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Case Report

Impact of renal transplantation on severe tricuspid regurgitation and pulmonary hypertension in end-stage renal disease

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ABSTRACT

The report presents a case of a patient with severe pulmonary arterial hypertension (PAH) and severe tricuspid regurgitation (TR) who was scheduled for a living donor renal transplantation. The focus of this report is on the importance of meticulous anaesthetic management and the impact of renal transplantation on the improvement of PAH and severe TR. Patient was a known case of end stage renal disease with grade 2 medical renal disease, splenomegaly and cirrhosis. The patient was monitored by assessment of stroke volume (SV), stroke volume variation (SVV), systemic vascular resistance (SVR), cardiac output (CO), cardiac index (CI) and CVP through Flo-trac to evaluate hemodynamic stability and guide appropriate management. Intraoperative course was uneventfull. After extubation, on Postoperative day 3, Milrinone infusion was stopped and 2D echocardiography was done which showed moderate PASP -54mm Hg, moderate TR, with normal LVEF. Further on Postoperative day 10, PASP came down to 40mm Hg with Mild TR and normal LVEF. Renal function was further improved and s. creatine became 1.14.

Keywords: Kidney transplantation, Pulmonary hypertension, Tricuspid regurgitation, Chronic kidney failure, Mean arterial pressure, Systemic vascular resistance

INTRODUCTION

End-stage renal disease (ESRD) is strongly associated with an elevated incidence of pulmonary arterial hypertension (PAH), which significantly contributes to increased mortality, morbidity, and a higher risk of early graft rejection in patients undergoing renal transplantation.[1-4]

Pulmonary arterial hypertension (PAH) represents a severe and progressive cardiac complication that has garnered considerable attention, being frequently observed in individuals with ESRD. In patients with chronic kidney disease (CKD), the estimated prevalence of pulmonary hypertension (PH) ranges from approximately 12% to 45%.[5]

The mechanisms underlying the development of PAH in ESRD remain subjects of ongoing investigation. Several key mechanisms have been proposed, including:[6-8] Pulmonary vascular stiffness and vasoconstriction observed in ESRD patients. An elevated cardiac output resulting from factors such as anaemia, hypervolemia, and the creation of an arteriovenous (AV) fistula.

The incidence of pulmonary hypertension (PH) is notably higher in patients undergoing haemodialysis (HD) compared to those receiving peritoneal dialysis.[9-11] Perioperative management in these patients, particularly those undergoing non-cardiac surgery, is critical to optimize outcomes. PH resulting from aetiologies other than left-sided heart disease, such as idiopathic pulmonary arterial hypertension (PAH) and PAH secondary to chronic kidney disease (CKD), may cause the

development of tricuspid regurgitation (TR) due to increased right ventricular (RV) afterload, RV dilation, and dysfunction.[12] The most prevalent form of functional tricuspid regurgitation (FTR) is closely associated with PH.

CASE REPORT

A 21year old male, weighing 54 kg with ESRD with systemic hypertension belonging to NYHA grade IV was scheduled for renal transplantation. He was on maintenance hemodialysis twice a week through left arteriovenous fistula (AV) since 2018. He had history of orthopnea and paroxysmal nocturnal dyspnea (PND).

Patient had features of right heart failure such as increased Jugular Venous Pressure (JVP), hepatosplenomegaly, mild ascites, pedal edema and fine crepts.

On pre anesthetic checkup his resting blood pressure was 160/90mm Hg and he was taking multiple oral antihypertensives including Tab Clonidine 0.1mg thrice a day, Tab Nicardipine 20mg once a day, Tab Metoprolol XL 50mg twice a day, Tab Prazocin XL 10mg twice a day. He was on Erythropoietin and Iron therapy for anemia. Patient was HCV positive (viral load 1462) since 2020, has taken treatment for 3months (tab Sofosbuvir 400 mg and tab Velpatasvir 100 mg).

