Keynote Lecture Series

Perioperative management of patients undergoing thoracic endovascular repair

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Thoracic endovascular aortic repair (TEVAR) is a less invasive method for treating thoracic and some thoracoabdominal aortic aneurysms, dissections of the thoracic aorta and blunt traumatic aortic injury, compared with conventional open surgery. Maximizing the likelihood of a successful outcome requires diligent multidisciplinary (surgical, critical care, nursing, pharmacy, nutrition and physical therapy) perioperative care. In this article, we discuss fundamentals for managing patients after endovascular aortic aneurysm repair. These principles focus on the transition between the operating room and the intensive care unit, prevention and management of spinal cord deficits (SCD), and vital neurological, respiratory, cardiovascular, renal, gastrointestinal and hematological concerns. The better the care team understands the expected postoperative course, the earlier that deviations can be recognized and the more likely that successful rescue can be achieved to reduce the incidence and severity of adverse outcomes. Achieving optimal results after TEVAR requires attention to detail across the preoperative, intraoperative and postoperative phases of care.

Keywords: Thoracic aortic endograft; thoracic aortic aneurysm; perioperative care; cerebrospinal fluid drainage

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Introduction

Thoracic endovascular aortic repair (TEVAR) is less physiologically demanding on patients than conventional open surgical repair. Nonetheless, significant morbidity and mortality can result from the procedure itself and patient comorbidities. Here, we discuss key perioperative management principles for the care of TEVAR patients, focusing on immediate postoperative management.

TEVAR can be used to treat fusiform and saccular descending thoracic aortic aneurysms (DTAAs), acute and chronic type B aortic dissection (TBAD) and blunt thoracic aortic injury. In more advanced forms, TEVAR with custom-made branched or fenestrated endografts can treat extensive thoracoabdominal aortic aneurysms (TAAAs). Each of these conditions reflects a different level of acuity and complexity. The better the care team understands the expected post-TEVAR course, the earlier that deviations can be recognized and the more likely that successful rescue from adverse events can be achieved (1).

Preoperative considerations

Individualized preoperative cardiovascular risk assessment is performed in elective settings consistent with current guidelines (2) focusing on age and physiological reserve, risk stratification, antiplatelet and anticoagulant medication cessation, tobacco cessation if necessary and optimization of major neurological, cardiovascular, respiratory and renal comorbidities, to reduce modifiable risks. Major preoperative comorbidities should be evaluated according to

acuity of presentation and clinical indication for TEVAR (3).

Carotid duplex ultrasonography evaluates for concomitant carotid artery disease in patients who are >60 years old or have a ≥20 pack-year smoking history. Carotid anatomy is especially relevant if left subclavian artery (LSA) coverage is necessary. Symptomatic carotid disease is treated before elective TEVAR. The size and takeoff of the vertebral arteries and the presence of a patent left internal mammary artery after previous coronary artery bypass grafting should be taken into account during revascularization before LSA coverage.

Patients with borderline pulmonary function should be optimized with pulmonary rehabilitation preoperatively before elective TEVAR. This includes exercise, weight loss, incentive spirometry training, bronchodilator therapy and mandatory tobacco cessation for at least 2–3 weeks. Any existing pleural effusion can be drained during the TEVAR.

Patients with acute TBAD needing urgent TEVAR for malperfusion are managed with anti-impulse therapy to maintain a systolic blood pressure <120 mmHg and a heart rate <70 beats per minute. Intravenous beta blockers (e.g., esmolol, labetalol) and vasodilators (e.g., nicardipine) are used while diagnostic and laboratory tests are performed. Patients with reduced creatinine clearance require adequate hydration before surgery or computed tomography (CT) imaging to reduce the risk of acute kidney injury (AKI).

