Case Report

Thoracic aorta aneurysm open repair in heart transplant recipient; the anesthesiologist's perspective

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ABSTRACT

Many years following transplantation, heart transplant recipients may require noncardiac major surgeries. Anesthesia in such patients may be challenging due to physiological and pharmacological problems regarding allograft denervation and difficult immunosuppressive management. Massive hemorrhage, hypoperfusion, renal, respiratory failure, and infections are some of the most frequent complications related to thoracic aorta aneurysm repair. Understanding how to optimize hemodynamic and infectious risks may have a substantial impact on the outcome. This case report aims at discussing risk stratification and anesthetic management of a 54-year-old heart transplant female recipient, affected by Marfan syndrome, undergoing thoracic aorta aneurysm repair.

Received: 24-07-15
Accepted: 06-10-15

Key words: Heart transplant recipient; Open repair; Thoracic aortic aneurysm

Access this article online

Website: www.annals.in

PMID: ***

DOI: ***

Quick Response Code:

INTRODUCTION

Non-cardiac surgical procedures in heart transplant recipients are becoming more numerous. Reported below is the first management description of a heart transplant recipient undergoing thoraco-abdominal aortic aneurysm repair, a procedure at high risk of morbidity and mortality. The anaesthetist's management together with a thorough knowledge of the underlying pathophysiological mechanisms are key aspects for an effective outcome. We took the cue from this individual patient to analyze tips and tricks for future anaesthesiologists facing such challenging cases.

CASE REPORT

A 54-year-old Marfan syndrome female was admitted to our department due to large (>7 cm) symptomatic dissecting aneurysm involving the thoracic aorta. Her history reveals acute type A aortic dissection 9 years earlier, with consequent aortic valve and ascending aorta replacement. Myocardial infarction and end-stage heart failure rapidly developed leading to orthotopic heart transplantation 2 years later.

Preoperative assessment

The patient was not a candidate for endovascular repair, lacking an adequate proximal aneurysm neck, and was listed for an open procedure [Figure 1].

Preoperative assessment included echocardiographic evaluation which reported mild left ventricular hypertrophy, normal function but altered diastolic

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Figure 1: Parasagittal angio-computed tomography of a 55-year-old Marfan patient with previous repair of type A dissection and orthotropic heart transplantation. The residual dissection of the descending thoracic aorta underwent gross aneurysmatic dilatation which is now over 7 cm in diameter and tender. There is no suitable neck for endovascular repair

pattern (ventricular septum 11 mm, posterior wall 10 mm, end-diastolic dimension 44 mm, end-systolic dimension 29 mm, tissue Doppler velocity 110 mL, telesystolic volume 48 mL, and left ventricular ejection fraction 56%); severe left atrial dilation (volume 100 mL); mild mitral regurgitation due to annular dilation and leaflet fibrosis; right atrial dilation, mild tricuspid insufficiency, normal right ventricular function (24 mm), and pulmonary pressures (30 mmHg).

Coronary angiography was performed with 60% stenosis of the left anterior descending artery. No other coronary artery had significant stenoses. Functional impact of the stenosis was not assessed, with dobutamine stress-test or computed tomography (CT), due to lack of adequate timing before surgery and overall assessment as not hemodynamically severe.

Brain CT, chest X-ray, functional respiratory evaluation, and carotid duplex scan were all negative.

Surgical intervention

A thoracic epidural catheter was placed (T7-8) for analgesia; an arterial access was gained cannulating the right humeral artery. General anesthesia was induced using propofol 200 mg, fentanyl 200 mcg, and cis-atracurium 20 mg. Intubation was performed via direct laryngoscopy using a double-lumen orotracheal tube (DLT) (robertshaw 37). A high-flow catheter (Fr12) was placed in the right internal jugular vein, together with a pulmonary arterial catheter (PAC) sheath (Fr8.5), and a quadri-lumen catheter in the right subclavian vein. Transesophageal echocardiographic (TEE) probe was also inserted.

