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# Advances in Anesthesia for Endovascular Aortic Aneurysm Repair (EVAR)

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## ABSTRACT

Over the past three decades, the number of patients who undergo endovascular aortic aneurysm replacement (EVAR) has increased. EVAR has becomed a promising alternative for open surgical graft replacement. This procedure is associated with lower perioperative morbidity and mortality rates compared to open surgical resection. It requires a multi disciplinary team composed of a vascular surgeon, an interventional radiologist and an anesthesiologist. Patients undergoing EVAR have a greater incidence of major co-morbidities and should therefore follow a comprehensive perioperative assessment and optimization by means of a multi disciplinary approach. Because the anesthesiologist is assuming an increasingly important role in the perioperative management of these cases, the aim of this chapter is to outline the anesthetic considerations related to EVAR: preoperative considerations, anesthetic techniques, complications (especially postoperative acute kidney injury) and protective strategies to decrease the morbidity in these patients.

**Keywords:** Endovascular aortic aneurysm repair (EVAR); Perioperative anesthetic management; Renal injury.

# INTRODUCTION

Approximately 80% of the patients presenting with an abdominal aortic aneurysm (AAA) are now a days primarily treated with EVAR. This procedure has disseminated rapidly as an alternative to open surgical repair to treat AAA. Due to its reduced invasiveness, low mortality and low morbidity rates, EVAR is preferred In high-risk elderly patients, especially when it is an atomically feasible. Early mortality is lower in EVAR as compared to open repair, however this benefit vanishes after three years postoperatively.

The use of EVAR in our institution has considerably increased in the last few decades. The development of a multi disciplinary program based on high-specialized professionals (vascular surgeon, interventional radiologist and anesthesiologist) has enabled this progress. On the other hand, there are numerous guidelines that have reviewed the literature relative to perioperative considerations of patients undergoing non-cardiac surgery in order to give recommendations for an optimal anesthetic assessment in these patients.

The purpose of this chapter is to show the advances in anesthesia for EVAR: preoperative considerations, intra operative anesthetic goals, perioperative complications and protective strategies directed to decrease the morbidity in these patients (highlighting strategies to manage renal injury, because these patients are at high risk for developping acute kidney injury postoperatively).

# PREOPERATIVE CONSIDERATIONS

Patients who undergo aortic aneurysm endovascular repair are often ancient and have numerous comorbidities. This is mainly based on the fact that endovascular repair is seen as less invasive but at the same time it requires a closer follow- up as this procedure has a higher incidence of re-interventions. Thus, when choosing the surgical technique, vascular surgeons tend to offer endovascular options to elderly patients and open repair to the youngest ones. Therefore, an optimal preoperative preparation is of utmost importance and a major concern for vascular anaesthesiologists. There are numerous guidelines that reviewed literature concerning the cardiovascular preoperative considerations of patients undergoing non-cardiac surgery, being the most well-known the American Heart Association (AHA) guidelines [1], the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA) guidelines [2]. More recently, the Canadian Cardiovascular Society guidelines [3] and the Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm [4] were published. In the following pages we will go through all of their recommendations for an optimal preoperative assessment (Table 1).

## Cardiovascular Risk Evaluation and Diagnostic Tests

**- Risk index:** may be a useful tool in the preoperative assessment. The Lee Index or "Revised Cardiac Risk" (RCR) index [5] and the newer American College of Surgeons National Surgical Quality Improvement Program (NSQIP) database [6] are among the most used. They were designed to predict postoperative cardiac events such as myocardial infarction (MI) or cardiac arrest.

**- Biomarkers:** guidelines advise to analyse troponin levels before and 48 to 72hours after surgery to detect ischemia damage [7]. NT-proBNP and BNP measurements should also be considered as important prognostic indicators for long-term mortality and postoperative cardiac events.

- A thorough **physical examination** is essential for a good preoperative assessment. Patients with any present **active cardiac condition** (such as symptomatic valvular disease or arrhythmia, acute cardiac failure, unstable angina or recent myocardial ischemia) should be referred to a cardiologist for treatment optimization [1-4].

**- Physical activity:** may be estimated using the quantification of the Metabolic Equivalent of Task (MET). Patients with exercise tolerance of at least 4MET (equivalent to climbing two flights of stairs uninterruptedly) correlate with low risk of cardiac complications during aortic interventions [8].

- The Society for Vascular Surgery guidelines recommends that preoperative **pulmonary function** studies should be performed in smokers, patients with chronic obstructive pulmonary disease or <4 MET [4]**.** AHA guidelines suggest that these tests could be useful in all patients undergoing high-risk procedures [1]. Treatment of patients with poor pulmonary function should be optimized prior to surgery [4].

**- 12-lead ECG:** is recommended for all patients [2].

**- Echocardiogram:** should only be performed in patients with previously documented heart conditions, worsening dyspnoea or dyspnoea of unknown aetiology. We must highlight that, despite these recommendations, European guidelines also consider the possibility of performing echocardiogram in asymptomatic patients for high-risk surgeries [2], not being the case in the AHA or Canadian guidelines [1,3].

