VASCULAR IMAGING INTERVENTIONAL RADIOLOGY

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Vascular Imaging
Vascular Imaging

**AIM**
to visualize the lumen, wall, and surroundings of arteries and veins

**METHODS**
- ultrasound / doppler sonography / CEUS
- plain X-ray
- fluoroscopy / DSA
- CTA
- MRA
by way of introduction

vessel = pipe

the wall of arteries and veins is build of 3 layers:

• inner membrane (tunica intima)
• middle membrane (tunica media)
• external membrane (tunica adventitia)
- **inner membrane** = the layer of single cells maintaining normal blood flow; inter alia preventing thrombosis; the most chemically active layer

- **middle membrane** = mainly consist of smooth muscles, % of them depends on the size of the vessel; smooth muscle contraction in response to a stressor that reduces vascular environment

- **external membrane** = connective tissue layer of different thickness of the membrane surrounding the middle membrane
the affected vessel wall leads to:

- weakening and widening
- aneurysm formation, cracking, bleeding
- form a pseudoaneurysm, arteriovenous fistulas
- thickening of the inner layer or the deposition of atherosclerotic plaque
angiography

Anatomical mapping of vascular structures and high contrast resolution images. The basic method of verification of diagnosis of vascular pathologies performed non-invasive methods such as the degree of stenosis, the extent of obstruction, evaluation of collateral circulation. Basis for the exercise of intravascular radiological treatments.
Common indications

1. Vascular malformations (with patent vessels).
2. Tumor supply / vascularity – for treatment guidance.
4. Aneurysms.
5. Bleeding.

Common applications

1. Atherosclerosis (carotids, iliacs, lower limbs).
2. Cerebral arterio-venous malformations.
3. Aortic aneurysms.
4. Pulmonary embolism.
5. GI bleeding.
Doppler ultrasound – indications: assessment of the vessel wall; suspicion of arterial stenoses; suspicion of the presence of vascular malformations; quantitative assessment of the flow. There are no contraindications.

Computed tomography - spiral with the administration of contrast agent, the possibility of three-dimensional reconstruction. Indications: suspected presence of dissecting aneurysm; assessment of tumor infiltration from the outside; suspicion of stenosis; the presence of vascular malformations; evaluation of complications after implantation of a vascular prosthesis.

Magnetic resonance imaging - no need to enter the contrast agent, possible to assess the direction of flow and velocity. Indications: Congenital anomalies of the aorta and its major branches (especially dissecting aortic aneurysm); evaluation of vascular malformations, assessment of the degree of stenosis or occlusion; control of endovascular treatment; study in patients with allergy to iodine-based contrast agents.
# Aorta

## Table 1 Clinical condition: pulsatile abdominal mass, suspected AAA

<table>
<thead>
<tr>
<th>Radiologic procedure</th>
<th>Rating</th>
<th>Comments</th>
<th>RRL&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>US aorta abdomen</td>
<td>9</td>
<td>Initial examination. May be limited by body habitus or acoustic window</td>
<td>O</td>
</tr>
<tr>
<td>CT abdomen without contrast</td>
<td>8</td>
<td>Preferred for symptomatic patients. Suitable for patients in whom US is not useful</td>
<td>★★★</td>
</tr>
<tr>
<td>CTA abdomen with contrast</td>
<td>7</td>
<td>Also enables preinterventional planning</td>
<td>★★★★</td>
</tr>
<tr>
<td>MRA abdomen without contrast</td>
<td>6</td>
<td>Alternative to CTA. Unable to detect calcium. Site-specific expertise important</td>
<td>O</td>
</tr>
<tr>
<td>MRA abdomen without and with contrast</td>
<td>6</td>
<td>Alternative to CTA. Unable to detect calcium. Site-specific expertise important. See statement regarding contrast in text under “anticipated exceptions”</td>
<td>O</td>
</tr>
<tr>
<td>Aortography abdomen</td>
<td>2</td>
<td>Essentially replaced by cross-sectional imaging for diagnostic purposes. May be used for preinterventional planning</td>
<td>★★★★</td>
</tr>
<tr>
<td>FDG-PET/CT abdomen</td>
<td>2</td>
<td></td>
<td>★★★★★</td>
</tr>
</tbody>
</table>

