Presse Med. 2011; 40: e437–e452

on line on
www.em-consulte.com/revue/lpm
www.sciencedirect.com

Management of peripheral arterial disease: Role of computed tomography angiography and magnetic resonance angiography

Gilles Soulez[1,2], Eric Therasse[1], Marie-France Giroux[1], Louis Bouchard[1], Patrick Gilbert[1], Pierre Perreault[1], Guy Cloutier[2], Vincent L. Oliva[1]

1. Department of Radiology, centre hospitalier de l'université de Montréal (CHUM)–hôpital Notre-Dame, Québec, Canada
2. CHUM-Research Center (CRCHUM), University of Montreal, Québec, Canada

Correspondence:
Gilles Soulez, Department of Radiology, CHUM–Notre-Dame, University of Montreal, 1560 Sherbrooke east, H2L 4M1, Montreal, Qc, Canada.
gilles.soulez.chum@ssss.gouv.qc.ca

Available online: 12 July 2011

Summary

The recent technological developments of CT and MR units enable fast angiographic acquisitions with an improved spatial and temporal resolution. With advanced 3D visualisation, image post-processing and vessel wall-imaging, these technologies are now almost replacing diagnostic angiography that is now mainly indicated in case of suboptimal computed tomography angiography (CTA) or magnetic resonance angiography (MRA) examinations. Catheter angiography is now used to guide endovascular therapy and the planning of endovascular intervention will rely mainly on CTA or MRA examinations. The relative indications of MRA and CTA for the assessment and follow-up of peripheral arterial disease are based on the clinical indication, potential contraindication and the accessibility. We will review in this chapter, the technical requirements to perform adequate CTA and MRA examination, the relative indications of both modalities for the diagnosis and management of peripheral arterial occlusive disease (PAOD) and abdominal and peripheral aneurysm diseases. The main imaging features observed in these patients will be detailed.

The incidence of peripheral arterial occlusive disease is increasing, reflecting longer life expectancy and prevalence of atherosclerosis. In the PARTNER study, the incidence of PAD in a large population of more than 70-year-old and 50 to 69-year-old with a history of smoking or diabetes was estimated at 29%. Most patients being asymptomatic [1] while 10 to 35% present an intermittent claudication and 1 to 2% a critical limb ischemia [2]. Measurement of ankle brachial index followed by color Doppler ultrasound is the first line examination to evaluate symptomatic patients [3]. However, in patients not responding to medical treatment, an accurate planning of atherosclerosis lesion distribution and distal run-off is necessary to plan endovascular or surgical interventions. For 10 years, magnetic resonance and computed tomography angiography

(respectively MRA and CTA) are progressively replacing invasive catheter angiography for the mapping of peripheral occlusive arterial disease [3]. Similarly, the prevalence of abdominal aortic aneurysms is also increasing and abdominal aortic aneurysms (AAA) are found in approximately 8% of men above 65 years [4,5]. CTA plays a major role in the planning and follow-up of aneurysm endovascular repair.

The relative indications of MRA and CTA for the assessment and follow-up of peripheral arterial disease are based on the clinical indication, potential contraindication and the accessibility. We will review in this chapter the basic technical requirements of CTA and MRA, the main imaging features and the relative indication of both technologies in the management of occlusive peripheral arterial disease and abdominal aortic and peripheral aneurysms.

Technique of abdominal aorta and peripheral arteries CTA acquisition

The fast technological evolution of CT units in the 10 last years allows the acquisition of high resolution images with an isotropic voxel size of less than 1 mm^3. Very adequate peripheral CT angiography can be performed with a 16-detector CT. Nowadays, most installed units have 64, 128 and sometimes 256 detectors. The typical coverage of a peripheral CTA includes

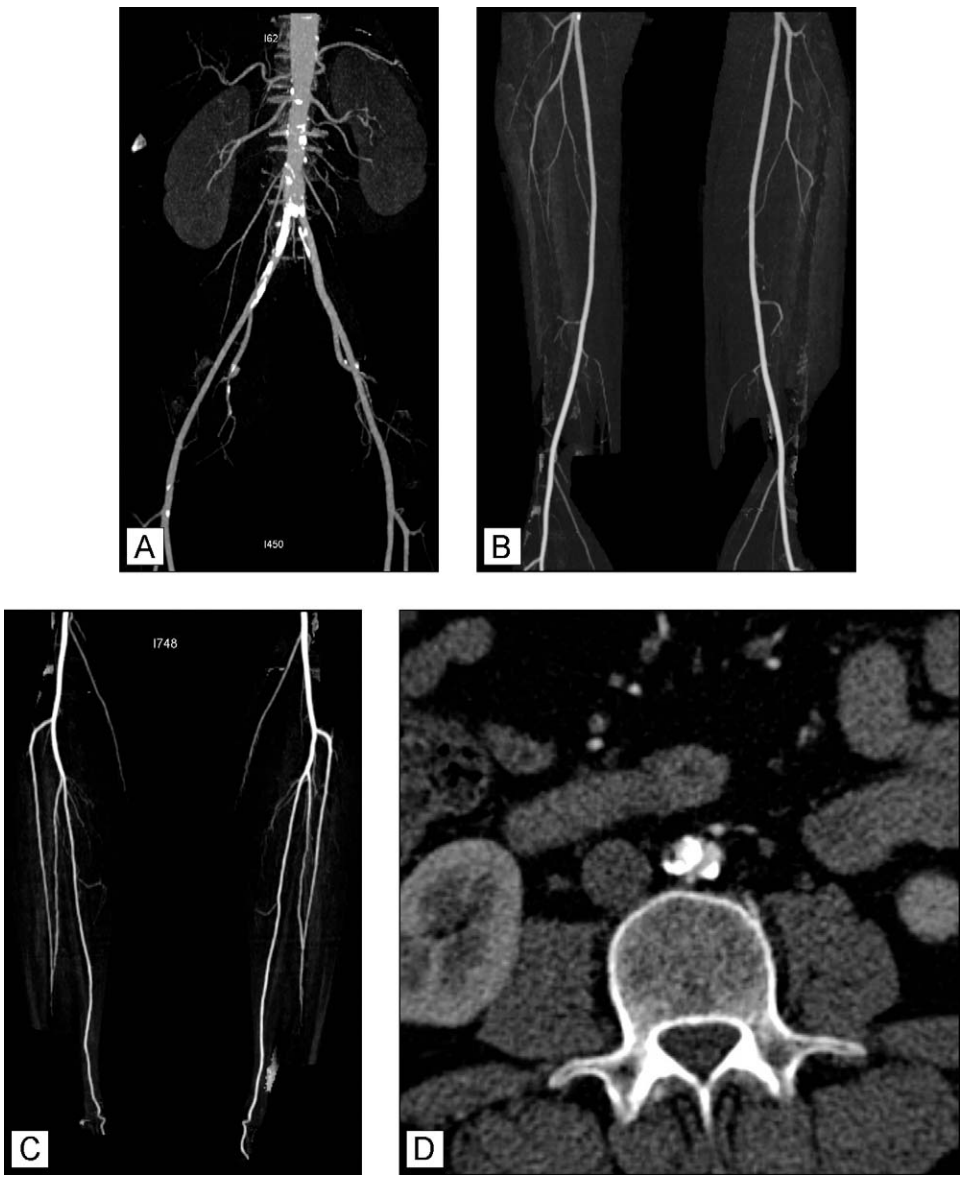

FIGURE 1

CTA examination of a 52-year-old women with a smoking history and a bilateral claudication

a: MIP reformation on the abdominal aorta and iliac arteries showing a calcified plaque at the aorto-iliac bifurcation indicating a Leriche syndrome; b, c: MIP reformation of the femoro-popliteal and infrapoliteal arteries without significant atherosclerotic lesion; d: axial source image at the level of iliac bifurcation showing a severe stenosis on the proximal right common iliac artery and significant but less severe stenosis on the left side.

the abdominal aorta from the celiac trunk down to the foot (average coverage 130 cm) (*figure 1*). To get an adequate vessel enhancement, it is important to synchronize properly the contrast bolus with the acquisition. The acquisition is started in a timely fashion with the arrival of the contrast bolus and the table speed need to be determined according to blood flow velocity.

Table speed is determined by the beam width, the rotation time and the pitch.

The beam width is a function of the number of detectors and the collimation [6]. For example, a 64 detectors CT with a collimation of 0.625 mm will give a beam width of 40 mm = 64 × 0.625 mm. The pitch is defined as the table travel per rotation divided by the beam width [7]. A pitch greater than 1 means there is no overlap in the acquisition. For example a typical peripheral CTA examination with a 64 detectors MDCT will have a table of speed of 48 mm/s = 40 mm beam width (64 channels detector × collimation of 0.625 mm) × pitch (0.6:1) and two rotations per second (rotation time 0.5s = 2 rotations per second). The time of acquisition is related to the table speed and the coverage. For a 130 cm coverage, the acquisition time for our example will be 27 s (130 cm/4.8 cm/s).

It is important to optimize the flow rate, the iodine concentration and the total amount of contrast agent injected to get an appropriate iodine concentration inside the vessel lumen to depict small vessels (1–2 mm) and differentiate vessel lumen from wall calcification. Typically, 90–100 mL of non-ionic contrast with an iodine concentration between 350 and 400 mg/L with a multiphasic injection: 70 at 5 mL/s, 30 at 4 mL/s flushed by 50 mL saline at 4 mL/s is injected. Saline infusion is necessary to avoid contrast retention in the veins of the arm not contributing to arterial enhancement [8]. The bolus arrival is automatically detected at the level of the proximal abdominal aorta and the acquisition is triggered with a delay varying between 6 and 10 s to be sure the distal arteries will be opacified properly. The median transit time between the aorta and the popliteal artery is estimated at 8 s and vary between 4 and 24 s. Furthermore blood flow is slower in the infrapopliteal arteries [9]. Hence, with recent units having 64 detectors or more, the table speed is reduced by decreasing the pitch or increasing the rotation time [10]. A table speed of 40–48 mm/s is suggested in patients with occlusive peripheral arterial disease [11]. If an aortic or femoro-popliteal aneurysm is suspected, a table feed of 30 mm/s is preferred [12]. A delayed acquisition covering knee, leg and foot is a good alternative when distal opcaification is likely to be suboptimal on the first pass.

