

CASE REPORT

Anesthetic management of a patient with dilated cardiomyopathy and end stage renal disease

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ABSTRACT

Cardiovascular disease is the leading cause of mortality among patients with end stage kidney disease (ESKD). Left ventricular hypertrophy (LVH) and left ventricular dilation (LVD) are independent risk factors for mortality and make the management of a patient with dilated cardiomyopathy (DCM) and ESKD stage 5. undergoing noncardiac surgery is a real challenging task as the perioperative course may be complicated by cardiac arrhythmias or cardiac failure at any time and associated with high perioperative morbidity and mortality. An adequate knowledge of the pathophysiology of these diseases and treatment modalities is essential to manage these cases successfully. Meticulous planning is the key to success. We stress that adequate preoperative preparation and a planned anesthesia leads to a successful management of dilated cardiomyopathy with ESKD.

Key words: Cardiomyopathy, Dilated; End stage kidney disease; Anesthesia; General anesthesia management

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INTRODUCTION

Patients with ESKD are at extreme cardiovascular risk. At least half of all patients starting dialysis therapy have overt cardiovascular disease such as new onset heart failure, peripheral vascular disease, ischemic heart disease and stroke. High level exposure to traditional risk factors such as smoking and dyslipidemia, hemodynamic overload and metabolic factors related to uremia all likely may play a role.¹ Cardiovascular disease accounts for over 40% of deaths in ESKD patients.² In general patients with even mild chronic kidney disease have greater burden of incident CVD compared with similar age control.³ There are various cardiovascular abnormalities that are commonly encountered in patients with chronic kidney disease or ESRD; these include LVH, LV dilatation, and LV systolic and diastolic dysfunction.⁴ We want to highlight the points related to anesthesia concern by reporting this case with dilated cardiomyopathy

and ESKD undergoing an emergency laparotomy for strangulated inguinal hernia.

CASE REPORT

A 33 year old male, weighing 55 kg, a known case of DCM with ESKD on multiple hemodialysis since 2 years, presented for emergency laparotomy with diagnosis of strangulated hernia. He had undergone dialysis 3 days back and was on following medications; torsemide 100 mg, clonidine 10 mg, moxonidine 0.3 mg, losartan 50 mg and prazosin 10 mg. On pre-anesthesia checkup his heart rate (HR) was 170/min and blood pressure (BP) was 202/140 mmHg. His RR was 25-30/min. Bilateral basal crepitations were present on systemic examination. Fresh ABG's showed pO₂ 83.1 mmHg, pCO₂ 34.3 mmHg, pH 7.44, HCO₃ 22.9 mEq/L, SaO₂ 96.6%. Arterio-venous fistula was seen on left cubital fossa. Preoperative investigations were; Hb was 9.7 g/dl, TLC 10,000 /mm³, Platelet count

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2,62000 /mm³, RBS 88 mg/dl, serum Na⁺ 135 mEq/L, K⁺ 4.2 mEq/L, Ca²⁺ 8.5 mg/dl, coagulation profile within normal limits, BU 143, creatinine 13.4 and creatinine clearance was <15 ml/min.

12 Lead ECG showed sinus tachycardia and severe LVH. A 2D-echo showed hypertensive heart disease, cardiomyopathy, global left ventricular hypokinesia, severe concentric hypertrophy, severe left ventricular systolic dysfunction, and an ejection fraction 25-30 %. Left atrium and ventricles were dilated. Chest x-ray revealed cardiomegaly with CT ratio 0.52 and bilateral pleural effusion. The patient's abdominal USG showed small and scarred kidneys, moderate amount of ascites with component of cystitis. Cardiology and nephrology consultations were taken. High risk consent and consent for post-operative ICU care was obtained. With the intention to optimize our patient preoperatively, inj fentanyl and inj metoprolol were administered IV slowly. After 10 mins of administering drugs HR and BP came down to 128/min and 150/100 mmHg respectively.

In OR standard monitors were attached. Under local anesthesia, an arterial line (right radial) and central venous pressure (CVP) line (in right internal jugular vein) were passed. Antiarrhythmic drugs (amiodarone, digoxin, lignocaine), vasodilators, vasopressors (adrenaline and noradrenaline) and dopamine were kept ready along with stand-by defibrillator. After preoxygenation with 100% oxygen for 3 min, general anesthesia was induced with a repeat dose of fentanyl 50 µg, etomidate 15 mg and atracurium 25 mg. Lignocaine 60 mg was used to blunt intubation response. Trachea was intubated with cuffed ETT 8 mm ID. General anesthesia was maintained with O₂/N₂O, isoflurane and atracurium. HR, ECG, invasive BP, SpO₂, EtCO₂, CVP, urine output, temperature, SE and ABG's were monitored and corrected. Surgery lasted for 60 min with a total blood loss of 350 ml. Metoprolol 2 mg (in graded dose of 1 mg) was used to keep heart rate below 100/min. Nitroglycerine (NTG) infusion was titrated (0.5-2 µg/kg/min) to maintain blood pressure around 140/90 mmHg and intra-operative CVP was kept between 7-10 cmH₂O with 0.9% normal saline and one unit of packed red blood cell. Urine output was 50 ml. Paracetamol 1 gm was given IV for postoperative pain relief. Blood pressure remained stable on NTG infusion. After completion of surgery, patient was shifted to intensive care unit for elective ventilation and monitoring with further cardiologic evaluation. In ICU patient was given fluids guided by CVP and

