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Perioperative anesthetic challenges in pulmonary endarterectomy for chronic thromboembolic pulmonary hypertension: A case series

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ABSTRACT

Pulmonary arterial hypertension as a result of the occlusion of the branches of the pulmonary vasculature is characterized by a rise in the mean pulmonary arterial pressure above 20 mmHg. Pulmonary endarterectomy is the preferred treatment modality for chronic thromboembolic pulmonary hypertension. Anesthetic challenges include management of right ventricular (RV) dysfunction, low baseline oxygen saturation, pre-operative deranged coagulation profile due to anticoagulant use, specific requirements for deep hypothermic circulatory arrest, and need for cerebral protection. Postoperatively, massive pulmonary hemorrhage, reperfusion pulmonary edema, residual pulmonary hypertension, and RV dysfunction may lead to prolonged intensive care unit stay and mechanical ventilation. We present a case series of the first seven endarterectomies in our institution, intending to highlight the perioperative anesthetic management of such patients.

Key words: Anesthetic management; Cardiopulmonary bypass; Endarterectomy; Pulmonary hypertension; Right ventricular dysfunction

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INTRODUCTION

Occlusion of either the major pulmonary arteries or smaller segmental and subsegmental arteries by post-embolic fibrotic material leads to chronic thromboembolic pulmonary hypertension (CTEPH)[1], incidence ranging between 0.1% and 11.8%[2,3] after an episode of acute pulmonary embolism. Despite proper anticoagulation for 3 months, the mean pulmonary artery pressure remains above 20 mmHg with a mismatched ventilation-perfusion (V/Q) scan.[4]

Pulmonary endarterectomy (PEA) remains the preferred treatment method for CTEPH with surgically accessible arterial occlusions.[5] The other treatment modalities include pulmonary arterial hypertension pharmacotherapy and balloon angioplasty[6] for those not deemed good surgical candidates. With a rise in the prevalence of this condition, more patients are likely to present for surgery. It is imperative that anesthetists should be aware of the pathophysiology, surgical technique, and potential complications associated with the procedure. We present a case series of the first seven PEA surgeries in our institution, intending to highlight the anesthetic management involved in the perioperative care of such patients.

CASE PRESENTATION

A thorough pre-operative assessment and diagnostic workup were carried out in all patients as listed in Table 1

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Table 1: Demographic parameters, presenting complaints, comorbidities, and pre-operative workup

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Age (years)	26	36	40	30	24	48	34
Gender	Male	Male	Male	Male	Male	Male	Male
Race	Indian	Indian	Indian	Indian	Indian	Indian	Indian
Diagnosis	CTEPH of left MPA, right descending interlobar artery with severe TR, Dilated RA/RV	CTEPH of RPA with severe PAH, Mild TR, LVEF-60%	CTEPH with RV thrombus and bilateral lower-limb DVT.	CTEPH\with severe TR with severe PAH	CTEPH with mass in RA and mild PAH	CTEPH of MPA extending to LPA with severe PAH	CTEPH, with severe TR with severe PAH
Profession/ Significant past history	Cook, history of standing for long periods	Nil	History of lower-limb DVT, IVC Filter placement	Traffic policeman by profession.	History of trauma necessitating bed-rest	Nil	History of hospitalization and ICU stay for pneumonia
Comorbidities	Right lower-limb varicose veins Impaired glucose tolerance (HbA1C-6.2) Hypothyroidism (TSH-4.76)	Right lower-limb chronic DVT, History of pulmonary Kochs, Hypothyroidism (TSH-9.87)	Right lower-limb DVT Post-IVC filter	Previous history of tuberculosis took full course of ATT	Morbid obesity	None	None
Anti-CCP/ANA/ RF/APLA/ CPKMB/beta2 -glycoprotein/ factor V leiden mutation/MTHFR	Normal	Normal	Inherited thrombophilia, protein C and protein S deficiency Protein C-40 (70-143) Protein S-41 (71-140) Factor V leiden mutation-None	Normal	Normal	Normal	Normal
Presenting complaints	Shortness of breath for 1 year with ordinary activity, walking on level ground B/L pedal edema	Shortness of breath and hemoptysis for 2 months, right pedal edema	Pedal edema and shortness of breath at rest after a symptom-free period of 2 years	shortness of breath and multiple episodes of hemoptysis for 3 months. History of occasional chest pain	History of shortness of breath for 10 days following an episode of trauma and bed rest.	Shortness of breath and chest pain on exertion for 3 years	Shortness of breath for 2 years
Pre-op NYHA class	II	III	IV	111	II	II	II
Post-op NYHA class	II	II	II	II	II	II	II
Pre-op room air SpO_2	88%	92%	92%	94%	95%	97%	97
Post-op room air SpO_2	90%	98%	97%	95%	96%	98%	96