The use of a lower tube voltage of 100 kV (compared to 120 kV) has been recommended to reduce the radiation exposure of the patient by 35% in comparison to 120 kV, without compromising image quality (except the increase of artifacts related to vascular calcification secondary to beam hardening) [13].

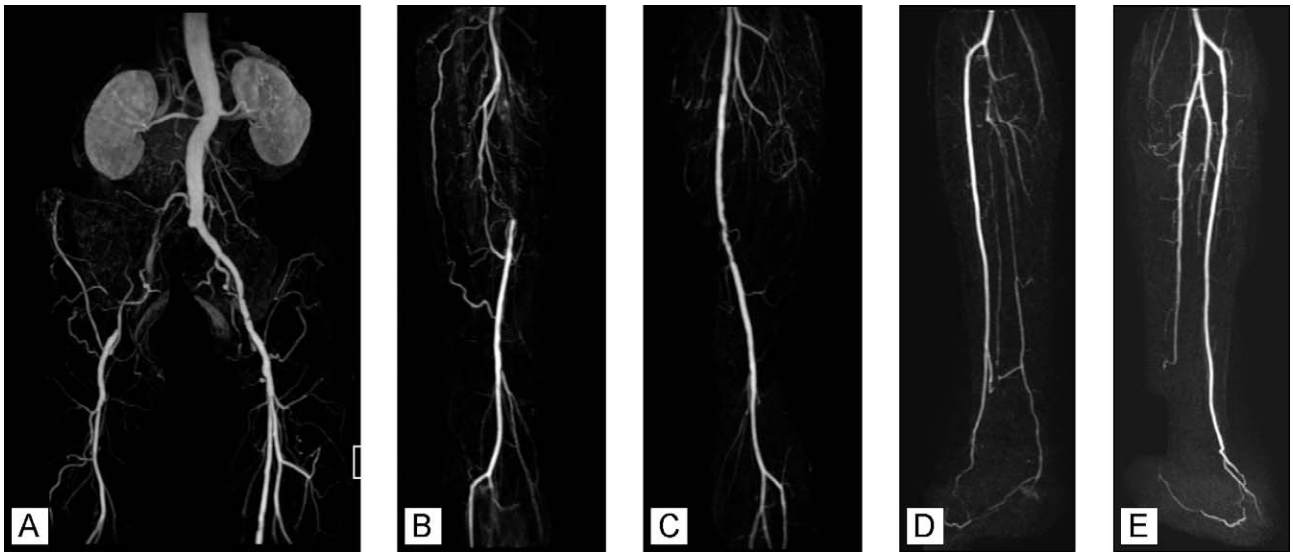

FIGURE 2

MRA examination of 61-year-old man with a stage II B claudication on the right side

a: MIP reformation of the abdominal aorta showing an occlusion of the right common and external iliac artery, a short stenosis of the distal left external iliac artery and a stenosis of the right renal artery; b: MIP reformation of the right femoropopliteal artery showing a long occlusion of the superficial femoral artery; c: MIP reformation of the left femoropopliteal artery showing a moderate stenosis of the mid superficial femoral artery; d,e: infrapopliteal station showing one patent infrapopliteal vessel (anterior tibial) on both sides.

Technique of MRA

There are three major families of MRA techniques. 1-The flow dependant techniques without contrast agent based on proton inflow (time of flight [TOF]) or 2-phase shift of the flowing protons (phase contrast [PC] angiography) and 3-contrast-enhanced (gadolinium) MRA. The TOF technique was used in the early stage of MRA, but not anymore for peripheral arteries because of its susceptibility to motion artifacts, stenosis overestimation and time consumption. The flow-independent non-contrast techniques were recently developed with the issue of nephrogenic systemic fibrosis (NSF) associated with gadolinium-based contrast agent. The magnetization-prepared balanced steady-state free precession (bSSFP) and ECG gated fast spin echo imaging techniques are under investigation but up to now there is no large scale clinical evaluation of the accuracy of this approach in peripheral arteries [14,15]. Finally, gadolinium-enhanced (GE) MRA combining fast imaging (time resolved imaging) and bolus injection of gadolium contrast agent is the technique presently used by most teams [16]. This technique involves the administration of a large (0.2 mmol/kg) dose of a gadolinium-chelated contrast agent during consecutive coronal 3D gradient echo acquisitions (stepping technique). To cover the aorta and lower limb arteries, three acquisitions are acquired sequentially to follow the progression of the contrast agent (*figure 2*). This technique provides an excellent signal with minimal motion artefact. Background suppression can be obtained with subtraction. High field MR unit (1.5 or 3 Tesla) equipped with high-speed gradients will give the better results especially for GE-MRA. With parallel imaging and high gradient capabilities, fast acquisition time can be obtained for each step depending of spatial resolution (between 12–20 s for step 1 and 2 and 25–40 s for step 3) [17]. Adequate timing of the bolus is critical to get adequate signal without venous enhancement. However, the timing of the last acquisition (infrapopliteal) is frequently in the late phase of contrast bolus progression leading to venous overlay and suboptimal visualization of infrapopliteal arteries [18]. Most team are presently using an hybrid technique combining a first series of consecutive acquisitions centered on the infrapoliteal arteries and feet with a single dose of gadolinium, followed by a stepping acquisition on the proximal arteries with a second contrast injection [19–21]. Finally, the last evolution of time-resolved imaging is based on the hypersampling of the central portion of the K-space to collect the contrast information of the image during the progression of the contrast bolus. The acquisition of the peripheral portion of the K-space which is related to spatial resolution will be acquired preferentially before and/or after contrast enhancement [22]. These techniques enable fast angiographic acquisitions comparable to digital subtraction acquisitions but with a lower spatial resolution. They are also proposed in combination with a standard bolus-chase technique to image infrapopliteal arteries [22].

Rendering techniques for 3D image processing

Post-processing of CTA-MRA data sets is crucial for adequate documentation and communication of anatomy and pathology. Post-processing is more demanding for CTA data, due to the high slice number, the need to remove bone structures and evaluate accurately vessel lumen in calcified vessels. Depending of the CT unit, between 1200 and 1600 axial slices (1–2 mm thickness) will be sent to the PACS. All the basic information (vessel lumen, vessel wall and surrounding structures) are included in this data set. To enable a 3D angiographic visualization of the vascular tree, several post-processing techniques are used. CTA post-processing now include bone and table removal to display only the vascular tree. The maximum intensity pixel projection (MIP) is a 2D angiographic projection of the 3D volume (*figure 3*). X- rays are simulated through the volume of reconstructed sections, and the maximum voxel value along each ray is selected. This voxel value is used in the final image while all other voxels along the ray are regarded as transparent (*figure 2*). This information will display the vessel lumen, and vascular calcification for CTA and only the vascular lumen for MRA since calcification are not visible. Usually several 2D projections separated by 15 to 20 degree intervals are sent to the PACS. In volume rendering technique (VRT), a 3D volume is created from a set of 2D slices, and the density value (a radiological entity) is translated into optical entities such as brightness, opacity, and color [23]. Thus, the vascular system is colored as a semi-transparent material and soft tissue nearly transparent. In contrast to the MIP, all voxels along a ray contribute to the displayed image (*figure 3*). VRT has superior accuracy compared with surface rendering in CT and MR angiography and produces relatively reproducible results with different operators [24]. Multiplanar reformation (MPR), are 2D reconstructions performed in sagittal, coronal or oblique views. By default, the radiologist can scroll the three orthogonal views (coronal, sagittal, axial) interactively on the visualization station. The curved multiplanar reformation is a new technique which is very convenient to evaluate vascular stenosis in CTA examinations. A curved plane is created along the central line of the vessel lumen. Then, the vessel can be rotated around this central line allowing stenosis quantification with the best angle of view (*figure 3*).

Adverse effects on contraindication of CTA and MRA

The effective radiation dose of peripheral CTA for 16-detector row CT angiography is estimated between 1.6–3.9 mSv and is lower than conventional DSA (6.4–16.0 mSv) [25]. Iodine

