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CASE REPORT

Right Ventricular Outflow Tract Obstruction in the Intensive Care Unit: A Case Report of 2 Patients

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Right ventricular outflow tract obstruction (RVOTO) is a rare cause of hemodynamic instability in the intensive care unit (ICU) after cardiac surgery. We report the first cases of RVOTO diagnosed in the ICU using continuous right ventricular pressure waveform monitoring. Our 2 cases reflect both mechanical and dynamic causes of obstruction, each of which require different approaches to treatment. Inotrope use can exacerbate RVOTO caused by dynamic etiology, whereas surgery is usually the treatment of choice for mechanical obstructions. Inability to recognize RVOTO or the correct etiology can lead to hemodynamic compromise and poor outcomes. (A&A Practice. 2021;15:e01532.)

GLOSSARY

2D = 2-dimensional; **3D** = 3-dimensional; **Ao** = aorta; **bpm** = beats per minute; **CABG** = coronary artery bypass graft; **CPB** = cardiopulmonary bypass; **HOCM** = hypertrophic obstructive cardiomyopathy; **HR** = heart rate; **ICU** = intensive care unit; **LA** = left atrium; **LV** = left ventricle; **MIS AVR** = minimally invasive surgical aortic valve replacement; **PA** = pulmonary artery; **Pfa** = femoral arterial pressure; **Ppa** = pulmonary artery pressure; **Prv** = right ventricular pressure; **RA** = right atrium; **RR** = respiratory rate; **RV** = right ventricle or ventricular; **RVH** = right ventricular hypertrophy; **RVOT** = right ventricular outflow tract; **RVOTO** = right ventricular outflow tract obstruction; **RVSP** = right ventricle systolic pressure; **SPAP** = systolic pulmonary artery pressure; **Spo_2** = oxygen saturation using pulse oximetry; **TEE** = transesophageal echocardiography; **TTE** = transthoracic echocardiography

Right ventricular outflow tract obstruction (RVOTO) is a rarely reported cause of hemodynamic instability in the intensive care unit (ICU).[1–3] RVOTO is diagnosed by a systolic gradient between the right ventricular pressure (Prv) and the pulmonary artery pressure (Ppa).[4] This can result from mechanical[5] or dynamic etiologies.[4,6,7] RVOTO may be identified on transthoracic (TTE), transesophageal (TEE),[4] or epicardial echocardiography.[8] Diagnosis can also be made with right ventricle (RV) cardiac catheterization[6] with Prv waveform display[9] using an opening at 19 cm from the distal tip (Thermodilution pace port catheter, Swan-Ganz P. ref 931F75; Edwards Lifesciences). This allows dual pressure monitoring of the Prv and Ppa. The adequacy of position is confirmed with a chest radiograph.

Severe RVOTO has been reported after lung transplantation, cardiac surgery, and cardiac catheterization, and has been called "suicide" RVOTO[1,3,7] as a result of acute reduction in RV afterload.[4,8] We describe the first cases of RVOTO diagnosed postoperatively in the ICU using continuous Prv waveform monitoring. Family of patient 1 gave written consent, and patient 2 gave written consent to report these cases.

Case 1

A 39-year-old male patient with known bicuspid aortic valve and moderate aortic stenosis presented with acute Stanford type A aortic dissection (Figure 1A; Supplemental Digital Content, Video 1A, http://links.lww.com/AACR/A460). Initial chest computed tomography showed dissection beginning at the root of the aorta, sparing the coronary arteries and extending through the aortic arch down to the renal arteries. Maximal diameter was 71 mm. The patient was urgently operated for replacement of the aortic hemiarch and aortic valve, performed under deep hypothermic circulatory arrest. After cardiopulmonary bypass (CPB) separation, anterior left ventricular (LV) wall akinesis was observed on TEE, requiring return to CPB for a coronary artery bypass graft (CABG) on the left anterior descending artery. CPB duration was 193 minutes, with 87 minutes of clamping and 22 minutes of circulatory arrest. Intraoperative volume balance was estimated to be 3135 mL greater than admission. A total of 1200 mL of packed red blood cells was transfused in addition to 500 mL of platelets, 1200 mL of fresh frozen plasma, and 200 mL of cryoprecipitate.

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Accepted for publication September 1, 2021.

Funding: Supported by the Montreal Heart Institute Foundation and the Richard Kaufman Endowment Fund in Anesthesia and Critical Care. The funding sources were not involved in this series.

Conflicts of Interest: See Disclosures at the end of the article.

