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Anaesthetic Management of Right Atrial Myxoma Extending into the Inferior Vena Cava: A Case Report

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Abstract: ***Background**: Cardiac myxomas are the most common benign primary cardiac tumours, most frequently arising in the left atrium. Right atrial myxomas are relatively uncommon and can present unique perioperative anaesthetic challenges, particularly when located near the inferior vena cava (IVC), where they may obstruct systemic venous return and complicate cardiopulmonary bypass (CPB) cannulation. **Case Presentation**: A 26-year-old male weighing 53 kg presented with progressive exertional dyspnoea. Transthoracic echocardiography (TTE) revealed a mobile right atrial mass measuring approximately 4.8 cm near the IVC–hepatic vein junction. Cardiac magnetic resonance imaging (CMR) demonstrated a lesion measuring 3.3 × 1.1 cm extending into the supradiaphragmatic IVC. Preoperative investigations revealed concomitant renal impairment (serum creatinine 2.70 mg/dL). Anaesthetic induction was performed with fentanyl, etomidate, and vecuronium. Intraoperative transoesophageal echocardiography (TEE) guided safe bicaval cannulation and confirmed complete tumour excision following right atriotomy under CPB. **Outcome**: The intraoperative course was uneventful without haemodynamic instability or embolic complications. The patient was successfully weaned from CPB with minimal inotropic support (dopamine 5 µg/kg/min), extubated at 8 hours postoperatively, and discharged on postoperative day 7. **Conclusion**: Right atrial myxomas extending into the IVC present distinct perioperative challenges including obstruction of venous return, risk of tumour embolisation, and complex cannulation strategy. Maintenance of adequate preload, preservation of systemic vascular resistance, judicious titration of PEEP, and intraoperative TEE guidance are the cornerstones of safe perioperative management.*

Keywords: right atrial myxoma; inferior vena cava extension; cardiac anaesthesia; cardiopulmonary bypass; transoesophageal echocardiography; haemodynamic management; tumour embolization

1. Introduction

Primary cardiac tumours are rare, with a reported autopsy incidence of 0.001–0.03%.[1] Among these, myxomas represent nearly 50% of all benign cardiac tumours and remain the most frequently encountered primary intracardiac neoplasm in adults. The left atrium accounts for approximately 75% of cases, with the fossa ovalis being the most common attachment site. Right atrial involvement accounts for only 15–20% of myxomas, and extension into the IVC is distinctly uncommon, reported in fewer than 50 cases in the published literature.[2, 3, 7]

The clinical manifestations of right atrial myxomas are variable and depend on tumour size, mobility, and anatomical relationships. The classic triad of obstruction, embolism, and constitutional symptoms may not always be present; dyspnoea on exertion is often the predominant symptom. When the tumour extends into the IVC, obstruction of systemic venous return is the principal haemodynamic consequence, and the risk of pulmonary embolism from tumour fragmentation is heightened.[4, 8]

From an anaesthetic standpoint, right atrial myxomas with IVC extension present a particularly challenging scenario. The anaesthesiologist must simultaneously manage a patient with reduced right ventricular preload reserve, ensure safe venous cannulation avoiding tumour dislodgement, and maintain vigilance for catastrophic haemodynamic decompensation at induction. Intraoperative TEE has emerged as an indispensable adjunct in this setting.[5, 6]

We report the perioperative anaesthetic management of a 26-year-old male with a right atrial myxoma arising near the IVC–hepatic vein junction and extending into the supradiaphragmatic IVC, and discuss the anaesthetic strategy in the context of recent literature.

2. Case Report

2.1 Clinical Presentation

A 26-year-old male weighing 53 kg presented with a several-month history of progressive exertional dyspnoea without syncope, chest pain, or palpitations. Clinical examination revealed stable haemodynamics with no peripheral oedema or features of congestive cardiac failure.

2.2 Preoperative Investigations

Preoperative laboratory findings are summarised in Table 1. Notably, renal function was impaired, with a serum creatinine of 2.70 mg/dL and blood urea of 63 mg/dL, consistent with chronic kidney disease. Serum electrolytes and coagulation profile were within acceptable limits. A mild normocytic anaemia was present (haemoglobin 10.9 g/dL). The activated partial thromboplastin time (APTT) was mildly prolonged at 40 seconds, noted for perioperative management.