- Regarding **cardiac exercise stress testing or imaging stress testing**, most guidelines recommend performing them in patients with more than two clinical risk factors and unknown or poor functional capacity (<4METS) in order to decide if a change of management is required [1,2].

- Routine preoperative **coronary angiography** is not recommended in all patients. It will just be performed in patients with acute myocardial ischemia (AMI or unstable angina) or unresponsive angina despite optimal medical treatment [9].

## Perioperative Treatment

**- Smoking cessation:** should be encouraged among all patients before undertaking surgery [3].

- Perioperative continuation of **β-blockers** is recommended in patients who are already on this treatment, as long as it is well tolerated and depending on the clinical condition of the patient. In patients with moderate or high risk of AMI or at least 3 revised cardiac risk index (RCRI) factors, initiation of β-blockers may be considered. In any case, is not recommended the initiation of this therapy at high doses without previous titration or within 24h of the intervention (as suggested by POISE trial results [10]).

**- Statins:** should be continued in patients who are currently receiving them. AHA and ESC/ESA guidelines recommend the initiation of statins at least two weeks prior to vascular surgery [2], whereas Canadian guidelines find the evidence too weak to support a recommendation [3].

- The use of **$\alpha_2$- agonists** for prevention of cardiac events is not currently recommended. In POISE-2 trial [11], clonidine did not have an effect on the reduction of the rate of death nor MI. Moreover, clonidine seemed to increase the rate of non-fatal cardiac arrest and clinically relevant hypotension.

- There is sparse data regarding **angiotensin-converting enzyme inhibitors** or **angiotensin II receptor blockers.** It is generally accepted that these treatments may be continued perioperatively in patients treated for heart failure. There are contradictory recommendations among different guidelines in withholding or not these therapies when prescribed solely for hypertension [1-3].

- Although there are similar contradictory recommendations regarding **calcium-channel blockers**, ESC/ESA guidelines suggest considering continuation or initiation of these drugs in patients with poor tolerance to beta-blockers [2].

**- Antiplatelet agents:** AHA guidelines recommend continuing dual anti platelet therapy when the surgery has to take place during the first 4 to 6 weeks after bare metal sent (BMT) or drug eluting stent (DES) implantation, unless the risk of bleeding outweighs the benefit of preventing acute stent thrombosis [1]. If possible, surgery should be postponed until 30 days after BMT or one year after DES implantation. When P2Y12 platelet receptor–inhibitor therapy must be discontinued, it is advisable to maintain aspirin treatment and restart the former as soon as possible. Initiating or continuing aspirin treatment is not beneficial unless the risk of thrombotic events outweighs the risk of bleeding [11].

**- Anticoagulant therapy:** should be discontinued before surgery following the recommendations for each anticoagulant regarding the time of cessation or need for bridge treatment [1,2].

- The Society for Vascular Surgery guidelines recommend **preoperative coronary revascularization** in patients with active coronary disease (MI or unstable angina) or unstable angina with left main coronary artery or three-vessels disease [4]. It should also be considered in two-vessels disease when the proximal descending artery is compromised, in patients with positive results for ischemia or when there is an ejection fraction of <50%. Surgery should be delayed by 30 days after BMT placement or Coronary Artery By-pass Surgery, if possible.

**Table 1:** Preoperative management of patients following EVAR.

| Diagnostic tests | Interventions |
|---|---|
| • Routine blood samples (blood count, biochemistry, coagulation) and BNP<br>• 12-lead ECG<br>• Chest XR/pulmonary function tests<br>• Echocardiogram (patients with documented LVEF <60%, recent dyspnea of unknown origin or worsening dyspnea)<br>• Cardiac stress testing (≥ 2RCRF or <4 METS) | • Encourage smoking cessation<br>• Optimize chronic medication<br>• Continue on chronic β-blocker therapy<br>• Initiate β-blockers in high-risk patients (consider in ≥3RCRI)<br>• Initiate statins 14 days before<br>• Coronary angiography |

BNP=brain natriureticpeptide.ECG=electro cardio gram. XR=X-ray. LVEF=left ventricle eyection fraction.

RCRF= revised cardiac risk factor. METS=metabolic equivalent of task.

# INTRAOPERATIVE ANESTHETIC MANAGEMENT

## Anesthetic Goals

The intraoperative anaesthetic management of an EVAR is a challenge for the anaesthesiologist. For an optimal intraoperative management, the following targets should be pursued [12]:

1. To ensure the right supply and intake of oxygen and to avoid the imbalance between oxygen intake and oxygen consumption of the myocardium.
2. To maintain the hemodynamic stability and, therefore, a good perfusion of vital organs, such as brain, heart, kidney, spinal cord or splanchnic region.
3. To minimize the intraoperative fluid loss and manage bleeding at an early stage.
4. To ensure normothermia.

## Work Environment and General Considerations

EVAR procedure should be carried out in a specialized theatre provided with appropriate interventional radiology equipment, angiography facilities, and also the ability to convert to an open procedure if required. Staff should avoid ionising radiation by wearing lead aprons, thyroid shields, and protective screens.

## Monitoring

EVAR is a High Risk procedure where blood loss may be occult and infra estimated [13]. Patients must be monitored with (Table 2):

-Pulse oximetry.