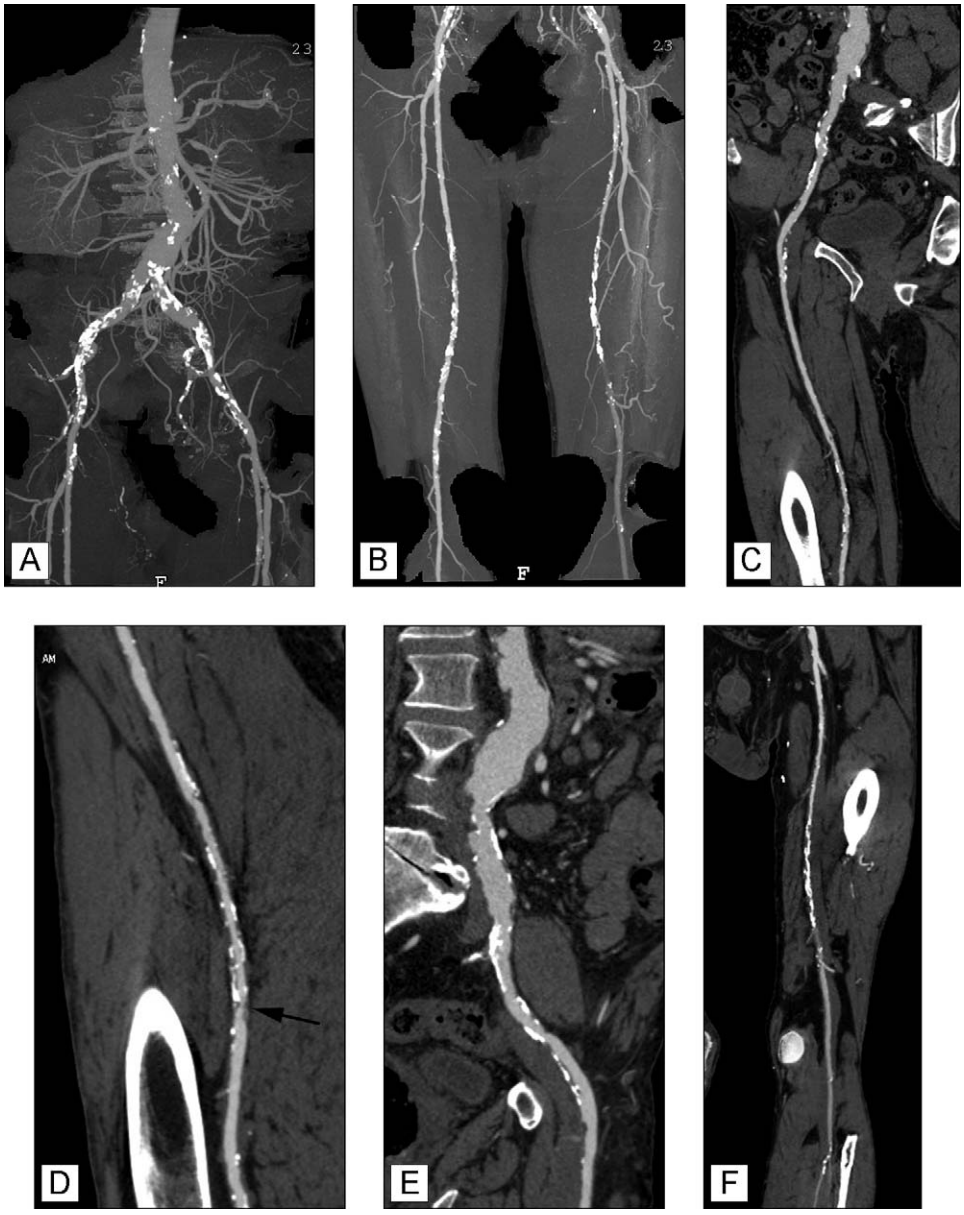

FIGURE 3

CTA examination in a 75-year-old man

a: MIP reformation of the aorta and iliac artery. Presence of extensive calcification impairing lumen patency evaluation; b: MIP reformation of the femoro-popliteal arteries. Extensive calcifications are seen on both side with multiple luminal stenoses on the right side and chronic occlusion on the left side; c, d: curved MPR reformations on the iliac and femoral arteries on the right side showing several moderate stenoses on the iliac artery and one severe stenosis on the distal superficial femoral artery (arrow); e, f: curved MPR reformation on the left side showing a moderate stenosis on the left common iliac and a long chronic occlusion of the mid and distal superficial femoral artery.

contrast induced nephropathy (CIN) is defined as an increase in serum creatinine level of more than 25% or 0.5 mg/dL [26]. Patients with baseline renal insufficiency, especially those with concomitant diabetes mellitus are at higher risk [26]. Nowadays, most centers are using low-osmolar contrast agent that are at lower risk for CIN than high-osmolar contrast agent [27]. Among low-osmolar contrast agents, it has been shown that iodixanol (iso-osmolar) may be less nephrotoxic than iohexol and ioxaglate but no difference has been found between iodixanol and iopamidol, iopromide or ioversol [28]. General screening program to identify patients at high risk for CIN is necessary to revaluate the clinical indication or prepare the patient by a saline hydration. Patient preparation combines hydration by serum saline or sodium bicarbonate combined or not with acetylcystein. Effectiveness of acetylcystein and sodium bicarbonate versus serum saline hydratation alone remains uncertain [29,30]. Allergic reactions to iodine contrast are observed in 5 to 8% most cases being mild. Corticosteroid

TABLE 1

Classification of peripheral arterial disease: Fontaine's stages and Rutherford's categories [36]

Fontaine		Rutherford		
Stage	Clinical	Grade	Category	Clinical
I	Asymptomatic	0	0	Asymptomatic
IIa	Mild claudication	I	1	Mild claudication
IIb	Moderate-severe claudication	I	2	Moderate claudication
		I	3	Severe claudication
III	Ischemic rest pain	II	4	Ischemic rest pain
IV	Ulceration or gangrene	III	5	Minor tissue loss
		IV	6	Ulceration or gangrene

preparation is still recommended in case of previous allergic reaction although its efficacy is still debated [31]. Gadolinium-based contrast agents (GBCA) have long been touted as non-nephrotoxic and MRA was frequently indicated as an alternative to angiography and CTA in patients with preexisting renal failure. Recently, the use of gadolinium in patients with renal impairment has been linked to the development of nephrogenic systemic fibrosis (NSF). This rare disease was mostly associated with two non-ionic linear GBCAs (Omniscan and Optimark), in patients with advanced renal failure having received high-dose of gadolinium [32]. The incidence of NSF in patients with chronic kidney disease (CKD) is low but caution is merited for dialysis patients and those with acute kidney injury, with relative caution for predialysis patients with stage 5 CKD [33]. No new case of NSF was reported in two large institutions over a 6-year period with the use of cyclic or ionic gadolinium agents, screening of high-risk patients and restriction of the use and dose of GBCA in patients with risk factors, [34]. In patients with renal failure, CTA or MRA indication should always be revaluated and the patient informed of the risk of each technique. In our practice, we are still doing MRA in stage 3 and 4 CKD, since in this population the risk of CIN is higher than the risk of NSF. For predialysis and dialysis patients depending on the indication, we will do a CTA if a proximal occlusive or an aneurysmal disease is suspected or a selective catheter angiography if a distal occlusive disease is suspected.

Investigation of occlusive peripheral arterial disease

Peripheral arterial occlusive disease (PAOD) is secondary to the apparition and progression of atherosclerotic plaque in the peripheral circulation leading to vascular stenosis or occlusion. This disease is more prevalent in the femoral and popliteal arteries (80–90% of symptomatic patients), tibial and peroneal arteries (40–50% of symptomatic patients) than in the aorta and iliac artery (30% of symptomatic patients) [35]. CTA or MRA examination are not indicated to make the diagnosis of PAOD which is based on clinical examination, ankle brachial index measurement and if necessary doppler ultrasound examination [3]. Following this clinical and physiological evaluation, patients will be classified according to the Fontaine or Rutherford classification (Table 1). According to these classifications, we will discuss separately patient with intermittent claudication (Fontaine stage II a and IIb and Rutherford category 1,23) and patients with critical limb ischemia (CLI) (Fontaine stage III, IV and Rutherford category 4,5,6) [36].

Intermittent claudication

PAOD patients with an intermittent claudication should be first treated by a control of risk factors (smoking cessation, weight reduction, control of blood pressure, diabetes and dyslipidemia), antiplatelet therapy and exercice program [3]. CTA or MRA examinations are indicated only if an invasive therapy is planned. For patient with intermittent claudication, endovascular treatement is indicated if there is a failure of medical therapy and if the limitation is affecting the quality of life and there is a suspicion of proximal lesion (iliofemoral) [37]. Surgical treatment is sometimes indicated in patients severely incapacited by their claudication. The goal of this examination is to map the distribution of atherosclerosis lesions and identify significant lesions that can be treated by endovascular approach or surgical bypass. The recommendations for endovascular or surgical approach are based on the Transatlantic Intersociety Consensus Classification (TASC), which is based on the localization, number and length of occlusive lesions and the quality of distal run-off [3] (*boxes 1 and 2*). TASC A and B lesions should be preferentially treated by endovascular approach and TASC D by surgical approach. TASC C lesions

Box 1

Transatlantic intersociety consensus classification of aorto-iliac lesions [3]

TASC A
- Unilateral or bilateral stenosis of CIA
- Short stenosis (<3 cm) of unilateral of bilateral EIA

TASC B
- Short stenosis of infrarenal aorta (<3 cm)
- Unilateral CIA occlusion
- Single or multiple stenoses totalling 3–10 cm involving EIA not extending into CFA
- Unilateral EIA occlusion not involving origin of internal iliac or CFA

TASC C
- Bilateral CIA occlusion
- Bilateral EIA stenosis 3–10 cm not extending to CFA
- Unilateral EIA stenosis extending into CFA
- Unilateral EIA occlusion that involves origin of internal iliac or CFA
- Heavily calcified unilateral EIA occlusion without involvement of origin of internal iliac or CFA

TASC D
- Infrarenal aortic occlusion
- Diffuse disease involving aorta and both iliac arteries requiring treatment
- Diffuse multiple stenoses of the CIA, EIA, and CFA
- Unilateral occlusion of CIA and EIA
- Bilateral occlusion of EIA
- Iliac stenoses in patients with AAA requiring treatment and not amenable to endograft placement

CIA: common iliac artery; EIA: external iliac artery; CFA: common femoral artery; AAA: abdominal aortic aneurysm.

Box 2

Transatlantic intersociety consensus classification of femoral-popliteal lesions [3]

TASC A
- Stenosis 10 cm or less in length
- Occlusion 5 cm or less in length

TASC B
- Stenoses or occlusions, multiple, each 5 cm or less
- Stenosis or occlusion, single, 15 cm or less not involving the infrageniculate popliteal artery
- Single or multiple lesions in the absence of continuous tibial vessels to improve inflow for a distal bypass
- Stenosis, single popliteal
- Occlusion, heavily calcified, 5 cm or less in length

TASC C
- Multiple stenoses or occlusions totaling 15 cm with or without heavy calcification
- Stenoses or occlusions, recurrent after 2 endovascular interventions

TASC D
- Chronic total occlusions of common femoral artery (CFA) or superficial femoral artery (>20 cm, involving the popliteal Artery)
- Chronic total occlusion of popliteal artery and proximal trifurcation vessels

CFA: common femoral artery.

are best treated with open revascularization with endovascular methods only used in patients at high risk for surgery. Usually, patients with intermittent claudication are younger and present a less advanced disease than patients with CLI. They are good candidate for CTA because they are less susceptible to present highly calcified vessels which is a major limitation of CTA examinations. In a recent meta-analysis, Met et al. reviewed the performance of CTA in PAOD population with a majority of intermittent claudication [38]. A 95% sensitivity (95% confidence interval [CI], 92–97%) for detecting more than 50% stenosis or occlusion and a 96% specificity (95% CI, 93–97%) were respectively reported [38]. Overstaging occurred in 8% of segments and understaging in 15%. In two studies with also a majority of patients presenting intermittent claudication, it has been shown that CTA provides the same information than MRA at a lower cost [39,40].

