Role of two-dimensional transoesophageal echocardiography in the management of a right ventricular tumour

Saul Lebovic MD, Robert Koorn MD, David L. Reich MD

Right ventricular outflow tract tumours are rarely reported. The perioperative management of a patient presenting for excision of an obstructing right ventricular outflow tract myxoma is described. Two-dimensional transoesophageal echocardiography was performed intraoperatively and was found to be of considerable value in understanding the pathophysiology of the tumour. Following excision of the tumour, right ventricular function improved and tricuspid regurgitation resolved.

Il est rare d'avoir affaire à une tumeur de la chambre de chasse du ventricule droit. Voici l'histoire d'un patient qui est venu à l'hôpital pour excision d'un tel myxome obstructif. L'usage peropératoire d'un échocardiographe bidimensionnel transoesophagien a permis de mieux comprendre la pathophysiologie de la tumeur. Après l'excision de cette dernière, il y eu amélioration de la fonction du ventricule droit et disparition de l'insuffisance tricuspide.

Tumours of the heart are uncommon and difficult to diagnose clinically. Although they were first described in the seventeenth century,¹ antemortem diagnosis was rare before 1950.² However, the introduction of angiography and echocardiography, as well as increased clinical awareness, have led to more frequent diagnosis of these

lesions.³⁻⁵ Two-dimensional transoesophageal echocardiography (2D-TEE) may be of considerable benefit both in the diagnosis and anaesthetic management of these patients.

We report a case of right ventricular outflow obstruction due to a tumour originating in the right ventricular outflow tract. The intraoperative management of this patient during excision of the tumour, and the additional information gained from intraoperative 2D-TEE are described.

Case report

A 58-yr-old woman presented for excision of a right ventricular mass. She had a history of hypertension, controlled with verapamil, and of chronic obstructive pulmonary disease with a bronchospastic component, possibly related to smoking. She had received oral theophylline preparations sporadically and inhaled albuterol for this problem. She presented with a one-week history of increasing shortness of breath, and a productive cough. During the evaluation of her persistent tachycardia and cardiac murmur, cardiac catheterization was performed, which revealed a large mobile mass within the right ventricular outflow tract. The mass was reported to be non-obstructive during angiography, but this could not be confirmed using transthoracic echocardiography because of a technically poor study.

On physical examination, she appeared to be in good general health, was 155 cm tall, weighed 56 kg, had a BP of 170/80, and a regular heart rate of 120 bpm. The lung fields were clear to auscultation and percussion. Auscultation of the heart revealed a grade II/VI systolic murmur best heard over the left upper sternal border, and a grade II/IV diastolic murmur was audible over the precordium. The pulmonic component of the second heart sound was muffled and delayed. The chest radiograph showed a normal cardiac silhouette and increased interstitial markings. The ECG was remarkable for sinus tachycardia, 0.1

Key words

ANAESTHESIA: cardiac;

MEASUREMENT TECHNIQUES: Doppler ultrasound, transoesophageal.

From the Department of Anesthesiology, Mount Sinai Medical Center, 1 Gustave L. Levy Place, Box 1010, New York, NY 10029-6574.

Address correspondence to: Dr. David Reich.

Accepted for publication 3rd June, 1991.

mV ST-segment depression and a flattened T wave in lead I only.

The patient received morphine, 5 mg, and scopolamine, 0.2 mg *im*, for preanaesthetic medication, and was transported to the operating suite with O₂ administered via nasal cannulae. A pulse oximeter attached to her right hand indicated a saturation of 96%. Monitoring included ECG leads II and modified V₅, and a right radial arterial catheter. The right internal jugular vein was cannulated with an 8.5 French introducer sheath, and a pulmonary artery catheter was advanced to 20 cm, so that the distal port monitored the central venous pressure (CVP).

