Urologic Surgery with Multisystem Comorbidities: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 **Upendra Maddineni**
ABCD 2 **Eric F. Worrall**
ABC 1 **Erik A. Baker**
ABCD 1 **Madhavi Earasi**
ABCDEF 1 **Francis Mathieu**
ABCD 3,4 **Georgi Guruli**

1 Department of Anesthesiology, McGuire Veterans Administration Medical Center, Richmond, VA, U.S.A.
2 Department of Anesthesiology, Virginia Commonwealth University, Richmond, VA, U.S.A.
3 Department of Urology, McGuire Veterans Administration Medical Center, Richmond, VA, U.S.A.
4 Department of Urology, Virginia Commonwealth University, Richmond, VA, U.S.A.

Corresponding Author: Upendra Maddineni, e-mail: upendra.maddineni@va.gov
Conflict of interest: None declared

Patient:	**Female, 70**
Final Diagnosis:	**Renal cell carcinoma**
Symptoms:	**Epistaxis • exertional dyspnea**
Medication:	**—**
Clinical Procedure:	**Radical nephrectomy**
Specialty:	**Urology**

Objective:	**Rare disease**
Background:	Originally implemented for colorectal surgery, enhanced perioperative protocols have been incorporated into many surgical fields in an effort to improve outcomes. The cornerstone of many strategies includes patient education, liberalized oral intake on the day of surgery, no routine bowel prep, targeted multimodal analgesia, cautious use of IV hydration, early extubation, avoidance of NG tubes and/or surgical drains, and encouraging early postoperative ambulation.
Case Report:	We report on the successful outcome of a single patient with a rare autosomal dominant disorder (hereditary hemorrhagic telangiectasia) with multisystem involvement including pulmonary, cardiac, hematologic, gastrointestinal, renal, oncologic, and hepatic comorbidities, scheduled for open nephrectomy.
Conclusions:	Prospective and retrospective studies are needed to specifically elucidate the role of similar management in higher-risk surgical candidates.

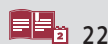|	
MeSH Keywords:	**Nephrectomy • Patient Education as Topic • Telangiectasia, Hereditary Hemorrhagic**
Full-text PDF:	https://www.amjcaserep.com/abstract/index/idArt/909554

📄 2598   ▦ 2   📊 —   📚 22

Background

Hereditary hemorrhagic telangiectasia, also known as Osler-Rendu-Weber syndrome, is an inherited autosomal dominant genetic disorder resulting in cutaneous telangiectasias and abnormal arterial and venous malformations (AVMs) that can often lead to bleeding and embolic sequela in fragile pulmonary, gastrointestinal, hepatic, spinal, and cerebral vessels [1–7]. Diagnosis is definite, possible, or unlikely based on the Curçao Criteria, including spontaneous recurrent epistaxis, telangiectasias, AVMs, and a familial inheritance pattern [1–3,5,6]. Disease subtype (1–4) is based upon the identified gene mutation, which includes active receptor-like kinase 1 gene (ALK1), endoglin gene (ENG), and mothers against decapentaplegic homolog 4 gene (MADH4), which are involved in the TGF-beta signaling pathway [1–3,5,7,8]. Epistaxis is common due to vascular malformations of the oropharyngeal mucosa [1]. Hemoptysis also occurs with less frequency due to the presence of pulmonary AVMs in 40–60% of patients [1,3,5]. Pulmonary hypertension recognized in patients either preceding or following the diagnosis of HHT can be a severe manifestation, for which the genetic basis remains undetermined [2,7,8]. Hemodynamics consistent with precapillary pulmonary hypertension in HHT includes low cardiac output, normal pulmonary capillary wedge pressure, and high PVR [2]. Advanced targeted therapy for pulmonary arterial hypertension includes prostacyclins, calcium channel blockers, endothelin receptor antagonists, and phosphodiesterase 5 inhibitors (sildenafil) [2,9–12]. The use of IV epoprostenol has been reported to be limited in HHT due to worsening of epistaxis [2].