Anesthesia maintenance was obtained with desflurane, Et 6–7%, and top-up 2 mg cis-atracurium every 60 min. Fenoldopam was infused for kidney protection and low-dose epinephrine, in the postclamp period, to maintain adequate cardiovascular performance.

Thoracic aorta aneurysm (TAA) repair was performed with left heart bypass (LHB) (biomedics centrifugal pump, superior left pulmonary vein to left femoral artery); the aorta was clamped proximally between left common carotid and left subclavian arteries. A 24 mm Dacron tube graft was used to replace the aneurysmal aorta.

Total intraoperative blood loss was 5500 mL, replaced by blood salvage and transfusion of packed red blood cells (800 mL) and fresh frozen plasma (1500 mL). No adverse events were recorded.

Postoperative setting

Due to significant blood loss (2000 mL), on the first postoperative day, from the left thoracic drainage, the patient was reopened, a hematoma was removed and all surgical anastomoses and possible bleeding sites were thoroughly checked. The patient then required low-dose epinephrine (maximum 0.02 mcg/kg/min) for approximately 18 h, and upon weaning transthoracic echocardiography was performed confirming good bi-ventricular function. Weaning from mechanical ventilation occurred 43 h from first intensive care unit (ICU) admission.

A clinical picture of acute-on-chronic kidney injury developed in the first few days, with spontaneous normalization of serum creatinine and a constantly maintained urinary output. Overall, the patient spent 3 days in ICU, was then sent to the vascular surgery ward, and on the eighth postoperative day was transferred to a rehabilitation center. At 6 months from the surgical procedure, the patient is alive and well.

DISCUSSION

Heart transplant recipients are greatly increasing worldwide with reported 90% and 50% survival at 1 and 5 years, respectively.[1] The prolonged life expectancy, linked to new pharmacological and interventional approaches, creates a new category of patients at increased risk of requiring noncardiac surgical procedures.[2] Cardiac transplant recipients represent

a unique challenge to nontransplant anesthesiologists for the altered physiology and increased complication rate.[3,4]

Risk stratification

Heart-transplant patients develop cardiac allograft vasculopathy, due to progressive and diffuse intimal hyperplasia that leads to coronary artery disease.[5] It is, therefore, advisable to perform adequate evaluation in the preoperative setting, with coronary angiography and functional tests. Brain CT is strongly suggested when, as in the present report, a history of neurological events is present. Chest X-ray is routinely performed to check the presence of tracheal distortion, which can lead to difficult orotrcheal intubation, atelectasis, or pneumonia. Functional respiratory evaluation can predict difficult oxygenation during one-lung ventilation. The findings of preoperative carotid stenosis, usually detected with carotid duplex scan, require higher mean arterial pressure during surgery, and improving brain perfusion.

Induction of anesthesia

A transplanted heart loses sympathetic, parasympathetic, and sensory innervation while preserving intrinsic cardiac mechanisms, making the heart strictly dependent on starling's pressure-volume relationship. Therefore, optimization of preload is essential to counteract vasodilation and maintain adequate stroke volume, as performed in our setting, with 1000 mL of crystalloid.

Monitoring

During TAA surgery, large volume shifts are expected and invasive hemodynamic monitoring and constant TEE imaging control are mandatory. For these reasons, we cannulated the humeral artery for invasive blood pressure measurement and an expert anesthesiologist in TEE was present during the surgery. We strongly recommend adding TEE monitoring to PAC in TAA repair with LHB because it allows more accurate assessment of biventricular function, new regional wall motion abnormalities, and optimized preload.

Airways management

A DLT was used to avoid intraprocedural dislocations. One-lung ventilation in TAA repair improves surgical exposure and decreases pulmonary contusion due to lung retraction, which may progress to hemorrhage during heparin administration. However, large TAAs may cause distortion of the left main bronchus rendering DLT placement difficult; in such cases a bronchial blocker or right-sided DLT can be possible options, and require correct positioning assessment with fiber-optic bronchoscope.