Antiplatelet agents (i.e., $P2Y_{12}$ inhibitors, aspirin) and antithrombotic therapy (i.e., warfarin, non-vitamin K oral anticoagulants) are discontinued without bridging therapy one week before surgery to allow for cerebrospinal fluid (CSF) drainage (4). This practice is individualized in patients with recent coronary stents or those unlikely to need CSF drainage. Patients requiring an urgent operation while receiving warfarin therapy are administered intravenous vitamin K or prothrombin complex concentrate to reverse anticoagulation, or else a CSF drain is not placed preoperatively.

After the procedure is completed during the recovery period, clear communication between the surgical, anesthesia, critical care and nursing teams are vital (5). Communication should focus on the extent of stent graft coverage, hemodynamic disturbances, airway or pulmonary concerns, whether planned or unplanned concomitant procedures were performed, anatomical concerns for malperfusion, whether femoral closure devices were deployed and distal pulse examination.

TEVAR-associated perioperative mortality is variable, depending on the acuity and indication of presentation and the patient's comorbidities. As with open surgical repair, TEVAR potentially affects multiple organ systems (*Figure 1*), hence detailed multidisciplinary attention is necessary for optimal outcomes (6).

Neurological considerations

Stroke

The pooled incidence of post-TEVAR stroke reported in a meta-analysis of ten studies and 2,594 patients was 4.1% (7). In the five studies that characterized LSA coverage, the incidence of stroke was 3.2% when the LSA was uncovered; when the LSA was covered, the incidence of stroke was lower when the LSA was revascularized (5.3%) versus not revascularized (8.0%). Conversely, another analysis of 2,346 patients found that the stroke incidence was higher (7.5%) with concomitant revascularization of the LSA than without (4.4%) and notably higher mortality (24%) after the stroke; risk factors included mobile atheroma in the aorta and previous stroke (8). A study of stroke after TEVAR (9) found a perioperative stroke rate of 3.8%, with similar distributions in the anterior and posterior cerebral circulation; all strokes were embolic in origin. Patients with chronic kidney disease and previous strokes were at highest risk.

Clinically, if a patient awakes from anesthesia with a focal neurological deficit, immediate brain imaging and neurological consultation is initiated to confirm that no hemorrhagic event necessitating neurosurgical intervention has occurred. Embolic strokes are managed supportively.

Spinal cord deficit (SCD)

SCD may be the most devastating TEVAR-related complication. A large meta-analysis reported that SCD incidence after TEVAR was 4% for endovascular repair, including 2% for DTAAs and 9% for TAAAs, with no difference between open and endovascular approaches when stratified by aneurysm extension (10).

TEVAR-associated SCD risk factors include extent of aortic coverage, LSA coverage, lengthy procedures with large-bore iliofemoral sheaths, perioperative hypotension, previous infrarenal aortic aneurysm repair, female sex and renal insufficiency (11). Risk for SCD may be reduced by using CSF drainage catheters, pharmacological adjuncts and hemodynamic management (*Table 1*).