Moderate-to-severe pulmonary arterial hypertension, group 1, as defined by the Fifth World Symposium in 2013, carries an independent increased risk for perioperative respiratory and cardiac complications, which is associated with increased morbidity and mortality in patients undergoing non-cardiac surgery [11–16]. In patients undergoing cardiac transplant, pulmonary hypertension and right heart failure account for a 19% mortality rate and a 50% morbidity rate [11]. In patients undergoing non-cardiac surgery, higher mortality, heart failure, sepsis, hemodynamic instability, respiratory failure, and intensive care and hospital length of stay have been reported [11,16]. In parturients, pulmonary hypertension is associated with a 25–30% mortality rate, approaching 50% in the presence of Eisenmenger physiology [12,17,18]. Standard preoperative screening includes assessment of functional class, cause, exercise tolerance, pulmonary vascular hemodynamics, and overall risks, benefits, and timing of the proposed intervention or surgery in an optimized patient [11,14]. Resting mean pulmonary artery pressures greater than 25 mmHg in the setting of pulmonary vascular resistance greater than 240 dyn s/cm^5 and pulmonary arterial occlusion pressure less than

or equal to 15 mmHG support the diagnosis of pulmonary artery hypertension [2,8–10,12,14,16,19].

Anesthetic management of patients with pulmonary hypertension should include preparation for the physiologic changes that can be detrimental, including impaired right ventricular function, changes in preload and afterload, and coronary artery perfusion [9,12]. Recommendations include avoidance of factors that increase pulmonary vascular resistance, including hypoventilation, hypoxemia, hypothermia, hypercarbia, hypervolemia, and acidosis, which can lead to acute right heart failure, hypotension, decreased cardiac output, and systemic oxygen delivery [9,11,13]. Emphasis is also placed on maintaining systemic vascular resistance and judicious management of hemodynamics during induction of general anesthesia, such as the sympathetic response to intubation, incision, and the decrease in preload from positive-pressure ventilation [13]. Regional anesthesia with peripheral nerve blocks under ultrasound guidance is beneficial, with improved numerical pain scores and minimizing the pulmonary and cardiac perturbations to which patients with moderate-to-severe pulmonary arterial hypertension are prone [11,13,19]. Neuraxial anesthesia is associated with decreased SVR, among other detrimental changes, and is generally contraindicated with the exception of parturients receiving a low dose or slowly titrated epidural, in which general endotracheal anesthesia poses a greater risk due to the presence of pulmonary hypertension [9,11,13,17,20].

Case Report

A 70-year-old woman (weight 61.2 kg; height 160 cm; BMI 23.9 kg/m^2) with hereditary hemorrhagic telangiectasia (HHT), severe pulmonary hypertension, diastolic heart failure, mild obstructive sleep apnea, hyperlipidemia, gastroesophageal reflux, and hypertension was scheduled for open radical nephrectomy for removal of her left kidney en bloc due to a 5.4-cm mass localized to the lower pole and concerning for malignancy. Three years prior, the patient underwent workup for severe exertional dyspnea, with left and right heart catheterization revealing angiographically normal-appearing coronary arteries, with the exception of findings suggestive of right coronary artery fistula. Invasive hemodynamics during right heart catheterization showed severe precapillary pulmonary hypertension with mean pulmonary artery pressure (mPAP) of approximately 45 mmHg (83/30), pulmonary capillary wedge pressure (PCWP) 8 mmHG, pulmonary vascular resistance (PVR) 7.8 WU, and cardiac index 3.0 L/min/m^2 (Table 1).

After she was started on sildenafil (REVATIO) for treatment of severe arterial pulmonary HTN, secondary to HHT, her WHO class III symptoms improved to class I or II. Subsequent follow-up with transthoracic echocardiogram showed RV

Table 1. Right and left heart catherization data.

Coronary angiography

Native vessels

Summary: normal coronary arteries
Dominance: mixed dominance
Narrative Description:
Left Main: large caliber vessel that appears normal
LAD: large caliber vessel
D1: moderate caliber vessel
D2: moderate caliber branching vessel
LCx: large caliber codominant vessel
OM1: moderate caliber vessel with a high takeoff
OM2: small caliber vessel
OM3: moderate caliber branching vessel
LPLB: small caliber branching vessel
RCA: moderate caliber codominant vessel
RPDA: small caliber vessel
RPLB: moderate caliber vessel