-Electrocardiogram: to be able to detect ischemic changes, a 5-lead electrocardiogram should be used.

-Invasive blood pressure: usually a radial arterial line is placed. It enables the anesthesiologist as well to take blood samples easily and use monitoring systems based on the pulse wave and the systolic variation. The arterial lines are usually placed in the right arm, because the left subclavian artery may be involved in the aneurysm and the surgeon may potentially need left axillary or left brachial artery access [14].

-To assess fluid therapy and monitor haemodynamics, devices based on continuous analysis of pulse waveform may be used. In some cases (complex surgeries, thoracic aneurysm, cardiac co morbidities), pulmonary artery catheter or transesophagueal echocardiography monitoring might be useful.

-Urine output should be measured every hour to detect changes in renal function and indirectly, in organ blood perfusion.

-It is of utmost importance to maintain normothermia. Thus, an esophagueal or vesical temperature catheter should be applied and fluid warmers and forced air warming devices should be used.

-Large bored venous access should be inserted in anticipation of a potential blood loss if conversion to an open procedure.

-A central venous access could be placed in long procedures or when vasoactive infusion is needed.

-If possible, access to thromboelastometry technology or to gasometry and haemoglobin testing in the surgical setting could facilitate fast diagnostic of electrolytic disturbances or guided treatment of bleeding. An ACT-machine testing is also needed to check for heparin levels.

-In procedures with high risk for spinal cord ischemia, surgeons might request the use of evoked potentials.

**Table 2:** Intraoperative monitoring during EVAR.

| |
|---|
| • Pulsioximeter |
| • 5-lead electrocardiogram |
| • Blood pressure (preferably invasive) |
| • Monitor of CO (CAPW, TEE, PAC) |
| • Urinary catheter |
| • Esophageal or urinary temperature catheter |
| • Tromboelastometer |
| • Lumbar CSF pressure monitoring (and drainage)* |
| • Somatosensory evoked potentials |

CO=cardiac output. CAPW=continuous analysis of pulse waveform.TEE=transesophageal echocardiography. PAC = pulmonary artery catheter.CSF= cerebrospinal fluid.

* Recommended in patients with high risk of spinal cord ischemia.

## Anesthetic Techniques

EVAR procedures can be performed under general anesthesia (GA), neuoraxial anesthesia [spinal (SA), epidural (EA), or both], and local anesthesia (LA) with conscious sedation (suitable for short infra-renal endovascular procedures).

There is still limited evidence and no consensus about which type of anesthesia is most suitable for EVAR patients, nevertheless the European and the American guidelines agree to recommend regional anesthesia with conscious sedation over GA whenever possible for these patients [15,16]. Less-invasive anesthetic techniques seem to be feasible and safe, and may ultimately be used as a primary strategy to significantly decrease the perioperative risk of patients undergoing EVAR.

Recommendations in favour of RA for EVAR are supported by the results published in an observational study from the National Surgery Quality Improvement Program (NSQIP), which analysed data from 6,009 patients who underwent EVAR between 2005 and 2008 [17]. They observed that over all procedures, GA was performed in the majority of cases (4868 cases, versus 419 patients under SA, 331 patients under EA, and 391 patients under LA). They also demonstrated that GA was associated with increased postoperative length of stay and pulmonary morbidity compared to SA and LA.

In addition, a meta-analysis from 2012 reviewed data from 13,459 patients who underwent EVAR [18] concluding that, despite of the increased age and burden of baseline cardiopulmonary comorbidity in the group of patients receiving LA, there was a significantly lower hospital stay and fewer postoperative complications in the mentioned group compared to the group who received GA.

A more recent retrospective study analysed 784 patients who underwent EVAR under local anesthesia (inguinal field block), observing that it was successful in almost all the procedures, requiring conversion to GA only in 1.4% of them [19].

As opposed to the evidence previously commented, a recent prospective, multicentre, non-randomized study with 1261 patients that underwent EVAR under GA, RA and LA [20] showed no significant differences in outcome nor 30-days mortality between the three groups. They also observed that GA was performed more frequently in sicker patients.

In regard to all previously mentioned, there are several intraoperative considerations in EVAR patients that may predispose the anesthesiologist to select more frequently GA over the rest of techniques:

- EVAR patients are frequently on antiplatelet medications preoperatively and will definitely require heparin intraoperatively. The safety interval of 1 hour required between the completion of regional anesthesia and the administration of heparin can be avoided if GA is performed.

- GA facilitates the patient´s comfort when arterial access is difficult, or should be placed in axillary or subclavian location.

- If aneurysm rupture occurs during the procedure and patient requires vasopressors and massive transfusion, the patient's airway is already secured. In EVAR of long duration, such as iliac bifurcated devices, complex fenestrated grafts or concomitant open surgery like femoro-femoral crossover grafts, GA may avoid patient´s discomfort.

-GA make it simpler to control patient´s brief periods of intermittent apnoea required to optimise the quality of angiographic images, justifying the use of endotraqueal tube better than laryngeal mask in this context.