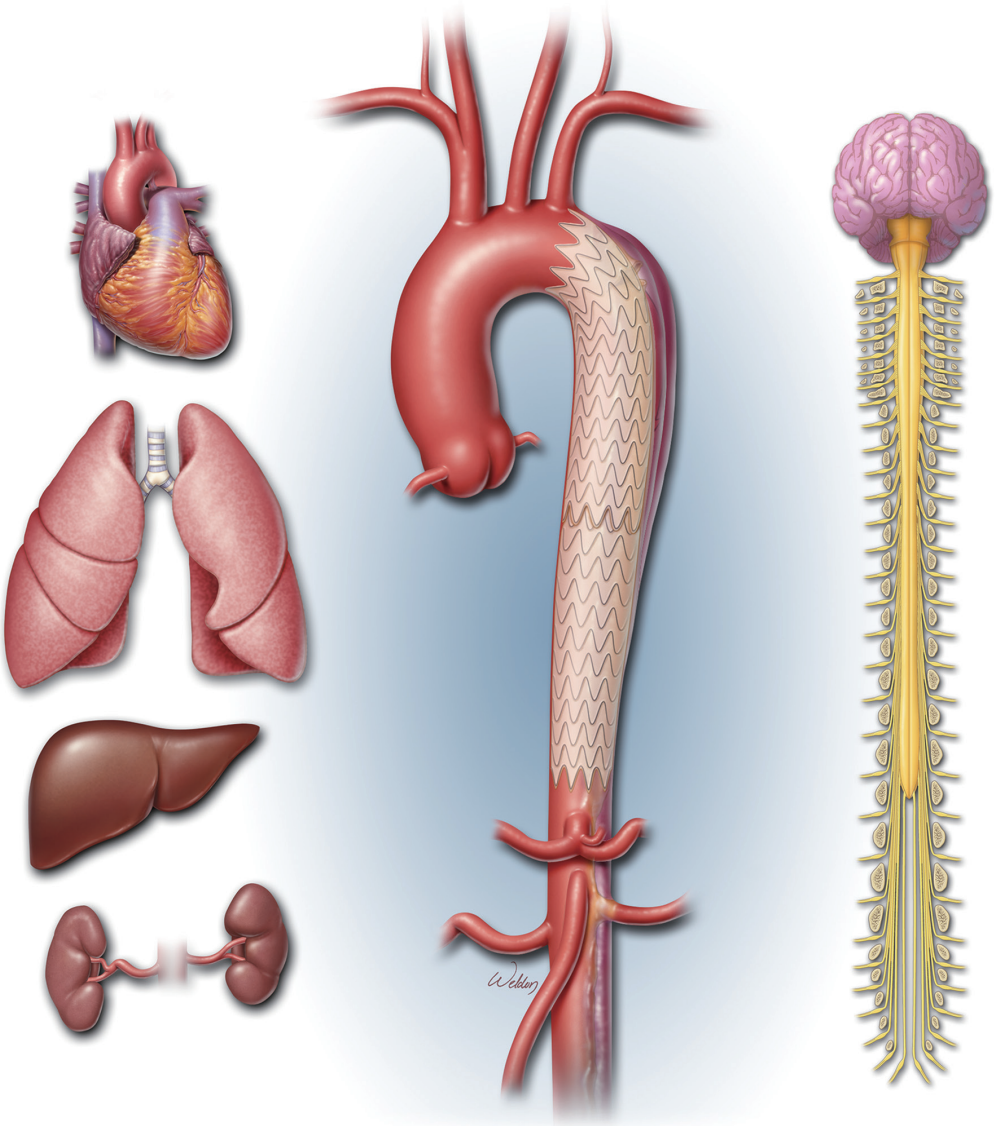

Figure 1 Major organ systems potentially affected after thoracoabdominal aortic aneurysm repair: cardiac, pulmonary, hepatic, renal and neurological (from left to right) (6). Printed with permission from Baylor College of Medicine.

Cerebrospinal fluid drainage

Although the benefits of CSF drainage in TEVAR procedures are not as clear for isolated DTAAs as they are for more extensive repairs, current guidelines do recommend CSF drainage for patients at risk of SCD during TEVAR (12,13). In our practice, this includes patients with planned full coverage of the descending thoracic aorta, those with previous abdominal aortic replacement (either endovascular or open) and those of specific concern. Drainage is facilitated by our anesthesiology colleagues, who have gained extensive experience during open TAAA procedures. The anesthesiologist routinely inserts a 14-gauge needle into the intrathecal space below the L2–L3 level and advances it 8–10 cm within the space. The drain is not placed until the end of the repair in hemodynamically unstable patients or in urgent cases when there is no time for preoperative CSF drainage (these patients may be awakened and assessed for leg strength post-TEVAR). CSF