Right heart catheterization
Pressures (mmHg)
RA mean: 8
RV: 80/8
PA: 83/30, mean 48
PCWP: 8
Mean BP: 86
Heart Rate: 96 bpm
Hemoglobin: 10.2
Cardiac Output (Q):
Q (Thermodilution): 5.11 L/min
Q (Fick): 4.6 L/min
CI (Thermodilution): 3.04 L/min/m2
CI (Fick): 2.74 L/min/m2
Saturations:
Arterial saturation: 96%
Mixed venous saturation: 61%
Vascular Resistance:
Pulmonary vascular resistance: 7.8
Systemic vascular resistance: 1221

enlargement with decreased function, moderate-to-severe eccentric TR, a moderately-to-severely dilated right atrium, and Doppler-derived estimated systolic pulmonary artery pressure >100 mmHg with low normal left ventricular function. Pulmonary function testing indicated normal volumes, spirometry, normal forced vital capacity (FVC), forced expiratory volume (FEV1), and FEV1/FVC ratio, with severely reduced diffusing capacity (43% of predicted). In addition, the patient's other sequela of HHT included frequent epistaxis and episodes of gastrointestinal bleeding due to arterial venous malformations, which required previous intervention with nasal and upper-gastrointestinal endoscopy. Her other comorbidities included mild OSA treated with CPAP, heart failure with preserved ejection fraction, anemia, a 30-pound weight loss, and synthetic liver dysfunction with an elevated PT INR and low albumin, which was discovered during preoperative evaluation.

During multidisciplinary perioperative consultation with anesthesia, cardiology, pulmonary, hematology, urology, and hepatology, the risks, benefits and alternatives were discussed, including an increased risk, albeit not prohibitive, due to her severe pulmonary hypertension. The patient consented to proceed with surgery given the likelihood for malignant renal cell carcinoma. She was deemed ASA physical status IV [21,22]. Preoperative transthoracic echocardiography was obtained, which showed similar findings to her previous echocardiogram. Chest x-ray

revealed cardiovascular silhouette at the upper limits of normal, with prominent pulmonary arteries. The patient was recommended to continue sildenafil, to which she had a good response, with exercise tolerance greater than 4 METS. Partial *vs.* radical nephrectomy was discussed and the patient elected to have removal of her entire left kidney. Preoperatively, she had minimal dyspnea with daily activities including house and yard work, occasionally experiencing lightheadedness during exertion, but with no angina. She denied significant orthopnea and paroxysmal nocturnal dyspnea, with no significant edema or hepatosplenomegaly on exam. She had frequent epistaxis episodes secondary to HHT. Based on results of a 1:1 mixing study, a dose of oral vitamin K was ordered in an attempt to correct her elevated PT/INR of 1.3. Subsequent outpatient workup did not show evidence of decompensated liver disease, and only low-grade fibrosis was seen on a fibroscan. She continued her other medications, including ferrous sulfate, Lasix, omeprazole, and albuterol.

The patient was admitted on the day of surgery. No significant changes to her medical condition were noted. Vitals were temperature 36.6 C, blood pressure 138/77 mmHG, respiratory rate 18/min, pulse oximetry 96% on room air, and pain 0/10. The airway exam was reassuring. She experienced no bleeding episodes other than frequent bouts of epistaxis. She denied new or worsening shortness of breath or chest discomfort.

Table 2. Laboratory profile.

Wbc	7.2	10^3/uL	4.8–10.8
Rbc	4.2 L	10^6/uL	4.7–6.1
Hemoglobin	11.2 L	g/dL	14–18
Hematocrit	35.4 L	%	42–52
Mcv	84.5	fL	80–94
Mch	26.7 L	pg	27–31
Mchc	31.6 L	g/dL	33–37
Rdw	15.9 H	%	11.5–14.5
Pltct	326	10^3/uL	130–400
Protime (PT)	15.2 H	SEC	11.7–14.4
Activated	PTT	34 SEC	23.4–36.1
Inr	1.3	Ratio	
Sodium	141 mmol/L	136–145	
Potassium	4.1 mmol/L	3.5–5.1	
Chloride	107 mmol/L	98–107	
Carbon dioxide	25 mmol/L	21–32	
Glucose	85 mg/dL	74–106	
Creatinine	0.7 mg/dL	0.55–1.3	
Blood urea nitrogen	12 mg/dL	7–18	
Calcium	9.0 mg/dL	8.5–10.1	
Phosphate	4.4 mg/dL	2.5–4.9	
Magnesium	2.1 mg/dL	1.8–2.5	
ANION GAP	9.0 mmol/L	8–15	
Albumin	3.5 g/dL	3.4–5.0	
EGFR	87.93	Ref: >60 mL/min/1.73 m²	

