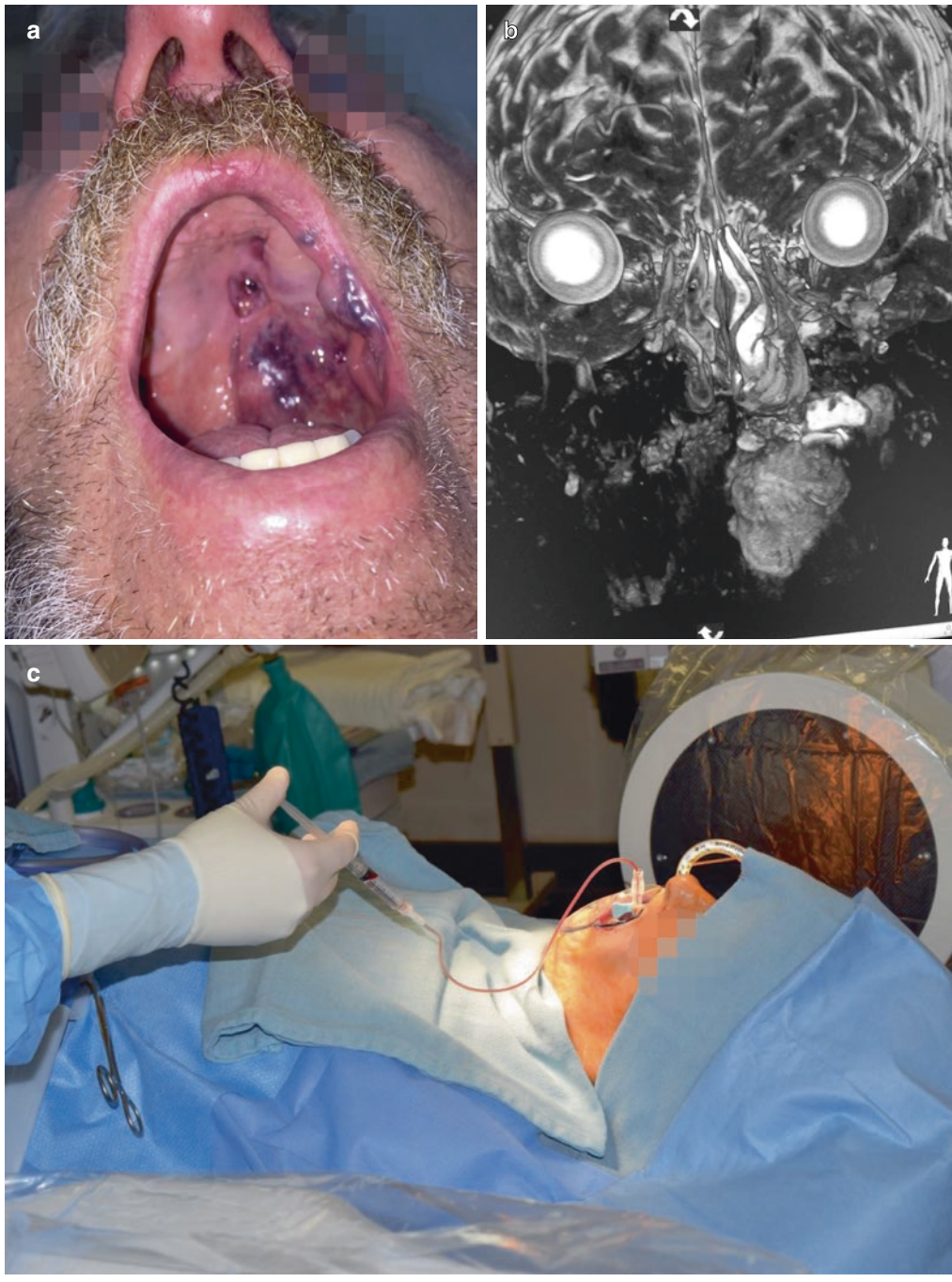


**Fig. 6.7** (**a**) Has extensive lesions of low flow quality scattered on his oral, palatal, and facial areas. The center of the palate has oro-nasal fistula. (**b**) A 3D rendition of the MRI shows the extent of this lesion from the left cavernous sinus, left nasal, peri orbital, palatal, and pharyngeal flow with contrast uptake. (**c**) Venous blood is freely aspirated from the lesion, confirming the vascular nature and flow pattern of filling of the lesion. Direct stick embolization setup intraoperatively with aspiration of the lesion revealing blood in the catheter and syringe. The lesion is then serially injected under fluoroscopic guidance with boluses of 1:1 3% sodium tetradecyl sulfate (sotradecol):ethiodized oil (ethiodol) and the amount of cc of sclerosing agent deployed is mindfully noted. Care is taken not to allow the sclerosing agent to enter into the draining veins. (**d**) Post injection of sclerosant


**Fig. 6.7** (continued)

**Fig. 6.8** Sagittal post contrasted MRI section reveals a blush on the tongue dorsum and floor of the mouth. Such isolated lesions are very amenable to direct stick embolization and excision

The procedure is then repeated for the venous malformation in the adjacent areas if any (idea is to do in increments) with similar 1:1 sotradecol:ethiodol total sclerosing agent deployed and similarly for the lesions in the oral mucosa, tongue, lip, upper airway, etc.

The needles were withdrawn from the venous malformations 5 min following completion of

**Fig. 6.9** These lingual vascular lesions are amenable to direct stick embolization with ultrasound or fluoroscopic exam. Surgically excision is amenable with proximal vascular clamp control as will be described in the surgical management section of the book with or without being sclerosed with cutaneous stick sclerotherapy

the injections and another 5 min of