After preoxygenation with 100% O₂, anaesthesia was induced with fentanyl, 2000 µg, and midazolam, 1 mg *iv*. Tracheal intubation was facilitated with vecuronium, 10 mg. At this time, there was a marked slowing of the heart rate (HR) from 120 to 60 bpm, associated with a decrease in MAP from 120 to 60 mmHg. Ephedrine, 10 mg, and phenylephrine, 160 µg, and 300 ml of crystalloid were administered. This resulted in increases in HR (to 90 bpm) and MAP (to 80 mmHg). Mechanical ventilation was instituted with 100% O₂, and the end-tidal CO₂ was adjusted to 4.0–4.7 kPa (30–35 mmHg) using a capnometer. Isoflurane was added 30 min after induction to control increases in MAP associated with surgical stimuli. It was noted that a tidal volume of 10 ml·kg⁻¹ was associated with a pronounced pulsus paradoxus. The paradox improved with a reduction in the tidal volume and an increased respiratory rate. The heart rate was maintained in the high 90s during the pre-cardiopulmonary bypass (CPB) phase. The haemodynamic variables are summarized in the Table.

The 2D-TEE probe was inserted after induction of anaesthesia and tracheal intubation. The right ventricle was noted to be markedly enlarged with moderate to severe tricuspid regurgitation during late systole. A prominent soft tissue mass was visible within the right ventricular outflow tract, with marked prolapse of the mass into the pulmonary artery during systole (see Figures 1 and 2). The mass completely obstructed the outflow tract during mid-systole. The duration of the obstruction (as a percentage of the cardiac cycle) and the severity of the tricuspid regurgitation increased during inspiration. From the long-axis view of the heart, the right ventricular ejection fraction area was calculated to be 38%, concomitant with a mid-papillary left ventricular ejection fraction area (short-axis view) of 48%.

After instituting hypothermic cardiopulmonary bypass (CPB), the right ventricular outflow tract and pulmonary artery were opened. A large pedunculated fleshy mass was found attached to the right ventricular outflow tract, and prolapsing through the pulmonary valve into the main

TABLE

	Pre-induct.	Post-induct.	Pre-CPB	Post-CPB
HR (bpm)	118	60	90	70
SBP (mmHg)	190	90	120	100
DBP (mmHg)	100	50	60	55
MAP (mmHg)	120	60	80	70
CVP (mmHg)	14	11	10	9
MPAP (mmHg)				27
PCWP (mmHg)				9
CO (L·min ⁻¹)				4.2
LVEFA (%)			48%	42%
RVEFA (%)			38%	50%

Treatment: ephedrine 10 mg; phenylephrine 160 µg; crystalloid 300 ml.

pulmonary artery nearly to the bifurcation. The leaflets of the pulmonary valve were severely damaged by mechanical trauma from the tumour. After excision of the mass, the pulmonary valve was replaced with a 29 mm tissue valve prosthesis, and the outflow tract reconstructed using a pericardial patch. The aortic cross-clamp time was 72 min, and the total duration of CPB was 142 min. A MAP of 55 mmHg was maintained during CPB, and anaesthesia was maintained with increments of pancuronium, fentanyl and midazolam.

The patient separated from CPB without difficulty, and the pulmonary artery catheter was advanced into the pulmonary artery at this time. The right ventricular ejection fraction area improved to 50% (in the absence of tricuspid regurgitation), and the left ventricular ejection fraction area was 42%. The ejection fraction calculations were made from identical echocardiographic views in the pre- and post-CPB periods. Three units of packed red blood cells were transfused, as well as autologous washed red cells scavenged from the surgical field.

The patient was transferred to the intensive care unit in stable condition. The trachea was extubated on the first postoperative day, but was reintubated the following day due to CO₂ retention. A transient oliguria responded to a low dose of dopamine and *iv* furosemide. Persistent hypertension was initially treated with sodium nitropruside, and later with prazosin. The trachea was again extubated on the third postoperative day, and arterial blood gas analyses remained within the normal range thereafter. At this time it was noted that liver function was abnormal (SGOT 3713 U·L⁻¹, SGPT 940 U·L⁻¹, total bilirubin 10.9 mg·dl⁻¹). After this initial peak, the values gradually decreased, and were normal on the ninth postoperative day (SGOT 33 U·L⁻¹, SGPT 72 U·L⁻¹, total bilirubin 2.5 mg·dl⁻¹). Her further postoperative course was unremarkable, and she was discharged from the hospital on the 15th postoperative day.