Supplemental digital content is available for this article. Direct URL citations are provided in the HTML and PDF versions of this article on the journal's website (www.cases-anesthesia-analgesia.org).

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DOI: 10.1213/XAA.0000000000001532

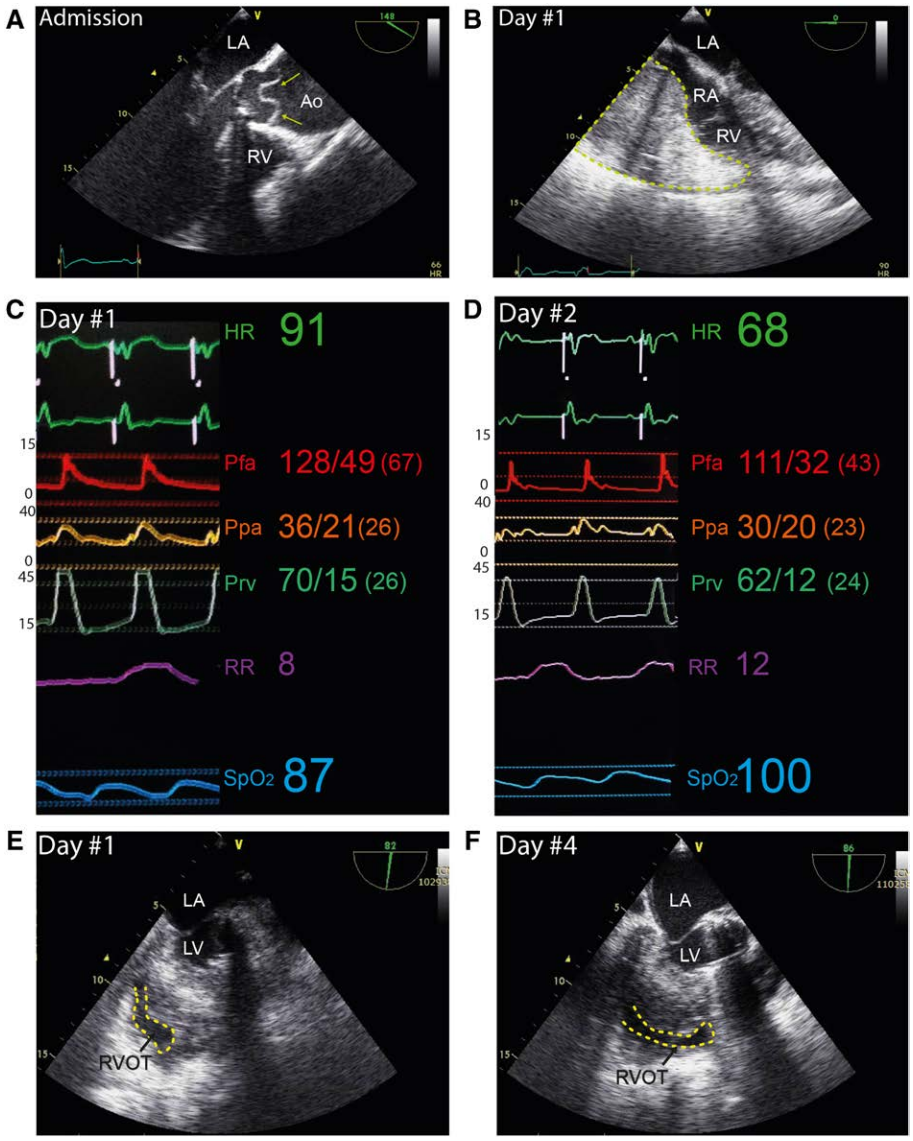

Figure 1. Patient #1. A, Midesophageal long-axis view showing Stanford type A aortic dissection. Note the dissecting flap (arrows). B, Midesophageal 4-chamber view showing compression of the right atrium (RA) following admission to the ICU by a large thrombus (dotted lines) (Supplemental Digital Content, Videos 1A and B, http://links.lww.com/AACR/A460, http://links.lww.com/AACR/A461). C, A significant systolic gradient of 34 mm Hg between the systolic Prv mm Hg (Prv = 70 mm Hg) and the systolic Ppa (Ppa = 36 mm Hg) was present on arrival in the intensive care unit after thrombus removal. D, Persistence of a 32-mm Hg gradient pressure on day 2. E, Midesophageal RV inflow-outflow view showing significant compression of the RVOT which was persistent (F) on day 2 (Supplemental Digital Content, Videos 1E and F, http://links.lww.com/AACR/A462, http://links.lww.com/AACR/A463). Ao indicates aorta; HR, heart rate; ICU, intensive care unit; LA, left atrium; LV, left ventricle; Pfa, femoral arterial pressure; Ppa, pulmonary artery pressure; Prv, right ventricular pressure; RA, right atrium; RR, respiratory rate; RV, right ventricle; RVOT, RV outflow tract; Spo_2, oxygen saturation using pulse oximetry.

