Journal of Clinical Research in Pain and Anaesthesia

Case Report *Volume 1; Issue 3*

Anaesthetic Management of a Patient with Severe Tricuspid Regurgitation Undergoing Live Donor Kidney Transplantation

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Received Date: August 05, 2019; **Published Date:** August 13, 2019

Abstract

Background*:* Isolated severe structural tricuspid regurgitation (TR) is a rare condition and intraoperative worsening of TR led to management dilemmas during live donor kidney transplantation (KT).

Case report*:* We are discussing anaesthetic management of a patient with isolated structural TR who underwent live donor kidney transplantation. Intraoperative use of noradrenaline caused significant derangements in haemodynamics and bradycardia produced prevented accurate noninvasive blood pressure monitoring.

Conclusion*:* A pure vasoconstrictor like noradrenaline causes adverse haemodynamic effects when used to increase blood pressure in a patient with severe TR.

Keywords: Severe TR; Pulmonary vascular resistance; Central venous pressure; Volume overload; Pressure overload; Noradrenaline; Dobutamine

Abbreviations: HD: Hemodialysis; TR: Tricuspid Regurgitation; TRPG: TR Pressure Gradient; ECG: Electro Cardio Gram; BP: Blood Pressure; HR: Heart Rate; PVR: Pulmonary Vascular Resistance; PPV: Positive Pressure Ventilation; RV: Right Ventricular; CVP: Central Venous Pressure.

Introduction

Tricuspid regurgitation (TR) could be functional due to right ventricular dilatation caused by cor pulmonale, myocardial infarction or pulmonary hypertension. Structural TR may occur with rheumatic heart disease, infective endocarditis, carcinoid syndrome, Ebstein's anomaly or due to chest trauma [1,2]. Isolated structural TR is a rare condition [2]. Currently no literature is available on incidence of TR in chronic kidney disease patients.

Preoperative assessment

Patient was a 46-year-old male, retired army officer with essential hypertension for 5 years duration. He was diagnosed to have chronic kidney disease in 2016 secondary to hypertension and gradually progressed to end stage kidney disease. He was haemodialysis (HD) dependent for one year's duration. HD was done via an A-

Citation: Rachini KW and Anuja Abayadeera. Anaesthetic Management of a Patient with Severe Tricuspid Regurgitation Undergoing Live Donor Kidney Transplantation. J Clin Res Pain Anaesthesia 2019, 1(3): 180013.

V fistula created on left upper limb, twice a week without any major complications. He did not give a history or symptoms suggestive of ischaemic heart disease, ureamic cardiomyopathy, pericardial effusion or heart failure. He had no symptoms of gastro-oeosophageal reflux disease. He was on erythropoietin treatment for the last one year's duration. He had no features suggestive of peripheral/ autonomic neuropathy or renal bone disease. He had good exercise tolerance of > 4 METs.

He had no other co-morbidities or significant surgical history. He was on prazosin 5mg t.d.s, methyldopa 250mg t.d.s, metoprolol 50mg b.d, atorvastatin 10mg nocte, $NaHCO_3$ 600mg t.d.s, frusemide 40mg b.d, $CaCO_3$ 500mg t.d.s, pantoprazole 40mg b.d, allopurinol 50mg b.d, $FeSO_4$ and folic acid supplementation. He was a non-smoker and non-alcoholic.

On examination his BMI was 23.3 kg/m^2. He had mild bilateral ankle oedema. Jugular venous pressure was not elevated. Pulse rate (PR) - 54/min, regular pulse, Blood pressure (BP) - 140/80 mmHg. On auscultation a pansystolic murmur was heard over left sternal border which increases with inspiration. Bilateral air entry was normal with no added sounds. He did not have tender hepatomegaly.

ECG showed sinus bradycardia. 2-D echocardiogram revealed ejection fraction of 50%, septal dyskinesia, mild impaired LV function. He was also found to have severe TR and mild AR. TR pressure gradient (TRPG) was 17mmHg. Right atrium and right ventricles were dilated with good right ventricular function. Trans-oesophageal echocardiogram was performed by cardiology team to exclude sinus venosus ASD as a cause for TR. All the other investigations were normal. HLA mismatching was 3/6. Both T and B cell matching were compatible.

Preoperative management

Informed written consent was obtained after explaining the risks and benefits. HD was done on day prior to surgery. Recommended fasting guidelines were adhered to. Routine antihypertensives and other drugs were continued.