very gentle pressure was applied to the puncture sites following removal of the needles. At the completion of the procedure the face, neck, and upper chest were re-cleansed with sterile saline solution. The skin of the neck and upper chest was no longer erythematous and without an urticarial reaction.

The butterfly needle can be used to access the other cystic lesion or pouches of the venolymphatic malformation with biplanar fluoroscopy aids. Free flowing blood aspiration out of the venolymphatic pouch, and a pouchogram is performed to ensure that you are in the venolymphatic pouch proper.

Once this was confirmed, flush the needle, and then inject a mixture of 1:1:1 of 3% Sotradecol,

Ethiodol and air for foam solution to sclerose any vascular island or pouch. This is done under biplane fluoroscopy. Additional pools or pouches in different planes and deeper are determined again by performing a pouchogram to ensure the access is within the pouch, and then injecting a sclerosing agent in the similar dilutions.

As needles are removed, pressure application is important for hemostasis. Notably, the ultrasound is used intraoperatively and was very important in finding these pouches, as well as the biplane fluoroscopy. The authors' advice caution with risk of scatter emboli in cerebral, pulmonary draining veins, etc. Direct stick embolization can be used for bleomycin and sclerosants too.

In conclusion, majority of vascular lesions are diagnosed by presentations clinically and can be managed in a multidisciplinary team approach, there are some which will need extensive interventional diagnostic and therapeutic techniques. These can help surgical approach, morbidity reduction, and optimal outcomes.

The multidisciplinary team of authors as reflected in a vascular anomaly clinic team have taken the liberty to tackle the other vascular cases and emergencies encountered in head and neck practice.