On ICU arrival, the patient had severe hemodynamic instability despite increased inotropic and vasopressor support with epinephrine 0.23 µg/kg/min, norepinephrine 0.74 µg/kg/min, and vasopressin 2.4 U/h (0.04 U/min). The cardiac index was estimated at 1.22 L/min/m^2. On the following day, TEE revealed significant pericardial collection causing compression (Figure 1B; Supplemental Digital Content, Video 1B, http://links.lww.com/AACR/A461). Decompressive sternotomy was performed, and the patient was reoperated for aortic anastomosis suture correction, which was identified as the source of bleeding. The patient remained unstable in the operating room as a result of RV dysfunction. After returning to the ICU, a systolic pressure gradient of 34 mm Hg between the Ppa and the Prv was noted (Figure 1C). Reduction of the RVOT size during systole was confirmed with TEE (Figure 1E; Supplemental Digital Content, Video 1E, http://links.lww.com/AACR/A462). The patient was also paced due to third-degree heart block.

On the second postoperative day, the patient still required vasoactive support and continuous renal replacement therapy. A 32-mm Hg gradient RVOTO remained despite optimization of volume status, pacing adjustment for 68 beats per minutes (bpm) (Figure 1D) and cessation of epinephrine. Bedside TTE showed RV hypokinesis. Because of the progressive deterioration of the patient, it was decided to proceed with urgent right CABG without preoperative angiography. Unfortunately, after the procedure, progressive cardiac failure and profound vasoplegia resulted in ongoing acute kidney injury and ischemic hepatitis. Over the next 2 days, RVOTO was persistent (Figure 1F; Supplemental Digital Content, Video 1F, http://links.lww.com/AACR/A463) and possibly due to both extrinsic cardiac compression by the closed sternum and intrinsic LV and RV myocardial edema and hypertrophy. As RV systolic function progressively declines, the patient developed refractory vasoplegia, rhabdomyolysis, and multiorgan failure, and died on postoperative day 4.

Autopsy showed dilated right atrium, patent right and left coronary artery ostia, and intact aortic root repair without evidence of dehiscence or significant deformation. There was a 70% focal stenosis of the mid-left anterior descending artery, with a transmural acute myocardial infarction of the

left posterior and posterolateral area. Multifocal subendocardial ischemia was present on the LV and RV. The CABGs were patent, with moderate stenosis on the right coronary artery venous graft. There was biventricular hypertrophy with edema and the heart weighed 615 g (normal <300 g). The RV outflow tract was small relative to the size of the heart. The aortic dissection originated from the ascending aorta and extended into the renal arteries. Severe hepatic ischemia with centrilobular congestion confirmed ischemic hepatitis. Congestive and hemorrhagic small bowel and colon with gallbladder edema, anasarca, and supratentorial cerebral edema were also present.

Case 2

A 52-year-old man with type I diabetes, hypertension, and dyslipidemia presented with non–ST-elevation myocardial infarction. Emergent triple-vessel CABG and right coronary endarterectomy were performed. Before CPB, TEE revealed an LV ejection fraction of 27% with normal RV function. Inhaled prostacyclin and milrinone were given, resulting in a transient 5 mm Hg RVOT gradient (Figure 2A). Total CPB duration and aortic cross-clamp were 72 and 47 minutes, respectively. CPB separation was difficult, requiring norepinephrine 0.12 µg/kg/min, intratracheal milrinone 5 mg, and a single asynchronous defibrillation attempt. Immediately after CPB separation and intratracheal milrinone administration, new-onset Prv to Ppa systolic pressure gradient up to 15 mm Hg was observed (Figure 2B). No hypotension was noted when the obstruction was present, and no potential mechanical causes of RVOTO, such as a compression by a thrombus, were seen on TEE. Post-CPB TEE revealed RVOTO, improved global LV function, and increased anterior wall contractility. On ICU arrival, the Prv-Ppa systolic pressure gradient increased up to 30 mm Hg. The patient was on vasopressor support (norepinephrine 0.02 µg/kg/min and epinephrine 0.05 µg/kg/min) when the obstruction was noted. Norepinephrine was weaned shortly after admission, and epinephrine was continued until the next morning. The systolic gradient persisted 2 hours later, without change in the patient's medications or clinical status. The evolution and continuous recording of the systolic Prv and Ppa gradient is shown in Figure 3. In addition, increased RV and LV change in pressure over time (dP/dT) was observed after administration on intratracheal milrinone, which persisted in the ICU. The patient was extubated 2 hours after admission and recovered after weaning him from vasoactive agents after 15 hours. He stayed 26 hours in the ICU and 8 days in the hospital. His postoperative fluid balance was neutral (2538 mL input and 2525 mL output).