FIGURE 1A A transoesophageal echocardiographic view of the right ventricular outflow tract with partial obstruction by the tumour (outlined) during early diastole.

FIGURE 2A A transoesophageal echocardiographic view of the pulmonary artery showing the tumour prolapsing from the right ventricular outflow tract during early systole.

FIGURE 1B A diagram of the echocardiographic view in Figure 1A demonstrating the left atrium, right atrium, aortic root, the right ventricular outflow tract, and the obstructing tumour.

FIGURE 2B A diagram of the echocardiographic view in Figure 2B demonstrating the ascending aorta, the right pulmonary artery, the main pulmonary artery, and the tumour prolapsing into the main pulmonary artery.

Pathological analysis of the tumour showed it to be a myxoma, most likely primary in origin.

Discussion

Primary tumours of the heart are rare, occurring with an incidence of less than 0.001 to 0.28%.^{6,7} They usually present as intracavitary lesions, of which more than 75% are benign.⁸ Current surgical techniques permit complete removal of most benign lesions. In contrast, malignant cardiac tumours have a poor prognosis.⁹ Secondary cardiac neoplasms occur 20 to 40 times more frequently than primary tumours of the heart.^{10,11} The increased

prevalence in recent years is possibly related to more vigorous surgical and radiation therapy in patients with primary neoplasms.¹² Of the benign tumours of the heart, intracardiac myxomas occur most frequently.¹³ Most (75%) are located in the left atrium, with 18% occurring in the right atrium and 4% occurring in the left and right ventricles, respectively. Other tumours also occur rarely in the right ventricle.¹⁴ Right ventricular tumours usually present with symptoms and manifestations of right-sided heart failure, syncope, unexplained fever, and a murmur consistent with pulmonary valvular stenosis.¹⁵

Whether involving primary or secondary tumours, neoplastic heart disease can only be expressed in a limited variety of ways. The occurrence of pericardial pain, effusion, tamponade, or constriction, rapid increase in heart size, new heart murmurs, electrocardiographic changes, atrial or ventricular arrhythmias, atrioventricular block, or unexplained heart failure are suggestive of secondary invasion of the heart.¹⁶ Intracavitary tumours, however, usually present with one or more effects of a triad of constitutional, embolic, or obstructive lesions. Ninety percent of these patients present with systemic illness characterized by weight loss, fatigue, fever, anaemia (often haemolytic), elevated sedimentation rate, and elevated serum immunoglobulin concentration formed in response to either tumour embolization, degenerative changes within the tumour itself, or changes in the normal cardiac muscle.^{3,4} Pulmonary embolization can also occur.¹⁷ Our patient's vague symptoms of asthma/COPD, increasing shortness of breath and general malaise are consistent with the constitutional symptomatology associated with an intracavitary lesion.

Preoperative echocardiographic assessment of cardiac morphology is particularly useful in these patients.¹⁸ A right-sided tumour (especially myxoma) is a relative contraindication to pulmonary artery catheter insertion because of the risk of embolization. As an alternative, the catheter may be inserted into a central venous location with a sterile sheath assembly, and advanced into the pulmonary artery after the tumour has been resected. Under these circumstances, 2D-TEE is especially well suited for the intraoperative monitoring of these patients.