Table 1 Evaluation of major organ systems, management goals and interventions

Organ system	Management goal	Diagnostic and therapeutic interventions
General	Normothermia	Warmed intravenous fluids, patient-warming devices
Neurological: brain	Early recognition of focal deficits	Diagnostic imaging when appropriate; neurology ± neurosurgery consultation
Neurological: spinal cord	Mean arterial blood pressure 85–100 mmHg	Volume resuscitation; vasopressors (norepinephrine, vasopressin); vasodilators (nicardipine, esmolol, labetalol)
	CSF drainage <15 mmHg	CSF pressure checks hourly with drainage (<10 mL/h); frequent exam checks
	Movement of lower extremity	If paraplegia is suspected, delayed-paraplegia algorithm initiation
Pulmonary	Ventilator management	Lung-protective ventilator settings (VT <6 mL/kg PBW, PEEP 8–10 mmHg); noninvasive alternatives to mechanical ventilation (high-flow oxygen, bi-level positive airway pressure)
Cardiovascular	Adequate cardiac output (CI >2.2 L/min/m^2)	Volume resuscitation; satisfactory hemodynamics (CVP 8–12 mmHg); satisfactory pulmonary artery diastolic pressure (12–16 mmHg) or POCUS; inotropes (epinephrine, dobutamine)
	Maintenance of sinus rhythm	Correction of electrolytes (K, Mg); antiarrhythmic therapy with amiodarone; low threshold for direct current cardioversion; systemic anticoagulation if CSF drain removed
Renal	Adequate volume resuscitation	Assessment of hemodynamics, urine output, base deficit, serum lactate
	Reduction of risk for acute kidney injury	Satisfactory hemodynamics; multidisciplinary rounding with pharmacy; avoidance of nephrotoxins, intravenous contrast, hyperglycemia; avoidance of peripherally inserted central catheters and subclavian lines
Gastrointestinal, hepatic	Prevention of stress ulcer	Stress ulcer prophylaxis
	Return of bowel function	Nasogastric tube if extensive procedure, cautious advancement of oral diet
Hematological	General	Resumption of antiplatelet/antithrombosis agents after CSF drain removal
	Avoidance of anemia	Hemoglobin transfusion to 9.5–10.5 g/dL
	Avoidance of thrombocytopenia	Platelet transfusion to ≥100,000 before CSF drain removal
	Avoidance of coagulopathy	Fresh frozen plasma transfusion to keep international normalized ratio ≤1.6 before CSF drain removal

Adapted from (1), with permission from Elsevier. CI, cardiac index; CSF, cerebrospinal fluid; CVP, central venous pressure; K, potassium; Mg, magnesium; PBW, predicted body weight; PEEP, positive end-expiratory pressure; POCUS, point-of-care echocardiography; VT, tidal volume.

drains are only placed in patients of concern for SCD.

The aim of CSF drainage is to achieve satisfactory spinal cord perfusion pressure [the difference between the mean arterial pressure (MAP) and CSF pressure]. We target a MAP of 85–100 mmHg and a CSF pressure of 10–15 mmHg to achieve a spinal cord perfusion pressure ≥70 mmHg. We avoid draining more than 10 mL per hour, 25 mL per four hours or 150 mL per 24 hours, as catastrophic central nervous system bleeding events can result from excessive drainage. Head elevation beyond 30° during mechanical ventilation is avoided as excessive elevation can cause CSF and cerebral perfusion pressure fluctuations.

Typically, the CSF drain is removed 24–48 hours after TEVAR, depending on repair complexity and whether reliable neurological examination is possible. We clamp the drain for six hours and conduct frequent neurological exams before removal. Proximal leg strength assessment should require the patient to raise their knee off the bed; foot dorsiflexion or hip rotation is insufficient. After drain removal, the patient remains on bedrest for four hours, with periodic neurological checks followed by assisted transfer to a chair.

Drainage complications are categorized as severe (intracranial hemorrhage, subarachnoid hemorrhage, epidural hematoma, meningitis, catheter or drainage-related neurological deficit), moderate (spinal headache, CSF leak requiring intervention, e.g., blood patch, drain fracture not requiring surgery), or mild (bloody spinal fluid, puncture-site bleeding, drain fracture, CSF leak not requiring intervention, catheter dislodgment or occlusion). A large meta-analysis (10) reported complication rates after endovascular DTAA or TAAA repair as severe (4.6%), moderate (0.7%) and mild (3.2%). We found that approximately 10% of patients experienced headaches with CSF drainage after open TAAA repair. Headache is initially managed with caffeine ingestion, and approximately one-third of these patients require an epidural blood patch for symptom resolution (14).

