Spinal Anesthesia for Emergent Abdominal Surgery in a Patient With a Tricuspid Valvectomy: A Case Report

Katherine Henderson Harold, DO, and Michael Webster, MD

Tricuspid valvectomy without replacement is an accepted treatment for drug-resistant endocarditis. The current intravenous drug abuse epidemic is leading to more occurrences of right-sided valvar endocarditis. This suggests that the incidence of tricuspid valvectomies may rise. Our academic hospital has seen 3 such patients in the past year alone. We review the implications of this pathophysiology and discuss the anesthetic management of a 33-year-old woman with previous tricuspid valvectomy presenting for emergent abdominal surgery. (A&A Practice. 2018;10:185–7.)

Described by Arbula in 1971, tricuspid valvectomy without replacement has remained a surgical treatment for intractable tricuspid endocarditis. Tricuspid regurgitation (TR), such as that created by a valvectomy, is an independent marker for increased mortality regardless of patient age, left ventricular ejection fraction, or pulmonary status. The current US drug epidemic is leading to more occurrences of right-sided valvar endocarditis. This suggests that the incidence of tricuspid valvectomies may rise. Our academic hospital has seen 3 such patients in the past year alone.

Written consent was obtained from the patient to present and publish this case report.

CASE DESCRIPTION

A 33-year-old woman with an incarcerated umbilical hernia presented for emergent exploratory laparoscopy. Medical and surgical history included hepatitis C, gastroesophageal reflux disease, intravenous (IV) drug abuse, congestive heart failure, and methicillin-susceptible Staphylococcus aureus endocarditis. She had a tricuspid valve replacement in 2013 with tricuspid prosthetic excision 1 year later for recurrent endocarditis. Her current medications were methadone 60 mg daily and furosemide 80 mg twice daily.

Transesophageal echocardiogram in 2015 revealed mild left ventricular systolic dysfunction with an ejection fraction of 40%–45%. The tricuspid valve was absent. The right atrium was severely dilated; the right ventricle was severely enlarged (Figure 1). Pulmonary artery pressure was normal. The tricuspid annulus was akinetic. The inferior vena cava was enlarged with <50% variation during inspiration.

Physical examination revealed an alert woman. Blood pressure was 104/64 mm Hg; heart rate 81 beats per min; respiratory rate 11 breaths per min; and oxygen saturation 97% on room air. Bilateral lower extremity pitting edema was present and a peritoneal fluid wave was demonstrated. She acknowledged the inability to walk more than 10 yards without having to rest because of dyspnea.

After discussion with the surgeon and patient, an open umbilical approach with spinal anesthesia was a safer option for this patient than a laparoscopic approach under general anesthesia. A low-dose norepinephrine infusion 1 μg/min was begun on arrival at the operating room. A spinal was placed using bupivacaine 15 mg alone. Minimal-to-moderate sedation was provided with midazolam 2 mg IV and a low-dose propofol infusion 25 μg/kg/min. Loss of sensation was assessed to the level of T4 by pinprick. The patient underwent open umbilical herniorrhaphy and small-bowel resection without complication. She complained briefly of nausea on bowel clamping but experienced no other problems. The norepinephrine infusion was weaned before conclusion of the procedure.

She was monitored for hemodynamic stability in the intensive care unit for 12 hours and transferred to the step down floor later the same day. Hydromorphone IV was used to treat initial postoperative pain. She was transitioned back on her home methadone approximately 6 hours after surgery. After return of bowel function and improvement of her heart failure symptoms, she was discharged home on postoperative day 2.

DISCUSSION

Tricuspid valvectomy presents unique physiologic consequences that the anesthesiologist must be prepared to manage. Excision of the valve leaflets essentially induces a Fontan physiology where blood flow is passively conducted to the pulmonic system. The effects of normal tricuspid leaflet impact on right ventricular (RV) flow is illustrated in Figure 2. Comparing this depiction with our patient’s transesophageal echocardiogram (Figures 1 and 3), severe unobstructed systolic regurgitation and bidirectional flow during diastole is seen. In these patients, especially those exhibiting right heart failure, venous return must be maintained and pulmonary vascular resistance (PVR) be minimized.

Laparoscopic surgery may have negative hemodynamic consequences for these patients. Abdominal insufflation diminishes venous return, while PVR is increased both by absorption
of carbon dioxide and from cephalad displacement of abdominal contents. The lesion in this patient prohibits standard ventilatory techniques to eliminate excess carbon dioxide (discussed further below). Laparoscopic surgery can however have the advantage of decreased postoperative pain and hospital length of stay. If one considers this patient’s physiology similar to Fontan physiology, there is evidence of a successful laparoscopic approach with a stable outcome. It was felt this patient would benefit most from minimizing fluctuations in her right heart afterload with an open surgical approach.

Anesthetic goals for a patient with TR and right heart failure include maintaining adequate preload, ensuring sinus rhythm, and minimizing RV afterload, namely PVR. Avoiding hypoxia, hypercarbia, and acidosis is paramount in these patients for their detrimental effects on PVR. We chose to perform a spinal anesthetic on this patient to maintain spontaneous ventilation. An epidural is a suitable alternative; however, in this intraabdominal case, we desired rapid and profound blockade.

Minimal sedation was provided to the patient with IV midazolam and low-dose propofol infusion. The choice of which anesthetic agent to use and the level of sedation should be tailored to each patient. Dexmedetomidine or ketamine would also have been suitable because they offer hemodynamic stability and decrease the chance of ventilatory depression. One should be cautious with sedation to the point of causing respiratory depression as we have discussed the detrimental effects of hypercarbia in the patients.