Rating scale: 1–3 usually not appropriate, 4–6 may be appropriate, 7–9 usually appropriate

<sup>a</sup> Relative radiation level
Aorta - variants
Aorta - variants

Coarctation of the aorta refers to a narrowing of the aortic lumen. It can be primarily divided into two types:

Infantile coarctation is characterised by diffuse hypoplasia or narrowing of the aorta from just distal to brachiocephalic artery to the level of ductus arteriosus, typically with a more discrete area of constriction just proximal to the ductus but distal to the origin of the left subclavian artery. Therefore the blood supply to the descending aorta is via the patent ductus arteriosus.

Adult coarctation in contrast is characterised by a short segment abrupt stenosis of the post-ductal aorta. It is due to thickening of the aortic media and typically occurs just distal to the ligamentum arteriosum (remnant of the ductus arteriosus).
Aorta - variants
Aorta - variants

clinical signs:
- chest pain
- difference in RR on both upper limbs
- pulmonary hypertension
Aorta - inflammation

**Aortitis** refers to a general descriptor that involves a broad category of infectious or noninfectious conditions where there is abnormal inflammation of the aortic wall.

**Pathology**

- **infectious**
  - syphilitic aortitis
  - tuberculous aortitis
  - pyogenic aortitis - especially with salmonella infection
  - infected (mycotic) aortic aneurysm

- **non infections**
  - giant cell arteritis
  - Takayasu arteritis
  - other rheumatologic disorders
    - rheumatoid arthritis
    - systemic lupus erythematosus
    - Wegener granulomatosis
  - idiopathic aortitis
  - radiation induced aortitis
Aorta - inflammation
Aorta - inflammation
Aorta - tumors
Aortic dissection is one of the acute aortic syndromes and a type of arterial dissection. It occurs when blood enters the medial layer of the aortic wall through a tear or penetrating ulcer in the intima and tracks along the media, forming a second blood-filled channel within the wall.

Epidemiology
The majority of aortic dissections are seen in elderly hypertensive patients. In a very small minority, and underlying connective tissue disorder may be present. Other conditions / predisposing factors may also be encountered, in which case they will be reflected in the demographics.

Examples include:
structural aortic abnormalities
- bicuspid aortic valve
- aortic coarctation
- abnormal connective tissues
  - Marfan syndrome
  - Ehlers-Danlos syndrome
Turner syndrome
pregnancy
intra aortic balloon pumps
Aorta - dissection

Clinical presentation
Patients are often hypertensive (although they may be normotensive or hypotensive) and present with anterior or posterior chest pain and a tearing sensation in chest.

Depending on the extent of dissection and occlusion of branches, end organ ischaemia may also be present (seen in up to 27% of cases), including:
- abdominal organ ischaemia
- limb ischaemia
- ischaemic or embolic stroke
- paraplegia: involvement of artery of Adamkiewicz

In some cases aortic rupture, involvement of coronary arteries may result in collapse and death. Symptoms of cardiac tamponade may also be seen.
Ascending aortic aneurysms are the most common subtype of thoracic aortic aneurysms, and may be true or false injuries. Aneurysmal dilatation is considered with the ascending aorta measures >4.0 cm in diameter.

Epidemiology
Ascending aortic aneurysms represent 60% of thoracic aortic aneurysms.

Clinical presentation
Typically ascending aortic aneurysms are an incidental finding and the patient is asymptomatic. Rarely the patient may present with symptoms and signs of rupture (e.g. pain, hypotension).

Aetiology
True aneurysms can be a result of a wide variety of conditions
atherosclerosis (uncommon)
connective tissue diseases (Marfan syndrome, Ehlers-Danlos syndrome)
aortitis (syphilis, Takayasu's disease, rheumatoid arthritis)
mycotic aneurysms
bicuspid aortic valve
cystic medial degneration

IDIOPATIC
Aorta - aneurysm

Normal Aorta  Aortic Aneurysms

Ascending  Thoracic  Abdominal

Fusiform aneurysm  Saccular aneurysm
Aorta - aneurysm
Aorta - aneurysm
Abdominal aortic aneurysms (AAA) are focal dilatations of the abdominal aorta that are 50% greater than the proximal normal segment or that is greater than 3 cm in maximum diameter.