Depending on the fixed or mobile nature of the tumour, the pathophysiological consequences of outflow obstruction caused by a right ventricular tumour could be managed in two different ways. If preload were maintained high, heart rate maintained low, and contractility diminished, then the increase in size of the right ventricular outflow tract would permit more flow past the lesion. This is analogous to the management scheme for an obstructive cardiomyopathy and might be useful in the management of fixed lesions.¹⁹ Alternatively, if preload were maintained high, heart rate maintained high, and contractility increased, then the increased number and strength of cardiac cycles would maintain the cardiac output in the situation where stroke volume is limited by a mobile, intermittently obstructing lesion. This is analogous to the management scheme for the "ball-valve" obstruction seen with atrial myxomas.²⁰ The hypotensive response to bradycardia, and the intermittent complete "ball-valve" obstruction seen with 2D-TEE suggest that this patient was best managed using the second alternative.

Heavy pre-anaesthetic medication was avoided, as this could have lead to venodilatation, which would reduce the

appropriately elevated preload necessary to overcome the outflow obstruction. The negative inotropy of the potent volatile anaesthetics is a theoretical disadvantage for the myocardium attempting to overcome outflow obstruction, unless this results in relaxation of the outflow tract. A more relevant drawback is the risk of arrhythmias, particularly junctional rhythm (with loss of atrial systole)^{21,22} and venodilatation, which would markedly diminish preload.

At the time of anaesthetic induction, we were not aware that the tumour totally obstructed the right ventricular outflow tract, and felt that the bradycardia associated with the fentanyl/vecuronium induction would improve flow past the lesion. Our error became apparent as the heart rate slowed, and the mean arterial pressure decreased. Hypotension was treated with phenylephrine and ephedrine which returned the heart rate and mean arterial pressure to acceptable values. We also noted the marked pulsus paradoxus, which improved with decreased tidal volume and increased respiratory rate.

Two-dimensional transoesophageal echocardiography and colour flow Doppler mapping revealed the "ball-valve" obstructive nature of the tumour, as well as the late systolic tricuspid regurgitation. The tricuspid regurgitation was most likely due to complete obstruction of the outflow tract by the tumour, with pressure and volume overload in the right ventricle. The haemodynamic consequences of the anaesthetic induction prompted us to adopt an anaesthetic technique relying on a faster heart rate. Further muscle relaxation was accomplished with pancuronium, and isoflurane was used to control hypertension. The pulsus paradoxus may be explained by the increased intrathoracic pressure during inspiration, which diminished venous return to the right heart. The colour flow Doppler jet of tricuspid regurgitation increased during inspiration, suggesting that diminished preload may have caused the tumour to obstruct earlier in the cardiac cycle. The post-CPB echocardiographic images revealed increased right ventricular ejection fraction (area) in the face of diminished tricuspid regurgitation. Although the right ventricle is an ellipsoid structure, the use of endocardial tracings of long-axis echocardiographic images to assess right ventricular function has been validated.²³ These indicated a marked improvement in right ventricular function following cardiopulmonary bypass.

Once surgery had commenced, we occasionally added low concentrations of isoflurane (0.2–0.4%) to facilitate blood pressure control. Although isoflurane is the least metabolized of the potent volatile agents, there have been isolated reports of postoperative hepatitis associated with its use.²⁴ It is unlikely that isoflurane was responsible for this patient's elevated transaminases and serum bilirubin.

A more likely aetiology was hepatic venous congestion due to right-sided heart failure. The existing hepatic dysfunction may have been exacerbated by non-pulsatile blood flow during cardiopulmonary bypass. Another factor that might have contributed to this transient complication was the administration of drugs known to decrease hepatic blood flow (propanolol, nitroprusside, prazosin and furosemide).²⁵

This patient might have been managed better. Pre-operative evaluation could have yielded evidence of increased symptomatology with position changes or exertion, and/or an awake transoesophageal echocardiographic examination would have identified the "ball-valve" obstructive nature of the tumour. Preoperative knowledge of the tumour pathophysiology would have led us to choose a different anaesthetic induction regimen such as ketamine or etomidate in combination with pancuronium.

This case report also illustrates the contribution made by two-dimensional transoesophageal echocardiography to the intraoperative care of a patient with a right ventricular intracavitary tumour. The pathophysiology of the tumour and the responses to changes in anaesthetic management were clarified by intraoperative echocardiography and colour-flow Doppler mapping.

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