Critical limb ischemia

Critical limb ischemia (CLI) is a manifestation of severe peripheral arterial disease with persistently recurring rest pain for more than two weeks, ulceration, or gangrene at the foot, with an ankle systolic pressure less that 50 mmHg [41]. In this stage multilevel arterial disease involvement is common. Approximately 5% of patients with intermittent claudication will progress to CLI over the next five years, and the incidence of CLI is estimated at one new patient per 1000 population per year [42]. The majority of patients with CLI will undergo some form of revascularization procedure, and this can be performed surgically or percutaneously. The BASIL trial compared bypass surgery and angioplasty in patients presenting with severe limb ischaemia due to infrainguinal disease suitable for surgery and angioplasty [43]. When both treatments are possible, endovascular therapy is preferred because of similar clinical outcomes and lower costs [43]. Despite revascularization, more than 50% will eventually die during the five next years [44]. In this population, an adequate visualization of infrapopliteal arteries is important because most patients have a poor run-off and distal revascularization is frequently indicated. There is no randomized trial comparing MRA and CTA in CLI patients.

However, since most patients with CLI are older and diabetic, CTA examination can be suboptimal due to the presence of small and calcified distal vessels [45]. In this population MRA with a biphasic protocol is very robust to document properly the arterial inflow and outflow and plan invasive therapy (*figure 4*). Finally, selective catheter angiography should be considered if MRA is suboptimal or in first intention in stage 5 CKD or dialysis patients [46].

Follow-up of patients with vascular stents

All metallic stents are susceptible to metallic artifacts in MRA. Balloon expandable stainless stents are ferromagnetic and will induce an important artefact impairing lumen visualization. Nitinol (self expandable) and chromium cobalt stents (balloon expandable) will create a shielding effect and a partial loss of signal inside the lumen, however lumen patency can still be evaluated on source images but lumen diameter can be underestimated [47]. Hence, in a patient with a previous history of peripheral stent insertion requiring further investigation, CTA should be preferred over MRA. Curved MPRs are particularly useful to evaluate stent lumen and quantify in-stent stenosis (*figure 5*).

Non-atheromatous peripheral occlusive diseases

Atherosclerosis is responsible for almost all cases of intermittent claudication or CLI, however several rare conditions, often present in younger patients, can lead to peripheral arterial obstructions.

Persistent sciatic artery

The persistent sciatic artery is a congenital anomaly characterized by the lack of regression of the axial limb sciatic artery. The ipsilateral ilio-femoral artery is underdeveloped and the limb supplied by the persisting sciatic artery which is prone to aneurysmal degeneration and thrombosis. CTA is the best examination to determine the presence and laterality of persistent sciatic artery and its associated vascular abnormalities, such as aneurysm, thrombus, distal thromboembolism, atherosclerotic change and its relationship with sciatic nerve, muscle, accompanying vein, and femoral artery [48].

Thromboangititis obliterans: Buerger's disease

This accelerated form of atherosclerosis affects young males (20–40-year-old) from Middle East and Asia. It involves mainly infrapopliteal arteries and lead to advanced CLI and frequent amputation. On CTA or MRA, they are characterized by multiple occlusions of infrapopliteal vessel with typical corkscrew collaterals. Patients with small corkscrew collaterals have a more advanced disease than patients with large corkscrew collaterals [49].

Cystic advential disease

This disease is characterized by the presence of an uni- or multilocular cyst with mucinous or gelationous content in the adventitial layer of the arterial wall and affect young men between 20 and 50-year-old. It is typically located in the popliteal artery and can occasionally communicate with articular cavity of the knee [50]. MRI is the best examination to visualize the cyst in the vessel wall, luminal compression and connections between cysts in the adventitia and the adjacent joint, which is important for successful treatment [50].

Fibromuscular dysplasia

Usually, fibromuscular dysplasia (FMD) is observed in renal or carotid arteries but rarely iliac lesion can be seen. Usually a typical aspect of string of bead is seen [51]. However, arterial dissection leading to rupture have been reported [52]. These FMD lesions can be well seen in CTA and MRA. Since CTA has a better spatial resolution, it is better suited to evaluate medial thickening observed in typical FMD lesions.

Popliteal artery entrapment syndrome (PAES)

Popliteal entrapment results from an aberrant relationship between the medial head of the gastrocnemius muscle (MGHM) and the popliteal artery. The current classification of embryological entrapment (types I to V) includes abnormal development of the MHGM (types I and II); abnormal fibrous, muscular, or tendinous bands usually derived from remnants of the MHGM (type III), and a primitive position of the distal popliteal artery posterior to the popliteus muscle (type IV). Popliteal vein entrapment with any of the above anomalies is referred to as type V [53]. Both CTA and MRI can show anatomic variations in the popliteal fossa and may be valuable in the diagnosis of PAES in young adults presenting with intermittent claudication [54,55]. However, accurate classification of the gastrocnemius medial head and lateral head anomaly is easier on MRI (*figure 6*) [53,56].

Iliac endofibrosis

High performance athletes, predominantly professional cyclists, can develop symptomatic arterial flow restriction in one or both legs during exercise caused by endofibrosis and/or kinking of the external iliac artery. On CTA or MRA wall thickening and luminal narrowing, without aneurysm formation can be seen. Cases of luminal thombosis have been also reported [57,58].

Acute limb ischemia

Acute limb ischemia is defined as any sudden decrease in limb perfusion causing a potential threat to limb viability [3]. The TASC II classification categorize acute limb ischemia in three categories [3]:

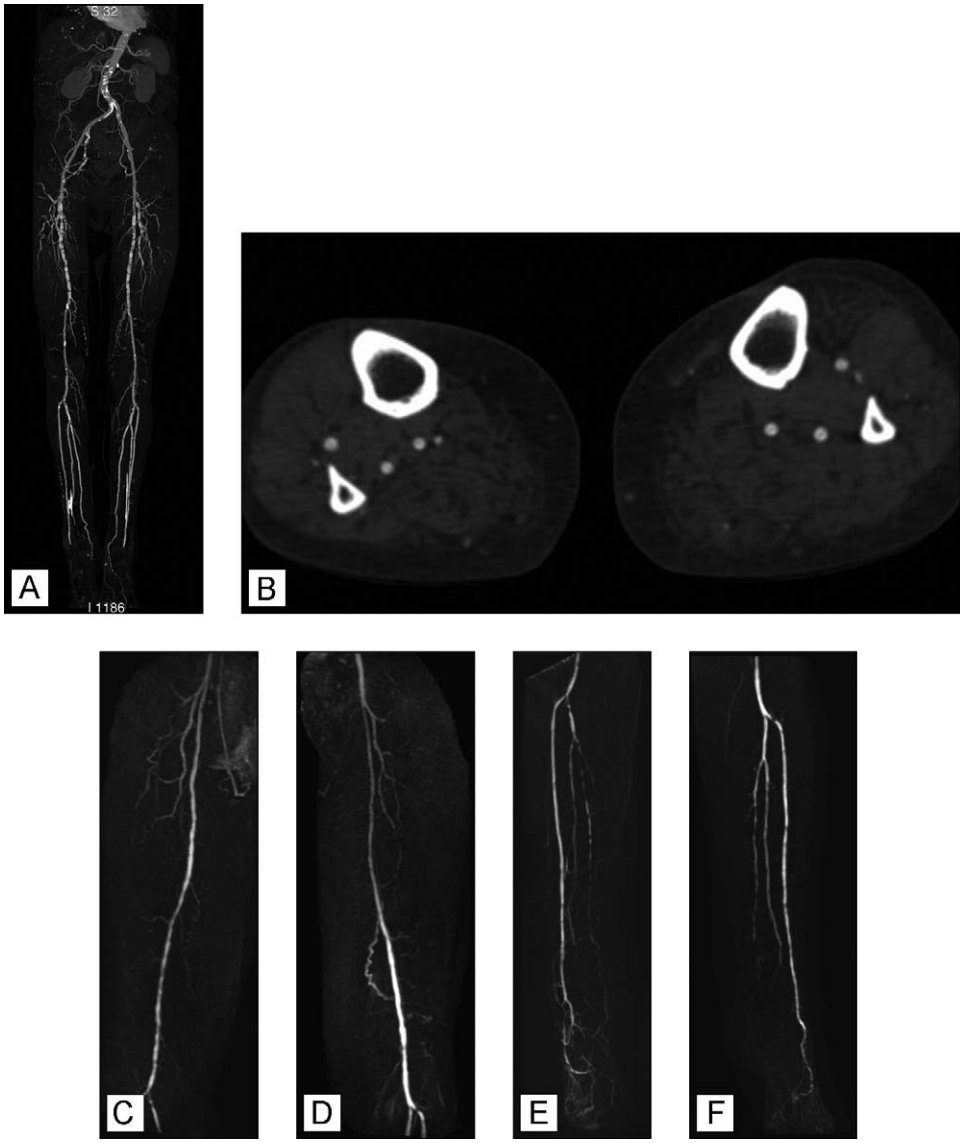

FIGURE 4

A 67-year-old women with diabetes, arterial calcinosis and critical limb ischemia on the left side

a: MIP reformation of a CTA examination showing diffuse vascular calcifications involving femoro-popliteal and infrapopliteal arteries impairing lumen visualization; b: Axial slice at the mid portion of the leg showing circumferential parietal calcification of the three infrapopliteal vessels with almost no visualization of iodine contrast MRA; c, d: MRA examination in the same patient. On the MIP reformation, a moderate infiltration of the distal femoral and proximal popliteal artery is observed on the right side and a diffuse infiltration of the proximal and mid portion of the superficial femoral artery on the left side; e,f: MIP on the infrapopliteal arteries showing patent tibial artery on the right side and a tight stenosis on the proximal portion of the tibial artery on the left side which is the only patent vessel to the foot.

I. viable: limb not immediately threatened;
II. threatened: IIa: marginally: salvageable if promptly treated;
IIb: immediately: salvageable if promptly treated;
III. irreversible.

In marginally threatened legs, the reduction in sensation is minimal whereas in immediately threatened limbs, the reduction is more profound. Loss of motor function indicates an immediately threatened limb. Imaging should not delay revascularization and management is based on the threat to limb survival. In stage I and IIa, there is still time for investigation. In these patients, CTA can be a good option but need to be performed immediately for stage IIa ischemia. If not, a peroperative angiography will be the best option. In stage IIB immediate revascularization is needed. CTA acquisition protocol should include a second delayed acquisition covering the popliteal and infrapopliteal arteries to compensate for distal flow compromise. CTA is also helpful to identify the source of emboli if located in the aorta or peripheral arteries (by example plaque thrombosis, aortic or popliteal aneurysm) (*figure 7*).