## Heparinization

Patients who undergo EVAR usually require administration of IV heparin (5000 IU) immediately after the surgical vessel cannulation. The anesthesiologist should check activated clotting time and ensure it is maintained at a range between 2 and 2.5 times the baseline (approximately 200-250s) in order to minimise the risk of thromboembolic complications [12]. Reversal with protamine might be required at the end of the procedure.

## Blood Pressure Control

Maintaining an adequate blood pressure during EVAR is crucial to ensure a correct blood flow and organ perfusion [12]. Hemodynamic instability is rare, unless aortic occlusion balloons are used (usually in ruptured aneurysms). Immediate control of hypertension is easily managed with nitrates and/or short-acting beta-blockers, such as esmolol. Hypotension is common after induction of anesthesia, as the magnitude of surgical stimulus is usually small. In these cases, infusion of low-dose vasopressors such as phenylephrine can be required.

## Fluid Therapy and Blood Loss

Most studies suggest that the optimal fluid management for major surgeries is based in restrictive strategies, which has been shown to reduce mortality and post-operative morbidities. Advanced haemodynamic monitoring to guide these strategies is recommended [21].

It is important to be prepared for potential complications, such as massive bleeding and conversion to open repair. Patients should always be cross-matched and rapid infusion devices should be always available on the surgery room. When infusion of large volume of fluids is necessary, warming devices and IV fluid warmers is strongly recommended. It is also important to have an easy vascular intravenous access if rapid infusion or vasopressor infusion is needed [12].

In case of haemorrhage, the European Society for Vascular Surgery [4] recommends permissive hypotension resuscitation with SBP between 50mmHg and 100mmHg. The European guidelines on the management of major bleeding and coagulopathy after trauma [22] recommend similar strategies but a goal SBP between 80mmHg and 90mmHg. Based on the IMPROVE trial [23], SBP should not be lower than 70mmHg for a prolonged period of time, especially in elderly or patients with important cardiovascular comorbidities.

Initially, bleeding should be treated with crystalloids and colloids. There is much controversy about which proportion should be used. The European Society for Vascular Surgery [4] recommends a combination of both (neither of them have demonstrated superiority versus the other), whereas the European guidelines [22] for major bleeding after trauma advocate for the use of balanced crystalloids and restrictive use of colloids due to their association with renal damage in critically ill patients.

The threshold for transfusion varies between 6 to 8g/dL, but clinical signs must guide the need for transfusion (haemodynamic instability, signs of organ ischemia, patient's cardiopulmonary reserve, amount of blood loss). When transfusion is necessary, the European Society for Vascular Surgery [4] recommends a ratio among blood, fresh frozen plasma and platelets of 1:1:1.

Ideally, thromboelastometry technology should be used to guide transfusion strategy. If this technology is not available, most studies recommend maintaining fibrinogen > 200mg/dL and administering 20-25mg/kg of tranexamic acid. Prothrombin complex concentrates could be used instead of fresh frozen plasma to reduce the risk of acute lung injury. Lastly, activated recombinant factor VII is generally only used in massive bleedings when conventional therapy has failed.

## Prevention of Spinal Cord "Ischemia" (SCI)

SCI is a devastating rare complication of EVAR that occurs in 0.21% of patients [24] and appears either in the intermediate or delayed postoperative period [25-27]. The pathogenesis of SCI in EVAR is not fully understood yet. There are several perioperative factors that may increase the risk of SCI, such as previous abdominal aortic aneurysm repair, a ruptured aneurysm, the complexity of suprarenal graft placement, acute section or occlusion of collaterals or feeder artery vessels for the spine cord [25, 26, 28, 29]. The risk of micro-embolism may be increased by a prolonged procedural time (>150 min), extensive intravascular manipulation of catheters and perioperative embolization of lumbar or hypogastric arteries [27, 30].

One of the best- established mechanisms of SCI is the occlusion of artery of Adamkiewicz, which supplies most of the anterior spinal arteries and hence perfuses the anterior third of the spinal cord. It arises from the aorta anywhere between T5 and L3 but most commonly from T9 to T12. Other collaterals that irrigate the spinal cord arise from the internal iliac, inferior mesenteric (IMA), and middle sacral arteries. It is important to bear in mind that IMA is invariably occluded during infra-renal EVAR. Besides, long stent grafts can occlude thoracic and lumbar collateral arteries, also predisposing to SCI [26, 29].

Intraoperative measures to treat SCI include CSF drainage, increase of arterial pressure (both increase perfusion pressure of the spinal cord blood), moderate hypothermia and steroids [31]. Spinal drains have proven to be effective in prevention and management of SCI in open repairs, where there is a higher risk of SCI [32, 33]. Preoperative spinal drains are occasionally inserted for complex abdominal EVAR if the patient is thought to be at particularly high risk of SCI [33, 34].

## PERIOPERATIVE COMPLICATIONS

In the last few decades, there has been an important reduction of the incidence of ruptured aneurysms, mainly because of two facts. Firstly, diagnostic tests for aortic aneurysms have much improved and secondly, the percentage of successful EVAR has increased as they have demonstrated to be at least as safe and effective as an open intervention. Nevertheless, it is important to highlight that this increase in patients undergoing EVAR procedure has come together with a rise of the rate of postoperative complications: it is estimated to be between 16% and 30%, whereas the percentage of re interventions is around 19%. A higher percentage (38%) of patients undergoing thoracic endovascular aortic aneurysm repair (TEVAR) suffer complications in the postoperative period, among which 24% will need re intervention.