(Contd,,,)

Table 1: ***(Continued)***

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Pre-op medication	Apixaban 2.5 mg Furosemide+ Spironolactone (10/50) ½ OD	Dabigatran 150 mg OD Torsemide+ Spironolactone (10+25 mg) Sildenafil 20 mg TDS	Warfarin 3 mg OD Riociguat 0.5 mg TDS Thrombolyzed with alteplase 15 days before surgery	Ambrisentan 5 mg OD Riociguat 1.5 mg BD Inj UFH 5000 U iv TDS	Tab Apixaban 5 mg BD	Torsemide+Spironolactone OD, Ambrisentan 5 mg Tadalafil 20 mg OD, Rivaroxaban 20 mg OD-stopped	Macitentan 10 mg OD Dytor 10 mg OD Rivaroxaban 20 mg OD Selexipag 200 mg TDS Riociguat 1 tds
Other blood investigations	D-Dimer-297 ng/mL NT-ProBNP-2696 Pg/mL	D-Dimer-356 ng/mL NT-ProBNP-3002 Pg/mL	D-Dimer-248 ng/mL NT-ProBNP-1165 Pg/mL	D-Dimer- 287 ng/mL NT-ProBNP-2202 Pg/mL	D-Dimer-344 ng/mL NT-ProBNP-1992 pg/mL	D-Dimer-302 ng/mL NT-ProBNP-1476 pg/mL	D-Dimer:326 ng/mL NT-ProBNP: 3460 Pg/mL
Pre-op ECG	NSR	S1Q3T3 pattern with RV strain	NSR	Sinus tachycardia, S1Q3T3 pattern with RV strain	NSR	NSR	NSR
Pre-op CXR	Cardiomegaly, blunting of left CP angle	Normal	Normal	Right lower zone haziness present.	Lung fields normal No cardiomegaly	Bilateral hilar prominence, no cardiomegaly	Normal lung fields No cardiomegaly
Pre-op transthoracic 2D echocardiography	Dilated RA/RV No RWMA Paradoxical IVS motion TAPSE 10 mm Tricuspid annulus 45 mm, Severe TR, RVSP=RAP+41 mmHg Pericardial effusion+	Moderate TR Severe PAH-RVSP=RAP+57 RA/RV dilated, TAPSE 13 mm Normal LV function, Tricuspid annulus 44 mm	Dilated RA/RV, RV thrombus present (2.3×2.3 cm) MPA >50% thrombus, saddle thrombus present, TAPSE 7 mm normal LV function.	Dilated RA/ RV, Severe TR, RVSP=RAP+62, LVEF=55–60%, TAPSE=12 mm, Intact IAS/IVS	Large RA thrombus (2.5×2.3 cm). No RWMA, mild TR, mild PAH Normal LV function	RA/RV dilated, moderate TR, RVSP=RAP+115, severe PAH, TAPSE=15 mm, RV systolic dysfunction+LVEF:60–65%	Dilated RA/RV, PA Severe TR RVSP=RAP+75 Jerky IVS, bulging towards left side. TAPSE=18 mm LVEF 55–60%
HRCT thorax	Atelectatic lesions in anterior basal segment of RLL, left lingular lobe, basal segment of left lower lobe. Subpleural thickening in anterior basal segment of RUL and RLL. Left pleural effusion	Large hypodense filling defect obliterating the lumen of the RPA and its branches The main pulmonary trunk and the LPA show normal homogenous opacification. Cavitating oblong infiltration of the right upper lobe. Mixed dense alveolar and ground glass infiltrates bilaterally. Mild left and minimal right pleural effusion.	Left lung cavitation, bronchiectatic changes, bilateral basal atelectasis	Both lungs show mosaic attenuation and right lower and middle lobe consolidation/ collapse.	Broad opacities towards pleura in the base of RLL with surrounding atelectatic changes. Obstruction of right pulmonary artery.	Consolidation with ground glass opacities in right lower lobe and few patchy areas of ground glass opacification in the left upper lobe.	Diffuse mosaic attenuation in bilateral lungs with fibrotic nodules and fibroatelectatic changes in the right lung