## References


1. Gandhi D, Gemmete JJ, Ansari SA, Gujar SK, Mukherji SK. Interventional neuroradiology of the head and neck. Am J Neuroradiol. 2008;29(10):1806–15. https://doi.org/10.3174/ajnr.A1211.
2. Broomfield S, Bruce I, Birzgalis A, Herwadkar A. The expanding role of interventional radiology in head and neck surgery. J R Soc Med. 2009;102(6):228–34. https://doi.org/10.1258/jrsm.2009.080240.
3. Tulunay-Ugur OE, Kocdor P, Rutledge JW, Akdol MS, Erdem E, Vural E. The role of interventional radiology in head and neck cancer. Otolaryngol Head Neck Surg. 2013;149(2_suppl):P192. https://doi.org/10.1177/0194599813496044a150.
4. Kulkarni SS, Shetty NS, Dharia TP, Polnaya AM. Pictorial essay: vascular interventions in extra cranial head and neck. Indian J Radiol Imaging. 2012;22(4):350–7. https://doi.org/10.4103/0971-3026.111490.
5. Storck K, Kreiser K, Hauber J, et al. Management and prevention of acute bleedings in the head and neck area with interventional radiology. Head Face Med. 2016;12:6. https://doi.org/10.1186/s13005-016-0103-3.
6. Cooke D, Ghodke B, Natarajan SK, Hallam D. Embolization in the head and neck. Semin Intervent Radiol. 2008;25(3):293–309. https://doi.org/10.1055/s-0028-1085929.
7. Manzoor NF, Rezaee RP, Ray A, Wick CC, Blackham K, Stepnick D, Lavertu P, Zender CA. Contemporary management of carotid blowout syndrome utilizing endovascular techniques. Laryngoscope. 2017;127:383–90. https://doi.org/10.1002/lary.26144.
8. Kovács AF, Turowski B. Chemoembolization of oral and oropharyngeal cancer using a high-dose cisplatin crystal suspension and degradable starch microspheres. Oral Oncol. 2002;38(1):87–95. https://doi.org/10.1016/s1368-8375(01)00088-4.
9. Cullen MM, Tami TA. Comparison of internal maxillary artery ligation versus embolization for refractory posterior epistaxis. Otolaryngol Head Neck Surg. 1998;118(5):636–42. https://doi.org/10.1177/019459989811800512.
10. Elden L, Montanera W, Terbrugge K, Willinsky R, Lasjaunias P, Charles D. Angiographic embolization for the treatment of epistaxis: a review of 108 cases. Otolaryngol Head Neck Surg. 1994;111(1):44–50. https://doi.org/10.1177/019459989411100110.
11. Moreau S, De Rugy MG, Babin E, Courtheoux P, Valdazo A. Supraselective embolization in intractable epistaxis: review of 45 cases. Laryngoscope. 1998;108:887–8. https://doi.org/10.1097/00005537-199806000-00018.
12. Levy EI, Horowitz MB, Cahill AM. Lingual artery embolization for severe and uncontrollable postoperative tonsillar bleeding. Ear Nose Throat J. 2001;80:208–11.
13. Siniluoto TM, Luotonen JP, Tikkakoski TA, Leinonen AS, Jokinen KE. Value of pre-operative embolization in surgery for nasopharyngeal angiofibroma. J Laryngol Otol. 1993;107:514–21. https://doi.org/10.1017/S002221510012359X.
14. Tikkakoski T, Luotonen J, Leinonen S, Siniluoto T, Heikkilä O, Päivänsälo M, et al. Preoperative embolization in the management of neck paragangliomas. Laryngoscope. 1997;107:821–6. https://doi.org/10.1097/00005537-199706000-00018.
15. Pletcher JD, Newton TH, Dedo HH, Norman D. Preoperative embolization of juvenile angiofibromas of the nasopharynx. Ann Otol Rhinol Laryngol. 1975;84:740–6. https://doi.org/10.1177/000348947508400603.
16. Tang IP, Shashinder S, Gopala Krishnan G, Narayanan P. Juvenile nasopharyngeal angiofibroma in a tertiary centre: ten-year experience. Singap Med J. 2009;50:261–4.
17. Persky MS, Berenstein A, Cohen NL. Combined treatment of head and neck vascular masses with preoperative embolization. Laryngoscope. 1984;94:20–7. https://doi.org/10.1002/lary.5540940105.

