ASK THE EXPERTS

Can Patients With CKD and TAAA Be Treated With an Endovascular Approach?

Considerations in clinical decision-making for thoracoabdominal aortic aneurysm repair in patients with impaired renal function.

With Linda M. Harris, MD, FACS, DFSVS; Matthew P. Sweet, MD, MS; Marcelo Ferreira, MD; Matheus Mannarino, MD; and Gabriela Velazquez-Ramirez, MD, FACS

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Patients with chronic kidney disease (CKD) have a different life expectancy and increased mortality following both endovascular and open procedures, and this should be factored into the decision of if, when, and how to repair a thoracic aortic aneurysm (TAA). In a study by Brown et al, 1-year survival was 48% in patients with end-stage renal disease (ESRD) who underwent thoracic endovascular aortic repair (TEVAR), 58% in patients with stage 4 CKD, 67% in those with stage 3 CKD, and 77% to 78% for stage 1/2 patients and those without renal dysfunction.[1] The main concern in performing endovascular procedures in the setting of CKD is a further decline in renal function due to contrast administration. This includes the use of preoperative and intraoperative imaging and postoperative surveillance. However, depending on the extent of the thoracoabdominal aortic aneurysm (TAAA), it may be possible to glean most information from plain CT scans, including the proximal landing zones, because the great vessels are easily identified without contrast. What is lost without contrast is vessel patency, thrombus burden, and assessment of the vertebral arteries. However, directionality of flow in the vertebral arteries and dominance can be easily assessed with noninvasive studies. Location/involvement of the celiac axis may be more difficult to assess without contrast. However, CTA of the chest may decrease the total contrast needed if fusion technology is utilized during the procedure. During TEVAR, all attempts at minimizing contrast should be employed, including fusion imaging, intravascular ultrasound (IVUS), and use of smaller, more dilute dye loads. After completion of the procedure, follow-up surveillance imaging can be problematic for CKD patients. However, plain CT of the chest with measurement of the sac diameter can be used without additional harm to the kidneys and provides a fairly accurate assessment of clinically significant

endoleaks by sac size changes. This then allows limited contrast utilization to those patients with potentially significant endoleaks.

So, why consider an endovascular approach with the inherent need for intraoperative contrast, as well as long-term follow-up imaging? Open surgery in "healthy" patients still has morbidity and mortality in the thoracic region, one of the main drivers of the move to endovascular interventions for thoracoaortic disease. Further, even with "simple infrarenal aneurysms," early renal function compromise has been shown to be greater with open repair and infrarenal clamping than with EVAR (P = .03), resolving by 1 year.[2] With suprarenal clamping, one would expect the changes in renal function to be even greater. Marques De Marino et al found that patients with EVAR had a greater decline in renal function after the initial perioperative period (P = .08), which was likely related to ongoing contrast exposure with surveillance. Not only is renal function initially compromised with open surgical repair, but mortality is also significantly higher than for TEVAR in ESRD patients (overall: 30% for open repair vs 15.7% for TEVAR; P = .002; 29.3% for elective open vs 14.3% for elective TEVAR).[3] However, survival was equivalent by 1 year.

It is important to be aware and appropriately alter the management of patients with CKD or ESRD in addition to TAAA, due to the increased risk of adverse outcomes compared with the normal population. In essence, the trade-off is an increased risk of early mortality and worsening of renal function with open repair compared with the potential late impact on renal function if continued contrast-based imaging is performed with endovascular intervention. Mortality is increased for moderate to severe CKD patients, and this should factor into decisions as to if and when to repair aneurysms versus when to observe. The threshold for the repair of TAAA should be shifted in patients with stages 3 to 5 CKD or ESRD. However, TEVAR should be considered the ideal method of treatment, assuming anatomic suitability.

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2. Marques De Marino P, Martinez Lopez I, Cernuda Artero I, et al. Renal function after abdominal aortic aneurysm repair in patients with baseline chronic renal insufficiency: open vs. endovascular repair. Int Angiol. 2018;37:377-383. doi: 10.23736/S0392-9590.18.04010-5
3. Liang NL, Yuo TH, Al-Khoury GE, et al. High mortality rates after both open surgical and endovascular thoracic aortic interventions in patients with end-stage renal disease. J Vasc Surg. 2017;66:991-996. doi: 10.1016/j.jvs.2016.12.144, 10.1016/j.jvs.2016.12.144

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It's essential to start from the perspective that operation for TAAA is almost always an elective procedure done as prophylaxis against rupture. As such, I would argue that a "good" outcome is only achieved with complete aneurysm exclusion, preservation of the patient's preoperative functional status, and avoidance of any new end-stage organ failure. Successfully repairing an asymptomatic aneurysm while rendering the patient dialysis-dependent is unlikely to meaningfully prolong the patient's life and certainly has serious consequences to the patient's quality of life and should, in my opinion, be considered treatment failure.

Given this premise, I think most surgeons who manage TAAA would agree that patients with CKD, specifically stage 3 or greater (estimated glomerular filtration rate [eGFR] < 60 mL/min), tend not to tolerate an open bicavitary repair well. As seen in numerous studies, CKD is the most consistent risk factor for adverse outcomes after open aneurysm repair. To answer this specific question, I think it is fairly simple to say that among patients with stage 3 CKD or greater, open repair of TAAA is high risk and it would be hard to expect most CKD patients to tolerate an open TAAA repair with a "good" outcome as described above. As such, among patients with TAAA and CKD, I would suggest that endovascular repair is strongly preferred over open repair.