If severe headache is accompanied by nausea and vomiting, CT imaging should be obtained expeditiously to exclude intracerebral hemorrhage. Asymmetric weakness may require imaging to assess for spinal hematoma. Urgent neurosurgical intervention is advised if a hematoma is present.

Delayed paraplegia or paraparesis

Patients can awaken from anesthesia with full motor function but later develop paraplegia or paraparesis, which is associated with greater risk for permanent disability and death. Delayed paraplegia is hypothesized by some to follow a 'second hit', whereby the 'first hit' occurs during surgery, leaving the spinal cord vulnerable, and the 'second hit' is caused by a hypoperfusion-induced ischemic event during the perioperative period (15). Much of our knowledge about delayed paraplegia is based on open repairs, with one study showing that in patients who developed delayed paraplegia after open TAAA repair, the mean MAP was 74 mmHg at diagnosis, but 95 mmHg during recovery (16).

Rescue maneuvers to increase spinal cord perfusion pressure and maximize oxygen delivery should be promptly initiated if delayed paraplegia or paraparesis develops (*Figure 2*) (6). The initial goal is to ensure a MAP of 90–100 mmHg. If a CSF drain is present, CSF pressure is reduced to 10–12 mmHg and drainage is increased up to 15 mL per hour or 50 mL per four hours. If no CSF drain is present, an anesthesiologist will quickly insert a new drainage catheter. Next, the patient is placed in the Trendelenburg position and the target MAP is increased to 100–115 mmHg. Adequate volume resuscitation targets a central venous pressure of 10–12 mmHg and a hemoglobin level of 9.5–10.5 g/dL. Excessive central venous pressure could raise intracerebral and CSF pressures and is thus avoided. Diligent physician monitoring is critical during rescue; increasing MAP and lowering CSF pressure must be done gradually to increase the likelihood of neurological recovery.

Adjunctive agents (e.g., intravenous mannitol 12.5 g, dexamethasone 10 mg) can be given every 12 hours for 48 hours. If no improvement is evident after 6–12 hours, rescue doses of intravenous lidocaine 100 mg and magnesium sulfate 4 g are administered. Glucocorticoid administration may induce hyperglycemia, thus close glucose monitoring is required and insulin infusion may be necessary. Similar management algorithms used in other institutions differ slightly from ours but agree on the broad principles (17,18).

Patients who awaken with paraplegia or paraparesis are unlikely to recover. For these patients, we increase the MAP to 100–110 mmHg, drain the CSF to approximately 10–15 mmHg, and treat with mannitol and dexamethasone. Because paraplegia lasting longer than 48 hours is probably permanent, venous thromboembolism prevention measures and bowel and bladder management protocols should be initiated.

Hemodynamic management

Blood pressure should be maintained between a 'floor' (lowest acceptable blood pressure before SCD risk increases) and a 'ceiling' (highest acceptable blood pressure before no further SCD prevention benefit remains and the patient risks harmful hypertension). Indications for TEVAR will influence the blood pressure target. For degenerative aneurysms, hypotension is the primary concern, whereas relative hypertension is well tolerated. Conversely, for TBAD with residual dissection, hypertension-induced exacerbation of the uncovered residual dissection is a risk. Stabilizing hemodynamics, avoiding labile blood pressure and hypotension, and maintaining satisfactory MAP are critical measures for avoiding SCD.

Patients are occasionally hypothermic (33–34 ℃) when they arrive in the intensive care unit (ICU). If the patient is shivering, demand ischemia and lower cardiac output can risk SCD. Rewarming to normothermia induces vasodilation and decreases MAP, which should be treated. Our typical vasopressor strategy is norepinephrine followed by vasopressin. We usually aim to maintain hemoglobin above 9 g/dL.