DISCUSSION

RVOTO is a rare complication occurring in 0.05% to 4% of cardiac surgeries,[4,10] with hemodynamic instability occurring in 91% of cardiac surgical patients.[4] The higher frequency in the latter study resulted from routine use of Ppa and Prv monitoring. Hemodynamic and echocardiographic methods can be used to detect and diagnose RVOTO.[4,9] The simplest and most precise method is to measure the pressure gradient between the RV and pulmonary artery. This can be done by pulling back the pulmonary artery catheter with the tip lying in the RV. It should not be permanently left in the RV due to the risk of ventricular arrhythmia. A normal systolic gradient between the Prv and Ppa should be <6 mm Hg. Any difference in peak systolic pressure ≥6 mm Hg signifies RVOTO. The obstruction is clinically significant when the pressure difference exceeds 25 mm Hg.[4,11] Diagnosis of RVOTO can be suspected with TTE and/or TEE using M-Mode, 2-dimensional (2D) and 3-dimensional (3D) modalities or color Doppler. On 2D TEE, dynamic RVOTO is seen as early systolic obliteration of the RVOT.[9] Other signs include reduced RVOT size, systolic or diastolic obstruction of the RVOT,[12] the presence of a surrounding mass or hematoma and RVOT color flow acceleration. An elevated pressure gradient across the tricuspid valve above the measured or estimated Ppa[4] should raise suspicion for RVOTO.

The etiology of RVOTO can be classified according to mechanical pathology or dynamic changes in the patient's

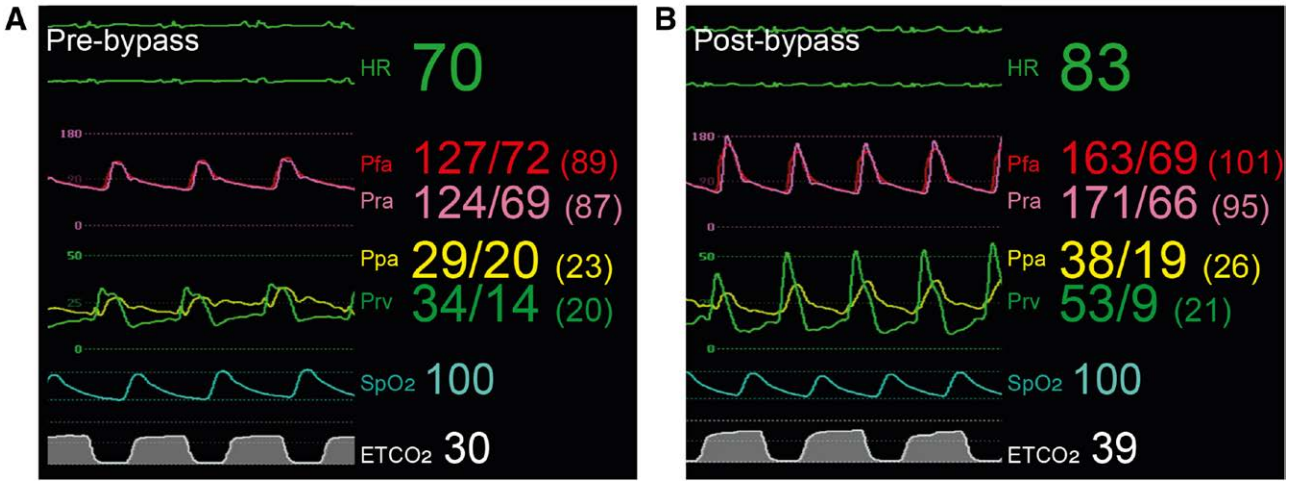

Figure 2. Hemodynamic waveforms before (A) and after (B) CPB. Note the significant increase in the 15 mm Hg systolic pressure gradient between the Prv and the Ppa after CPB. CPB indicates cardiopulmonary bypass; $ETco_2$, end-tidal carbon dioxide; HR, heart rate, Pfa, femoral arterial pressure; Ppa, pulmonary artery pressure; Pra, radial arterial pressure; Prv, right ventricular pressure; Spo_2, oxygen saturation using pulse oximetry.