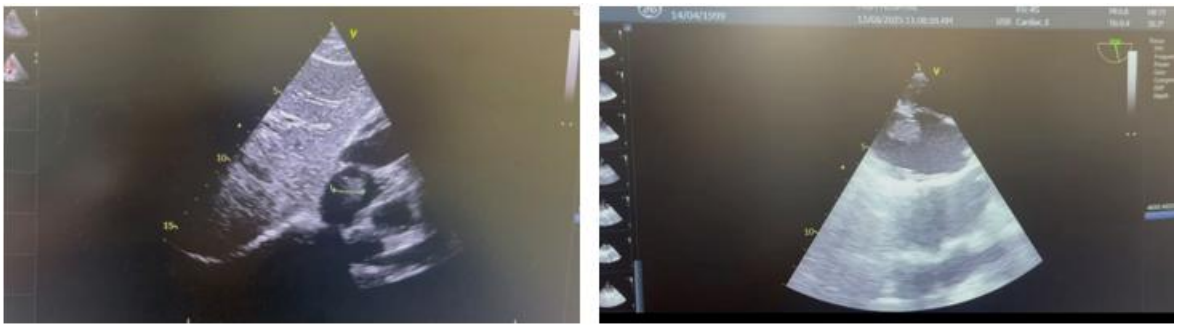
Table 1: Preoperative Laboratory Investigations

Investigation	Value	Reference Range
Haemoglobin	10.9 g/dL *	13.0–17.0 g/dL
Total Leucocyte Count	8688 /mm³	4000–11000 /mm³
Platelet Count	3.16 ×10⁵ /mm³	1.5–4.0 ×10⁵ /mm³
Serum Creatinine	2.70 mg/dL *	0.7–1.2 mg/dL
Blood Urea	63 mg/dL *	7–20 mg/dL
Random Blood Glucose	98 mg/dL	70–140 mg/dL
Total Bilirubin	0.40 mg/dL	0.1–1.2 mg/dL
SGOT (AST)	39 U/L	10–40 U/L
SGPT (ALT)	105 U/L *	7–56 U/L
Sodium (Na^+)	137 mEq/L	135–145 mEq/L
Potassium (K^+)	4.5 mEq/L	3.5–5.0 mEq/L
Prothrombin Time	11.3 s	11–13 s
INR	0.94	0.85–1.15
APTT	40 s *	25–35 s

** Values outside normal reference range. SGOT: serum glutamic oxaloacetic transaminase; SGPT: serum glutamic pyruvic transaminase; INR: international normalised ratio; APTT: activated partial thromboplastin time.*

2.3 Imaging

Transthoracic echocardiography (TTE) demonstrated a mobile, heterogeneous right atrial mass measuring approximately 4.8 cm, arising near the junction of the IVC and hepatic veins (Figures 1A and 1B). The mass prolapsed towards the tricuspid valve during diastole. Left ventricular systolic function was preserved with a calculated ejection fraction of 65%. Cardiac magnetic resonance imaging (CMR) confirmed the mass and demonstrated a lesion of 3.3 × 1.1 cm extending into the supradiaphragmatic IVC, morphologically consistent with a myxoma and excluding thrombus or malignancy.



(a) **(b)**

Figure 1: Preoperative transthoracic echocardiography. (A) Subcostal view demonstrating the right atrial mass with measurement calipers. (B) Modified apical view showing the echogenic mass within the right atrium near the IVC–hepatic vein junction.

2.4 Anaesthetic Management

Following multidisciplinary review by the cardiothoracic surgical and anaesthesiology teams, the patient was scheduled for elective tumour resection under CPB. Written informed consent was obtained from the patient for both surgery and academic publication.

On the day of surgery, standard ASA monitoring was established, including five-lead electrocardiography, pulse oximetry, and end-tidal carbon dioxide monitoring. Invasive arterial pressure monitoring was secured via right femoral artery cannulation prior to induction, given the haemodynamic unpredictability anticipated. A central venous catheter was inserted into the right internal jugular vein under real-time ultrasound guidance, with the catheter tip deliberately positioned in the superior vena cava (SVC) to avoid contact with or displacement of the tumour.

Anaesthetic induction was performed with slow intravenous fentanyl (3 µg/kg) to attenuate the sympathetic response to laryngoscopy, followed by etomidate (0.3 mg/kg) and vecuronium (0.1 mg/kg). Etomidate was specifically selected for its haemodynamic neutrality: it preserves SVR and myocardial contractility, and does not cause histamine release, making it particularly appropriate in patients with impaired venous return.[10, 11] Propofol was deliberately avoided given its established vasodilatory properties and reduction in preload. Tracheal intubation was performed under direct laryngoscopy without haemodynamic compromise.

Anaesthesia was maintained with an oxygen–air mixture (FiO_2 0.5) and sevoflurane at 0.8–1.0 minimum alveolar concentration (MAC), supplemented with intermittent fentanyl boluses and vecuronium. Positive end-expiratory pressure (PEEP) was deliberately minimised throughout, as elevated PEEP increases intrathoracic pressure and further impairs venous return, worsening IVC obstruction.[3, 6] Normovolaemia was maintained by close attention to the CVP waveform and intraoperative TEE findings.