**Epidemiology**
Its prevalence increases with age. Males much more commonly affected than females (with a male:female ratio of 4:1).

**Clinical presentation**
- unruptured - abdominal or back pain, pulsatile mass, none,
- ruptured - severe abdominal or back pain, hypotension, shock, death (59-83%).

The risk of rupture is proportional to the size of the aneurysm and the rate of growth.

**Causes**
- atherosclerosis (most common)
- inflammatory abdominal aortic aneurysm
- chronic aortic dissection
- vasculitis, e.g. Takayasu arteritis
- connective tissue disorders, (Marfan syndrome, Ehlers-Danlos syndrome)
- mycotic aneurysm
Aorta - aneurysm
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Aorta - aneurysm
Aorta - aneurysm
Aorta - atherosclerosis
Aorta - atherosclerosis

**Aortic atherosclerotic lesions** have been referred to in several different ways in the medical literature. These include atheromas, protruding atheromas, atherosclerotic debris, and plaque. For the purposes of this review, we define these lesions as plaques. The mobile components to these plaques have been called mobile debris, mobile plaque, and superimposed thrombi. These mobile lesions are most often thrombi. Clinical data show that the plaques with high risk for embolization are those that are ≥4 mm thick. We refer to these lesions as severe plaques. Finally, the term complex plaque has been used in the literature to refer to those plaques that are ≥4 mm thick (called severe here), contain mobile elements (most often thrombi), or both.

**Consequences:**
- distal embolization
- vessel occlusion
- aneurysm
- dissection
- rupture
Aorta - atherosclerosis
Aorta - atherosclerosis
Aorta - atherosclerosis
Aorta - atherosclerosis
Thoracic aortic injury is a life threatening, and often life ending event. It can result from either blunt or penetrating trauma:

- blunt trauma (more common)
  - rapid deceleration (e.g., motor vehicle accident, fall from great height)
  - crush injury
- penetrating trauma
  - stab wound
  - gun shot wound

Clinical presentation
Approximately 70% of patients with thoracic aortic injury die at the scene of the trauma. In those who make it to hospital, clinical diagnosis is difficult. The signs and symptoms are non-specific and distracting injuries are often present. Clinical presentation may include chest or mid-scapular back pain, signs of external chest trauma or haemodynamic instability. Clinical suspicion is usually based on mechanism and severity of the injury, the haemodynamic status of the patient and/or the presence of related injuries. The diagnosis ultimately relies on appropriate imaging.
Aorta - trauma
Aorta - trauma
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Aorta - trauma
Aorta - trauma
**Pulmonary embolism** refers to occlusion of the pulmonary arteries or its branches, usually via venous thrombus (other: air, fat, amniotic fluid).

A thrombus may lodge at the bifurcation of the main pulmonary artery causing haemodynamic compromise, while smaller thrombi may be located more distally.

**Risk factors:**

- deep vein thrombosis,
- immobilisation,
- recent surgery,
- malignancy,
- paralysis,
- smoking,
- obesity.
Pulmonary embolism

Clinical presentation:
• dyspnoea either at rest or on exertion,
• pleuritic chest pain, cough,
• orthopnoea and haemoptosis,
• calf/thigh pain and swelling,
• tachypnoea and tachycardia
• cyanosis and decreased oxygen saturation
• elevated D-dimer level
Pulmonary embolism

Imaging:

- **CXR:** usually either normal or show non-specific findings – pleural effusion, cardiomegaly, atelectasis, Hampton hump (shallow wedge shaped opacity in lung periphery) and Westermark sign (sharp pulmonary vessel with distal hypoperfusion)