Intraoperative management

Monitoring of ECG with ST analysis, noninvasive BP, S_PO_2 were established. AVF was protected. Patient was induced with i.v. morphine 6mg, propofol 120mg and atracurium 40mg. He was intubated with size 8mm endotracheal tube and volume-controlled ventilation with tidal volume of 6ml/kg at a rate of 12/min was started. Anaesthesia was maintained with 1.2% isoflurane, 30% O_2 and air. Paralysis was maintained with i.v. atracurium boluses as indicated. Antibiotic prophylaxis with i.v. *meropenem* 1g was given. Patient was catheterized. Right side TAP block was given under ultrasound guidance with 0.25% plain bupivacaine 20ml. Right internal jugular central venous line was inserted under ultra sound guidance. Initial CVP was noted as 17mmHg. His BP was maintained around pre-operative BP and heart rate (HR) was persistently 45-50/min. $EtCO_2$ was maintained 35-40mmHg. Ringer's lactate 2000ml was infused intravenously before perfusion of the kidney. With the fluid management gradual rise in CVP was noted. Intravenous methylprednisolone 1g infusion was started. Noradrenaline intravenous infusion was started to maintain MAP> 90mmHg to ensure adequate renal perfusion.

A significant bradycardia and a rise in CVP up to 40mmHg were noticed. This made noninvasive BP measurement difficult which required right radial artery cannulation and invasive BP monitoring. Dobutamine infusion was started and noradrenaline was tailed off. MAP was maintained with dobutamine $5\mu g.kg^{-1}.min^{-1}$. With start of dobutamine HR improved up to 65-70/min and CVP gradually decreased. By the end of surgery CVP was almost at initial starting value. Patient was extubated on table without any complications and was transferred to intensive care unit. After extubation CVP was 11mmHg. Post-operative period was uneventful.

Discussion

TR may result from structural alterations of any or all of the components of the tricuspid valve apparatus. Isolated TR is a rare condition. This patient had an isolated TR diagnosed as structural by the cardiology team with a cause not found. In TR, backward flow occurs through the tricuspid valve from right ventricle to right atrium during ventricular contraction. The regurgitant volume combines with normal RA volume and returns to the RV during each diastole. This result in RV volume overloads [1]. Severe TR can cause both RA and RV dilatation.

RV can well compensate for volume overload. Therefore, isolated TR is well tolerated [3]. This patient was asymptomatic. On the other hand, a pressure load is not well tolerated by the RV. Most symptoms associated with TR are directly related to an increased RV afterload which is determined by pulmonary vascular resistance. Therefore, when TR is associated with pulmonary hypertension, the impedance to RV ejection produces significant clinical deterioration from decreased right

ventricular stroke volume and cardiac output [4]. This patient had a pulmonary arterial pressure of 27mmHg which was determined by TRPG. Pulmonary hypertension is defined as pulmonary arterial pressure of > 30mmHg.

Our aims during anaesthesia were to prevent further increase in TR, maintain adequate right ventricular stroke volume and to prevent right ventricular failure.

To provide adequate forward flow, preload augmentation is desirable. Tachycardia is desirable to decrease diastolic time and hence regurgitation duration. A low pulmonary vascular resistance (PVR) should be maintained to reduce right ventricular (RV) afterload thereby facilitating forward flow and reducing RV workload. Positive pressure ventilation (PPV) and peak end expiratory pressure increases PVR. Other factors which increase PVR are hypoxia, hypercarbia, acidosis and pain which were avoided. Systemic vascular resistance has little effect on TR [3].

RV failure is the primary cause for clinical deterioration in patients with TR. Because the RV is designed geometrically to accommodate volume but not pressure loads, it may require perioperative inotropic support, especially in the setting of PPV or elevated PVR. Any suppression of contractility with myocardial depressants may induce severe RV failure [3].

In view of KT, adequate preload is important to maintain renal perfusion. In the setting of severe TR, CVP becomes an unreliable indicator of fluid status. Due to backward flows through the tricuspid valve this patient had a high CVP of 17mmHg at start which further increased by administration of intravenous fluids. Noradrenaline causes pulmonary vasoconstriction which increases RV afterload. This increases RV pressure, further increasing CVP. Noradrenaline causes venocontriction which increases preload as well. Both factors contributed for high CVP observed during intraoperative period. With the significant rise in CVP up to 40mmHg there was a risk of right ventricular failure.

This patient was on metoprolol (started as an anti-hypertensive) which was the possible cause for bradycardia which was worsened by the administration of noradrenaline. The bradycardia worsened TR and caused difficulty in measuring noninvasive BP as the oscillometer relies upon a regular cardiac cycle, with no great differences between successive pulses [5].

Dobutamine is an inodilator which reduces pulmonary vascular resistance and improves right ventricular contractility. It also has chronotropic effects. All these factors favour dobutamine as a good option compared to noradrenaline to achieve an adequate BP in the setting of severe TR [2].

Conclusion

Knowledge of pressure and volume dynamics of TR would have helped to manage this patient without the dilemmas we faced. We learnt a lesson to think of the haemodynamics in using vasoactive agents.

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