A multiplane TEE probe was inserted following tracheal intubation. Intraoperative TEE served four critical functions: (i) characterisation of tumour dimensions, attachment site, and IVC extent; (ii) real-time guidance for safe bicaval venous cannulation to avoid tumour fragmentation; (iii) continuous monitoring of right ventricular volume and function during CPB; and (iv) immediate post-excision confirmation of complete tumour removal, unobstructed tricuspid valve inflow, and satisfactory biventricular function.[4, 5]

After systemic heparinisation (300 IU/kg, target ACT >480 seconds), CPB was established via aortic and bicaval cannulation. The IVC cannula was introduced with particular caution under direct TEE guidance to prevent tumour dislodgement. Myocardial protection was achieved with cold

crystalloid cardioplegia. A right atriotomy was performed, and the tumour- identified as a gelatinous, lobulated mass attached near the IVC–hepatic vein junction- was excised in toto. The pedicle attachment site was cauterised to reduce the risk of local recurrence.

2.5 Postoperative Course

The patient was successfully weaned from CPB on dopamine infusion at 5 µg/kg/min. Haemodynamic stability was maintained, and the dopamine infusion was discontinued within 12 hours. The trachea was extubated approximately 8 hours postoperatively. Postoperative renal function was closely monitored given the preoperative renal impairment; creatinine did not deteriorate significantly from baseline. The patient was discharged from the intensive care unit on postoperative day 2 and from hospital on postoperative day 7, without neurological, thromboembolic, or wound complications.

Gross examination of the excised specimen revealed a gelatinous, lobulated mass with haemorrhagic foci measuring approximately 3.5 × 2.0 × 1.5 cm (Figure 2). Histopathological examination confirmed a cardiac myxoma: characteristic stellate and polygonal cells embedded in an abundant myxoid matrix, with no evidence of malignancy.

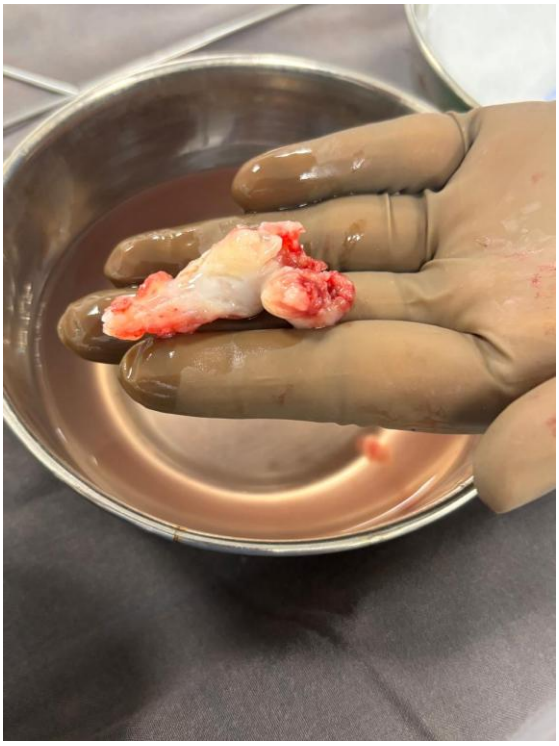


Figure 2: Gross specimen of the excised right atrial myxoma demonstrating gelatinous consistency, lobulated surface, and haemorrhagic foci. Dimensions approximately 3.5 × 2.0 cm.

3. Discussion

This case report illustrates the anaesthetic management of a young patient with a right atrial myxoma extending into the supradiaphragmatic IVC- a rare and surgically demanding variant of an already uncommon tumour. The case is instructive for several interconnected reasons: the young age of the patient, significant preoperative renal impairment, the IVC extension complicating cannulation strategy, and the successful avoidance of intraoperative haemodynamic deterioration.

3.1 Haemodynamic Considerations

The pathophysiology of right atrial myxoma with IVC involvement differs fundamentally from its left-sided counterpart. While left atrial myxomas obstruct mitral valve inflow and may simulate mitral stenosis, right-sided lesions impair systemic venous drainage and reduce right ventricular preload and cardiac output.[3, 8] The consequence is a 'fixed preload' state: any further reduction in venous return- from vasodilatation, high PEEP, or hypovolaemia- precipitates rapid cardiovascular decompensation.

Published reports of right atrial myxoma anaesthesia consistently emphasise the imperative of maintaining normovolaemia and avoiding agents that reduce SVR or venous return.[1, 6] Qu et al. (2024), reporting a case of giant right atrial myxoma at altitude, specifically highlighted adequate volume preload therapy and continuous echocardiographic monitoring as the principal means of preventing haemodynamic compromise.[1] In our patient, intraoperative CVP monitoring and serial TEE assessments of right ventricular volume together guided fluid administration throughout the procedure.