- **pulmonary CT angiography (CTA):** is a method of choice for definitive diagnosis; emboli cause filling defects in the branches of the pulmonary arterial system; if there is a central filling defect that spans the main pulmonary artery where it branches into left and right pulmonary arteries, it is called a **saddle embolus.**
Pulmonary embolism

Imaging continued:

- **Ventilation/perfusion scan** – are performed where CT pulmonary angiography is contraindicated such as in severe renal failure, pregnancy or contrast allergy; PE is expressed by lung areas that are ventilated but not perfused,

- **Pulmonary angiography** – the historical gold standard for PE diagnosis; nowadays it is reserved for patients where CT pulmonary angiography or V/Q scans are non diagnostic; a filling defect or vessel occlusion is diagnostic of pulmonary embolism.
Pulmonary embolism
Pulmonary embolism
Pulmonary embolism
Pulmonary embolism
Pulmonary embolism
Pulmonary embolism
Interventional Radiology
# Interventional Radiology

**AIM**
to treat pathologies using image guidance

**OPTIONS**

<table>
<thead>
<tr>
<th>Imaging</th>
<th>Angiography / Arteriography / Venography</th>
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</thead>
<tbody>
<tr>
<td>Fluid evacuation</td>
<td>Puncture</td>
</tr>
<tr>
<td>Repair of narrowings</td>
<td>Angioplasty, Stenting</td>
</tr>
<tr>
<td>Repair of occlusions</td>
<td>Recanalization</td>
</tr>
<tr>
<td>Closure of foramens / canals</td>
<td>Obliteration, Embolization</td>
</tr>
<tr>
<td>Closure of vessels</td>
<td>Embolization</td>
</tr>
<tr>
<td>Drug delivery</td>
<td>Targeted Therapy, Chemoembolization, Radioembolization</td>
</tr>
<tr>
<td>Vessel lumen repair</td>
<td>Stenting, Stentgraft Implantation</td>
</tr>
</tbody>
</table>
DSA
Additional processing features of DSA
- last image hold
- road mapping
- pixel shifting
- 3D DSA
Arteriography - contraindications

absolute:

• lack of consent for research

• unstable patient with multiple organ failure

relative:

• coagulopathy (blood platelet counts below 50000/μl, prothrombin time exceeds 16 s)

• recent myocardial infarction arrhythmia and renal electrolyte levels

• serious side reaction to contrast agents in an interview

• renal failure

• failure to remain in a lying position because of heart failure or respiratory

• barite-presence in the gastrointestinal tract

• pregnancy
Seldinger method
equipment

- catheters
- guidewires
- balloons
- stents
- stentgrafts
- coils
- closing devices
- embolization particles
- vena cava filters
equipment

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examples

1. Angioplasty for the renal artery stenosis.
2. Cerebral aneurysm embolization.
3. Stentgraft implantation for the thoracic aorta aneurysm.
4. Embolization for the liver tumors.
Renal artery stenosis (RAS) refers to a narrowing of a renal artery. When the process occurs slowly, it leads to secondary hypertension. Acute renal artery stenosis does not lead to hypersecretion of renin.

When the stenosis occurs slowly, collateral vessels form and supply the kidney. The kidney wrongly senses the reduced flow as low blood pressure (via juxtaglomerular apparatus) and releases large amount of renin, that converts angiotensinogen to angiotensin I. Angiotensin I is then converted to angiotensin II with the help of angiotensin converting enzyme (ACE) in lungs. Angiotensin II is responsible for vaso-constriction and release of aldosterone which causes sodium and water retention, thus resulting in secondary hypertension.
Renal artery stenosis may be caused by several pathological processes:

- atherosclerosis ~75% involves proximal renal artery
- fibromuscular dysplasia ~20% involves distal renal artery
- vasculitides (especially polyarteritis nodosa, Takayasu arteritis, radiation)
- neurofibromatosis type 1
- abdominal aortic coarctation
- aortic dissection
- segmental arterial mediolysis
renal artery stenosis
renal artery stenosis

Case courtesy of Dr Charlie Chia-Tsong Hsu, Radiopaedia.org

https://www.youtube.com/watch?v=Ilg-1xT3o1U
renal artery stenosis
Effectiveness of Management Strategies for Renal Artery Stenosis:
A Systematic Review

Ethan Balk, MD, MPH; Gowri Raman, MD; Mel Chung, MPH; Stanley Ip, MD; Athina Tatsioni, MD; Alvaro Alonso, MD; Priscilla Chew, MPH; Scott J. Gilbert, MD; and Joseph Lau, MD

Background: Atherosclerotic renal artery stenosis is increasingly common in an aging population. Therapeutic options include medical treatment only or revascularization procedures.