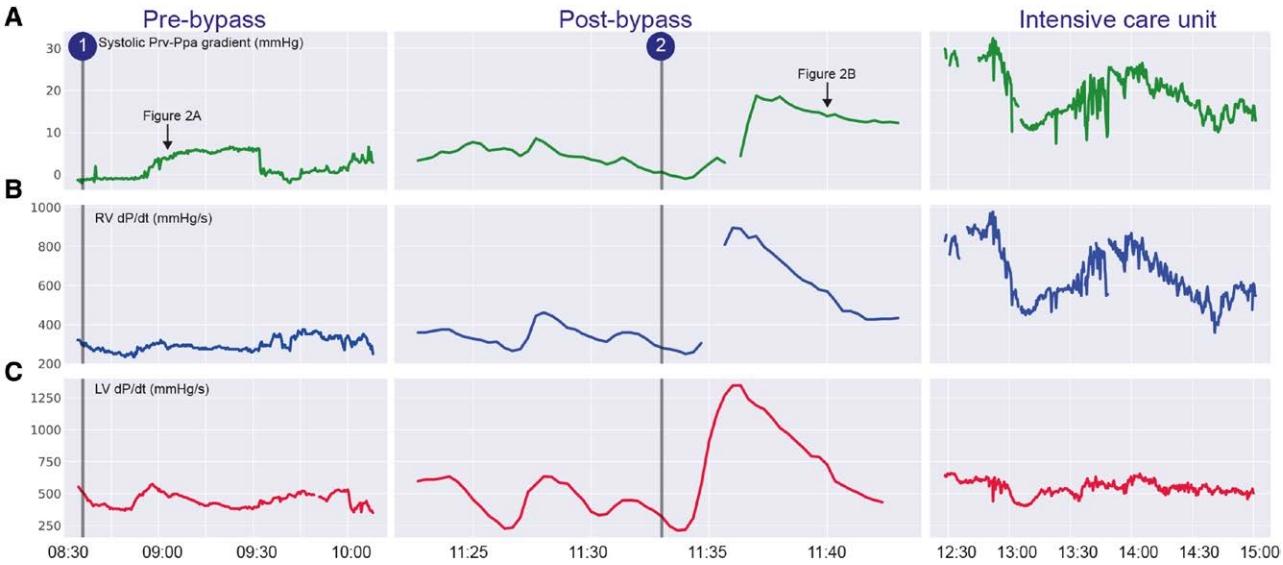

Figure 3. Patient #2. A, Continuous recording of changes in Prv and Ppa systolic gradient. B, Continuous recording of changes in systolic Prv over time (RV d*P*/d*t*) and (C) LV changes in systolic pressure over time (LV d*P*/d*t*) before CPB, after CPB and in the ICU. Note the mild increase in the systolic Prv and Ppa gradient following administration of combined inhaled milrinone and prostacyclin (1) before CPB but the more significant increase after the administration of 5 mg of intratracheal milrinone (2). The RV systolic pressure gradient persisted in the ICU. Note also the parallel increase in both RV and LV d*P*/d*t* after intratracheal milrinone that also persisted also in the ICU. CPB indicates cardiopulmonary bypass; ICU, intensive care unit; LV, left ventricular; Ppa, pulmonary artery pressure; Prv, right ventricular pressure; RV, right ventricular.

physiology. Mechanical RVOTO may be intrinsic, as seen in patients with hypertrophic cardiomyopathy, or extrinsic due to external compression. In adult populations, extrinsic mechanical causes of RVOTO can include mediastinal hematomas and tumors, iatrogenic obstruction after various cardiac surgery procedures, RV aneurysm or tension pneumothorax.[10,13] Treatment is often surgical and determined by the underlying pathology.

So far, there have been 233 studies examining RVOTO, 229 of which are case reports and series, and 4 of which were retrospective and prospective observational studies.[10] Approximately 90% are of mechanical origin, with 58% being intrinsic causes and 42% extrinsic. Extracardiac tumor metastasis represents the most commonly reported cause of RVOTO.