His investigations were as follows- Hb- 8 gm/dl,

TLC- 6,160, Platelet count- 68,000, S. Urea / S. Creatinine- 77/7.09. Other investigations like Liver Function Test, S. Na/K- 135/3.69, PT/INR- 13.6/ 1.06, S. albumin- 3.94 and Blood sugar and thyroid profile was within normal limits. ECG showed right ventricular hypertrophy, right axis deviation, P pulmonale and tall R waves in lead V1. Echocardiography revealed concentric LVH, mild MR, trivial AR, mild PR, severe TR, severe PAH, PASP-84 mm Hg, LV diastolic dysfunction grade III, LVEF-60%. no regional wall motion abnormality detected. On chest X ray we found straightening of right heart border and cardiomegaly.

Pulmonary function test revealed mixed pattern with FEV1/FVC-90%, FEV1-46%, PEF-30%. Color Doppler USG revealed right sided patent communication between common femoral artery and vein. On USG whole abdomen, we found grade 2 medical renal disease with small right kidney, hepatomegaly with grade 1 fatty changes, splenomegaly and mild ascites. Upper GI endoscopy revealed gastroduodenitis. On fibro scan cirrhosis was present (14/F4) and was advised increased risk of ascites and hepatic encephalopathy in post operative period by the gastromedicine specialist.

We advised incentive spirometry, steam inhalation and nebulization with duolin and budecort for optimization of pulmonary function. The patient was advised to continue all antihypertensive medication till morning of surgery except tab prazosin which was stopped a day before surgery.

Following a detailed evaluation by a multidisciplinary team which included the nephrologist, transplant surgeon, anesthesiologists, cardiologist, pulmonologist, renal transplantation was scheduled under general anesthesia. After seeking reference from Cardiologist, they advised same treatment and added Tab Dytor 40 mg and conveyed to get the patient in high-risk consent. Patient was transfused 1unit of SDP and 1 unit of PRBC one day prior to surgery.

After taking high risk and postoperative ventilatory consent, patient was shifted to operative room. Invasive arterial monitoring in radial artery and central venous catheter was secured in right IJV.

The patient was monitored with Flo Trac monitor and the stroke volume (SV), stroke volume variation (SVV), systemic vascular resistance (SVR), cardiac output (CO), cardiac index (CI) and CVP were assessed to evaluate hemodynamic stability and guide appropriate treatment to augment vascular volume, reduce anesthetic administration or use of vasopressors or inotropes.

After preoxygenation for 3 min, inj Midazolam 2 mg iv, inj Fentanyl 2.5 mcg/kg body weight iv was given. Patient was induced with inj Etomidate 0.3 mg/kg body weight and was intubated with inj Cis atracurium 0.2 mg/kg body weight with 8 mm ETT and was put on mechanical ventilation with Volume control mode (VCM) having TV-400, RR-14, I: E-1:2, PEEP-5. The anesthesia was maintained with 50% Oxygen+ 50 % air, isoflurane and intermittent boluses of fentanyl and cis-atracurium. Fluid administration was managed with the help of Vigileo-Flotrac monitor assessing SVR, SVV and CO. Intraoperative blood loss was about 500 ml and 2 l of balanced salt solution was transfused. Systemic and Pulmonary Hypertension were managed with inj Nitroglycerin (5-10mcg/min) and inj Milrinone (0.375mcg/kg/min) infusion to keep MAP around 110mm Hg with normal ranges of SVR and CO. Serial ABG monitoring was done and found to be normal or near normal. Rest of the intraoperative period was uneventful. After anastomosis of vessels, urine output was started immediately and continued perioperatively.

Patient was extubated with reversal agent (neostigmine and glycopyrrolate) He was haemodynamically stable with pulse rate 90/min, BP -180/90 mm Hg, Sp02–100% on room air. Patient was then shifted to nephrology ICU with infusion of inj Milrinone.

Post transplantation day 1, renal function improved and creatinine went down to 1.51 with sufficient urine output. Dyspnea, chest congestion and fluid overload subsided.

On postoperative day 3, milrinone infusion was stopped and 2D echocardiography was done which showed

moderate PASP -54mm Hg, moderate TR, with normal LVEF. Further on Postoperative day 10, PASP came down to 40mm Hg with Mild TR and normal LVEF. Renal function was further improved and s. creatine became 1.14 (Table 1).