(Contd,,,)

Table 1: *(Continued)*

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Pre-op CTPA study (Figure 1)	Chronic occlusive thrombus in left main pulmonary artery Chronic recanalized thrombus in anterior segmental artery of RUL, lateral segmental artery of RML, superior segmental artery, posterior basal segmental artery, lateral basal segmental and subsegmental artery of RLL. Peripheral juxtapleural consolidation in lateral and anterior segment of RLL and lateral basal segment of LLL-suggestive of infarct. Cardiomegaly with pericardial effusion with RV/LV ratio >1 Left pleural collection	Grossly stable large occlusive thrombus in the right pulmonary artery extending into upper lobar artery and descending into interlobar arteries. Patchy ground glass opacities in lateral basal segment of left lobe. Small cavitation nodule in right upper lobe with subpleural nodule in right upper lobe.	Acute on chronic pulmonary thromboembolism with extension and involvement of pulmonary trunk, RA, RV, azygous vein and SVC thrombus, left empyema with cavitation and bronchiectatic changes	Dilated main pulmonary artery with non-visualization of left pulmonary trunk. Severe attenuation of distal most right pulmonary artery. CTEPH of LPA and it's branches and right interlobar artery and it's segmental branches of right middle and lower lobe with multiple MAPCAS.	Chronic pulmonary artery thromboembolism involving right distal main pulmonary artery extending into right lobar PA and segmental branches. RA mass (likely myxoma). Multiple subpleural wedge-shaped fibrotic bands.	Saddle-shaped non-enhancing hypodense filling defect within arterial wall noted at pulmonary artery bifurcation, and extending into left pulmonary artery, luminal narrowing-60%, extending into lower lobe segmental and subsegmental branches. Few dilated MAPCAs seen.	Non-enhancing intraluminal filling defect in distal part of RPA, segmental and subsegmental branches of right middle and lower lobes with eccentric wall thickening of right upper lobe segmental arteries and dilated main pulmonary artery

Anti-CCP: Anti-cyclic citrullinated peptide, ANA: Antinuclear antibody, APLA: Antiphospholipid antibody, BD: Twice daily dosing, CPKMB: Creatinine phosphokinase-myocardial band, CTEPH: Chronic thromboembolic pulmonary hypertension, CP angle: Costophrenic angle, CXR: Chest X-ray, CTPA: Computed tomography pulmonary angiogram, DVT: Deep vein thrombosis, DVT: Deep vein thrombosis, ECG: Electrocardiogram, HbA1C: Hemoglobin A1c (glycosylated hemoglobi), HRCT: High resolution computed tomography scan, IVC: Inferior vena cava, IVS: Interventricular septum, LPA: Left pulmonary artery, LV: Left ventricle, LVEF: Left ventricular ejection fraction, LLL: Left lower lobe, MTHFR: Methylenetetrahydrofolate reductase gene, MPA: Main pulmonary artery, NYHA: New York Heart Association, NT-ProBNP: N-terminal Pro B-type natriuretic peptide, OD: Once daily dosing, PAH: Pulmonary arterial hypertension, RA: Right atrium, RPA: Right pulmonary artery, RV: Right ventricle, RVSP: Right ventricular systolic pressure, RF: Rheumatoid factor, RWMA: Regional wall motion abnormality, RUL: Right upper lobe, RLL: Right lower lobe, RML: Right middle lobe, SVC: Superior vena cava, SpO_2: Oxygen saturation of blood on pulse oximetry, TSH: Thyroid-stimulating hormone, TDS: Thrice daily dosing, TAPSE: Tricuspid annular plane systolic excursion, TR: Tricuspid regurgitation, UFH: Unfractionated heparin, ICU: Intensive care unit