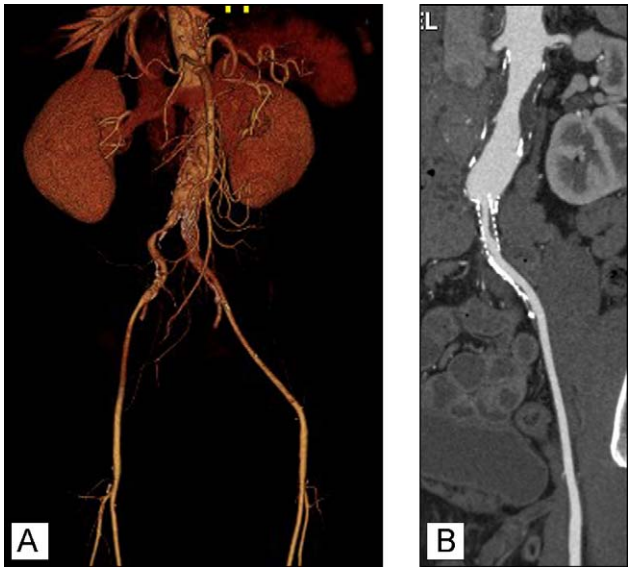

FIGURE 5

CTA acquisition in a patient with a previous history of left iliac stenting and apparition of claudication on the right side

a: VRT reformation showing a stenosis on the right common iliac artery. The left iliac stent is visible but assessment of lumen patency is not possible; b: curved MPR of the left iliac artery showing a moderate stenosis in the stent related to intimal hyperplasia.

Peripheral aneurysms

An aneurysm is defined by a 50% diameter increase of an artery compared to its expected size. A threshold of more than 7 mm for a popliteal aneurysm and 10 mm for a femoral aneurysm is usually recognized [59]. Popliteal artery aneurysms are the most common of peripheral aneurysms and exhibit a strong male predominance. In 40% of the time, they are associated with aortic aneurysms and are bilateral in 50 to 70% of cases. Diameter greater than 2 cm is often stated as being an indication for elective operation in asymptomatic popliteal aneurysms [60]. They are almost exclusively atherosclerotic but can rarely be related to trauma, cystic advential disease, entrapment syndrome or infection. The usual clinical presentation is aneurysm thrombosis and distal embolization, or both with resultant acute limb ischemia. Digital necrosis secondary to microemboli or claudication can be observed in 30 to 45% of patients [61]. Aneurysm rupture is unlikely and lead more often to ischemia from arterial compression by the contained haematoma than exsanguination. Doppler ultrasound is the first imaging modality to make the diagnosis. Both CTA and MRA are helpful to delineate the aneurysm and evaluate thrombosis extension and distal run-off. In emergency, CTA is preferred. A second delayed acquisition covering the popliteal and infrapopliteal arteries is helpful to image properly the distal run-off and compensate for slow flow secondary to aneurysmal disease and popliteal thrombosis (*figure 8*).

Abdominal aortic aneurysm

An abdominal aortic aneurysm (AAA) is defined by a diameter of more than 3 cm. The prevalence of AAAs (5%) is expected to increase with aging of the population [62]. Aortic aneurysm is the 15th leading cause of death in the United States and most of these deaths are due to rupture of AAAs [63]. AAAs enlarge at an average rate of 1 cm every three years until symptoms of rupture develop, usually after the AAA is 6 cm [64,65]. Screening for AAAs with ultrasound of high risk-population (male more than 65-year old, smoking history) has become accepted

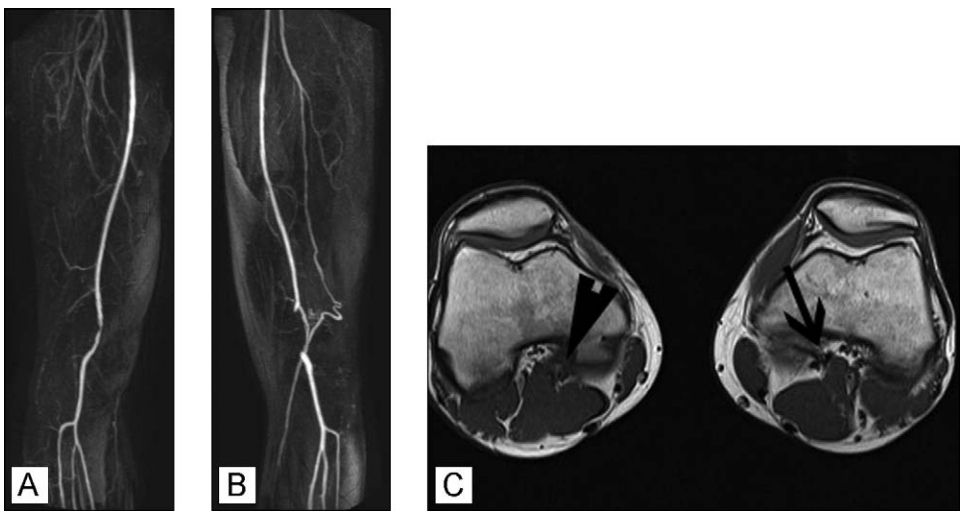

FIGURE 6

MRA examination of a 28-year-old male with an history of bilateral claudication

a,b: MIP of on the right and left femoropopliteal artery showing a stenosis and medial deviation of the right popliteal artery and on the left side an occlusion of the popliteal artery; c: T1 weighted axial acquisition showing the compression of the artery by the medial head of the gastrocnemius (MHGNM) on the right side (arrowhead) and the thrombosed artery on the left side and its relation the MHGNM (arrowhead).

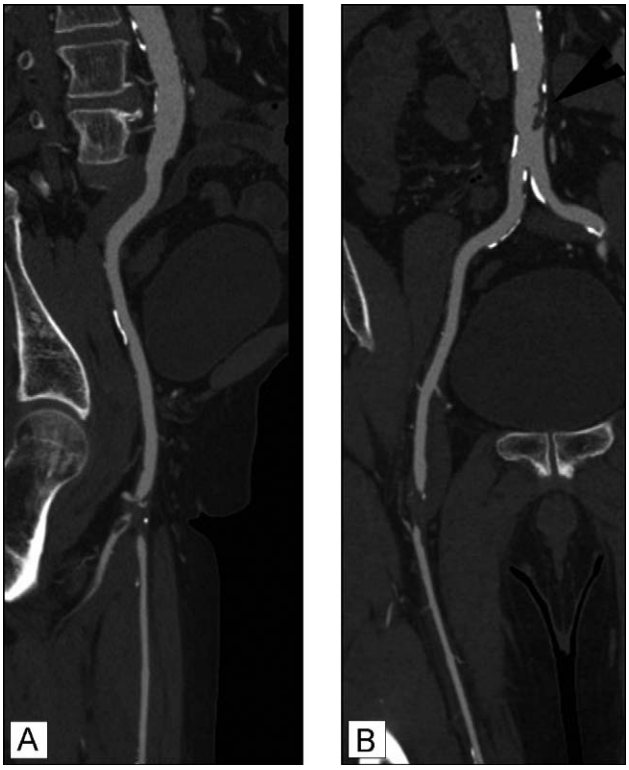

FIGURE 7

CTA examination in a 73-year-old patient with a grade 1 acute ischemia of the right lower limb

a: CPR of the aorta right iliac and common femoral artery showing a thrombus sitting in the femoral bifurcation; b: other angulation of the CPR reformation showing the source of the emboli related to an aortic plaque with a floating thrombus (arrowhead).

practice in the United States [66]. Elective repair by open or endovascular repair (EVAR) is indicated when aneurysm diameter exceeds 5–5.5 cm [64,67]. Open repair can be carried out with relatively low operative mortality (2 to 5%) by experienced surgeons. EVAR with stent-graft (SG) is a promising alternative with reduced rates of immediate mortality/morbidity [68,69]. Not all patients with an AAA are suitable for endovascular repair. CTA will play a pivotal role to evaluate anatomic eligibility for EVAR or plan open repair. First, this examination will confirm in a reproducible way the maximum AAA diameter. This measurement should be taken outer wall to outer wall in lateral and anteroposterior projection. There is still some controversy if measurement should be taken on axial slices or longitudinal reformation perpendicularly to aneurysm central line [70–72]. Then CTA will display the relation ship of AAA with renal, mesenteric and celiac arteries and aneurysm extension in iliac arteries. Several anatomic features will be evaluated on CTA to ensure EVAR can be feasible. The major criteria for anatomic suitability are:

- proximal landing zone (aneurysm neck): the distance between renal arteries and the AAA (proximal neck) should be at least 15 mm. The neck should be less than 32 mm in diameter and present an angulation less than 90°C. Finally, the shape of the neck will be analyzed. A conical neck, the presence of mural thrombus or extensive calcifications can constitute a contraindication for EVAR;
- distal landing zones (iliac arteries): a minimal length of 15 mm is necessary in the common iliac artery to land the distal component of the graft. If there is an iliac aneurysm precluding a landing above the origin of the internal iliac artery, the graft can be extended into the external iliac artery. However, bilateral coverage of internal iliac artery should be avoided because of potential risk of colonic necrosis and hip claudication. The distal aortic lumen should be more than 15 mm in diameter to accommodate both SG limbs. Smaller diameter of distal aorta can lead to SG limb collapse. Hence, an aorto-uniliac design can be preferred;
- delivery device accessibility: a minimal iliac diameter less than 6–7 mm is necessary to accommodate SG delivery system depending of SG brand and diameter. Excessive vascular tortuosity and presence of extensive vascular calcification should be noted, since this can lead to EVAR contraindication or influence the choice of access side to deliver the main body of the stent-graft or the contralateral limb. Finally, once the feasibility is assumed, curved and stretch MPRs will be created along the lumen central line to enable the sizing of the stent-graft according to patient anatomy.

