Case Report

Anaesthetic management of a patient with severe pulmonary arterial hypertension for renal transplantation

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Access this article online

Website: www.ijaweb.org

DOI: 10.4103/0019-5049.199854

Quick response code

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ABSTRACT

We describe a patient with severe pulmonary arterial hypertension scheduled to undergo live-related renal transplantation. We emphasise on meticulous anaesthetic management and early renal transplantation to prevent the progression of disease which would become refractory to treatment, leading to right ventricular failure. Regional (continuous epidural) anaesthesia has been used as technique of choice, where the selective advantages of this technique have been put to good use.

Key words: Anaesthesia, end-stage renal disease, pulmonary arterial hypertension, renal transplantation

INTRODUCTION

End-stage renal disease (ESRD) is associated with higher incidence of pulmonary arterial hypertension (PAH), leading to increased chances of mortality, morbidity and early rejection in patients slated for renal transplantation.[1-4] The prevalence of PAH among the patients receiving long-term haemodialysis (HD) ranges from 16% to 58%.[4,5] Early renal transplantation is required in these patients as the pro-inflammatory proteins and oxidative stress markers are not adequately excreted through HD, which is the cause for cardiovascular compromise.[6] In this article, we report management of a patient with PAH for renal transplantation and stress on the need for early renal transplantation and tailored anaesthetic management.

CASE REPORT

A 51-year-old Nigerian male with ESRD and associated systemic hypertension belonging to New York Heart Association Functional Classification III was scheduled for live-related renal transplantation. He was on regular HD thrice weekly through left radio-cephalic arteriovenous (AV) fistula. On routine pre-transplant evaluation, his body mass index was 23 kg/m^2. His resting blood pressure was 220/110 mmHg and he was receiving multiple oral antihypertensives including amlodipine 10 mg twice daily, clonidine 0.2 mg four times daily, telmisartan 40 mg once daily, prazosin 5 mg twice daily and metoprolol 50 mg twice daily. He had features suggestive of right heart failure such as raised jugular venous pressure, hepatosplenomegaly, moderate ascites and basal crepitations. Breath holding time was 20 s and oxygen saturation was 91% on room air. His electrocardiogram revealed right bundle branch block. Echocardiography showed

How to cite this article: Chidananda Swamy MN, Mukherjee A, Rao LL, Pandith S. Anaesthetic management of a patient with severe pulmonary arterial hypertension for renal transplantation. Indian J Anaesth 2017;61:167-9.

severe concentric left ventricular hypertrophy, Grade I left ventricular diastolic dysfunction with pulmonary arterial systolic pressure (PASP) 70 mmHg and left ventricular ejection fraction (LVEF) 55%. His contrast tomogram pulmonary angiogram showed mildly dilated central pulmonary arteries with no evidence of pulmonary thrombo-embolism. Pulmonary function test revealed severe restrictive and obstructive pulmonary disease. Pre-operative lower limb Doppler revealed normal flow in the iliac vessels. In addition, our patient was retroviral positive on anti-retroviral therapy (ART). All haematological and biochemical parameters were within normal range.

Following a detailed evaluation by a multidisciplinary team which included the nephrologist, transplant surgeons, anaesthesiologist, cardiologist and pulmonologist, renal transplantation was planned under epidural anaesthesia. After connecting standard monitors, intravenous (IV) access was secured in the right upper limb. With patient in sitting position, under local anaesthesia, an 18-gauge epidural catheter was placed in T9–T10 interspace. Graded epidural anaesthesia was commenced with 5 ml boluses of 0.5% bupivacaine. A total of 10 mL was given over 15 min, after which epidural infusion of 0.25% bupivacaine and 2 μg/mL fentanyl was started at 3–5 mL/h. Adequate padding was done to protect AV fistula and other pressure points.

Right radial artery and right internal jugular vein were cannulated with 20-gauge arterial catheter and triple lumen 7.5 Fr catheter, respectively under local anaesthesia with ultrasound guidance. A loading dose of dexmedetomidine 1 μg/kg was infused over 20 min and then titrated at 0.2–0.5 μg/kg/h to reach a Ramsay Sedation Score 4. Systemic and pulmonary hypertensions were managed with nitroglycerin (5–10 μg/min) and milrinone (0.375 μg/kg/min) infusion titrated to keep target mean arterial pressure around 90 mmHg. Intraoperative blood loss was about 300 mL and 2 L of balanced salt solution was transfused to maintain central venous pressure around 8–10 mmHg. The rest of the intraoperative course was uneventful. Dexmedetomidine and nitroglycerin infusions were stopped at the end of surgery, and he was shifted to the Intensive Care Unit. Epidural infusion of 0.125% with 2 μg/mL of fentanyl was continued into the post-operative period for pain management.