As postoperative complications are relevant and not uncommon in patients undergoing EVAR, we will focus next on its early detection and treatment. For this, we will classify the complications in two groups: the group of systemic complications and the group of complications related to endovascular access [15, 16, 35-38] (Table 3):

### Systemic complications

The incidence of systemic complications in EVAR is between 3 and 12%. Globally, EVAR is associated with a lower incidence of systemic complications than open surgery.

**1. Post Implantation Syndrome (PIS):** The incidence of PIS following EVAR ranges between 13 and 60%. This syndrome is common but usually benign and represents an immunological response of the endothelium to the prosthetic material of the endovascular devices, which manifest clinically asa systemic inflammatory response without sepsis. Patients with PIS present with fever, leukocytosis, platelet activation and elevated inflammatory biomarkers, including C-reactive protein, endotoxin, tumour necrosis factor and interleukin 6. Some studies have shown that procalcitonin usually remains low. It is important to exclude any potential infective complication

before diagnosing this syndrome. Most of the PIS cases are self-limited and disappear after two weeks of surgery. Treatment of PIS is exclusively symptomatic, managed with intravenous fluids and antipyretics. Administration of antibiotics is not recommended.

**2. Ischemic Complications:** There is a greater incidence (around 9%) of ischemic events following EVAR compared to open surgery. This may be caused by arterial thrombosis, embolism, arterial dissection or arterial obstruction, being the latter complication produced by a bad prosthetic placement, which may compromise the vascularisation of several organs and tissues. The organs most commonly affected are the kidneys, the intestine, the pelvic organs and the muscles.

**Kidney Ischemia** occurs in 0.7-18% of the abdominal EVAR. Recent studies have shown lower incidence of kidney damage in endovascular procedures compared to open surgery.

**Intestinal Ischemia** after EVAR is presented in around 1 to 3% of cases. It is produced by a prosthetic obstruction at the level of the inferior mesenteric artery and it mainly affects the colon. These patients usually manifest abdominal pain and rectal bleeding within the first 30 days after the procedure. Previous surgical embolization of internal iliac arteries increases the risk of this complication.

**Pelvic Ischemia** occurs as a result of the internal iliac arteries embolization. Patients with iliac aneurysms that require complex endovascular prosthetic insertion are at high risk of developing this complication. Clinical manifestations include claudication and pain of the buttocks, rectus ischemia, erectile dysfunction, and perineal skin lesions.

As for the **spinal cord ischemia** (SCI), it is an uncommon but devastating complication following toraco-abdominal EVAR, involving up to 16% of these patients. There are around 14 cases of SCI published so far. Typical symptoms of SCI usually appear within the first 12 hours after surgery and may include paraplegia, lower limb hypoesthesia and urine retention. However, SCI may appear anytime during the postoperative period. Long duration of the procedure, intraoperative hypotension, complex and long endovascular devices, left subclavian artery obstruction, previous aortic surgery, or previous renal dysfunction are some of the main risk factors for developing SCI.

**3. Cardiothoracic Complications:** Predictors of perioperative cardiac risk should be detected and medically optimized prior to the intervention, as cardiovascular complications are the most common cause of perioperative morbidity and mortality in patients following EVAR. Its incidence ranges from 1.8% to 5.4%. The risk of patients with aortic aneurysm is considered equivalent to the one observed in patients with coronary disease.

Regarding pulmonary complications it is important to point out that they are significantly lower after endovascular repair compared to open surgery, occuring around 2.9 to 3.3% of the patients. Elderly patients and those with chronic obstructive pulmonary disease (COPD) are at higher risk of developing these complications.

**4. Complications related to the use of contrasts:** The use of significant amount of iodinated contrast is a common practice during EVAR. It predisposes to the establishment of acute kidney injury (AKI) and the appearance of allergic reaction to contrasts. Prevention and management of these complications is explained in greater detail in the next section.

**5. Cerebrovascular Events:** The incidence of embolic cerebrovascular events oscillates between 4 and 8% of the cases, and it is similar to open surgery rates. Proximity of the proximal portion of the endovascular device to the vertebral or carotid arteries, existence of atherosclerotic plaques at this level or previous acute ischemic attack (AIT)increase the risk of suffering this complication.

**6. Abdominal Compartment Syndrome (ACS):** This complication is due to an increase in intra-abdominal pressure, which may end up generating organ dysfunction, such as AKI. Evidently, the incidence climbs up in cases of emergent surgery: approximately 10% of patients following endovascular repair of ruptured aneurysm may develop ACS.

## Complications Related to the Different Endovascular Accesses

**1. Endoleaks** are the most frequent complication following endovascular procedures. They are defined as the persistence of blood flow in the aneurysm sack, and they are regarded as a "*non fully occluded aneurysm*".