EVAR follow-up

The main limitation of EVAR is the durability of aneurysm exclusion and the occurrence of endoleaks [73]. Endoleaks are defined by the persistence of blood flow perfusing the aneurysm and are observed in 10 to 36% of cases (median 25%) [74–80]. In the same series, 6 to 20% of EVAR patients eventually require a reintervention most being endovascular [74–80]. Since endoleak can lead to aortic rupture, a life-long surveillance is required. CTA is the best examination to detect and classify endoleaks. They have been classified into five types [81,82]. Type I leaks are related to perigraft flow around the proximal (type IA) or distal end (type IB) of the graft. Type II leaks are associated with retrograde flow through collateral branches within the aneurysmal sac, such as the lumbar or inferior mesenteric arteries (*figure 9*). Type III and IV leaks are related to device failure. Type III leaks are characterized by persistent blood flow secondary to graft dislocation or fabric tears, whereas type IV leaks are due to graft porosity. Type V "leaks" (endotension) are a continued growth of the aneurysmal sac visualized on imaging studies without evidence of endoleaks [78,82]. Type I and III endoleaks are clearly related to

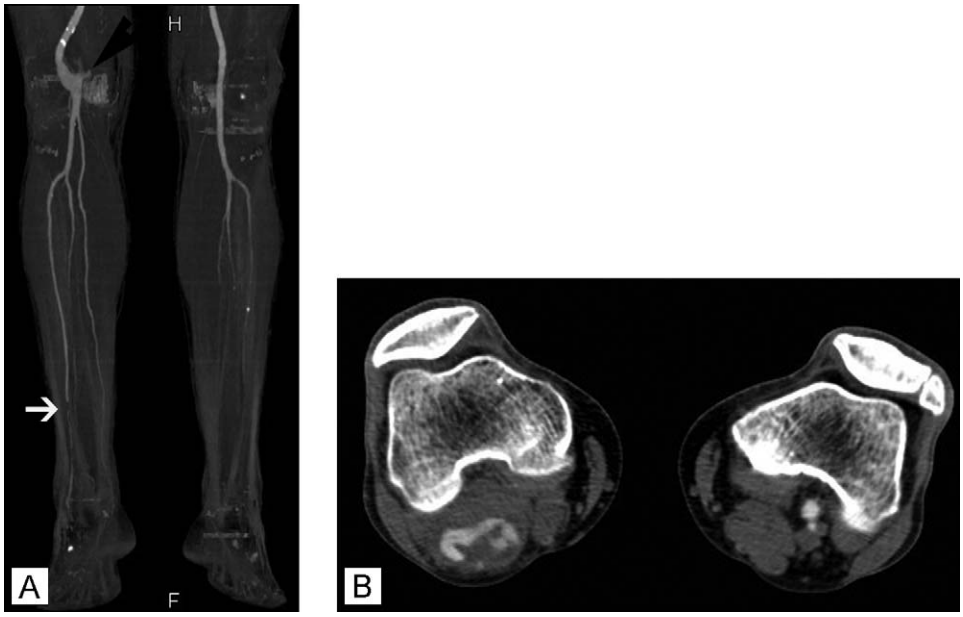

FIGURE 8

CTA examination in patient with suspicion of popliteal aneurysm

a: MIP of the popliteal and infrapopliteal region showing the lumen of the popliteal aneurysm (arrowhead) and an embolic occlusion of the distal anterior tibial artery (arrow); b: The lumen and the thrombosed portion of the popliteal aneurysm are well seen on the axial image.

aneurysmal rupture and require immediate treatment [83]. Type II endoleaks with AAA expansion can also lead to aortic rupture and require embolization or surgical conversion [75,84]. Usually, a CTA is performed three month after EVAR and every year thereafter if no endoleak is detected. This surveillance increases the cost and exposes the patient to the hazard of ionizing radiation and iodine contrast nephrotoxicity [85,86]. To minimize contrast injection, only one acquisition during the arterial phase is recommended [87]. However, acquisition in venous phase can be more sensitive to detect slow type II endoleaks [88]. Other authors have proposed aneurism sac volume measurement on an unenhanced study and complete by contrast study only if aneurism sac volume progression is observed [89]. MRA is sometimes indicated to follow patients with renal failure or severe allergy to iodine contrast. However, only patient having nitinol SG implanted can be imaged properly whereas stainless steel SG induces too much ferromagnetic artifacts to detect endoleaks.

Inflammatory aortic disease

Inflammatory abdominal aortic aneurysm (AAA) represents 5 to 10% of all AAA cases. These aneurisms are painful and soft tissue infiltration surrounding the aneurysm due to adventitial thickening can be seen on MRA and CTA (*figure 10*). Medical therapy combines smoking cessation with corticosteroids or immunosuppressive therapies. Surgical or endovascular repair is indicated for painful aneurysms and when diameter exceeds 5.5 cm as for atherosclerotic aneurysms [90]. Takayasu arteritis involves the abdominal less-frequently than thoracic aorta. The diagnosis can be made when a stenosis involving the proximal abdominal aorta, visceral trunk and proximal iliac artery is observed in a young to middle-aged female patient without evidence of atherosclerosis. In the acute phase, a thickening of the aortic wall with an enhancement after contrast injection can be observed on CTA and MRA [91]. In the chronic phase, luminal stenosis and calcifications are seen and mural thickening is less pronounced.

Giant cell arteritis usually affects predominantly young women. Characteristic angiographic features include symmetric bilateral stenoses and post-stenotic aneurysmal dilatation associated with profuse collateral arterial blood supply. Evans et al. found that patients with giant cell arteritis were 17.3 and 2.4 times more likely to develop thoracic and abdominal aneurysms, respectively, when compared with the general population [92]. Such aneurysms can be either focal or diffuse along the thoraco-abdominal aorta and are often associated with visceral and renal artery occlusive disease.

Mycotic aneurysm are aneurysms secondary to embolization from an infected cardiac vegetation or hematogenous seeding of atherosclerotic plaque. A focal aortitis with dissolution of the aortic wall and a false aneurysm will develop. Imaging features of infected aneurysms on CTA and MRA include a lobulated vascular mass, an indistinct irregular arterial wall, perianeurysmal edema, and a peri-aneurysmal soft-tissue mass [93].

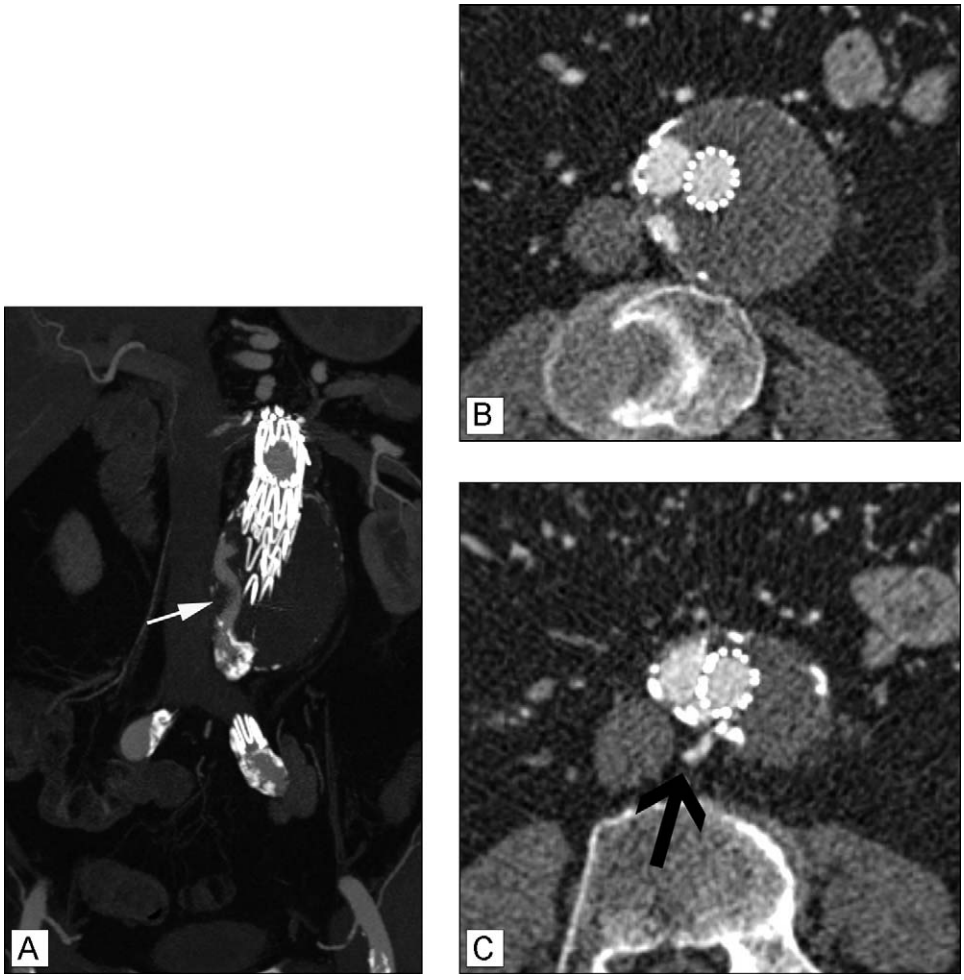

FIGURE 9

Follow-up CTA examination in a patient 3 months after EVAR

a: coronal MPR showing contrast extravasation in the right portion of the aneurysm sac (arrow); b, c: axial image showing the endoleak and its connection with the right lumbar artery (arrow) suggesting a type II endoleak.

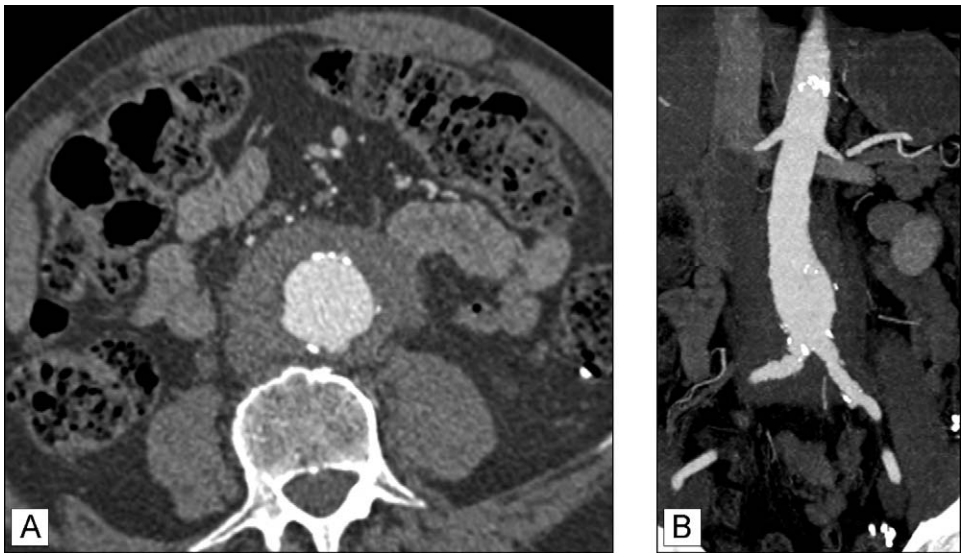

FIGURE 10

CTA examination in a 58-year-old patient with a painful AAA

a: axial image showing the aneurysm and a circumferential thickening of the adventitia; b: coronal reformation showing the extension of the AAA and adventitial inflammation indicating an inflammatory aortic aneurysm.

