

Anaesthesia for non-cardiac surgery in a cardiac transplant recipient

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ABSTRACT

Cardiac transplantation has become the standard therapy for idiopathic dilated cardiomyopathy and end-stage ischaemic heart disease. With the introduction of newer immunosuppressants, together with better patient selection, improved perioperative monitoring and care, the overall survival of recipients has improved. An increasing number of patients who received a transplant present for either elective or emergency non-cardiac surgery. We hereby discuss the perioperative management of such a patient who came to our set-up for bipolar haemiartroplasty.

Key words: Anaesthesia, cardiac transplant, graft function, immunosuppressants, non-cardiac surgery

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INTRODUCTION

Ever since the first human cardiac transplant was performed in 1967 by Christian Bernard, now, the average frequency of this procedure is approximately 1% of the population with heart failure.^[1,2] With improved graft function and survival, these patients are now presenting to hospitals for various non-cardiac procedures. The information regarding the physiological and pharmacological interactions in a denervated allograft heart, the side-effects of immunosuppression, the risk of infection and the potential for rejection is essential to the anaesthetist managing such patients in hospitals that are not otherwise involved in transplantation procedures.^[3] Following these lines, we now describe the perioperative management of a patient with cardiac transplant who came to our hospital for bipolar haemiartroplasty.

CASE REPORT

A 57-year-old male patient of average height and built, weighing 65 kg, sustained intertrochantric fracture of the left femur and was posted for bipolar haemiartroplasty. Other than the current orthopaedic

problem, he was diabetic type-2 for 5 years on NPH insulin with good control. He was a documented carrier of hepatitis-B antigen, on chronic Lamivudine (Hepatovir) therapy. He had undergone orthotopic heart transplant 3 years back. Presently, he was NYHA-II. He was on immunosuppressant – FK-506 (Prograf-tacrolimus) and Mycophenolate (Cellcept) – in addition to Diltiazem and pantoprazole. On admission, a complete haemogram, coagulation profile and all biochemical parameters (including hepatic and renal functions, lipid profile, electrolytes) were checked and found to be normal. His echocardiogram showed ejection fraction of 55% with mild diastolic dysfunction and no post-transplant complication like tricuspid regurgitation. He was undergoing regular myocardial biopsies, the last being done 3 months back, with no documented evidence of graft rejection. On the day of surgery, the patient was given his morning doses of immunosuppressants, insulin and diltiazem. After a standard fasting period of 8 h, he was premedicated with alprazolam 0.25 mg and pantoprazole 40 mg orally. Antibiotic prophylaxis (Inj. Ceftriaxone with sulbactam 1.5 g IV) was given 30 min prior to surgery. Standard monitoring with electrocardiogram (ECG), pulse oximetry and end tidal carbon dioxide (ETCO₂) was established. For beat to beat blood pressure and

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central venous pressure monitoring, the radial artery and subclavian vein, respectively, were cannulated. The patient handling and all invasive lines were performed taking strict aseptic precautions. BIS monitoring was also undertaken to ensure early recovery from anaesthesia and to keep tight control over the use of anaesthetic drugs. Pre-induction, his opening CVP was 4 mmHg, heart rate (HR) was 85/min and blood pressure (BP) was 142/80 mmHg. CVP was built-up to 8 mmHg after preloading with infusion of 1 L normal saline. An opioid-based general anaesthesia was planned for the patient. An epidural catheter was inserted in the L2-L3 space. Inj. morphine 3 mg was given through an epidural catheter for perioperative analgesia. Nor-epinephrine and isoprenaline infusions were kept ready before the start of anaesthesia. After administration of morphine (0.10 mg/kg), fentanyl (3 µg/kg) and midazolam (0.02 mg/kg), anaesthesia was induced with titrated doses of Inj propofol (1 mg/kg) and maintained with isoflurane (0.2–0.6%), nitrous oxide and oxygen mixture (60:40). The patient was intubated after achieving adequate muscle relaxation with Inj. vecuronium (0.15 mg/kg) and put on IPPV and surgery commenced in the right lateral position. Throughout the surgery, we encountered only two hypotensive episodes. The first was immediately post induction, when the HR decreased to 74/min and the BP fell to 108/76 mmHg. This hypotensive episode responded to a fluid bolus of about 200 ml normal saline. In addition, we gave Inj. mephentermine 6 mg, but its effect became apparent only after 90 s. The second episode occurred with blood loss during long bone reaming, which was managed with an infusion of crystalloid and colloid without vasopressor. The surgery lasted for 3.5 h. HR strictly remained in the range of 74–76 beats/min and no dysarrhythmias were noted perioperatively. CVP was maintained between 8 and 10 mmHg, Bispectral index was kept within the range of 40–60, with isoflurane titration and intermittent fentanyl boluses (1 µg/kg). Blood sugar was monitored and maintained between 120 and 150 mg %. ABG was also normal. At the end of surgery, the patient was reversed with neostigmine (0.05 mg/kg) and atropine (0.02 mg/kg) and extubated after return of airway reflexes. After emergence from anaesthesia, his HR again picked up to 84–85/min. Inj. morphine 3 mg was again given, through an epidural catheter, 10 h after the first dose for post-operative analgesia. DVT prophylaxis was given with subcutaneous LMW heparin. The patient was kept in

the Intensive Care Unit for 24 h and discharged after 7 days of uneventful stay in hospital.