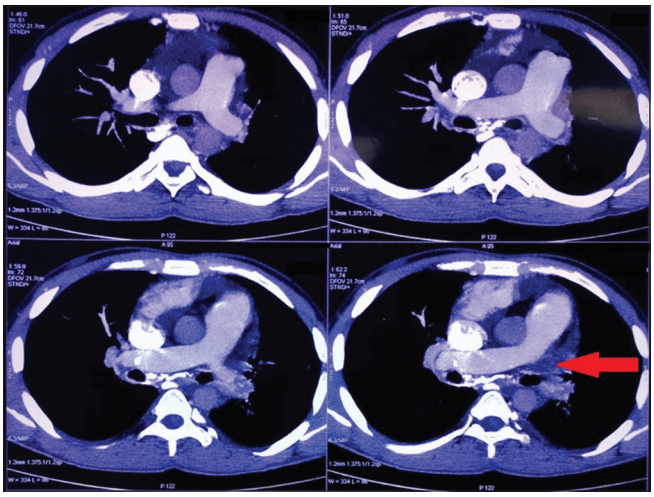

Figure 1: Computed tomography pulmonary angiogram image showing filling defect in the left pulmonary artery

and Figure 1. All the surgeries were performed by a senior surgeon and the anesthesia protocol was done as per institutional practice.

Protocol for anesthetic management

Oral anticoagulant medications were omitted 1 week before surgery and bridged with unfractionated heparin (UFH). No sedative premedication was given in the pre-operative holding area to avoid any respiratory depression. Inside the operating room, all standard American Society of Anesthesiologists monitors were attached along with external defibrillator pads and two wide bore cannulas were secured. Invasive monitoring was established with a 20G radial arterial line and 7Fr, triple lumen right internal jugular vein under local anesthesia. Induction was done after thorough pre-oxygenation with injection midazolam 1 mg, injection fentanyl 3–5 μg/kg (titrated), and titrated doses of injection etomidate. Vecuronium bromide was the muscle relaxant of choice. Phenylephrine boluses of 20 μg were used to maintain the systemic vascular resistance. A transesophageal echocardiography (TEE) probe was placed after induction along with core temperature monitoring and urinary catheterization. A 16G femoral arterial line was secured as a central to peripheral temperature gradient is very common post-bypass.[7,8] The patients were put on pressure control mode of ventilation with a 50% air-oxygen mixture, higher positive end expiratory pressure (PEEP), and inspiratory pressure adjusted to achieve a tidal volume of 6 mL/kg. Near infrared spectroscopy (NIRS) was used to monitor cerebral oxygenation.[9] Injection methylprednisolone 1 g, injection tranexamic acid, and vitamin K 10 mg were supplemented.

Surgical management

The aim of PEA is to improve the blood flow in the proximal blocked part of the pulmonary artery by taking out the clot. This helps in proper distribution of pulmonary blood flow.[10] The patients were positioned supine with hands tucked by the side. Midline sternotomy was performed and heparin was administered to achieve an activated clotting time of >480 s. The patients were cooled till a core temperature of 25°C before applying the aortic cross clamp (AXC). Deep hypothermic circulatory arrest (DHCA) was achieved at 18–20°C along with surface cooling using icepacks over the head.[11] Thiopentone boluses were given while on bypass for neuroprotection. Selective cerebral perfusion was not performed in any case.[2,3,12] Post-correction, inotropic support mainly included dopamine, milrinone, adrenaline, and noradrenaline. A leak test[6,13] was performed after correction to check for any gas leaks by inflating the lungs and flushing the field with saline. Post-correction TEE findings were recorded and the patient was shifted to the intensive care unit (ICU) for elective ventilation. The details of the surgery, intraoperative and post-operative data, and course of stay in the ICU have been tabulated in Table 2.

