Laparoscopic cholecystectomy in a cardiac transplant recipient

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ABSTRACT

An increasing number of cardiac transplants are being carried out around the world. With increasing longevity, these patients present a unique challenge to non-transplant anesthesiologists for a variety of transplant related or incidental surgeries. The general considerations related to a cardiac transplant recipient are the physiological and pharmacological problems of allograft denervation, the side-effects of immunosuppression, the risk of infection and the potential for rejection. A thorough understanding of the physiology of a denervated heart, need for direct vasoactive agents and post-transplant morbidities is essential in anesthetic management of such a patient. Here, we describe a case of a heart transplant recipient who presented for a cholecystectomy at our center.

Key words: *Heart transplantation, immunosuppression, post-transplant surgery, recipient, transplant*

INTRODUCTION

The number of patients receiving organ transplants is increasing around the world and in India. The Registry of the International Society of Heart and Lung Transplantation estimates that the number of heart transplants being performed world-wide exceeds 5000/year.[1] With newer and better immunosuppression, improvement in surgical technique and advancement in organ preservation, the lifespan of these patients is constantly improving. With increasing longevity, cardiac transplant recipients are more likely to be operated for incidental non cardiac, transplant or immunosuppression related surgery. These patients present a unique challenge to non-transplant anesthesiologists.

Here, we present a cardiac transplant recipient who came for a cholecystectomy at our center.

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CASE REPORT

A 43-year-old male weighing 85 kg was admitted with symptomatic cholelithiasis for laparoscopic cholecystectomy. He had undergone a cardiac transplant in 2004 in South Africa after viral myocarditis induced cardiomyopathy and heart failure. He was asymptomatic as regards his cardiac symptoms and was on 3 drug oral maintenance immunosuppressive therapy with tacrolimus, mycophenolate mofetil and prednisolone with normal serum drug levels. Yearly angiography and myocardial biopsies had shown no abnormality. He was on amlodipine and perindopril for control of hypertension. Physical examination showed a pulse rate of 86 beats/min and a blood pressure of 130/80 mmHg. Airway examination revealed no abnormality. Heart sounds were normal with no murmurs. Investigations showed a normal complete blood count, normal fasting and post-prandial blood sugar levels, normal liver profile except for a raised Sr. Amylase (129 U/L) raised creatinine (1.5 mg/dl) with normal electrolytes. Electrocardiogram (ECG), X-ray chest and pulmonary function tests showed no abnormality. Echocardiography showed an ejection fraction of 55% with the increase in left atrial and right atrial size. Dobutamine stress echocardiography showed no evidence of inducible ischemia.

Pre-operatively patient was given a proton pump inhibitor the night before and on the morning of surgery.

Immunosuppressant and antihypertensive medication was administered orally on the morning of surgery and hydrocortisone 100 mg was given intravenously. Antibiotic prophylaxis was started prior to surgery. Monitoring consisted of 5 lead ECG with ST (S wave, T wave) segment monitoring, left radial arterial blood pressure, plethysmography, capnography, bisbectral index (BIS) and neuromuscular monitoring (TOF-train of four). Large bore intravenous access was secured. Anesthesia was induced with fentanyl 200 mcg and midazolam 3 mg intravenously followed by etomidate titrated to loss of consciousness followed by atracurium 50 mg. Cuffed endotracheal tube and orogastric tubes were placed thereafter. Anesthesia was maintained with sevoflurane in an oxygen air (FiO2-0.5) mixture titrated to keep BIS between 40 and 60. With the creation of pneumoperitoneum a hypertensive response was seen which was tackled with fentanyl 50 mcg i.v. No subsequent doses of muscle relaxant were required and the patient was stable hemodynamically. At the completion of surgery, patient was warm and awake with a TOF ratio >0.9. We decided against the administration of neostigmine considering case reports of neostigmine induced bradycardia and asystole. Extubation and recovery were uneventful and patient was discharged on the 3rd day after surgery.

DISCUSSION

Anesthesia for cardiac transplant recipients involves understanding the post-transplant morbidities, using the direct vasoactive agents and the multiple side-effects of immunosuppressive regimens.