Conclusion

CTA and MRA are now playing a pivotal role in the management of peripheral occlusive atherosclerotic disease and peripheral aneurysms. These imaging modalities should be requested in symptomatic patients (occlusive disease) or patients with a therapeutic indication (aneurysm disease) who require a therapeutic planning. Clinical examination and Doppler ultrasound remain the first line investigation to make the diagnosis of PAOD and aneurysm. CTA and MRA have almost replaced diagnostic angiography and enable appropriate therapeutic planning for a majority of patients.

Disclosure of interest: Gilles Soulez receives research grant from Siemens Medical and Bracco Diagnostic.
GS is supported by a clinical research scholarship from Fonds de la recherche en santé du Québec (FRSQ). No other potential conflict of interest for other co-authors.

References

[1] Hirsch AT, Criqui MH, Treat-Jacobson D *et al.* Peripheral arterial disease detection, awareness, and treatment in primary care. JAMA 2001;286:1317-24.
[2] Weitz JI, Byrne J, Clagett GP *et al.* Diagnosis and treatment of chronic arterial insufficiency of the lower extremities: a critical review. Circulation 1996;94:3026-49.
[3] Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG. Inter-society consensus for the management of peripheral arterial disease (TASC II). J Vasc Surg 2007;45:S5-67.
[4] Melton LI, Bickerstaff L, Hollier L. Changing incidence of abdominal aortic aneurysms: a population-based study. Am J Epidemiol 1984;120:379-86.
[5] Newman AB *et al.* Cardiovascular disease and mortality in older adults with small abdominal aortic aneurysms detected by ultrasonography: the cardiovascular health study. Ann Intern Med 2001;134:182-90.
[6] Foley WD, Stonely T. CT angiography of the lower extremities. Radiol Clin North Am 2010;48:367-96 ([ix]).
[7] Saini S. Multi-detector row CT: principles and practice for abdominal applications. Radiology 2004;233:323-7.
[8] Schoellnast H, Tillich M, Deutschmann HA *et al.* Abdominal multidetector row computed tomography: reduction of cost and contrast material dose using saline flush. J Comput Assist Tomogr 2003;27:847-53.
[9] Fleischmann D, Rubin GD. Quantification of intravenously administered contrast medium transit through the peripheral arteries: implications for CT angiography. Radiology 2005;236:1076-82.
[10] Siriapisith T, Wasinrat J, Mutirangura P, Ruangsetakit C, Wongwanit C. Optimization of the table speed of lower extremity CT angiography protocols in different patient age groups. J Cardiovasc Comput Tomogr 2010;4:173-83.
[11] Albrecht T, Meyer BC. MDCT angiography of peripheral arteries: technical considerations and impact on patient management. Eur Radiol 2007;17(Suppl 6):F5-15.
[12] Meyer BC, Oldenburg A, Frericks BB *et al.* Quantitative and qualitative evaluation of the influence of different table feeds on visualization of peripheral arteries in CT angiography of aortoiliac and lower extremity arteries. Eur Radiol 2008;18:1546-55.
[13] Wintersperger B, Jakobs T, Herzog P *et al.* Aorto-iliac multidetector-row CT angiography with low kV settings: improved vessel enhancement and simultaneous reduction of radiation dose. Eur Radiol 2005;15:334-41.
[14] Lim RP, Hecht EM, Xu J *et al.* 3D non-gadolinium-enhanced ECG-gated MRA of the distal lower extremities: preliminary clinical experience. J Magn Reson Imaging 2008;28:181-9.
[15] Cukur T, Lee JH, Bangerter NK, Hargreaves BA, Nishimura DG. Non-contrast-enhanced flow-independent peripheral MR angiography with balanced SSFP. Magn Reson Med 2009;61:1533-9.
[16] Leiner T. Magnetic resonance angiography of abdominal and lower extremity vasculature. Top Magn Reson Imaging 2005;16:21-66.
[17] Tang A, Cloutier G, Therasse E *et al.* Optimization of spatial resolution for peripheral magnetic resonance angiography. Acad Radiol 2007;14:54-61.
[18] von Kalle T, Gerlach A, Hatopp A, Klinger S, Prodehl P, Arlart IP. [Contrast-enhanced MR angiography (CEMRA) in peripheral arterial occlusive disease (PAOD): conventional moving table technique versus hybrid technique]. Rofo 2004;176:62-9.
[19] Tongdee R, Narra VR, McNeal G *et al.* Hybrid peripheral 3D contrast-enhanced MR angiography of calf and foot vasculature. AJR Am J Roentgenol 2006;186:1746-53.
[20] Schmitt R, Coblenz G, Cherevatyy O *et al.* Comprehensive MR angiography of the lower limbs: a hybrid dual-bolus approach including the pedal arteries. Eur Radiol 2005;15:2513-24.
[21] Meissner OA, Rieger J, Weber C *et al.* Critical limb ischemia: hybrid MR angiography compared with DSA. Radiology 2005;235:308-18.
[22] Andreisek G, Pfammatter T, Goepfert K *et al.* Peripheral arteries in diabetic patients: standard bolus-chase and time-resolved MR angiography. Radiology 2007;242:610-20.
[23] Johnson PT, Heath DG, Kuszyk BS, Fishman EK. CT angiography with volume rendering: advantages and applications in splanchnic vascular imaging. Radiology 1996;200:564-8.
[24] Addis KA, Hopper KD, Iyriboz TA *et al.* CT angiography: in vitro comparison of five reconstruction methods. AJR Am J Roentgenol 2001;177:1171-6.
[25] Willmann JK, Baumert B, Schertler T *et al.* Aortoiliac and lower extremity arteries assessed with 16-detector row CT angiography: prospective comparison with digital subtraction angiography. Radiology 2005;236:1083-93.
[26] Josephs SC, Rowley HA, Rubin GD. Atherosclerotic Peripheral Vascular Disease Symposium II: vascular magnetic resonance and computed tomographic imaging. Circulation 2008;118:2837-44.
[27] Rudnick MR, Goldfarb S, Wexler L *et al.* Nephrotoxicity of ionic and nonionic contrast media in 1196 patients: a randomized trial. The Iohexol Cooperative Study. Kidney Int 1995;47:254-61.
[28] Reed M, Meier P, Tamhane UU, Welch KB, Moscucci M, Gurm HS. The relative renal safety of iodixanol compared with low-osmolar contrast media: a meta-analysis of randomized controlled trials. JACC Cardiovasc Interv 2009;2:645-54.
[29] Ferrario F, Barone MT, Landoni G *et al.* Acetylcysteine and non-ionic isosmolar contrast-induced nephropathy – a randomized controlled study. Nephrol Dial Transplant 2009;24:3103-7.
[30] Trivedi H, Daram S, Szabo A, Bartorelli AL, Marenzi G. High-dose N-acetylcysteine for the prevention of contrast-induced nephropathy. Am J Med 2009;122(874):e879-915.
[31] Tramer MR, von Elm E, Loubeyre P, Hauser C. Pharmacological prevention of serious anaphylactic reactions due to iodinated

contrast media: systematic review. BMJ 2006;333:675.

[32] Weinreb JC, Abu-Alfa AK. Gadolinium-based contrast agents and nephrogenic systemic fibrosis: why did it happen and what have we learned? J Magn Reson Imaging 2009;30: 1236-9.

[33] Chrysochou C, Power A, Shurrab AE *et al.* Low risk for nephrogenic systemic fibrosis in nondialysis patients who have chronic kidney disease and are investigated with gadolinium-enhanced magnetic resonance imaging. Clin J Am Soc Nephrol 2010;5: 484-9.

[34] Altun E, Martin DR, Wertman R, Lugo-Somolinos A, Fuller ER.3rd, Semelka RC. Nephrogenic systemic fibrosis: change in incidence following a switch in gadolinium agents and adoption of a gadolinium policy – report from two U.S. universities. Radiology 2009;253:689-96.

[35] Meru AV, Mittra S, Thyagarajan B, Chugh A. Intermittent claudication: an overview. Atherosclerosis 2006;187:221-37.

[36] Hirsch AT, Haskal ZJ, Hertzer NR *et al.* ACC/AHA 2005 Practice Guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease): endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. Circulation 2006;113:e463-654.

[37] Hiatt WR. Medical treatment of peripheral arterial disease and claudication. N Engl J Med 2001;344:1608-21.

[38] Met R, Bipat S, Legemate DA, Reekers JA, Koelemay MJ. Diagnostic performance of computed tomography angiography in peripheral arterial disease: a systematic review and meta-analysis. JAMA 2009;301:415-24.

[39] Ouwendijk R, de Vries M, Pattynama PM *et al.* Imaging peripheral arterial disease: a randomized controlled trial comparing contrast-enhanced MR angiography and multidetector row CT angiography. Radiology 2005;236:1094-103.

[40] Ouwendijk R, de Vries M, Stijnen T *et al.* Multicenter randomized controlled trial of the costs and effects of noninvasive diagnostic imaging in patients with peripheral arterial disease: the DIPAD trial. AJR Am J Roentgenol 2008;190:1349-57.

[41] Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). J Vasc Surg 2007;45(SupplS):S5-67.

[42] Second European Consensus Document on chronic critical leg ischemia. Circulation 1991; 84:IV1-26

[43] Adam DJ, Beard JD, Cleveland T *et al.* Bypass versus angioplasty in severe ischaemia of the leg (BASIL): multicentre, randomised controlled trial. Lancet 2005;366:1925-34.