Moderate hypertension (systolic blood pressure of 140–

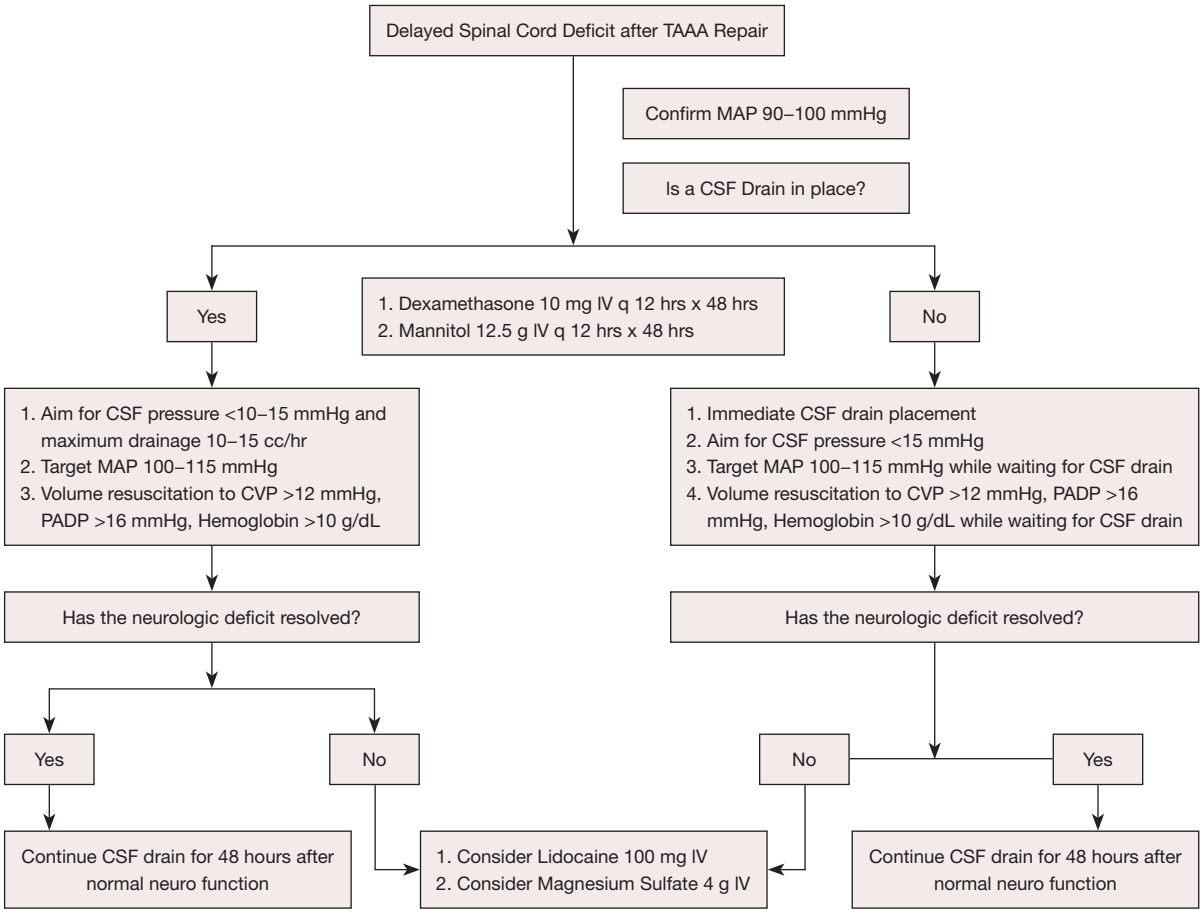

Figure 2 Algorithm for managing delayed paraplegia after thoracoabdominal aortic aneurysm repair (6); also used for managing delayed paraplegia after TEVAR. Reprinted from (6), with permission from Elsevier. CSF, cerebrospinal fluid; CVP, central venous pressure; IV, intravenous; MAP, mean arterial pressure; PADP, pulmonary artery diastolic pressure; TAAA, thoracoabdominal aortic aneurysm; TEVAR; thoracic endovascular aortic repair.