An electrocardiogram (ECG) showed normal sinus rhythm, rate 95; premature ventricular complexes; and inferior and anterolateral T-wave changes consistent with previous ECGs. Laboratory data (Table 2) included hemoglobin of 11.2 g/dl and hematocrit of 35.4%. A large-bore IV access and radial arterial line for hemodynamic monitoring was placed prior to transferring the patient to the operating room. For induction of general anesthesia, the patient was placed in supine position with standard ASA monitors. After adequate pre-oxygenation, we administered IV fentanyl, etomidate, and lidocaine, followed by rocuronium. The patient was intubated with a 7.0-mm endotracheal tube using a MAC 3 blade showing a Cormack-Lehane grade I view. No telangiectasias were observed during direct laryngoscopy. Endotracheal tube position was confirmed with the presence of equal bilateral breath sounds and positive end-tidal CO_2. Pulse oximetry, heart rate, and arterial blood pressure remained stable.

Next, a Swan-Ganz catheter was placed after several attempts with difficulty due to severe tricuspid regurgitation via right internal jugular venous 9 fr introducer with initial pulmonary artery pressure of 96/46 mmHG, mean pulmonary artery pressure of 65 mmHG, and systemic arterial pressure of 125/74 mmHG. Initial arterial blood gas was pH 7.33, pCO2 43 mmHG, pO2 145 mmHG, and HCO3 22.7 mEq/L. After correction of hypercarbia and mild acidosis, inhaled nitric oxide (INO) 20 ppm was initiated with improvement in pulmonary artery pressures to 70–80s/30–40s mmHG to a nadir of 67/29 mmHG and mean pulmonary artery pressure of 44 mmHG, which was coincident with a systemic arterial pressure of 123/67 mmHG. Fluid balance was maintained with adequate urine output and replacement of blood loss that was less than 100 cc.

Prior to incision, transversus abdominal plane block was performed using real-time ultrasound guidance with a 1: 1 mixture of 0.25% bupivacaine and 1.3% liposomal bupivacaine for a total of 20 cc local infiltration. General anesthesia was maintained with sevoflurane. Total surgical time after positioning in right lateral decubitus position was less than 60 min using open technique with a flank incision. The patient remained hemodynamically stable on inhaled nitric oxide. Final arterial blood gas analysis in the operating room was pH 7.38, pCO2 38 mmHG, and pO2 182 mmHG on 50% FiO2. The patient was transferred to the surgical intensive care unit, and inhaled nitric oxide was weened over 3 h as cardiac output and pulmonary artery pressures reached baseline values (PAP 70–80s/30–40s mmHG, CO 3–4 L/min) without evidence of pulmonary arterial crisis in the perioperative period.

The patient was extubated on postoperative day 0 to non-invasive positive pressure ventilation and weened to nasal cannula and was able maintain pulse oximetry saturation at 93–96%. Pain was adequately controlled based on a numerical rating scale <4 using a multimodal regimen including PO and IV acetaminophen, Ultram, and incremental doses of fentanyl. The patient had the expected return of bowel function without placement of nasogastric tube and was able to tolerate oral diet within 24 h after the operation. On postoperative day 1, the patient was transferred to a step-down unit and was able to ambulate with assistance. She was discharged to home in stable condition on postoperative day 3. Pathologic specimens returned as renal cell carcinoma, clear cell type, with high-grade histologic pathology, classification pT1bG4, with clear margins. On follow-up, the patient continued to progress as expected and was able to resume normal daily activities. Of note, she turned 71 years old during her 3-day hospitalization.

Discussion

Due to the presence of bleeding and other comorbidities, patients with HHT may require a number of procedures that require special perioperative considerations [1]. Care is required

during induction of anesthesia to minimize hypertension, which theoretically can result in rupture of arterial venous malformations, leading to potentially catastrophic hemorrhage [1]. Previous case reports have recommended precautions be taken during direct laryngoscopy to minimize the airway trauma due to the frequent presence of hazards (e.g., telangiectasias) in the path of airway apparatus such as endotracheal tubes and Mac and Miller blades [1]. A routine induction dose of lidocaine and narcotic is recommended to blunt the sympathetic response to laryngoscopy, as well as an anesthetic plan tailored to maintain coronary perfusion [1]. Pertinent laboratory values that should be reviewed by the perioperative physician include hemoglobin, hematocrit, platelets, and coagulation factors [1]. Based on clinical evaluation, additional workup may be undertaken for the presence of cerebral, pulmonary, gastrointestinal, and hepatic AVMs [1]. Pulmonary hypertension (PH) may be present in patients with HHT either in post-capillary PH, due high-output heart failure secondary to arterial venous and or portovenous shunting, and, less commonly, as precapillary pulmonary arterial hypertension [2].