DISCUSSION

Anesthetic management of PEA has always been challenging owing to several factors. Reduced cardiopulmonary physiological reserve results from pulmonary hypertension, right ventricular (RV) dysfunction, and tricuspid regurgitation (Figure 2), with concomitant left ventricular dysfunction, low baseline oxygen saturation, V/Q mismatch, pre-operative deranged coagulation profile due to anticoagulant use, presence of intimal irregularities, specific requirements for DHCA, and cerebral protection. Along with PEA, the patients might also be candidates for tricuspid valve annuloplasty, patent foramen ovale repair, and coronary artery bypass grafting in patients with associated coronary artery disease. RV failure may also predispose to hepatic congestion and reduced renal perfusion. Cardiopulmonary bypass (CPB), DHCA, and a hypercoagulable state may increase the risk of post-operative stroke. Goals of anesthetic management are to make the symptoms due to RV dysfunction more tolerable for the patient; to prevent any further progression of RV dysfunction; to avoid any surge in pulmonary vascular resistance (PVR) due to hypoxia, hypercarbia, and acidosis; and to treat dyspnea. A balanced anesthetic induction and maintenance is preferred to blunt the sympathetic response and further rise in PVR at the same time minimizing hypotension due to the vasodilatory effects of anesthetics. Possible post-operative complications of PEA include massive pulmonary hemorrhage, reperfusion pulmonary edema, residual pulmonary hypertension, and RV dysfunction.[14,15]

Table 2: Details of the surgery, perfusion data, intraoperative and post-operative data, course of stay in the intensive care unit, and complications

Parameters	Case 1	Case 2	Case 3	Case4	Case 5	Case 6	Case 7
Operation performed	PEA	RPA thromboembolectomy with RPA plasty	PEA	PEA	PEA and RA mass excision	PEA	PEA with RPA plasty
Initial CVP (mmHg)	35	12	20	15	12	15	24
Initial RV pressure (mmHg)	50/24	56/22	46/21	40/19	42/18	44/18	44/22
Total CPB time (min)	191	174	257	200	140	143	230
Total AXC time (min)	116	121	187	124	65	72	100
Total DHCA sessions	2	1	3	3	1	1	2
Length of individual DHCA session (min)	35, 24	20	4, 6, 8	15,3,21	26	23	22, 20
Intraoperative TEE finding	Dilated RA (80×84 mm) Severe TR, VC-1.3 cm Tricuspid annulus-40 mm TAPSE-8 RVSP-RAP+23 RV and MPA dilated Mild MR LVEF-50%	RA/RV dilated Mod TR VC-0.5 Severe PAH-RVSP=RAP+54 Tricuspid annulus 42 mm TAPSE-9 Normal LV function	Dilated RA/RV, RV apical thrombus present (2.6×2.4 cm) attached to endocardium, TAPSE 7 mm Moderate TR-VC-0.64 RVSP= RAP+48 normal LV function LVEF-55%	Dilated RA/RV, Thrombus seen in main PA extending to right PA. A part of thrombus seen extending to left PA. TAPSE 10 mm Moderate TR-VC-0.53 RVSP=RAP+16	Dilated RA/RV, RA thrombus (2.2×2.8 cm). Moderate TR, VC-0.5 TAPSE-10 EF-60%, RVSP-RAP+35	Moderate TR VC-0.4, RVSP 59+24 RV dysfunction present, TAPSE- 11 LVEF-60%, Trace MR Hyperechoic mass present in main pulmonary artery extension couldn't be appreciated	Dilated RA/RV, Thrombus seen near origin of right PA. TAPSE 14 mm Moderate TR-VC-0.5 RVSP=RAP+20
Post-op TEE finding	Moderate TR, VC-0.6 cm TAPSE-10 on ionotropic support LVEF-50%	Mod TR VC-0.4 Severe PAH-RVSP=RAP+45 RA/RV dilated Normal LV function TAPSE-10 on ionotropic support	No residual thrombus seen in RV RVSP=RAP+45 TAPSE 8 mm on ionotropic support Moderate TR, VC-0.63 LVEF-54%	No residual thrombus seen in RV RVSP=RAP+12 TAPSE 10 mm on ionotropic support Moderate TR-0.5 LVEF 50%	No residual thrombus seen in RA or pulmonary artery. RVSP-RAP+14 TAPSE-10 on ionotropic support. Mild TR, VC-0.3	Mild TR, VC-0.3 TAPSE-12 mm LVEF-60%	No residual thrombus seen in pulmonary artery. RVSP-RAP+14 TAPSE-11 on ionotropic support. Mild TR, VC-0.28 Normal LV function
Post-PEA CVP	21	15	12	12	14	13	16
Post-PEA RV pressure (mmHg)	48/24	60/22	58/24	39/14	38/12	38/14	
Pacemaker needed postoperatively	No	No	No	No	No	No	No
Post-op complications	Severe PAH RV dysfunction	Severe PAH Mild TR	Severe PAH Mild TR, RV dysfunction	Moderate TR	Mild TR	RV dysfunction	Moderate PAH, RV dysfunction
Ionotropic support post bypass	Dopamine, Adrenaline, noradrenaline, milrinone, Lasix	Dopamine, Adrenaline, noradrenaline, milrinone	Dopamine, Adrenaline, noradrenaline	Dopamine, Adrenaline	Dopamine	Dopamine and Milrinone	Dopamine and Milrinone