150 mmHg) is not treated aggressively. We recommend waiting 4–6 weeks after TEVAR before strict hypertension control. Severe hypertension (MAP >120 mmHg) can cause complications, especially for patients with TBAD. We use systemic vasodilators (labetalol followed by nicardipine or esmolol) to treat severe hypertension.

Respiratory considerations

Patients who undergo elective TEVAR are typically extubated in the operating room, whereas in urgent cases, patients may require ongoing mechanical ventilation. We target lung-protective ventilation with a goal tidal volume of 6 mL/kg of predicted body weight and a positive end-expiratory pressure of 8–10 mmHg. In borderline candidates, high-flow oxygen and bi-level positive airway pressure can facilitate extubation. After extubation, coughing, deep breathing, bronchodilator therapy and other pulmonary toilet measures are encouraged, especially in patients with pre-existing moderate to severe pulmonary comorbidities (*Table 1*). In a Nationwide Inpatient Sample study with 8,255 descending thoracic aortic aneurysm repair patients and 712 TEVAR patients, unadjusted postoperative respiratory complication rates were 5.6% *vs.* 2.4%, respectively (19).

Cardiovascular considerations

An arterial line and central venous catheter are typically used for hemodynamic monitoring. For more extensive

repairs, a pulmonary artery catheter and continuous cardiac output monitor may be used if the preoperative left ventricular ejection fraction is ≤35%. Adequate cardiac output is achieved with volume resuscitation and, if needed, epinephrine for augmenting both cardiac output and blood pressure. During resuscitation, we generally monitor cardiac output (if available), mixed venous oxygen saturation, serum lactate and urine output.

Close monitoring for postoperative atrial fibrillation is necessary, although the reported incidence is considerably lower after TEVAR (9%) (20) than after open TAAA repair or routine cardiac surgery (approximately 23% for both) (21). Postoperative atrial fibrillation may cause hypotension secondary to loss of atrial kick, which in turn can cause SCD. Preventive strategies include electrolyte monitoring and correction. Rate control with atrioventricular nodal agents can risk hypotension and SCD, so a cautious approach is advised; we prefer amiodarone for aggressive rhythm control. For TEVAR patients, who often have preexisting respiratory disease, amiodarone during the index hospitalization is well tolerated and unlikely to produce pulmonary toxicity (22). Cardioversion may be appropriate for patients experiencing prolonged or symptomatic dysrhythmias. Sustained postoperative atrial fibrillation may warrant anticoagulation, however this decision is individualized, with recent preference leaning towards use of direct oral anticoagulants.

Post-TEVAR vascular examinations should be conducted in the operating room and postoperatively. Major vascular complications can occur in up to 12% of patients with iliofemoral vascular size discrepancies (23). These complications have been associated with a 13% mortality rate and require prompt recognition for prevention and optimal management. Early intervention may be necessary for limb salvage to avoid compartment syndrome. Ischemic complications can be caused by arterial thrombosis, embolism, aortic dissection or endograft malpositioning (24). Device-related complications (endoleaks, endograft migration or collapse, kinking or stenosis of an endograft limb, graft infection) require vigilant monitoring, as 19–24% of cases require some type of secondary intervention (24). Finally, retrograde type A aortic dissection after TEVAR occurs in 1–3% of cases and is associated with a mortality rate of almost 40% (25,26).

Postimplantation syndrome is a unique complication of endovascular repair that occurs in 13–60% of patients (24). Thought to be an inflammatory, immune-mediated response, it is characterized by fever, leukocytosis and elevated levels of inflammatory markers such as C-reactive protein. Pleural effusions have been noted in 37–73% of TEVAR cases (27). Treatment is largely supportive (aspirin and surveillance); antibiotics have not been proven beneficial.

Renal considerations

Preventing AKI is a major priority after TEVAR. Endovascular TAAA repair was associated with a high rate (32%) of postoperative renal insufficiency, defined as a 50% increase in serum creatinine (28) consistent with moderate AKI, which compromises survival (29).