18. Valavanis A. Preoperative embolization of the head and neck: indications, patient selection, goals, and precautions. AJNR Am J Neuroradiol. 1986;7:943–52.
19. Gemmete JJ, Ansari SA, McHugh J, Gandhi D. Embolization of vascular tumors of the head and neck. Neuroimaging Clin N Am. 2009;19(2):181–98.
20. Wittekind C. Prognostic factors in patients with squamous epithelium carcinoma of the head and neck area and their evaluation. Laryngorhinootologie. 1999;78:588–9. https://doi.org/10.1055/s-1999-12971.
21. Greve J, Bas M, Schuler P, Turowski B, Scheckenbach K, Budach W, et al. Acute arterial hemorrhage following radiotherapy of oropharyngeal squamous cell carcinoma. Strahlenther Onkol. 2010;186:269–73. https://doi.org/10.1007/s00066-010-2114-5.
22. Lowe LH, Marchant TC, Rivard DC, Scherbel AJ. Vascular malformation: Classifications and terminology the radiologist needs to know. Semin Rotengenol. 2012;47:106–17.
23. Armstrong DC, ter Brugge K. Selected interventional procedures for pediatric head and neck vascular lesions. Neuroimaging Clin N Am. 2000;10:271–92.
24. Erdmann MW, Jackson JE, Davies DM, Allison DJ. Multidisciplinary approach to the management of head and neck arteriovenous malformations. Ann R Coll Surg Engl. 1995;77:53–9.
25. Arat A, Cil BE, Vargel I, Turkbey B, Canyigit M, Peynircioglu B, et al. Embolization of high-flow craniofacial vascular malformations with onyx. AJNR Am J Neuroradiol. 2007;28:1409–14.
26. Berenguer B, Burrows PE, Zurakowski D, Mulliken JB. Sclerotherapy of craniofacial venous malformations: Complications and results. Plast Reconstr Surg. 1999;104:1–11.
27. Cabrera J, Cabrera J Jr, Garcia-Olmedo MA, Redondo P. Treatment of venous malformations with sclerosant in microfoam form. Arch Dermatol. 2003;139:1409–16.
28. Ryu CW, Whang SM, Suh DC, Kim SM, Jang YJ, Kim HJ, et al. Percutaneous direct puncture glue embolization of high-flow craniofacial arteriovenous lesions: a new circular ring compression device with a beveled edge. AJNR Am J Neuroradiol. 2007;28:528–30.
29. Wakhloo AK, Juengling FD, Van Velthoven V, Schumacher M, Hennig J, Schwechheimer K. Extended preoperative polyvinyl alcohol microembolization of intracranial meningiomas: assessment of two embolization techniques. AJNR Am J Neuroradiol. 1993;14:571–82.
30. Pérez Higueras A, Saura Lorente P, García Alonso J, de las Heras García JA. Diagnosis and treatment of head and neck paragangliomas. Uses of angiography and interventional radiology. Acta Otorrinolaringol Esp. 2009;60:53–67.
31. Gruber A, Bavinzski G, Killer M, Richling B. Preoperative embolization of hypervascular skull base tumors. Minim Invasive Neurosurg. 2000;43:62–71.
32. Chaloupka JC, Putman CM, Citardi MJ, Ross DA, Sasaki CT. Endovascular therapy for the carotid blowout syndrome in head and neck surgical patients: diagnostic and managerial considerations. AJNR Am J Neuroradiol. 1996;17:843–52.
33. Goodman DN, Hoh BL, Rabinov JD, Pryor JC. CT angiography before embolization for hemorrhage in head and neck cancer. AJNR Am J Neuroradiol. 2003;24:140–2.
34. Morrissey DD, Andersen PE, Nesbit GM, Barnwell SL, Everts EC, Cohen JI. Endovascular management of hemorrhage in patients with head and neck cancer. Arch Otolaryngol Head Neck Surg. 1997;123:15–9.
35. Sittel C, Gossmann A, Jungehülsing M, Zähringer M. Superselective embolization as palliative treatment of recurrent hemorrhage in advanced carcinoma of the head and neck. Ann Otol Rhinol Laryngol. 2001;110:1126–8.
36. Chang FC, Lirng JF, Luo CB, Guo WY, Teng MM, Tai SK, et al. Carotid blowout syndrome in patients with head-and-neck cancers: Reconstructive management by self-expandable stent-grafts. AJNR Am J Neuroradiol. 2007;28:181–8.
37. Dubose J, Recinos G, Teixeira PG, Inaba K, Demetriades D. Endovascular stenting for the treatment of traumatic internal carotid injuries: expanding experience. J Trauma. 2008;65:1561–6.
38. Santhosh J, Rao VR, Ravimandalam K, Gupta AK, Unni NM, Rao AS. Endovascular management of carotid cavernous fistulae: observation on angiographic and clinical results. Acta Neurol Scand. 1993;88:320–6.
39. Gobin YP, Garcia de la Fuente JA, Herbreteau D, Houdart E, Merland JJ. Endovascular treatment of external carotid-jugular fistulae in the parotid region. Neurosurgery. 1993;33:812–6.