By 10 years after cardiac transplantation, surviving recipients have hypertension (97%), severe renal insufficiency (14%), hyperlipidemia (93%), diabetes (39%) and angiographic coronary artery vasculopathy (CAV) (52%).[1] Rejection presents with a decreasing functional capacity, malaise and fever. Endomyocardial biopsy confirms the diagnosis.[1,2] CAV, an accelerated form of atherosclerosis, has an 8% incidence at 1 year, 30% at 5 years and more than 50% at 10 years. Angiography and intravascular ultrasound are used for annual surveillance and a pre-operative dobutamine stress echocardiography is a better predictor of subsequent cardiac events.[1,2]

Maintenance immunosuppression regimens usually consist of two or more drugs from the following 3 classes: Calcineurin inhibitors (cyclosporine and tacrolimus), antimetabolites (azathioprine [AZA] and mycophenolate mofetil) and corticosteroids.

Cyclosporine (and to a lesser extent tacrolimus) causes nephrotoxicity, hypertension, hyperlipidemia, hyperglycemia, tremor, paresthesias, cholelithiasis and osteoporosis. Cyclosporine also potentiates the neuromuscular block produced by vecuronium and atracurium.[3] Side-effects of AZA are myelosuppression, pancreatitis, hepatitis, hepatic veno-occlusive disease and skin malignancy. Mycophenolate mofetil causes less myelosuppression. Steroids have the largest number of long-term adverse effects including hypertension, peptic ulcers, myopathy, weight gain, hyperlipidemia, salt and water retention, hyperglycemia and osteopenia.[4]

The transplanted heart has no sympathetic, parasympathetic or sensory innervation with any response to carotid sinus massage or valsalva maneuver. Loss of vagal tone results in a higher resting heart rate (91-101 bpm).[3] There is a lag period in the hemodynamic response because cardiac output is increased secondary to catecholamine release rather than neural reflexes. Exaggerated responses to catecholamines have been observed, which are attributed to the increase in receptor density (up-regulation) and a greater proportion of available local catecholamines, which are not removed by neuronal uptake.[5] Epinephrine and norepinephrine have an augmented inotropic effect. Isoproterenol and dobutamine are more effective inotropes than dopamine in the denervated heart. Indirectly acting drugs have blunted responses on blood pressure and heart rate. Atropine is ineffective in increasing the heart rate.[3]

Pre-operative assessment and investigations should include a thorough work-up of cardiac function, adequacy of immunosuppression and its effects on other organ function. All pre-operative drug therapy should be continued. If a pacemaker is present, its proper functioning should be confirmed. A transcutaneous or transvenous pacemaker, defibrillator should be immediately available. Antibiotic prophylaxis and strict asepsis have immense importance. Invasive monitoring is not indicated for short minor surgical procedures. However because these patients are preload dependent, invasive hemodynamic monitoring is useful in major surgeries with fluid shifts.[3] General anesthesia is generally preferred due to the impaired response to hypotension with spinal or epidural anesthesia. The goal of anesthesia is to avoid significant vasodilatation and acute decrease in preload.[3] In laparoscopic surgeries, pneumoperitoneum causes an increase in central venous pressure and pulmonary artery occlusion pressure, but no significant change in cardiac index or systemic vascular resistance and is well-tolerated in patients with normal left ventricular function.[5,6] There are multiple case reports of neostigmine induced bradycardia and asystole,[7-8,9,1011] which could be due to some degree of parasympathetic reinnnervation[7] or denervation supersensitivity to the cholinergic agonist effect of neostigmine.[9] Ongoing episode of rejection increases the chances of arrhythmias

with neostigmine.[8] Therefore, avoiding the use of muscle relaxants where antagonism of the block will be necessary or using short acting agents that permit adequate recovery of neuromuscular function should be considered[8] with close monitoring and availability of resuscitative equipment.

CONCLUSION

Cardiac transplant recipients can safely undergo a surgical procedure. With 9 year survival exceeding 50%, we as anesthesiologists are very likely to encounter such patients. With a thorough understanding of the denervated heart, appropriate pharmacology, judicious use of invasive lines and strict asepsis, these patients present an acceptable anesthetic risk.

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