The incidence of AKI in open TAAA repair series ranges from 5–29% (depending on how AKI is defined), with 4–17% needing renal replacement therapy (RRT) (29). In TEVAR, the published incidence of AKI is approximately 10%, with less need for RRT (30). In severe AKI, RRT is initiated for volume overload, refractory acidemia or electrolyte abnormalities (31). Fundamental AKI management guidelines include routine preventive strategies (e.g., multidisciplinary rounding with pharmacists) (32).

The risk of contrast nephropathy after TEVAR is related to higher contrast volume and aortic dissection extending into the renal arteries (33). To reduce this risk, intravenous hydration is administered before imaging commences (34). Due to the risk of hyperchloremic metabolic acidosis, normal saline is avoided in favor of isotonic solutions for volume resuscitation.

Meticulous care of vascular access catheters is necessary to reduce the risk of catheter-associated bloodstream infections that could infect the TEVAR graft. For this reason, peripherally inserted central catheter lines are avoided in patients with AKI (35). When longer-term RRT appears necessary, conversion to a tunneled dialysis catheter can reduce the infection risk associated with temporary catheters. Future dialysis access needs should be considered in patients with chronic kidney disease (*Table 1*).

Gastrointestinal and nutritional considerations

Most patients are able to tolerate a regular diet within 24 hours. Patients who undergo TEVAR for TAAA or TBAD occasionally benefit from continued nasogastric tube drainage after extubation. With TBAD, careful vigilance is needed to monitor for gut malperfusion (36), as indicators (e.g., acidemia, increased intravenous fluid requirements) may be subtle. In patients who continue to be mechanically

ventilated, enteral feeds are begun 48–72 hours after surgery. Routine stress ulcer prophylaxis should be administered, and gastrointestinal bleeding not responding to higher-dose proton-pump inhibitors or lavage should be evaluated endoscopically.

Bleeding and hematological considerations

Early postoperative bleeding from femoral access sites can induce hemorrhagic shock. An unexplained drop in hemoglobin may necessitate CT imaging to identify hemorrhage in the chest or abdomen, especially if hypotension persists. Vigilance is necessary, and early return to the operating room for exploration, evacuation and surgical control may be needed.

For patients who were taking warfarin for previous mechanical valve prostheses, we typically resume warfarin 2–3 days postoperatively without bridging anticoagulation, given the potential risk for bleeding from the CSF drainage site. For patients who may need additional procedures (e.g., tracheostomy) due to a prolonged ICU course, unfractionated heparin is started 48–72 hours after the CSF drain is removed. Oral anticoagulation is resumed when no further surgical procedures will be needed. Chemical prophylaxis against venous thromboembolism is initiated 24 hours after CSF drain removal. Aspirin is started the day of CSF drain removal, whereas $P2Y_{12}$ receptor blockers are resumed 24 hours after drain removal.

Miscellaneous considerations

Early mobility and removal of intravascular or urinary catheters is encouraged. Postoperative aortic imaging provides a baseline for ongoing surveillance but is best done after serum creatinine level normalizes. Nonetheless, more than half of TEVAR patients are lost to follow-up and not compliant with routine surveillance imaging (37). Continued communication with primary and referring cardiologists is necessary to ensure optimal long-term survival.

Conclusions

The prevention, early detection and appropriate management of post-TEVAR complications depends on diligent, multidisciplinary care. Close attention across the perioperative, intraoperative and postoperative treatment phases makes achieving successful outcomes more likely.

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Footnote

Conflicts of Interest: Dr. SC has participated in advisory boards for Edwards Lifesciences & La Jolla Pharmaceutical Corp. Dr. OP provides consultation for and participates in clinical trials with Medtronic and W.L. Gore & Associates. Dr. JSC participates in clinical trials with and/or consults for Terumo Aortic, Medtronic, and W.L. Gore & Associates and receives royalties and grant support from Terumo Aortic. The other author has no conflicts of interest to declare.

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