NTG infusion was titrated and stopped gradually. Trachea was extubated after 48 h, then he was shifted to HDU of surgery unit and subsequently discharged on twice weekly hemodialysis.

DISCUSSION

The aim of this case report is to provide brief review of the management goals with particular attention given on the management of perioperative arrhythmias and prevent further complications intended to get a good outcome. In the absence of intrinsic heart disease, LV enlargement is most probably attributable to chronic volume/flow overload associated with 3 principal factors: Anemia, the presence of arteriovenous shunts and sodium water retention.⁵ Along with these factors, hypertension which is leading cause of LVH⁶, is also a common and challenging entity, for anesthesiologist to manage. All these findings are present in our patient, so particular emphasis was given on: careful induction of general anesthesia, avoidance of myocardial depression, maintain normovolemia and normothermia, reduction in ventricular preload and afterload, avoidance of intra-operative tachycardia and hypertension, avoidance of hypoxia, hypercarbia and metabolic acidosis, avoidance of nephrotoxic drugs, maintenance of adequate renal perfusion pressure, avoidance of drugs requiring renal metabolism for termination of action which might worsen the clinical condition of the patient. For induction of general anesthesia inj etomidate had been chosen as it is like ketamine and narcotics having minimal depressive effects on cardiac function. During maintenance of anesthesia, the dose dependent direct myocardial depression produced by volatile anesthetics must be considered. It is recommended that fluid therapy and pharmacological management be guided by the use of pulmonary artery catheter and determination of cardiac filling pressure but this facility was not available in our hospital during emergency services, so we relied on CVP that was monitored throughout the perioperative period to avoid volume overload. We used NTG infusion mainly to reduce afterload, it is a venodilator at low doses and arterial dilator at a high dose, lowering intracardiac pressure and alleviating pulmonary congestion. It also dilates coronary arteries making it useful in patients with heart failure and myocardial infarction (MI). Lignocaine, amiodarone and/or defibrillation must be used to treat any arrhythmias if required⁷, which usually occur due to reduced levels of K⁺ and Mg²⁺ that should be assessed and

corrected pre-operatively. For this reason we kept these drugs and defibrillator ready by the side.

Patients with CKD surgical stimulation may produce undesirable increases in heart rate, which may be treated with beta-antagonists such as metoprolol or esmolol, keeping in mind the potential for these drugs to cause cardiac depression. Adequate oxygenation and ventilation were closely monitored with SpO₂ and EtCO₂. Metabolic acidosis is very common in CKD patients but initiation of dialysis typically results in improvement of metabolic acidosis due to addition of base load delivered in dialysate. As we had already put an arterial line, ABG monitoring during ICU stay became easy. Increased sympathetic nervous system activity in form of tachycardia and hypertension is present in the vast majority of these patients and plays a key role in progressive deterioration of renal function and in the exceedingly high rate of cardiovascular events, which represent the primary cause of morbidity and mortality in this patient group.

Anemia (Hb levels 6-12 g/dl) is an independent predictor of mortality in dialysis patients,¹ therefore, a Hb level equivalent to 13-14 g/dl has been recommended.⁸ Cardiac output may be improved by inotropes, biventricular synchronized

pacings or an aortic balloon pump.

Regional anesthesia for example epidural anesthesia, may be an alternative to general anesthesia in selected patients. Clinical experience is limited, however and caution is indicated to avoid an abrupt onset of blockade of sympathetic nervous system innervation. Since the patient's general condition was very poor with signs of pulmonary hypertension, uncontrolled hypertension and tachycardia, we relied on general anesthesia. With meticulous monitoring, judicious use of required drugs and proper knowledge, general anesthesia can be induced safely with improved outcome.

CONCLUSION

During perioperative period, various important factors should be kept in mind before giving anesthesia to a patient with DCM complicated with ESKD; therefore, we must be aware of pathophysiology, signs and symptoms, diagnostic evaluations and treatment modalities to manage these cases successfully.

Conflicts of interests: None

Authors' contribution: Both authors took equal part in the management of the case and the manuscript preparation.

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