(Contd,,,)

Table 2: *(Continued)*

Parameters	Case 1	Case 2	Case 3	Case4	Case 5	Case 6	Case 7
Extubated on	POD 3	POD 1	POD 2	POD 2	POD 1	POD 1	POD 2
Need for post-op NIV	Yes	Yes	Yes	No	No	No	No
Oral Vasodilators used postoperatively	Tab Sildenafil 25 mg TDS	Tab Sildenafil 25 mg TDS	Tab Sildenafil 25 mg TDS	Tab Sildenafil 25 mg TDS	Tab Sildenafil 25 mg TDS	Tab Sildenafil 25mg TDS	T Sildenafil 25 mg tds T Bosentan 62.5 mg BD

AR: Aortic regurgitation, AXC: Aortic cross-clamp, CVP: Central venous pressure, CPB: Cardiopulmonary bypass, DHCA: Deep hypothermic circulatory arrest, FFP: Fresh frozen plasma, MR: Mitral regurgitation, PEA: Pulmonary endarterectomy, PRBC: Packed red blood cells, PRP: Platelet rich plasma, POD: Post-operative day, TEE: Transesophageal echocardiography, VC: Vena contracta

Oral anticoagulants either direct factor Xa inhibitors (tablet apixiban), direct thrombin inhibitors (tablet dabigatran), or oral vitamin K antagonist (tablet warfarin) must be discontinued before scheduled surgery[16] and bridged with UFH. A central-peripheral gradient in blood pressure is very common post-bypass,[7] and hence, two arterial lines, one radial and one femoral, were secured. A study by Hong et al.,[9] showed how intraoperative cerebral oxygen desaturation could help in predicting neurological deficits postoperatively and guiding the management intraoperatively, thus warranting the use of NIRS.

DHCA has been reported to be mandatory to achieve a bloodless surgical field to perform endarterectomy down to the subsegmental branches of the pulmonary vessels[6] (Figure 3). Hypothermia itself is extremely important for neuroprotection.[17] During periods of DHCA, additional doses of methylprednisolone and thiopentone were given for cerebral protection. Langley et al.,[18] in their

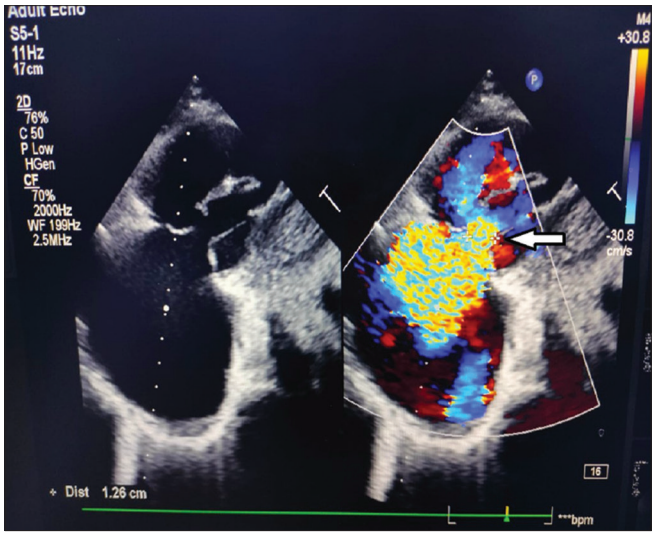

Figure 2: Echocardiographic image showing severe tricuspid regurgitation with a vena contracta 1.26 cm