The more challenging question is when is CKD an important contraindication to repair of a TAAA using a branched endograft? As seen in one large cohort study, about one-third of patients with TAAA have some sort of end-stage organ failure.[1] In general, end-stage organ failure, including ESRD, is a contraindication for a branched endograft because the patient's overall survival is likely limited and aneurysm rupture risk rarely exceeds the risk of that condition. How to manage patients with more modest stage 3 CKD (eGFR 30-60 mL/min) is more nuanced.

In my practice, I look at both anatomic and physiologic risk factors for repair, and CKD is an important physiologic risk factor. I place increasing importance on this with worsening disease. Mild CKD (stage 2 to 3A, eGFR 45-90 mL/min) is very common among elderly patients with degenerative TAAA and is not considered a significant risk factor for endovascular repair. Stage 3B CKD (eGFR 30-44 mL/min) is also seen somewhat frequently and I consider this a risk factor, but in isolation, I would not change the decision for repair presuming the renal anatomy is amenable to revascularization. In a patient with hostile renal artery anatomy or multiple other advanced comorbidities, this may push me to advise the patient against repair. Stage 4 or 5 CKD is a strong relative contraindication for me, as I think the likelihood of achieving a successful treatment with a reasonable medium-term survival is low. Certainly, rarely, there are individuals with stage 4 or 5 CKD who might reasonably be expected to survive in the medium term with a successful aneurysm repair, but these patients are few and far between in my experience. Among patients treated with t-Branch and custom-made devices (Cook Medical) as reported in the United States Aortic Research Consortium, only 1.5% of nearly 1,000 patients were dialysis-dependent prior to endovascular repair.

1. Kang PC, Bartek MA, Shalhub S, et al. Survival and patient-centered outcome in a disease-based observational cohort study of patients with thoracoabdominal aortic aneurysm. J Vasc Surg 2019;70:1427-35. doi: 10.1016/j.jvs.2019.02.033

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At first glance, one could associate renal dysfunction only to the contrast media used in endovascular procedures, but it's clear that even open or endovascular treatment for TAAAs has a similar rate of acute kidney injury (AKI), and CKD is a well-recognized and documented risk factor for both techniques.[1]

First, AKI is a common complication of open surgery for TAAA, affecting approximately 10% to 30% of patients.[2] Even centers with large volume and great expertise in open surgery for TAAA report AKI rates of 12.7% and 9.6%,[2,3] indicating that AKI during these procedures is multifactorial. Although history of CKD is a risk factor, various other factors are deleterious to kidney function including the development of visceral or limb ischemia during the procedure, amount of bleeding, amount of contrast, visceral microembolization, hypotensive periods, use of high doses of vasopressors, and low volume input.

In addition, several studies have shown no evidence of contrast-induced nephropathy regardless of CKD stage, whereas others found evidence of contrast-induced nephropathy only in patients with severely reduced kidney function.[4] With this and considering published results of the endovascular approach and our results,[5,6] we conclude that patients with CKD and TAAA can safely undergo endovascular repair with the caveat that CKD is a recognized risk factor for both open and endovascular approaches. Importantly, patients with CKD should be prepped adequately, hydrated with saline, and whenever possible, transesophageal echocardiography should be utilized to assess the need for adequate fluid replacement during the endovascular repair, which usually produces an important inflammatory response. Finally, of note, the chemistry of the contrast media has changed over the past 10 years, with procedures now requiring less contrast, and image quality has also substantially improved.

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5. Oderich GS, Ribiero M, de Souza LR, et al. Endovascular repair of thoracoabdominal aortic aneurysms using fenestrated and branched endografts. J Thorac Cardiovasc Surg. 2017;153:S32-S41.e7. doi: 10.1016/j.jtcvs.2016.10.008
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This question is one that makes us pause when evaluating patients for repair of TAAAs; it's not a straightforward answer but more of a gestalt. The presence of baseline CKD alone should not be a hard contraindication for endovascular repair of TAAA; however, several aspects should be taken into consideration as part of the preoperative discussion.

CKD is a significant risk factor for increased perioperative morbidity and mortality as well as decreased long-term survival if it progresses to needing renal replacement therapy. These are the helpful points of discussion:

- What is the baseline CKD stage and risk of progression to hemodialysis postoperatively? Approximately 20% to 30% of patients undergoing endovascular repair of TAAA will have deterioration of their baseline CKD. This is defined as either change to a worse CKD category or > 25% decline in baseline eGFR.[1,2] This is an important discussion to have with the patient. If their risk of progressing to needing hemodialysis after surgery is imminent, one must discuss its effect on long-term survival as well as consider all other risk factors and risk of rupture based on size. This is probably the most important conversation to have when considering whether or not to pursue aneurysm repair.
- Is there intrinsic atherosclerotic renovascular disease contributing to the baseline CKD? This scenario is unlikely but something to consider if TAAA repair could improve their kidney function by treating hemodynamically significant renal artery stenosis.
- The need for perioperative renal protection strategies should be emphasized. The use of catheter-directed CTA and low-iodine CTA protocols has significantly reduced the amount of contrast needed without compromising imaging quality, especially for patients with stage 3 CKD.[3] To prepare for surgery, the patient should be given preoperative hydration with either saline or sodium carbonate, N-acetyl cysteine can be used (even though its use is controversial), and nephrotoxic medications should be held. There should be judicious and careful planning to reduce the amount of contrast agent needed intraoperatively.
- Long-term follow-up imaging protocols are important as well, especially for patients who do not have significant progression of CKD and are not on dialysis. Use of noncontrast CT scans and mesenteric and renal artery duplex ultrasounds are standard follow-ups for these patients, with the understanding that evaluation may be somewhat limited, especially for endoleaks.

The presence of CKD should not be a contraindication for consideration of endovascular repair of TAAs, but it should be more of a significant point for discussion when weighing risks and benefits of repair in the context of risk of rupture, progression to dialysis, and survival. ■

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