**2. Device migration:** represents the most frequent cause of re intervention in endovascular procedures. It is usually produced because of the dilatation of the proximal part of the neck of the aneurysms. If this complication is not treated on time, leak or rupture of the aneurysm may occur.

**3. Endograft infection:** This complication is observed in between 0.4 and 3% of patients following EVAR and it associates high mortality rates (up to 25 to 50%). Preventive strategies are similar to those carried out in open surgery and they include preoperative administration of antibiotics.

**4. Access site complications:** Their incidence oscillates between 9 and 16% of patients following EVAR. The most common ones are presented locally (hematoma, dissection or thrombosis, fistula, pseudoaneurysm but it can also be manifested distally) or distally (manifested as thromboembolic events).

## Reconversion to Open Surgery

The percentage of conversion of the endovascular procedure to open surgery ranges between 0.6 and 4.5%. Reconversion is performed in unsuccessful EVAR, in cases of symptomatic type V endoleak (also referred as "endotension") or when there is significant involvement of the endovascular device. In some of the complex cases, where the removal of endovascular device and placement of another one is required, the surgical risk increases considerably.

**Table 3:** Potential complications in patients following EVAR.

| Systemic complications | Local complications |
|---|---|
| • Postimplantation syndrome<br>• Thromboembolic events*<br>• Pulmonary complications<br>• Contrast-induced-nephropathy<br>• Allergic reaction to iodinated contrast | • Endoleak<br>• Device migration<br>• Endograft infection<br>• Access site complications (hematoma, thrombosis, fistula, pseudoaneurysm) |

*Thrombembolic events produce acuteischemia in different organs: mainly in myocardium, brain, bowel, kidney, pelvis.

# STRATEGIES TO MANAGE RENAL INJURY IN EVAR

Endovascular procedures are safe and effective for treatment of abdominal aortic aneurysm. In contrast to open procedures, EVAR is a low invasive procedure which avoids aortic cross clamping, hemodynamic instability, and ischemia-reperfusion syndrome. Despite of this, several trials [39] have suggested that renal function may be deteriorated to a greater extent in patients following EVAR compared to open surgery. An initial postoperative benefit in preservation of renal function is observed in patients undergoing EVAR compared to open procedures, however this effect is vanished over the subsequent 12 months after surgery. This deterioration may be a result of repeated postoperative contrast enhanced CT scans and angiograms in EVAR patients [40,41].

## Physiopathology of AKI in EVAR

Among the potential causes of acute kidney injury (AKI) establishment in patients following EVAR, the most relevant are:

1. Contrast-induced nephropathy.
2. Suprarrenal endograft fixation.
3. Micro embolization of renal arteries.
4. Ischemia-reperfusion of lower limbs.
5. Others: fluid depletion, inflammatory or/and oxidative stress biomarkers.

Over the past decade, endovascular procedures associated with intra-arterial contrast administration has become an established method for aortoiliac aneurysm repair. The use of ionized contrast agents can cause impaired renal function, which can even lead to end-stages of renal disease. These agents produce hemodynamic changes in the kidney within 24 to 48 hours postoperatively, consisting in renal vasoconstriction and interference intubular absorption of water and sodium, leading to an activation of the tubuloglomerular feedback mechanism and a significant decrease of glomerular filtration rate (GFR). Other mechanisms related to the

pathogenesis of contrast-induced nephrotoxicity are a reduction of medullar blood flow, an increase in oxidative stress and urinary viscosity and tubular obstruction.

Besides, chronic renal insufficiency prolongs the elimination half-life of the contrast agents, leading to further renal exposure to contrast therapy and further nephrotoxicity.

The association between contrast volume and nephrotoxicity remains controversial. Several strategies must be adopted to reduce the amount of contrast agent used during EVAR as bony lumbar vertebral landmarks to approximate the level of the renal arteries (usually L1-L2) and the position the main body of the modular stent graft. Automated power injectors are used as well to infuse low volumes of contrast at high flow rate when marking the position of the renal arteries.

## Outcomes of AKI

Up to 25% of patients undergoing elective EVAR develop AKI, which is associated with short and long-term morbidity and mortality. Patients at risk of this condition are those with chronic obstructive pulmonary disease and intraoperative factors such as urgency of presentation, suprarenal clamping and long operative times. However, it is important to point out that most of the present evidence regarding postoperative AKI included predominantly open aneurysm repairs. Thus, the real impact of renal dysfunction on mortality and the predictors of such complications following endovascular procedures remains unclear. Furthermore, there is no high-quality of evidence for preventing perioperative AKI in EVAR, and this is partly due to the complexity of the mechanism underlying renal injury in EVAR, as described above.

Saraztis et al. [42] documented that elective EVAR can lead to AKI in up to 18.8% of patients (in a cohort of 149 patients), and they also observed that preoperative renal function is the main predictor of AKI in these patients. It is therefore essential to establish risk factors preoperatively in order to direct strategies of renal protection in order to decrease the incidence of AKI after elective EVAR.