A norepinephrine infusion was initiated to maintain preload and counter the hemodynamic consequences of spinal blockade. While a fluid bolus compensates for vasodilatation from spinals, we were hesitant to administer one because of this patient’s right-sided fluid overload. In a review of management options for RV dysfunction, norepinephrine in doses <0.5 µg/kg/min was found to increase mean arterial pressure without increasing PVR, making it a good option for this case. An increase in PVR can have detrimental effects on RV ejection and negatively impact overall outcomes. Milrinone is a good option in this patient population for its ability to increase chronotropy while decreasing pulmonary and systemic vascular resistance. It can however lead to hypotension from decreased venous return and venous pooling. If available, inhaled pulmonary vasodilators such as nitric oxide and epoprostenol may be ideal.

Maintaining spontaneous ventilation with a spinal has potential hemodynamic advantages in this patient. Spontaneous inspiration creates a negative intrathoracic pressure gradient favoring venous return into the right heart. This is supported by evidence that TR has been shown to be dynamic throughout the respiratory cycle with forward blood flow augmented on inspiration. The physiologic cost of positive pressure ventilation (PPV) negates this hemodynamic effect that may be critical in a patient exhibiting right heart failure and severe TR.

PVR is known to increase as lung volume and positive end-expiratory pressure (PEEP) rise with the institution of PPV. Jardin et al illustrated that, at physiologic tidal breaths, no significant change occurred in PVR. However, a progressive
increase in RV pressure was necessary to eject blood into the pulmonic system in the face of increasing lung volumes and the institution of PEEP. Therefore, if general anesthesia and PPV are necessary, we advocate the use of low tidal volumes, low rates with minimal PEEP of 1–3 mm Hg. In the case of inadequate anesthesia from our spinal, we would have induced general anesthesia while maintaining the above ventilatory parameters. A milrinone infusion would have been our inotrope of choice given signs of decreased cardiac output.

In conclusion, we have illustrated neuraxial anesthesia with efforts to maintain preload and minimize PVR as a safe option for this difficult patient population. We are left with the need for further investigation into the implications that this surgical technique has on right heart function and how to best serve these patients in the operating room.

**DISCLOSURES**

**Name:** Katherine Henderson Harold, DO.

**Contribution:** This author was the primary writer and investigator.

**Name:** Michael Webster, MD.

**Contribution:** This author was the co-author and investigator.

This manuscript was handled by: Raymond C. Roy, MD.

**REFERENCES**

1. Arbula A, Thomas NW, Chiscano A, Wilson RF. Total tricuspid valvulectomy without replacement in the treatment of pseudo-monas endocarditis. *Surg Forum*. 1971;11:162–164.

2. Arbula A, Holmes RF. Tricuspid valvectomy without replacement. Twenty years’ experience. *J Thorac Cardiovasc Surg*. 1991;102:917–922.

3. Nath J, Foster E, Heidenreich PA. Impact of tricuspid regurgitation on long-term survival. *J Am Coll Cardiol*. 2004;43:405–409.

4. Kim JB, Ejiofor JI, Yammine M, et al. Surgical outcomes of infective endocarditis among intravenous drug users. *J Thorac Cardiovasc Surg*. 2016;152:832–841.e1.

5. Nayak S, Booker PD. The Fontan circulation. *BR J Anaesth*. 2008;8:26–30.

6. Fredriksson AG, Zajac J, Eriksson J, et al. 4-D blood flow in the human right ventricle. *Am J Physiol Heart Circ Physiol*. 2011;301:H2344–H2350.

7. Sheehan F, Redington A. The right ventricle: anatomy, physiology and clinical imaging. *Heart*. 2008;94:1510–1515.

8. Perrin M, Fletcher A. Laparoscopic abdominal surgery. *Conti Educ Anaesth Crit Care Pain*. 2004;4:107–110.

9. Pans SJ, van Kimmenade RR, Ruurda JP, Meijboom FJ, Sieswerda GT, van Zaane B. Haemodynamics in a patient with Fontan physiology undergoing laparoscopic cholecystectomy. *Neth Heart J*. 2015;23:383–385.

10. Radosevich MA, Brown DR. Anesthetic management of the adult patient with concomitant cardiac and pulmonary disease. *Anesthesiol Clin*. 2016;34:633–643.

11. Price LC, Wort SJ, Finney SJ, Marino PS, Brett SJ. Pulmonary vascular and right ventricular dysfunction in adult critical care: current and emerging options for management: a systematic literature review. *Crit Care*. 2010;14:R169.

12. Jardin F, Vieillard-Baron A. Right ventricular function and positive pressure ventilation in clinical practice: from hemodynamic subsets to respiratory settings. *Intensive Care Med*. 2003;29:1426–1434.

13. Topilsky Y, Tribouilloy C, Micheletta HJ, Pislaru S, Mahoney DW, Enriquez-Sarano M. Pathophysiology of tricuspid regurgitation: quantitative Doppler echocardiographic assessment of respiratory dependence. *Circulation*. 2010;122:1505–1513.

14. Di Mauro M, Bezante GP, Di Baldassarre A, et al; Italian Study Group on Valvular Heart Disease Italian Society of Cardiology. Functional tricuspid regurgitation: an underestimated issue. *Int J Cardiol*. 2013;168:707–715.