DISCUSSION

Pre-operative assessment of any transplant recipient undergoing non-cardiac surgery should focus on graft function and rejection, risks of infection and function of other organs, particularly those that may be compromised due to either immunosuppressive therapy or dysfunction of the transplanted organ itself and drug interactions.^[4] The patient was assessed for any evidence of late graft rejection, as high morbidity has been noted if surgery is performed during the rejection period.^[5] His previous myocardial biopsy was normal. Echo showed a well-functioning graft with good LV function and no regional wall motion abnormality thus ruling out any late-onset vasculopathy. There was no post-transplant complication like tricuspid regurgitation, which is reported to be as high as 47–98% following heart transplantation.^[2] Dysrhythmias, probably due to a lack of vagal tone, rejection and increased endogenous catecholamine concentrations, can occur in over 50% of the patients.^[6] The sinus node may have an increased refractory period and atrial conduction may be prolonged. Thus, first-degree atrioventricular block and right bundle-branch block are common. As many as 20% of the heart-transplant patients may require a pacemaker for bradyarrhythmias.

A brief review of the pathophysiology of heart transplant would be necessary to understand its anaesthetic management. The transplanted heart has no sympathetic, parasympathetic or sensory innervation, and the loss of vagal influence results in a higher than normal resting HR.^[4] Unlike the normal heart, which increases cardiac output via neural stimuli causing increased HR and contractility, the denervated heart lacks the ability to respond acutely to hypovolaemia or hypotension with reflex tachycardia but responds to stress primarily by an increase in stroke volume by circulating catecholamines. The increase in cardiac output is dependent on venous return, with an initial increase in left ventricular end-diastolic volume, which mediates an increase in stroke volume and ejection fraction by means of the Frank-Starling mechanism. That is why heart transplant patients are said to be “preload dependent.”^[6] Keeping this goal of avoiding acute vasodilatation, hypotension and hypovolemia, sole central neuraxial blockade was avoided and general anaesthesia was preferred, although a variety of

anaesthetic techniques (general, regional, neuroleptic) have been successfully used in patients with a transplant history.^[7,8] Slow induction and titrated doses of anaesthetic agents and fluid pre-loading also helped in smooth induction of anaesthesia. Although an epidural catheter was inserted, no local anaesthetic bolus was administered, for obvious reasons. Only two episodes of hypotension (post-induction and during femur reaming) were noticed, which responded well to intravenous fluids and mephentermine bolus. Another implication of loss of neural control is blunted chronotropic response due to sympathetic stimulation secondary to hypoxia, hypercarbia, hypotension, laryngoscopy and inadequate anaesthetic depth.^[9] Therefore, we monitored BIS, blood gases and sugar throughout the surgery. In the transplanted heart, the HR shows no response to drugs like muscle relaxants (pancuronium, gallamine), anticholinergics (atropine, glycopyrrolate and scopolamine) and anticholinesterases (neostigmine, edrophonium, pyridostigmine, physostigmine). Our patient's awake HR was 82–85/min and, under anaesthesia, it remained 74–76/min. Because vagolytic drugs such as atropine are ineffective in increasing HR, other positive chronotropic and direct beta-adrenergic stimulating (ephedrine, isoproterenol) drugs should be readily available. Epinephrine and norepinephrine have an augmented inotropic effect in heart transplant recipients.

In transplant recipients, immunosuppressive drugs in common use are cyclosporine A, azathioprine, antilymphocyte globulin, monoclonal antibodies and steroids. Newer drugs such as tacrolimus (FK506) and mycophenolate mofetil are replacing cyclosporine A and azathioprine, respectively. The side-effects of immunosuppressives that have a direct impact on anaesthetic and perioperative management are anaemia, leucopaenia, thrombocytopaenia, hyperkalaemia, hypomagnesaemia, hypertension, diabetes, neurotoxicity, renal insufficiency, anaphylaxis and fever. Immunosuppressed patients are at risk of infections that may be bacterial, viral, fungal or protozoal.^[4] It is imperative to realize that the immunosuppressed patient does not present with the typical signs and symptoms of sepsis – fever and leucocytosis. A very high index of suspicion is therefore required. Our patient was a carrier of hepatitis B, but

was on chronic lamivudine therapy. Lamivudine is a nucleoside reverse transcriptase inhibitor and it works by acting as a chain terminator of DNA synthesis, thereby stopping the spread of hepatitis B virus. The high incidence of fatal infections with invasive lines outweighs the benefits derived from invasive monitoring. But, our surgical procedure involved major blood loss and large volume shifts and chances of air or fat embolism during long bone reaming; hence, such invasiveness was warranted for haemodynamic management. Appropriate perioperative antibiotic prophylaxis should be used. To prevent post-operative sepsis, our post-operative care included aggressive respiratory care, aseptic wound handling and early post-operative removal of CVP/arterial lines, epidural and urinary catheters.

CONCLUSION

In general, if the transplanted heart is functioning satisfactorily, these patients present few problems in elective, acute or even emergency non-cardiac surgery, provided the anaesthetist has some understanding of the pathophysiology of the transplanted and denervated heart.

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