Zettervall et al. [43] studied a sample of 4.503 patients, from which the majority of patients (3869) underwent EVAR and only 634 patients had an open repair. Renal complications ocurred less frequently in patients who followed endovascular intervention (1% of patients after EVAR vs. 5% of patients who followed an open repair). Besides, dialysis was rarely initiated after EVAR (0.6% of patients following EVAR compared to 4% of patients after open repair). Among the group of patients who underwent EVAR, both morbidity and mortality rates were significantly increased when renal complication was noted. Specifically, 30-day mortality was 55% in patients with renal complications whereas it was much lower (1%) in patients who did not develop them. Major complications were also higher among patients with renal complications, including myocardial infarction (21% vs. 1%), pulmonary complications (49% vs. 2%), ischemic colitis (15% vs. 0.3%) and lower extremity ischemia (15% vs. 1%). The hospital stay was also increased in patients with renal dysfunction (8 days long compared to 2 days in patients without renal impairment). The authors of the study also established several predictive factors of postoperative renal impairment:

such as a preoperative GFR <60ml/min, AAA diameter, open repair, transfusion and prolonged operative time.

Chronic renal insufficiency can also increase the incidence of contrast-induced nephrotoxicity. High osmolar contrast agents, dehydration, nephrotoxic drugs and diabetes mellitus can further increase the risk of renal failure.

In opposition to the above, Mehta et al. [44] conducted a prospective randomized study where they demonstrated that perioperative renal protection measures (such as adequate intravenous hydration, use of low osmolar contrast agents, restriction of nephrotoxic drugs, and use of mannitol to promote diuresis) were associated with low rates of postoperative renal failure impairment in patients with preexisting chronic renal insufficiency compared to patients with normal renal function. Thus, optimizing perioperative management of patients with chronic renal insufficiency with appropriate strategies, which mainly consist in avoiding perioperative hypotension and limiting the volume of iodinated contrast agents, have enabled to perform EVAR safely in these patients.

## Early Detection of AKI in EVAR

AKI is characterized by a rapid decline in GFR or an increase in creatinine of >0.3mg/dL within the first 48 hours of the insult. Traditional diagnostic tests for AKI include laboratory determinations (determination of the serum creatinine, blood urea nitrogen, creatinine clearance, urinary electrolytes, microscopic examination of the urine sediment) and radiological imaging. Nevertheless, these indicators are insensitive, non-specific, as well as non useful for an early detection of the disease.

Several biomarkers have recently demonstrated to be much reliable during early-stages of AKI and consequently they can better guide therapeutic decisions in patients following EVAR. Among them, the most important ones are described below:

**1. Cystatin C:** is a non-glycosylated protein. After glomerular filtration, it is fully catabolized in the proximal renal tubule and thus, is has no renal reabsorption. Unlike serum creatinine, levels of cystatin C are unaffected by gender, age, race, protein intake or muscle mass. For these reasons, serum cystatine C has been suggested to be an ideal endogenous marker for GFR [45].

**2. Neutrophil gelatinase-associated lipocalin (NGAL):** is an ion-transporting agent produced in the distal part of the nephron and its synthesis is up regulated in response to AKI. NGAL is therefore a marker of renal injury and also of kidney disease progression [46].

**3. Kidney Injury Molecule-1 (KIM-1):** is also an early biomarker associated with human renal damage in the proximal tubule [47].

**4**. **Liver-type fatty acid binding protein (L-FABP)** and **interleukin-18 (IL-18):** are some of the other remarkable markers that can be feasible to detect AKI in these patients.

Nevertheless, the ideal marker still has not been yet found and further robust studies are needed to clarify to improve the diagnosis and management of AKI in patients following EVAR.

In the past the evidence of studies concerning this field was not reliable and showed heterogeneous results, considering there was a lack of a global definition of AKI [41, 48]. This problem has been approached after an expert meeting, where authors developed standardized criteria for a definition, classification, prevention and treatment of the AKI, creating the RIFLE (Risk, Injury, Failure, Loss, and End Stage Kidney) (Table 4). One of the main characteristics of this scale is that it has 3 severity levels regarding the level of creatinine, the urine output or both.

**Table 4:** RIFLE Criteria for diagnosis of AKI.

| | ↑SerumCreatinine | ↓Glomerular Filtration | ↓Urinary Output (UO) |
|---|---|---|---|
| **Risk** | 1.5 x basal creatinine | 25% | UO <0.5 ml/Kg/h in 6h |
| **Injury** | 2 x basal creatinine | 50% | UO <0.5 ml/Kg/h in 12h |
| **Failure** | 3 x basal creatinine or >4mg/dl | 75% | UO <0.3 ml/Kg/h 24h or anuria in 12h |
| **Loss** | Complete loss of kidney function >4 weeks | | |
| **End-stage** | End-stage kidney disease >3 months | | |

## Strategies for Prevention AKI in EVAR (Table 5)

It seems evident that prevention of AKI following EVAR requires a multi factorial approach:

**1. General Precautions:** include avoidance of hypo perfusion, adequate hydration, avoidance of nephrotoxic drugs or use low osmolarity contrast agents.

**2. Remote ischemic preconditioning (RIPC):** describes an innate tissue adaptation to ischemia, which involves humoral mediators and other metabolic pathways. The evidence of the potential benefits of this strategy on the prevention of AKI in patients following EVAR is low and only based in underpowered studies.