On the other hand, dynamic RVOTO represents only 10% of reported cases (n = 29).[10] Dynamic RVOTO is due to changes in patient physiology. These include changes in volume status and use of inotropic agents,[4,9] particularly in the context of hypertrophic cardiomyopathy.[13] Dynamic RVOTO may also occur after lung transplantation[3] and pulmonic stenosis correction.[8] The term "suicide" RV has been used to describe dynamic RVOTO occurring after acute reduction of RV afterload, such as after lung transplantation or due to RVOT spasm during catheterization.[1,3,7] Figure 4 summarizes the various etiologies and mechanisms of RVOTO.

Our first patient had a complex postoperative course with compromised coronary circulation and RV failure, culminating in multiorgan failure. Combined intraoperative and postoperative RVOTO has never been previously reported in the context of aortic dissection. In this case, the observed phenomenon likely had both mechanical and dynamic factors. After surgery, inotropes used for LV dysfunction might have contributed to the development of RVOTO. However, even as these were withdrawn and the heart rate was lowered by epicardial pacing,[14] persistent RVOTO was still observed, supporting an origin of RVOTO that is more mechanical than dynamic. The associated hemodynamic instability, prolonged hospitalization, and mortality[4] with significant intraoperative RVOTO are consistent with the evolution of this patient. RV dysfunction has already been reported after relief of tamponade. The mechanism is thought to be related to RV volume overload from an increase in venous return, which, if severe, can lead to a decompression syndrome.[15–17] In addition, elevated pericardial pressure and reduced aortic pressure will result in reduction in coronary perfusion pressure. This can lead to subendocardial ischemia and myocardial dysfunction.[18,19] This is consistent with the autopsy report showing multifocal areas of subendocardial ischemia. Finally, biventricular myocardial depression as a result of bowel edema and bacterial translocation could also be an addition mechanism of hemodynamic instability in this patient.[20,21] Further, high-dose vasopressors for a prolonged period combined with low cardiac output as described in case 1 promote mesenteric and bowel ischemia and are further associated with high mortality.[22] It is possible that RV failure was more important than RVOTO in determining the outcome of this patient, and the use of RV mechanical assistance should have been considered at an earlier stage.[23] However, the obstruction created by the RVOTO did not facilitate RV function and recovery. The autopsy confirmed the reduced size of RV outflow tract, the increased weight of the heart from biventricular hypertrophy and edema, as well as bowel edema and hemorrhage. In patients with mechanical RVOTO, particularly those with extrinsic obstruction, the treatment is mostly surgical. However, in this case, the intrinsic obstruction played a more important role than the extrinsic obstruction because of its persistence on sternal opening.

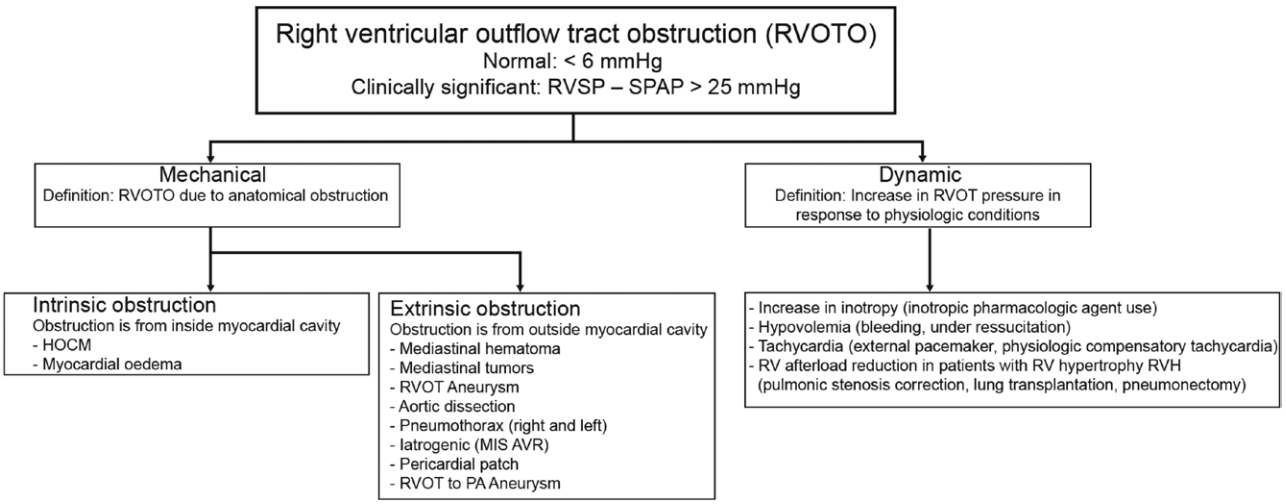

Figure 4. Classification of RVOTO by etiology. HOCM indicates hypertrophic obstructive cardiomyopathy; MIS AVR, minimally invasive surgical aortic valve replacement; PA, pulmonary artery; RV, right ventricle; RVH, right ventricular hypertrophy; RVOT, right ventricle outflow tract; RVOTO, right ventricular outflow tract obstruction; RVSP, right ventricle systolic pressure; SPAP, systolic pulmonary artery pressure.