Pulmonary arterial hypertension (PAH) often presents preoperatively and during the physical exam, with exertional dyspnea, angina, syncope, peripheral edema, and hepatomegaly from right heart strain, and is associated with a higher rate of operative complications than with other classification groups (2–5) of pulmonary hypertension [14,16]. The prevalence of PAH is 15–60 persons/million, involving pathophysiologic remodeling of all layers of the pulmonary vasculature and resulting in increased pulmonary vascular resistance and right ventricular hypertrophy, pressure overload, RV and LV failure, shock, and death [9,17]. Differentiation from among the other WHO clinical groups can involve laboratory testing, pulmonary function, imaging, sleep studies, cardiac evaluation with surface echo, and invasive right heart catherization to demonstrate increased pulmonary artery pressures and treatment response to agents such as nitric oxide and prostacyclin.

Therapeutic options include oxygen, diuretics, and pulmonary vasodilators (e.g., epoprostenol, bosentan, nitric oxide, and sildenafil), which can be administered through single or combination dose escalation by oral, inhalational (which is preferred), or intravenous route, based on WHO functional classification [10,11,14]. Perioperatively, patients should remain on vasodilators, oxygen, diuretics, inhaled beta-agonist or anticholinergics, steroids if indicated, and CPAP or BiPAP if required for optimization of ventilation [11]. In addition, maintaining sinus rhythm, optimizing preload, and coronary perfusion help to augment right ventricular function [9]. Post-capillary pulmonary hypertension, the most common form in both the general population and patients with HHT, is distinguished by elevated pulmonary arterial occlusion pressure and also requires optimization of left-sided heart function [8–10,14]. Patients presenting with

class IV symptoms may be considered to be at prohibitive risk for elective surgery [14]. Extracorporeal membrane oxygenation (ECMO) and heart-lung transplant are final options for patients that meet criteria for end-stage heart failure [9,17].

Morbidity due to heart failure, arrhythmias, and respiratory failure associated with a specific functional class is difficult to quantify and is not always governed by the degree of elevation in mean pulmonary artery pressure [14]. However, in general, patients that are functional class I without significant symptoms at baseline activity are best able tolerate elective procedures [14]. Pulmonary arterial hypertension is associated with higher surgical morbidity and mortality in comparison to other WHO categories of pulmonary hypertension, which encompass a range of disorders divided into 5 groups [9,14]. Cardiac index, right atrial pressure, 6-min walk test, BNP, and troponin are useful parameters in assessing therapeutic response and survival [14]. Increased risk is associated with surgery that is emergent, intermediate-to-high risk, and or involving significant hemorrhage requiring transfusion [14]. Lung resection, which impairs vascular anatomy and orthopedic surgery with embolic phenomenon, can precipitate pulmonary hypertensive crisis in patients with already-compromised right ventricular function [14]. Standard ECG, pulse oximetry, pre-induction invasive arterial blood pressure monitoring, large-bore IV and multi-lumen central venous access, carefully de-aired, is recommended [9,11]. Intraoperative monitoring of cardiac output (CO), PCWP, and mixed venous saturation with a pulmonary artery catheter or continuous transesophageal echo can be used to guide management of pulmonary artery pressures and right heart function with inhaled nitric oxide (INO), nebulized prostacyclins (iloprost or epoprostenol), or intravenous milrinone [9,11,12,14,17]. Systemic hypotension, intra- or postoperative, can also be detrimental due to decreased coronary perfusion and loss of right ventricular function, requiring volume expansion, vasopressors, or inotropes [9,11,14,15]. Postoperative weening of short-acting pulmonary vasodilators such as INO can cause rebound pulmonary hypertension and can be attenuated with sildenafil [11].

Conclusions

We report on the successful outcome of a patient with a rare genetic disorder and a predisposition to bleeding, undergoing major intraabdominal surgery. Future retrospective and prospective studies are necessary to further elucidate the role of similar management in patients with higher perioperative risk profiles due to the presence of multisystem comorbidities.

Acknowledgement

Department and Institution where work was done

Department of Anesthesiology, McGuire Veterans Administration Medical Center, Richmond, VA, USA.

