Are EVAR complications always an emergency? A practical MDCT approach.

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Learning objectives

To provide the radiologist with a practical approach in order to differentiate urgent EVAR complications from deferrable complications.
Background

AAA is a focal dilatation of the abdominal aorta, exceeding the normal diameter of the vessel with 50% or larger than 3 cm of the vessel’s maximum diameter. This pathological condition affects 2-8% of men and 1-2% of women and is therefore to be recognized as highly relevant (1-2).

The main and most fatal complication related to AAA is rupture of the aorta. The mortality rate is high both in case of absence of surgery (85-90%) as well as when urgent surgery is performed (50-70%). Therefore, the target is to perform the programmed intervention before rupture occurs (1-2).

Although in the early 90’ introduced merely as alternative for the high-risk patient, Endovascular Aneurysm Repair (EVAR) has become the gold standard in today’s AA pathology. EVAR of the abdominal aorta involves the placement of a stent graft within the abdominal aortic lumen/the aneurysm. Separating the aneurismal sac from the aortic blood flow, the endograft creates an alternative conduit for the blood flow and reduces the pressure on the vascular walls, preventing the aneurysm’s enlargement and possible rupture.

Comparison of endovascular and open procedures has shown that EVAR results in less periprocedural complications and a lower mortality rate; nonetheless EVAR doesn't exclude local perivascular complications and often requires secondary treatment during the follow-up phase (3,4,5). Nowadays the role of the radiologist is not limited to identification of complications in general. Additionally, the radiologist has to confront correlated problems in order to estimate the emergency of the situation and evaluate whether or not immediate response is necessary.

Therefore, it is crucial for radiologists to distinguish between on one hand complications that expose the patient to a major risk of death and on the other hand complications which are not so dangerous and can therefore at least temporarily be managed using more conservatively.

Patients with EVAR complications arriving at First Aid, suffering clinical symptoms unequivocally related to possible aortic rupture, should accordingly be considered urgent.
Findings and procedure details

Computed Tomography Angiography (CTA) is the gold standard in the preoperative assessment and in the follow-up of AAA.

According to our clinical experience we recommend the acquisition of an unenhanced phase, followed by an arterial phase performed with the aid of "bolus tracking" technique. The venous/delayed phase is to be performed in all cases before EVAR procedure and during the first follow-up; subsequently only in few selected cases.

To simplify the discussion we decided to divide the post-EVAR complications into two main categories: endoleak complications and non endoleak complications.

This division makes it easier to distinguish between urgent and deferrable complications.

Endoleak Complications

Endoleak is a common complication of endovascular aortic repair (EVAR). It is defined as a persistent blood flow outside the endograft but within the aneurysm sac. It is the most frequent complication of EVAR. Four different types of endoleak are known (6,7):

- Type I

Type I endoleak occurs when there is a leak of blood at the attachment site of an endograft due to inadequate seal at the graft ends. Type I endoleak is subdivided in Type IA, IB, and IC depending on the occurrence at proximal and distal end of the endograft, or iliac occluder, respectively.

- Type II

Type II occurs when there is retrograde flow into the aneurysm sac from the excluded aortic branch; the most frequent sources of endoleak type II are patent lumbar arteries and inferior mesenteric artery.

They are further differentiated into Type IIA when they are related to only one patent branch and Type IIB when they are complex with two or more patent branches and creating a flow-through situation.

- Type III occurs when there is a structural failure of the endograft.
This includes modular disconnection (IIIA) or fabric disruption with endograft fractures or holes (III B)

- Type IV is caused by porosity of the prosthesis; there is passage of blood through the fabric of the graft immediately after placement of the endoprosthesis with the patient being fully anticoagulated.

- Until recently the phenomenon of endotension was considered as a Type V endoleak, whereas modern radiologists do no longer consider this complication as endoleak complication. Endotension occurs when there is an enlargement of an aneurysm sac without an identifiable Type I-IV endoleak on imaging.

The risk that an endoleak evolves into an aneurysmal rupture is directly related to the flow and pressure of the endoleak. In case the endoleak flow and pressure are equal to the aortic flow and pressure, thus exposing the diseased aneurysmal wall to a mechanical trauma comparable to the one before treatment, the risk of aneurysmal rupture is higher. Therefore, in case of endoleak Type I (A and B) and Type III (prosthesis disconnection or rupture) an urgent treatment is generally required. In such endoleak the diagnosis is obtained during the arterial phase with evidence of a synchronous and isodense opacification of the excluded lumen compared to the lumen of the aorta. As a consequence, because their opacifications are synchronous, their flows and pressure regimes have the same features. (Fig.1,2,3)

In case of Type II endoleak the decision on the follow up procedure is less obvious. The pressure regime and blood flow entering the excluded lumen are not comparable with the aortic ones, since the blood flow and pressure regime in the aortic lumen are much higher. This does not mean, however, that a Type II endoleak cannot evolve into an aneurysmal rupture.

Therefore, it is crucial to distinguish between Type II A (Fig. 4,5,6) and Type II B (Fig.7):

- Type II A is characterized by only one patent branch, with low pressure and flow, which has a tendency to self-limitation, especially after reduction of antiplatelet therapy.

- Type II B is characterized by an afferent vessel and one or more efferent vessels, creating a flow-through situation, with as consequence a rare physiological regression.

The decision whether to intervene or not doesn't depend on the subcategory of Type II endoleak (A or B) but is related to the increase in size of the aneurysm. In case of first
evidence of a Type II endoleak the strategy is conservative, unless there is an increase in size of the aneurysm compared to preoperative dimension.

In case of evidence of increased dimensions of the aneurysm and/or endoleak at any point of the follow up, treatment is necessary.