In the second case, a systolic pressure gradient between the RV and the pulmonary artery developed after CPB following administration of intratracheal milrinone, which was associated with increased in RV and LV performance.[24] Dynamic RVOTO after CPB and continuing into the ICU period without significant hemodynamic instability has not been reported. Dynamic RVOTO is unlikely to require any further investigation or treatment except withdrawing the inotropic agent, which can make it worse.

In a recent systematic review, the overall prevalence of RVOTO in cardiac surgery is estimated at 4% (1%–9%).[10] An ongoing current prospective study using RV pressure monitoring will determine the exact prevalence of RVOTO in the operating room and in the ICU (NCT04092855). The diagnosis of RVOTO can be made instantaneously using RV pressure waveform monitoring and should be considered in any hemodynamically unstable patient after cardiac surgery, particularly those who deteriorate after inotropic medication or chest closure.

CONCLUSIONS

Two cases of RVOTO in adult patients observed in the ICU after cardiac surgery with different outcomes were presented. The obstruction was persistent and mechanical in the first case, while it was dynamic and transient in the second case. Recognition of RVOTO is important because the hemodynamic and surgical management is unique to this etiology.

DISCLOSURES

Name: Yu Hao Zeng, MD.
Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and drafting and revising the article critically for important intellectual content.
Conflicts of Interest: None.
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Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and drafting and revising the article critically for important intellectual content.
Conflicts of Interest: None.
Name: William Beaubien-Souligny, MD, PhD.
Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and drafting and revising the article critically for important intellectual content.
Conflicts of Interest: None.
Name: Matthew P. Aldred, MBBS, FANZCA.
Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and drafting and revising the article critically for important intellectual content.
Conflicts of Interest: None.
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Conflicts of Interest: None.
Name: Ali Hammoud, BScN.
Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and drafting and revising the article critically for important intellectual content.
Conflicts of Interest: None.
Name: Cristhian Potes, MSc, PhD.
Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and drafting and revising the article critically for important intellectual content.
Conflicts of Interest: None.
Name: Yoan Lamarche, MD.
Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and drafting and revising the article critically for important intellectual content.
Conflicts of Interest: None.
Name: André Y. Denault, MD, PhD.
Contribution: This author helped with the conception and design, data acquisition, analysis, and interpretation, and

drafting and revising the article critically for important intellectual content.

Conflicts of Interest: A. Y. Denault is on the speakers bureau for CAE Healthcare Inc. (2012), Masimo (2017), and received an equipment grant from Edwards (2019).

This manuscript was handled by: Kent H. Rehfeldt, MD.