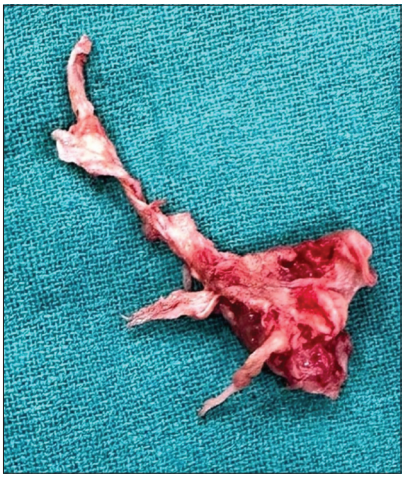

Figure 3: Fibrosed clot removed from pulmonary artery

randomized trial, have demonstrated better recovery of global and regional cerebral blood flow in the group receiving methylprednisolone post-DHCA and significant recovery in cerebral metabolic rate of oxygen consumption at 77.9% of the pre-DHCA levels. To reduce the risk of neuropsychiatric complications due to release of emboli after removal of the AXC, barbiturates have shown promising results.[19] Bypass was reinitiated for 10 min in between subsequent rounds of DHCA. Maintenance of selective cerebral perfusion was not performed in any case.[17]

Pressure control mode of ventilation having the advantage of early detection of decrease in tidal volume in the face of endobronchial hemorrhage was employed to achieve some degree of hyperventilation at a PEEP of 10 cm H_2O.[6] In the post-operative course, maintenance of a negative fluid balance is a must for a favorable outcome. Repeated transthoracic echocardiography is repeated in the post-operative period daily to look for residual RV dysfunction.

Post-operative hypoxia and atelectasis are known complications after CPB. There are high chances of V/Q mismatch due to loss of protective hypoxic pulmonary vasoconstriction. Thromboendarterectomy may cause redistribution of pulmonary arterial resistance leading to a pulmonary vascular "steal" phenomenon[20] wherein the newly endarterectomized segments of the lung are better perfused than the normal lung segments. Hence, many of these patients need supplemental oxygen for a prolonged period.

None of the seven patients had surgical hemorrhage or reperfusion pulmonary edema; however, some amount of residual TR and RV dysfunction[7,14,15] leading to high pulmonary arterial pressures were seen in all three patients. An incidence of 5–35% has been reported for this complication.[14,15] Small embolic fragments in the distal vasculature along with small vessel vasculopathy which may not be treated by surgical removal of the clots may cause irreversible PH.[21] Braams et al.,[22] studied 25 patients undergoing PEA and noticed a significant fall in the RV mass and diastolic stiffness post-surgery. However, they have also reported a relatively lesser decrease in the matrix volume than the cellular volume which may contribute to persistent RV dysfunction. Hence, long-term treatment with oral anticoagulants and sildenafil is recommended. Ionotropic support to maintain RV function may be needed for several days postoperatively. Combination of inhaled nitric oxide and inhaled iloprost has shown to reduce PH.[23]

Pulmonary hemorrhage can be identified on table and corrected surgically. It may need lung isolation using bronchial blocker, reversal of heparin, and correction of coagulation disorders. In severe cases, extracorporeal membrane oxygenation may be needed.[24]

Supportive treatment is needed in cases of reperfusion pulmonary edema, mainly using diuretics to reduce the pulmonary congestion, ventilating with low tidal volumes,[14] and supplementing steroids as this is usually a consequence of inflammation.[25]

In a study of 10 patients undergoing PEA, Chen et al.,[24] have reported subdural hematoma as the most common complication in 60% patients. However, this was not seen in any of the three patients in our study.

CONCLUSION

Complicated and rare surgeries like PEA must be undertaken at experienced centers having a multidisciplinary team approach consisting of surgeons, anesthesiologists, radiologists, intensive care physicians, and respiratory physicians. Many possible complications may still be unrecognized. As per the difference in demographics, no fixed protocol can be established for all patients. Based on the expertise of the anesthesiologist and institutional practices, the available protocols may be modified. Higher success rates with fewer complications may be seen with studies in a larger population and with better expertise.

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