[44] Bradbury AW, Adam DJ, Bell J *et al.* on behalf of the BASIL trial. Bypass versus Angioplasty in Severe Ischaemia of the Leg (BASIL) trial: An intention-to-treat analysis of amputation-free and overall survival in patients randomized to a bypass surgery-first or a balloon angioplasty-first revascularization strategy. J Vasc Surg 2010;51:5S-17S (Participants, Birmingham and Edinburgh, United Kingdom).

[45] Meyer BC, Werncke T, Foert E *et al.* Do the cardiovascular risk profile and the degree of arterial wall calcification influence the performance of MDCT angiography of lower extremity arteries? Eur Radiol 2010;20:497-505.

[46] Pomposelli, Pomposelli F. Arterial imaging in patients with lower extremity ischemia and diabetes mellitus. J Vasc Surg 2010;52:81S-91S.

[47] Letourneau-Guillon L, Soulez G, Beaudoin G *et al.* CT and MR imaging of nitinol stents with radiopaque distal markers. J Vasc Interv Radiol 2004;15:615-24.

[48] Jung AY, Lee W, Chung JW *et al.* Role of computed tomographic angiography in the detection and comprehensive evaluation of persistent sciatic artery. J Vasc Surg 2005;42:678-83.

[49] Fujii Y, Soga J, Nakamura S *et al.* Classification of corkscrew collaterals in thromboangiitis obliterans (Buerger's disease): relationship between corkscrew type and prevalence of ischemic ulcers. Circ J 2010;74:1684-8.

[50] Maged IM, Turba UC, Housseini AM, Kern JA, Kron IL, Hagspiel KD. High spatial resolution magnetic resonance imaging of cystic adventitial disease of the popliteal artery. J Vasc Surg 2010;51:471-4.

[51] Thevenet A, Latil JL, Albat B. Fibromuscular disease of the external iliac artery. Ann Vasc Surg 1992;6:199-204.

[52] Honjo O, Yamada Y, Kuroko Y, Kushida Y, Une D, Hioki K. Spontaneous dissection and rupture of common iliac artery in a patient with fibromuscular dysplasia: a case report and review of the literature on iliac artery dissections secondary to fibromuscular dysplasia. J Vasc Surg 2004;40:1032-6.

[53] Pillai J. A current interpretation of popliteal vascular entrapment. J Vasc Surg 2008;48:61S-5S ([discussion 65S]).

[54] Hai Z, Guangrui S, Yuan Z *et al.* CT angiography and MRI in patients with popliteal artery entrapment syndrome. AJR Am J Roentgenol 2008;191:1760-6.

[55] Housseini AM, Maged IM, Abdel-Gawad EA, Hagspiel KD. Popliteal artery entrapment syndrome. J Vasc Surg 2009;49:1056.

[56] Kim HK, Shin MJ, Kim SM, Lee SH, Hong HJ. Popliteal artery entrapment syndrome: morphological classification utilizing MR imaging. Skeletal Radiol 2006;35:648-58.

[57] Kral CA, Han DC, Edwards WD, Spittell PC, Tazelaar HD, Cherry KJJr. Obstructive external iliac arteriopathy in avid bicyclists: new and variable histopathologic features in four women. J Vasc Surg 2002;36:565-70.

[58] Vink A, Bender MH, Schep G *et al.* Histopathological comparison between endofibrosis of the high-performance cyclist and atherosclerosis in the external iliac artery. J Vasc Surg 2008;48:1458-63.

[59] Diwan A, Sarkar R, Stanley JC, Zelenock GB, Wakefield TW. Incidence of femoral and popliteal artery aneurysms in patients with abdominal aortic aneurysms. J Vasc Surg 2000;31:863-9.

[60] Galland RB. Popliteal aneurysms: from John Hunter to the 21st century. Ann R Coll Surg Engl 2007;89:466-71.

[61] Bouhoutsos J, Martin P. Popliteal aneurysm: a review of 116 cases. Br J Surg 1974;61:469-75.

[62] Melton LJ3rd, Bickerstaff LK, Hollier LH *et al.* Changing incidence of abdominal aortic aneurysms: a population-based study. Am J Epidemiol 1984;120:379-86.

[63] Hoyert DL, Arias E, Smith BL, Murphy SL, Kochanek KD. Deaths: final data for 1999. Natl Vital Stat Rep 2001;49:1-113.

[64] The U.K. small aneurysm trial participants. Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. Lancet 1998;352:1649-55.

[65] Lederle FA, Wilson SE, Johnson GR *et al.* Immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002;346:1437-44.

[66] Fleming C, Whitlock EP, Beil TL, Lederle FA. Screening for abdominal aortic aneurysm: a best-evidence systematic review for the U.S. Preventive Services Task Force. Ann Intern Med 2005;142:203-11.

[67] Rutherford RB. Management of abdominal aortic aneurysms: which risk factors play a role in decision-making? Semin Vasc Surg 2008;21:124-31.

[68] Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. Lancet 2004;364:843-8.

[69] Prinssen M, Verhoeven EL, Buth J *et al.* A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med 2004;351: 1607-18.
[70] Dillavou ED, Buck DG, Muluk SC, Makaroun MS. Two-dimensional versus three-dimensional CT scan for aortic measurement. J Endovasc Ther 2003;10:531-8.
[71] Abada HT, Sapoval MR, Paul JF, de Maertelaer V, Mousseaux E, Gaux JC. Aneurysmal sizing after endovascular repair in patients with abdominal aortic aneurysm: interobserver variability of various measurement protocols and its clinical relevance. Eur Radiol 2003;13:2699-704.
[72] Wever JJ, Blankensteijn JD, van Rijn JC, Broeders IA, Eikelboom BC, Mali WP. Inter- and intraobserver variability of CT measurements obtained after endovascular repair of abdominal aortic aneurysms. AJR Am J Roentgenol 2000;175:1279-82.
[73] Buth J, Laheij RJ. Early complications and endoleaks after endovascular abdominal aortic aneurysm repair: report of a multicenter study. J Vasc Surg 2000;31: 134-46.
[74] Hiramoto JS, Reilly LM, Schneider DB, Sivamurthy N, Rapp JH, Chuter TA. Long-term outcome and reintervention after endovascular abdominal aortic aneurysm repair using the Zenith stent graft. J Vasc Surg 2007;45:461-5 ([discussion 465–466]).
[75] Gelfand DV, White GH, Wilson SE. Clinical significance of type II endoleak after endovascular repair of abdominal aortic aneurysm. Ann Vasc Surg 2006;20:69-74.
[76] Leurs LJ, Buth J, Laheij RJ. Long-term results of endovascular abdominal aortic aneurysm treatment with the first generation of commercially available stent grafts. Arch Surg 2007;142:33-41 ([discussion 42]).
[77] Steingruber IE, Neuhauser B, Seiler R *et al.* Technical and clinical success of infrarenal endovascular abdominal aortic aneurysm repair: a 10-year single-center experience. Eur J Radiol 2006;59:384-92.
[78] Veith FJ, Baum RA, Ohki T *et al.* Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. J Vasc Surg 2002;35:1029-35.
[79] Brewster DC, Jones JE, Chung TK *et al.* Long-term outcomes after endovascular abdominal aortic aneurysm repair: the first decade. Ann Surg 2006;244:426-38.
[80] Torsello G, Osada N, Florek HJ *et al.* Long-term outcome after Talent endograft implantation for aneurysms of the abdominal aorta: a multicenter retrospective study. J Vasc Surg 2006;43:277-84 ([discussion 284]).
[81] White GH, Yu W, May J. Endoleak – a proposed new terminology to describe incomplete aneurysm exclusion by an endoluminal graft. J Endovasc Surg 1996;3:124-5.
[82] Gilling-Smith G, Brennan J, Harris P, Bakran A, Gould D, McWilliams R. Endotension after endovascular aneurysm repair: definition, classification, and strategies for surveillance and intervention. J Endovasc Surg 1999;6:305-7.
[83] Fransen GA, Vallabhaneni SRSr, van Marrewijk CJ, Laheij RJ, Harris PL, Buth J. Rupture of infra-renal aortic aneurysm after endovascular repair: a series from EUROSTAR registry. Eur J Vasc Endovasc Surg 2003; 26:487-93.
[84] van Marrewijk C, Buth J, Harris PL, Norgren L, Nevelsteen A, Wyatt MG. Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: the EUROSTAR experience. J Vasc Surg 2002;35:461-73.
[85] Jones C, Badger SA, Boyd CS, Soong CV. The impact of radiation dose exposure during endovascular aneurysm repair on patient safety. J Vasc Surg 2010;52:298-302.
[86] Brown LC, Brown EA, Greenhalgh RM, Powell JT, Thompson SG. Renal function and abdominal aortic aneurysm (AAA): the impact of different management strategies on long-term renal function in the UK EndoVascular Aneurysm Repair (EVAR) Trials. Ann Surg 2010;251:966-75.
[87] Macari M, Chandarana H, Schmidt B, Lee J, Lamparello P, Babb J. Abdominal aortic aneurysm: can the arterial phase at CT evaluation after endovascular repair be eliminated to reduce radiation dose? Radiology 2006;241:908-14.
[88] Iezzi R, Cotroneo AR, Filippone A, *et al.* Multidetector CT. in abdominal aortic aneurysm treated with endovascular repair: are unenhanced and delayed phase enhanced images effective for endoleak detection? Radiology 2006;241:915-21.
[89] Bley TA, Chase PJ, Reeder SB *et al.* Endovascular abdominal aortic aneurysm repair: non-enhanced volumetric CT for follow-up. Radiology 2009.
[90] Hellmann DB, Grand DJ, Freischlag JA. Inflammatory abdominal aortic aneurysm. JAMA 2007;297:395-400.
[91] Charrada-Ben Farhat L, Miaoui A, Askri A *et al.* [Imaging features of Takayasu's arteritis]. J Radiol 2009;90:465-8.
[92] Evans JM, O'Fallon WM, Hunder GG. Increased incidence of aortic aneurysm and dissection in giant cell (temporal) arteritis. A population-based study. Ann Intern Med 1995;122:502-7.
[93] Lee WK, Mossop PJ, Little AF *et al.* Infected (mycotic) aneurysms: spectrum of imaging appearances and management. Radiographics 2008;28:1853-68.