It is important to remember that in case of Type II endoleak the venous phase is the most important phase and it must always be performed because especially in the endoleak from mesenteric vessels, the full opacification of the sac only takes place in the venous phase.

In case of Type IV endoleak, which are extremely rare, the pathophysiological mechanism is self-limiting. Type IV endoleak therefore don't require additional treatment and resolve spontaneously when the antiplatelet therapy is finished.

In case of endotension, a poorly understood phenomenon, treatment is urgent only in case of symptomatic patients.

**Non endoleak complications**

Non endoleak complications are often related to mistakes during the technical planning or execution of the EVAR and frequently require immediate exams to evaluate the post-operative situation. In general, the urgency of these complications is not merely linked to the complication itself but above all to the clinical conditions of the patient.

We identified four main categories, which often may coexist and may cause an endoleak.

- **structural alterations of the endograft**

- **graft migration**

- **graft infection**

- **graft occlusion**

Structural alterations of the endograft: even in the absence of a structural rupture of the prosthesis, its deformation can induce an alteration of the blood flow within the endograft.

In most cases these flow changes are not even clinically perceived and therefore they don't require special care. In rare cases such alterations may determine more significant
flow alterations that can generate sharp gradients upstream and downstream of the twisted segment of the endograft with significant reduction of the downstream flow.

In those cases, treatment is never urgent because life of the patient is not at risk. Additionally, the CTA does not allow an evaluation of flows and therefore structural alterations should only be reported and when possible evaluated with color Doppler examination.

The migration of the graft is another extremely rare event and is to be managed with absolute caution (8). Distal migration of the endograft cranial extremity causes a Type I A endoleak and therefore requires immediate treatment (Fig.1). Another rare complication is the proximal migration of the graft with as a consequence coverage of renal arteries (Fig.8).

In some cases, the migration of the graft occurs simultaneously with the structural deformation of the graft.

The infection of the graft is the most serious post-EVAR complication (9). The enhancement of the periprosthetic perianeurysmatic granulation tissue and the simultaneous presence of fluid and air are best recognized in the venous phase, therefore the venous phase is a crucial moment for identification of the graft infection.

The treatment is usually surgical and should not be performed in emergency, except in case of fistulas with adjacent structures.

The occlusion of the graft is the most frequent non endoleak complication (0,5-11%) (10,11). In general, whatever the cause of the occlusion, an urgent treatment is necessary only when the occlusion is related to significant perfusion deficit of a downstream organ. The finding of an acute ischemic syndrome of the lower limb is frequent in the immediate postoperative phase consequence of the occlusion of a branch. After establishing the emergency of the treatment, the cause should be identified. In most cases the cause is linked to kinking, migration, dislocation of an endograft, incomplete expansion of the prosthesis or excessive oversizing of the graft in relation to the native vessels (Fig.9).
Fig. 1: Sagittal reformatted CT image (B) shows a type I endoleak with distal dislocation of the body of aortic endoprostheses. 3D VR images show endoleak before (A) and after (C) treatment.

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Fig. 2: Axial CT image (A) shows a voluminous endoleak; coronal MPR image (B) shows disconnection and of the right limb. 3D VR confirm the voluminous type III endoleak.

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Fig. 3: Same patient shown in figure 2 3D VR and 3D MIP show the endoleak occlusion after the placement of a stent-grat at the level of the disconnection

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Fig. 4: Axial Ct image (A) shows type II endoleak (arrow) with a voluminous aneurysm (diam > 7cm). 3D VR (B) shows hypertrophy of the Riolano’s arcade (arrowhead) with retrograde revascularization of the IMA with consequent type II endoleak .

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Fig. 5: Same patient shown in figure 4. Selective angiography of Riolano’s arcade confirm type II endoleak (arrow). Superselective microcatheterism (B) of the endoleak (arrowhead). Final check after embolization with Onyx shows embolization of the endoleak and of the origin of Inferior Mesenteric Artery (IMA). The angiographic control shows regular opacification of sigmoid-colic arcade (D) despite the presence of Onyx microemboli migrated in the IMA.

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Fig. 6: Same patient shown in figure 4 Axial CT image (A) shows occlusion of IMA (arrow); axial CT image (B) shows complete embolization with Onyx of the endoleak (arrow); 3D VR shows normal opacification of the Riolano's arcade, the endoleak embolization (ring) and the Onyx microfragment (arrow) migrated in the medial part of IMA.

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Fig. 7: Axial CT image shows type II endoleak (A; arrow); 3-month follow up: axial CT image (B) shows increased dimensions of the endoleak (arrowhead). Digital subtraction angiography (DSA) of the ileolumbar circle (C) and the aneurysm sac (D) with placement of a microcatheter (arrow) shows two lumbar arteries that supply the endoleak. DSA after embolization shows complete occlusion of the endoleak.

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**Fig. 8**: A coronal reformatted CT image (A) shows the migration of the proximal extremity of the stent-graft covering the origin of the renal artery thus causing its steno-occlusion. Superselective angiography of the renal artery confirms the steno-occlusion of the vessel (B) with subsequent impossibility to catheterize it.

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Fig. 9: A pre-treatment coronal reformatted CT image (A) shows a severely angulated and narrow neck of the aneurysm; post-treatment 3D MIP (B) and VR (C) images show a compression of the right stent graft limb (arrow). Post treatment axial images show compression (arrow) of the stent-graft (D) with complete thrombotic occlusion (arrow) of the distal part of stent graft limb (E).

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Conclusion

The radiologist's capacity to differentiate an urgent complication from a deferrable one facilitates the correct clinical-therapeutic follow-up of the patient.
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