3.2 Choice of Induction Agent

The selection of etomidate for anaesthetic induction in this case was based on its well-documented haemodynamic neutrality. Unlike propofol, etomidate does not significantly reduce SVR, cardiac preload, or myocardial contractility, making it the preferred agent in patients with reduced cardiovascular reserve.[10, 11] A randomised controlled trial by Hannam et al. (2019) demonstrated that propofol caused a significantly greater reduction in mean arterial pressure than etomidate during cardiac surgical induction, confirming the haemodynamic superiority of etomidate in this context.[11]

It is important to acknowledge that etomidate suppresses adrenocortical function via inhibition of 11β-hydroxylase, even after a single bolus dose.[12] This may blunt the stress response for up to 24–48 hours postoperatively. However, a randomised trial by Morel et al. demonstrated that this did not translate into increased vasopressor requirements after cardiac surgery.[12] In patients with pre-existing renal impairment- as in the present case- the risks of haemodynamic instability at induction outweigh the theoretical concern of transient adrenocortical suppression, and etomidate remains the preferred induction agent.

3.3 Role of Intraoperative Transoesophageal Echocardiography

Intraoperative TEE is now considered an indispensable tool in the management of intracardiac tumours undergoing surgical resection. Its role extends beyond diagnosis to real-time guidance of venous cannulation, monitoring of right ventricular function during CPB, and confirmation of complete tumour excision.[4, 5] These functions are especially

critical when the tumour extends into the IVC, where the risk of cannula-induced tumour fragmentation is substantial.

Darwazah et al. emphasised that IVC cannulation in the presence of a tumour at the IVC–right atrial junction presents a unique surgical challenge, and that alternative strategies- including femoral venous cannulation or temporary removal of the IVC cannula under hypothermia- may be required.[8] Yashiro et al. (2024) employed venous cannulation via the main pulmonary artery combined with moderate hypothermic circulatory arrest to achieve a tumour-free operative field in a similar case.[4] In the present case, bicaval cannulation was safely achieved under continuous real-time TEE guidance, precluding the need for hypothermic arrest.

3.4 Risk of Tumour Embolisation

Cardiac myxomas are gelatinous, friable lesions prone to fragmentation. In right-sided tumours, embolisation carries the risk of pulmonary embolism, which may be acutely life-threatening.[2, 3] The risk is greatest during periods of haemodynamic flux- at induction, laryngoscopy, patient positioning, and initiation of CPB. In our patient, measures to minimise embolisation risk included slow haemodynamically gentle induction, avoidance of external chest manipulation, early TEE probe insertion, and meticulous TEE-guided cannulation. The case reported by Mittal et al.- a right atrial myxoma with IVC extension complicated by pulmonary embolism and right-to-left shunting via a patent foramen ovale- underscores the catastrophic potential of tumour embolisation when perioperative precautions are not observed.[5]

3.5 Renal Considerations

The preoperative serum creatinine of 2.70 mg/dL in this 26-year-old patient warrants specific commentary. Renal impairment in the context of right atrial myxoma may reflect chronic elevations in systemic venous pressure causing renal venous congestion and reduced effective renal perfusion- analogous to cardiorenal syndrome type 2. CPB independently confers a significant risk of acute kidney injury, particularly in patients with pre-existing renal dysfunction, due to non-pulsatile flow, haemodilution, and microembolic injury.[6] In this patient, adequate CPB perfusion pressure was maintained, nephrotoxic agents were avoided, and renal function was monitored postoperatively without deterioration- consistent with a successful renal protection strategy.

4. Conclusion

This case illustrates the multidimensional perioperative challenges posed by right atrial myxomas extending into the inferior vena cava. Key anaesthetic principles include: maintenance of adequate intravascular volume and right ventricular preload; avoidance of agents that reduce SVR or impair venous return; minimisation of PEEP to preserve right heart filling; and vigilance for tumour embolisation during all phases of induction and cannulation. Etomidate, combined with a synthetic opioid, remains the induction regimen of choice in this haemodynamically vulnerable population. Intraoperative TEE is indispensable for real-time tumour characterisation, guidance of safe bicaval cannulation, and confirmation of complete excision. Concomitant renal impairment necessitates additional perioperative vigilance. A coordinated multidisciplinary approach, structured preoperative planning, and meticulous intraoperative execution are the cornerstones of successful management in this rare but challenging clinical entity.

Declarations

Ethics Approval and Consent to Participate: Written informed consent was obtained from the patient for surgical intervention, publication of this case report, and reproduction of accompanying clinical images. Patient anonymity has been maintained throughout.

Competing Interests: The authors declare no competing interests.

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