Purpose: To compare the effects of medical treatment and revascularization on clinically important outcomes in adults with atherosclerotic renal artery stenosis.

Data Sources: The MEDLINE database (inception to 6 September 2005) and selected reference lists were searched for English-language articles.

Study Selection: The authors selected prospective studies of renal artery revascularization or medical treatment of patients with atherosclerotic renal artery stenosis that reported mortality rates, kidney function, blood pressure, cardiovascular events, or adverse events at 6 months or later after study entry.

Data Extraction: A standardized protocol with predefined criteria was used to extract details on study design, interventions, outcomes, study quality, and applicability. The overall body of evidence was then graded as robust, acceptable, or weak.

Data Synthesis: No study directly compared aggressive medical therapy with angioplasty and stent placement. Two randomized trials compared angioplasty without stent and medical treatments. Eight other comparative studies and 46 cohort studies met criteria for analysis. Studies generally had poor methodologic quality and limited applicability to current practice. Overall, there was no robust evidence. Weak evidence suggested no large differences in mortality rates or cardiovascular events between medical and revascularization treatments. Acceptable evidence suggested similar kidney-related outcomes but better blood pressure outcomes with angioplasty, particularly in patients with bilateral disease. Improvements in kidney function and cure of hypertension were reported among some patients only in cohort studies of angioplasty. Available evidence did not adequately assess adverse events or baseline characteristics that could predict which intervention would result in better outcomes.

Limitations: The evidence from direct comparisons of interventions is sparse and inadequate to draw robust conclusions.

Conclusions: Available evidence does not clearly support one treatment approach over another for atherosclerotic renal artery stenosis.
renal artery stenosis

- Reviewed 55 studies
- “Almost two thirds of the studies that we reviewed were of poor methodologic quality; none was deemed to be of good quality.”
- “More than half of the studies had limited applicability to patients commonly seen in practice or to modern management strategies.”
- “No study directly compared angioplasty with stent placement and "aggressive" medical treatment with currently available antihypertensive, antiplatelet, and lipid-lowering agents.”
renal artery stenosis

<table>
<thead>
<tr>
<th>Study</th>
<th>Device</th>
<th>N</th>
<th>Cure</th>
<th>Improved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Klinge</td>
<td>stent</td>
<td>134</td>
<td>10%</td>
<td>68%</td>
</tr>
<tr>
<td>Lossino</td>
<td>stent</td>
<td>153</td>
<td>12%</td>
<td>51%</td>
</tr>
<tr>
<td>DRASTIC balloon</td>
<td></td>
<td>106</td>
<td>7%</td>
<td>68%</td>
</tr>
<tr>
<td>Rocha</td>
<td>stent</td>
<td>150</td>
<td>6%</td>
<td>50%</td>
</tr>
<tr>
<td>Dorros</td>
<td>stent</td>
<td>145</td>
<td>1%</td>
<td>52%</td>
</tr>
</tbody>
</table>
cerebral aneurysm embolization

epidemiology
• 1.7-3.1% of the general population
• ADPK, family history, atherosclerosis, art. hypertension, smoking, females, genetic factors
• 87-92% in the anterior circulation
• 68-77% ≤ 6 mm
• in 10-30% multiple aneurysms

symptoms
• none, detected accidentally
• subarachnoid hemorrhage (8-10 / 100 000 yearly)
• vision defects, deficits from cranial nerves, seizures, headache, TIA
cerebral aneurysm embolization

surgical
- clipping
- packing
- trapping

radiological = endovascular
- embolizacja
cerebral aneurysm embolization

DISADVANTAGES OF COILING

- primarily incomplete filling – 24-53%
- aneurysm recurrence (recanalization) – 21-34%
- rebleeding – 0,1-1,8%
- necessity for retreatment – 6-10%
cerebral aneurysm embolization
cerebral aneurysm embolization
cerebral aneurysm embolization
Endovascular aneurysm repair (EVAR) was first pioneered in the early 1990s. Since then technology of the devices has rapidly progressed and EVAR is now widely used as treatment of thoracic and abdominal aortic aneurysms (AAA).