Conflicts of interest

None.

References:

1. Robinson D, Rogers B, Kapoor R et al: Anesthetic considerations for a patient with hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome) undergoing a five-box thoracoscopic maze procedure for atrial fibrillation. J Investig Med High Impact Case Rep, 2014; 2(4): 2324709614553669

2. Ishiwata T, Terada J, Tanabe N et al: Pulmonary arterial hypertension as the first manifestation in a patient with hereditary hemorrhagic telangiectasia. Intern Med, 2014; 53(20): 2359–63

3. Dupuis-Girod S, Cottin V, Shovlin CL: The lung in hereditary hemorrhagic telangiectasia. Respiration, 2017; 94(4): 315–30

4. Magalhães-Costa P, Chagas C: Hereditary haemorrhagic telangiectasia. BMJ Case Rep. 2015; 2015: pii: bcr2014208826

5. Sacco KM, Barkley TW Jr.: Hereditary hemorrhagic telangiectasia: A primer for critical care nurses. Crit Care Nurse, 2016; 36(3): 36–48

6. Butt MU, Yoo E, Khan U: Osler-Weber-Rendu syndrome.QJM, 2016; 109(9): 615–16

7. Vorselaars VM, Velthuis S, Snijder RJ et al: Post MC1 pulmonary hypertension in hereditary haemorrhagic telangiectasia. World J Cardiol, 2015; 7(5): 230–37

8. Revuz S, Decullier E, Ginon I et al: Pulmonary hypertension subtypes associated with hereditary haemorrhagic telangiectasia: Haemodynamic profiles and survival probability. PLoS One, 2017; 12(10): e0184227

9. Rex S, Devroe S: Anesthesia for pregnant women with pulmonary hypertension. Curr Opin Anaesthesiol, 2016; 29(3): 273–81

10. Hill NS, Preston IR, Roberts KE: Inhaled therapies for pulmonary hypertension. Respir Care, 2015; 60(6): 794–802

11. Kaw RK: Spectrum of postoperative complications in pulmonary hypertension and obesity hypoventilation syndrome. Curr Opin Anaesthesiol, 2017; 30(1): 140–45

12. Pilkington SA, Taboada D, Martinez G: Pulmonary hypertension and its management in patients undergoing non-cardiac surgery. Anaesthesia, 2015; 70(1): 56–70

13. Shinohara H, Hirota K, Sato M et al: Monitored anesthesia care with dexmedetomidine of a patient with severe pulmonary arterial hypertension for inguinal hernioplasty. J Anesth, 2010; 24(4): 611–13

14. Yang EI: Perioperative management of patients with pulmonary hypertension for non-cardiac surgery. Curr Rheumatol Rep, 2015; 17(3): 15

15. Lai HC, Lai HC, Wang KY et al: Severe pulmonary hypertension complicates postoperative outcome of non-cardiac surgery. Br J Anaesth, 2007; 99(2): 184–90

16. Kaw R, Pasupuleti V, Deshpande A et al: Pulmonary hypertension: An important predictor of outcomes in patients undergoing non-cardiac surgery. Respir Med, 2011; 105(4): 619–24

17. Meng ML, Landau R, Viktorsdottir O et al: Pulmonary hypertension in pregnancy: A report of 49 cases at four tertiary North American sites. Obstet Gynecol, 2017; 129(3): 511–20

18. Patel S, Weierstahl KL, Shah S, Fidkowski CW: Anesthetic management for cesarean delivery in a patient with pulmonary emboli, pulmonary hypertension, and right ventricular failure. A A Case Rep, 2016; 7(7): 146–49

19. Swamy MC, Mukherjee A, Rao LL, Pandith S: Anaesthetic management of a patient with severe pulmonary arterial hypertension for renal transplantation. Indian J Anaesth, 2017; 61(2): 167–69

20. Sen S, Chatterjee S, Mazumder P, Mukherji S: Epidural anesthesia: A safe option for cesarean section in parturient with severe pulmonary hypertension. J Nat Sci Biol Med, 2016; 7(2): 182–85

21. Gerlach RM, Sweitzer BJ: Overview of preoperative assessment and management. Anesthesiology. Longnecker DE, et al. (eds.), New York, NY. McGraw-Hill

22. Doyle DJ, Garmon EH: American Society of Anesthesiologists Classification (ASA Class). SourceStatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, 2017