**3. Targeted Renal Therapy (TRT):** involves the administration of fenoldopam inside the renal arteries using a bifurcated urinary catheter. Fenoldopam is a short-acting selective agonist of dopamine-1 receptor and is known to be an effective renal arterial vasodilator, and subsequently it is capable to increase renal blood flow [49]. Although this effect may be effective inpreventing contrast media-induced nephrotoxicity, randomized controlled trials are required to confirm the role of TRT.

**4. N-Acetylcysteine (NAC):** is an antioxidant that is usually used to prevent worsening of renal function in patients undergoing administration of intravascular contrast agents. It is effective at doses of 600 mg orally twice daily on the day before and the day of the exposure to contrast agent.

Evidence in favour of the administration of NAC as a renal protection strategy in patients who undergo EVAR is controversial. Moore et al. [50] conducted a prospective randomized study to compare several parameters of renal function, such as retinol-binding protein (RBP) and median

urinary albumin/creatinine ratio (ACR) between patients groups of patients exposed to standard fluids and patient who received fluids and NAC during EVAR. They demonstrated that EVAR causes a significant acute subclinical renal injury in most patients, with significant elevations of both RBP and ACR. However, RBP and ACR levels where similar between the two groups. Otherwise, in a meta-analysis of Kelly et al. [51], they found NAC was more protective of renal function than hydration alone.

**5. Mannitol:** is an osmotic diuretic with antioxidant and renovascular effects that might be beneficial in preserving renal function after EVAR. In contraposition, it induces an increase in urinary loss and subsequent dehydration in cases where fluids are inadequately replaced. Where mannitol plays an important role or not in renal protection in these group of patients has not been evaluated yet using the new sensitive indices of renal injury such as cystatin-C and neutrophil gelatinase-associated lipocalin (NGAL). Kalimeris et al. [52] compared hydration intervention versus hydration plus mannitol intervention for prevention of renal dysfunction after EVAR by evaluating changes in levels of creatinine, cystatine-C and NGAL, as well as other traditional biomarkers such as urea and albuminuria. They determined that mannitol plus hydration improved postoperative creatinine and cystatin-C values, indicating a small but significant benefit of this strategy when compared to hydration alone in prevention of AKI. Among the potential mechanisms implicated on this effect are an up-regulation of prostaglandine-I2 (PG-I2) synthesis, an increase in the renal blood flow (especially noted in ischemic kidneys), and reversal of renal vasoconstriction, which is a fundamental mechanism of contrast-induced nephropathy. Furthermore, mannitol can scavenge free radicals released by contrast media and ischemia-reperfusion produced from the lower limbs. Besides, its osmotic effect maintains a high urine flow, inhibiting formation of tubular casts and swelling of endothelial cells. This can also lead to a secondary benefit of avoiding furosemide. A recent meta-analysis showed that furosemide doubled the risk of contrast-induced nephropathy; possibly by reversing blood flow within the kidney and rendering the vulnerable outer medulla ischemic. Despite the early favourable outcomes observed with mannitol therapy, these were not maintained after 24 hours. This can be explained at least partially by the postoperative development of renal microembolizations or bilateral renal artery stenosis, as well as the realization of CT scans during the follow-up period.

**6. Prevention of contrast induced nephropathy:**

- Intravenous hydration induces a high urine flow rate to decrease tubule exposure to contrast.
- Theophylline: may also reduce the risk for contrast induced nephropathy, although the association has not been significant in the literature [51].
- Diuretics have been shown to increase the risk for AKI.
- Intravenous sodium bicarbonate alkalizes urine, protecting against oxidative stress.
- NAC can provide renal protection through scavenging free radicals and increasing nitric oxide production.

In a recent study of 2018, Saratzis et al. [53] developed a specific reno-protective strategy of urinary alcalinization with a bolus dose (1mg/Kg) of intravenous 8, 4% sodium bicarbonate together with perioperative intravascular volume expansion. This study is a pilot phase that must be confirmed in definitive and large RCT.

**Table 5:** Strategies to prevent AKI in EVAR.

| STRATEGIES TO PREVENT AKI IN EVAR | | |
|---|---|---|
| **Preoperatively** | **Intraoperatively** | **Postoperatively** |
| • Identify high-risk patients<br>• Hydration + N-acetylcysteine<br>• Surgical planning<br>• Stop nephrotoxic drugs<br>• Targeted renal therapy<br>• Remote ischemic preconditioning | • N-acetylcysteine<br>• Bicarbonate<br>• Limit use of contrast<br>• Limit balloon dilation<br>• Use smaller-diameter sheaths; axillo-bifemoral BP to limit ischemia | • Adequate hydration<br>• Perform laparotomy if abdominal compartment syndrome<br>• Close patient monitoring for sepsis and limb ischemia<br>• Considering measuring subclinical markers of AKI |

# CONCLUSION

The introduction of EVAR has led to substantial improvements in management of AAA. However, despite this innovation, further research will be necessary to optimize the anesthetic management of these patients. Conduction of a future fast-track EVAR program could be feasible, safe, and could improve efficiency of health care resource allocation in selected patients undergoing EVAR.

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