Anesthetic management during the surgery

Loss of vagal influence results in increased resting heart values, 90–110 bpm approximately; on the contrary, tachycardia in response to stressful stimuli (tracheal intubation, inadequate anesthetic depth, pain, and hypovolemia) is weakened, therefore adequate analgesic coverage throughout the entire procedure is of maximal importance.[6] In the present report, we adopted epidural analgesia with the aim of achieving excellent analgesia both during surgery and postoperatively.

A few concerns regarding the altered pharmacodynamics: Reinnervation of transplanted heart muscle may occur, in a casual manner, leading to an altered and unpredictable response to some drugs.[7] This is the case of epinephrine and norepinephrine that have enhanced inotropic effect with respect to vasoconstrictor one. Therefore, epinephrine is effective in such patients with heart failure to improve contractility;[7] in our patient low-dose epinephrine was administered for moderate left ventricular dysfunction. On the contrary, atropine is not effective in increasing heart rate, and when neostigmine is administered as neuromuscular blocker (NMB) reversal agent, at the end of surgery, it can lead to bradycardia and asystole. This seems to be due to allograft parasympathetic reinnervation, intrinsic sinoatrial node dysfunction, and denervation hypersensitivity of muscarinic myocardial receptors to neostigmine.[8] Thus, the use of rocuronium and subsequent NMB reversal with sugammadex should be considered when an early extubation is planned.[9] In our patient, we decided for cis-atracurium during surgery because of chronic renal failure, and the admission to ICU after TAA without NMB reversal.

Furthermore, in these patients, the risk of arrhythmias remains high related both to denervation and intrinsic conduction abnormalities, especially atrioventricular blocks and right bundle branch block. Therefore, in our patient, a temporary pacemaker was inserted via the femoral vein to treat possible brady-arrhythmias.

Postoperative period

Immunosuppressive therapy requires appropriate management, gaining an acceptable equilibrium between the risk of rejection and the risk of developing infections.

To conclude, the following aspects should be taken into consideration for a positive and straightforward perioperative outcome:

- Preoperative risk stratification of allograft vasculopathy
- Intraoperative TEE-directed hemodynamic optimization
- Prevention and prompt treatment of brady-arrhythmias using both intracardiac and epicardial temporary pacemakers
- Minimization of immunosuppressive regimen interruption and careful evaluation of early signs of infection (temperature and white blood cells count).

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Taylor DO, Edwards LB, Boucek MM, Trulock EP, Waltz DA, Keck BM, *et al*. Registry of the International Society for Heart and Lung Transplantation: Twenty-third official adult heart transplantation report-2006. J Heart Lung Transplant 2006;25:869-79.
2. Valerio R Jr, Durra O, Gold ME. Anesthetic considerations for an adult heart transplant recipient undergoing noncardiac surgery: A case report. AANA J 2014;82:293-9.
3. Blasco LM, Parameshwar J, Vuylsteke A. Anaesthesia for noncardiac surgery in the heart transplant recipient. Curr Opin Anaesthesiol 2009;22:109-13.
4. Kostopanagiotou G, Smyrniotis V, Arkadopoulos N, Theodoraki K, Papadimitriou L, Papadimitriou J. Anesthetic and perioperative management of adult transplant recipients in nontransplant surgery. Anesth Analg 1999;89:613-22.
5. Prada-Delgado O, Estévez-Loureiro R, Paniagua-Martín MJ, López-Sainz A, Crespo-Leiro MG. Prevalence and prognostic value of cardiac allograft vasculopathy 1 year after heart transplantation according to the ISHLT recommended nomenclature. J Heart Lung Transplant 2012;31:332-3.
6. Spann JC, Van Meter C. Cardiac transplantation. Surg Clin North Am 1998;78:679-90.
7. Bristow MR. The surgically denervated, transplanted human heart. Circulation 1990;82:658-60.
8. Beebe DS, Shumway SJ, Maddock R. Sinus arrest after intravenous neostigmine in two heart transplant recipients. Anesth Analg 1994;78:779-82.
9. Gómez-Ríos MÁ, López LR. Use of combination of rocuronium and sugammadex in heart transplant recipients. Anaesth Intensive Care 2012;40:903-4.