REFERENCES

1. Kroshus TJ, Kshettry VR, Hertz MI, Everett JE, Bolman RM III. Suicide right ventricle after lung transplantation for Eisenmenger syndrome. *Ann Thorac Surg*. 1995;59:995–997.
2. Denault A, Ferraro P, Couture P, et al. Transesophageal echocardiography monitoring in the intensive care department: the management of hemodynamic instability secondary to thoracic tamponade after single lung transplantation. *J Am Soc Echocardiogr*. 2003;16:688–692.
3. Gangahanumaiah S, Scarr BC, Buckland MR, Pilcher DV, Paraskeva MA, McGiffin DC. Suicide right ventricle after lung transplantation for pulmonary vascular disease. *J Card Surg*. 2018;33:412–415.
4. Denault AY, Chaput M, Couture P, Hébert Y, Haddad F, Tardif JC. Dynamic right ventricular outflow tract obstruction in cardiac surgery. *J Thorac Cardiovasc Surg*. 2006;132:43–49.
5. Tardif JC, Taylor K, Pandian NG, Schwartz S, Rastegar H. Right ventricular outflow tract and pulmonary artery obstruction by postoperative mediastinal hematoma: delineation by multiplane transesophageal echocardiography. *J Am Soc Echocardiogr*. 1994;7:400–404.
6. Kirshbom PM, Tapson VF, Harrison JK, Davis RD, Gaynor JW. Delayed right heart failure following lung transplantation. *Chest*. 1996;109:575–577.
7. Singhal A, Kumar S, Kapoor A. Sudden iatrogenic suicidal right ventricle. *Indian Heart J*. 2015;67:406–408.
8. Munirathinam GK, Kajal K, Jayant A, Dogra N, Singh H. Epicardial echocardiography as rescue modality for detection of dynamic right ventricular outflow tract obstruction in post pulmonary valve stenosis repair. *A A Pract*. 2019;13:396–398.
9. Raymond M, Grønlykke L, Couture EJ, et al. Perioperative right ventricular pressure monitoring in cardiac surgery. *J Cardiothorac Vasc Anesth*. 2019;33:1090–1104.
10. Zeng YH, Calderone A, Rousseau-Saine N, et al. Right ventricle outflow tract obstruction in adults: a systematic review and meta-analysis. *CJC Open*. 2021. Accessed April 8, 2021. https://doi.org/10.1016/j.cjco.2021.03.011.
11. Therrien J, Dore A, Gersony W, et al; Canadian Cardiovascular Society. CCS Consensus Conference 2001 update: recommendations for the management of adults with congenital heart disease. Part I. *Can J Cardiol*. 2001;17:940–959.
12. Vegas A, Denault A, Royse C. A bedside clinical and ultrasound-based approach to hemodynamic instability - part II: bedside ultrasound in hemodynamic shock: continuing professional development. *Can J Anaesth*. 2014;61:1008–1027.
13. Malik R, Maron MS, Rastegar H, Pandian NG. Hypertrophic cardiomyopathy with right ventricular outflow tract and left ventricular intracavitary obstruction. *Echocardiography*. 2014;31:682–685.
14. Aldred MP, Elhaj B, Zeng YH, et al. Right ventricular epicardial pacing postcardiac surgery can cause dynamic right ventricular outflow tract obstruction: a case report. *A A Pract*. 2020;14:e01346.
15. Pradhan R, Okabe T, Yoshida K, Angouras DC, DeCaro MV, Marhefka GD. Patient characteristics and predictors of mortality associated with pericardial decompression syndrome: a comprehensive analysis of published cases. *Eur Heart J Acute Cardiovasc Care*. 2015;4:113–120.
16. Chung J, Ocken L, Wolo E, Herman CR, Goldhammer JE. Acute right ventricular failure after surgical drainage of pericardial tamponade: a case report of pericardial decompression syndrome and review of the literature. *J Cardiothorac Vasc Anesth*. 2019;33:768–771.
17. Ricarte Bratti JP, Brunette V, Lebon JS, Pellerin M, Lamarche Y. Venoarterial extracorporeal membrane oxygenation support for severe pericardial decompression syndrome: a case report. *Crit Care Med*. 2020;48:e74–e75.
18. Armstrong WF, Feigenbaum H, Dillon JC. Acute right ventricular dilation and echocardiographic volume overload following pericardiocentesis for relief of cardiac tamponade. *Am Heart J*. 1984;107:1266–1270.
19. Anguera I, Paré C, Perez-Villa F. Severe right ventricular dysfunction following pericardiocentesis for cardiac tamponade. *Int J Cardiol*. 1997;59:212–214.
20. Sandek A, Swidsinski A, Schroedl W, et al. Intestinal blood flow in patients with chronic heart failure: a link with bacterial growth, gastrointestinal symptoms, and cachexia. *J Am Coll Cardiol*. 2014;64:1092–1102.
21. Sundaram V, Fang JC. Gastrointestinal and liver issues in heart failure. *Circulation*. 2016;133:1696–1703.
22. Auchet T, Regnier MA, Girerd N, Levy B. Outcome of patients with septic shock and high-dose vasopressor therapy. *Ann Intensive Care*. 2017;7:43.
23. Saeed D, Maxhera B, Kamiya H, Lichtenberg A, Albert A. Alternative right ventricular assist device implantation technique for patients with perioperative right ventricular failure. *J Thorac Cardiovasc Surg*. 2015;149:927–932.
24. Gebhard CE, Rochon A, Cogan J, et al. Acute right ventricular failure in cardiac surgery during cardiopulmonary bypass separation: a retrospective case series of 12 years' experience with intratracheal milrinone administration. *J Cardiothorac Vasc Anesth*. 2019;33:651–660.