The advantages of endovascular repair over open repair is that they are less invasive than open surgery, have a lower surgical morbidity and mortality rate, and they reduce the length of post-operative stay in hospital. One disadvantage is the need for life-long follow-up imaging, and the long-term durability of graft material is yet to be proven.

**Indications**

EVAR is performed in patients undergoing elective aneurysm repair as well as patients undergoing emergency repair (e.g. traumatic aortic injury and ruptured AAA).
stentgraft

https://www.youtube.com/watch?v=_zpT8RJwE10
stentgraft
Complications

- endoleak: occurs in 30-40%
- continued enlargement of the aneurysm sac without endoleak
- delayed aneurysm rupture
- graft migration
- branch vessel occlusion with end-organ ischaemia/infarction
- infection
- stent-graft structural breakdown
- groin complications

Patients require life-long imaging surveillance to monitor for endoleak, aneurysm expansion and graft integrity. This is most commonly performed via CTA. MRA is an alternative but stainless steel stents cause major susceptibility artefact that limits its usefulness in such cases.
stentgraft
liver embolization
liver embolization

**Tumor embolization** is a procedure to shut down the blood supply to cancer cells in order to promote their reduction or complete cell death. It can be subdivided into three groups of procedures:

- bland embolization
- chemoembolization
- embolization with radioactive particles (radioembolization)
liver embolization

Is a method of treating liver tumors (primary or secondary) in patients in whom surgery is not an option, such as:

- unresectable hepatic metastatic disease
- unresectable hepatocellular carcinoma (HCC)
- hepatic tumour progression despite treatment
- symptoms related to hepatic tumor bulk or hormonal excess (Neuroendocrine tumors)
- "bridge to transplant": stop tumor progression while awaiting liver transplant
- life expectancy > 90 days
- liver-dominant tumor burden
Primary and metastatic hepatic malignancies derive 80-100% of their blood supply from the hepatic artery unlike normal liver that receives only 20% from the arterial supply. This allows the use of higher doses or internal radiation or chemotherapy than the normal liver can tolerate.

The transcatheter hepatic artery infusion of chemotherapeutic / radioisotope delivers high local tumoral doses, while sparing the surrounding healthy liver parenchyma (or at least only a low, tolerable dose).
Figure 1. A 56-year-old female patient with symptomatic gastrinoma liver metastasis that progressed despite systemic chemotherapy. She received 4 sessions of conventional transarterial chemoembolization (TACE) that led to complete symptomatic remission and disease control. Computed tomography scan before TACE shows large tumor burden in both lobes (A). Computed tomography scan performed 5 years later shows excellent oncologic result (B).
Radioembolization

It generally considered efficacious in patients with hepatocellular cancer, neuroendocrine and colorectal liver metastases. It generally involves a single delivery of $^{90}$yttrium micro-spheres into the hepatic artery. Preferential uptake is achieved into liver tumours, because of their predominant hepatic arterial blood supply. Average tumour doses of radiation in excess of 200 Gy are achieved.

It`s achieved by the intraarterial injection of Yttrium 90 (beta emitter) labelled glass or resin microspheres as an interventional radiology procedure.

https://www.youtube.com/watch?v=LPMGLs6eVZs
liver embolization

Potential complications

- nausea, vomiting, fever, diarrhea and abdominal pain
- transient lab abnormalities including liver function, hemoglobin and platelet levels
- acute pancreatitis, pneumonitis, gastritis, hepatitis, acute cholecystitis

Main advantage

potentially repeatable