On the 1st post-operative day (POD_1), echocardiography showed PASP 45 mmHg. Milrinone infusion was stopped on day 2. On day 6, saturation on room air was 98% and echocardiography showed PASP 25 mmHg and LVEF 50%. He was discharged on POD 10.

DISCUSSION

PAH is defined as persistent elevation of the mean pulmonary arterial pressure (mPAP) >25 mmHg at rest or >35 mmHg with exercise.[7] mPAP of 25–40 mmHg is classified as mild PAH, 41–55 mmHg is moderate PAH and >55 mmHg is severe PAH.[8] Pulmonary hypertension is seen in patients with ESRD and the causes are said to be prolonged duration of renal replacement therapy, extra-osseous pulmonary calcification, high cardiac output due to AV fistula, anaemia, fluid overload, hormonal and metabolic imbalances associated with uraemia and impaired endothelial function.[9]

Human immunodeficiency virus-related PAH is a rare complication of HIV infection and the incidence is estimated to be 0.5%,[10] which is approximately 2500 times greater than that of primary pulmonary hypertension (PPH) in the general population.[11] Highly active ART down-regulates viral replication leading to a decrease in abnormal T-cell activation.[12] ART by decreasing chronic stimulation of the immune system might also decrease the (still unknown) driving force behind the development of PPH.[13]

In ESRD, there is significant elevation of the pro-inflammatory proteins interleukin-6, tumour necrosis factor-α and C-reactive protein. In addition, oxidative stress markers such as plasma protein carbonyls and F_2-isoprostanesare are also elevated. HD is ineffective in controlling the levels of these factors thus leading to significant rise in pulmonary pressure. After renal transplantation, due to the use of immunosuppressant and restoration of normal kidney function, these levels decline over a period of 2 months. This leads to significant improvement in pulmonary functions and pulmonary artery pressure.[14]

Our patient underwent dialysis through AV fistula and had high cardiac output in a non-compliant heart, hypertrophied left ventricle and enlarged left atrium with pulmonary congestion, which has led to inevitable severe pulmonary hypertension.[15]

Medical line of management for pulmonary hypertension in ESRD has minimal role, and thus, it was decided to treat the cause surgically. Recommended intraoperative treatment for pulmonary hypertension are inhaled nitric oxide at 10–40 ppm, milrinone (phosphodiesterase 3 inhibitor) used as infusion of 0.25–0.75 μg/kg/min (initial 50 μg/kg bolus), inhaled epoprostenol (continuous) 10–50 ng/kg/min and IV prostacyclin 4–10 ng/kg/min.

Our anaesthetic goals in this patient were to maintain systemic vascular resistance and optimal preload to the heart, to maintain sinus rhythm and to avoid rise in mPAP due to hypoxaemia, hypercarbia and pain.

In severe pulmonary hypertension, anaesthetic technique of choice should be regional wherever possible. Direct laryngoscopy during general anaesthesia causes increased sympathetic outflow leading to increased pulmonary vascular resistance and thereby precipitating acute right heart failure. Over use of narcotics, inhalational agent or muscle relaxants to control sympathetic response can inadvertently reduce cardiac output and hence coronary perfusion.

We could successfully manage this patient with severe pulmonary hypertension using epidural anaesthesia and dexmedetomidine infusion to sedate and allay anxiety. To control his pulmonary vascular resistance, adequate ventilation and oxygenation were ensured, and milrinone infusion in titrated doses was instituted till POD 2. Following renal transplantation, our patient showed significant decrease in mPAP from 70 mmHg to 25 mmHg over a period of 6 days.

CONCLUSION

Regional anaesthesia either alone or in combination with plays an important role in the anaesthetic management of complex patients with pulmonary hypertension presenting for renal transplantation.

Acknowledgement

We would like to thank the patient and his family.

We would also like to thank Dr Anil Kumar, Senior Consultant, Department of Nephrology, BGS Global Hospitals, Bengaluru in helping us in the preparation of manuscript and publication.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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