Table 1: Investigations of patients.

Date	2021-08-09	2021-09-10	2021-09-18	2021-09-19	2021-09-20	2021-09-21	2021-09-22	2021-09-23	2021-09-24	2021-09-27
Hb	10.4	11.6	11.2	11.3		10.8	10	9.7	10.7	10.4
TLC	6150	9100	11200	10900		12500	12400	10800	8500	10000
Platelet	68	106	102	95		100	90	104	132	143
PT/INR	13.8/1.06	14.2/1.3	15.6/1.3	13.8/1.02						
Na/K	135/3.7	130/4.4	133/4.1	131/4.2		132/3.8	128/3.7	130/3.75	133/3.7	135/3.6
Urea/Creatinine	31/4.2	38/2	44/1.8	76/1.8		68/2.2	76/2.5	79/2.24	78/1.9	71/1.6
LFT	-	-	-	-	-	-	-	-	-	-
S. Bilirubin T/D	0.76/.16	-	0.23/.12	-	0.21/0.14	-	-	-	-	0.28/0.18
SGOT	27	-	24	-	48	-	-	-	-	42
SGPT	35	-	31.9	-	30	-	-	-	-	35
ALP	95	-	190	-	161	-	-	-	-	146
S. Albumin	3.94	-	3.6	-	3.8	-	-	-	-	3.9

DISCUSSION

PAH is characterized by a persistent elevation in the mean pulmonary arterial pressure (mPAP), defined as greater than 25 mmHg at rest or greater than 35 mmHg with exercise.[13] PAH is classified based on the mPAP as follows: mild PAH (25–40 mmHg), moderate PAH (41–55 mmHg), and severe PAH (>55 mmHg).[14] In patients with ESRD, impaired endothelial function also plays a key role in the pathogenesis of PAH in these patients.[15] In patients with ESRD, there is a marked increase in pro-inflammatory biomarkers, such as interleukin-6, tumor necrosis factor-alpha (TNF-α), and C-reactive protein, alongside elevated oxidative stress markers like plasma protein carbonyls and F2-isoprostanes. HD does not adequately reduce these factors, contributing to persistently high pulmonary arterial pressure. After renal transplantation, immunosuppressive therapy and restoration of normal renal function led to a gradual decrease in these biomarkers over approximately two months, resulting in significant improvements in pulmonary function and pulmonary artery pressure.[10]

FTR results from structural changes in the right heart, leading to dilation of the tricuspid annulus and/or tethering of the leaflets, which impairs proper leaflet coaptation. PH from causes other than left-sided heart disease such as idiopathic PAH, pulmonary embolism, chronic pulmonary diseases, and ESRD can contribute to FTR by increasing RV afterload, causing RV dilation and dysfunction. Among these, PH is the most common underlying cause of FTR.[12]

In this particular case, our anaesthetic goals were focused on maintaining systemic SVR and optimal preload to the heart, ensuring sinus rhythm, and preventing an increase in mPAP due to hypoxia, hypercapnia, or pain. Our specific objectives during anaesthesia included preventing further exacerbation of TR, maintaining adequate right ventricular stroke volume, and avoiding RV failure. To optimize forward flow, preload augmentation was prioritized, and tachycardia was encouraged to reduce diastolic time, thereby limiting the duration of regurgitation. A low pulmonary vascular resistance (PVR) was maintained to reduce RV afterload, which in turn facilitated forward flow and alleviated RV workload.

In this patient, severe TR was most probably due to PH and was FTR and PH was due to ESRD due to various causes, discussed earlier. Therefore, after successful renal transplantation, on postoperative day 1, symptoms of fluid overload subsided. On postoperative day 3, PASP came down to 54 mmHg from 84 mm Hg preoperatively and TR becomes moderate on Echocardiographic findings. Finally at postoperative day 10, PASP comes down to 40 mm HG and TR became of mild state.

CONCLUSION

With this case report, it was conclude that severe FTR due to high PASP caused by ESRD can be reversed with successful cause removal that is renal transplantation.

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