



Anaesthetic Management of Renal Cell Carcinoma with Cavo-Atrial Tumour Thrombus: A Case Report

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Abstract: Removal of Inferior Vena Cava and right atrial tumour thrombi during radical or cytoreductive nephrectomy for renal cell carcinoma implicates an aggressive surgical technique with high perioperative morbidity and mortality. Our case report is one such interesting presentation of renal cell carcinoma with involvement of the right atrium. Radical nephrectomy was done with right atrial thrombectomy under cardiopulmonary bypass. Although reports of renal tumour extending to the inferior vena cava or one of the heart chambers are plenty in the literature, what is truly lacking is the information on challenges in anaesthetic management.

In this report we have tried to enumerate the anaesthetic implications of renal cell carcinoma with cavo-atrial thrombus and its management during surgical resection.

Keywords: Renal cell carcinoma, cavo-atrial thrombus, Tumour embolization, Cardiopulmonary Bypass, Transoesophageal Echocardiography.

Introduction: Renal cell carcinoma (RCC) accounts for approximately 3% of all adult malignancies and over 90% of primary renal tumours.¹ Every year, approximately 12, 000 people die from RCC in the United States.² The tumour is notorious for the systemic manifestations that may have adverse effects on the perioperative management. To make things worse RCC has the tendency to infiltrate into the venous system (renal vein and then into the inferior vena cava). Major causes of perioperative mortality are pulmonary embolism, myocardial infarction and complications of cardiopulmonary bypass (CPB). However, with better perioperative management and standardization of the surgical and vascular bypass techniques, the mortality rate has decreased considerably.

Case Report: A 38 yrs old female weighing 68 kg presented with gross haematuria and left groin pain. Her haemoglobin was 9 gm% and kidney function tests were within normal limits. Computed tomography scan showed a mass in the left kidney extending to the renal vein and inferior vena cava (IVC). Echocardiography showed concentric left ventricular hypertrophy and a mass in right atrium (RA) 3.04 x 2.70 cm in size in continuity with the IVC mass. Pre-anaesthetic assessment showed an otherwise healthy patient, with arterial pressure (BP) of 120/90 mmHg, and heart rate of 70 beats per minute. A decision of left open radical nephrectomy with IVC and right atrial thrombus excision under CPB was made. In the operation theatre, patient was premedicated with Midazolam 1 mg and Fentanyl 100 µg. Following right radial artery cannulation, patient was induced with Fentanyl 400 µg, Midazolam 2 mg, and oxygen-air-Isoflurane mixture. Vecuronium 6 mg was used for tracheal intubation. Central venous catheter was inserted via right Internal Jugular vein taking particular care of not introducing the guide wire too far in and securing the catheter at 12 cm mark. A Transoesophageal Echocardiography (TEE) probe was then inserted and presence of tumour thrombus in the RA (Fig 1) and position of catheter tip was confirmed by TEE.

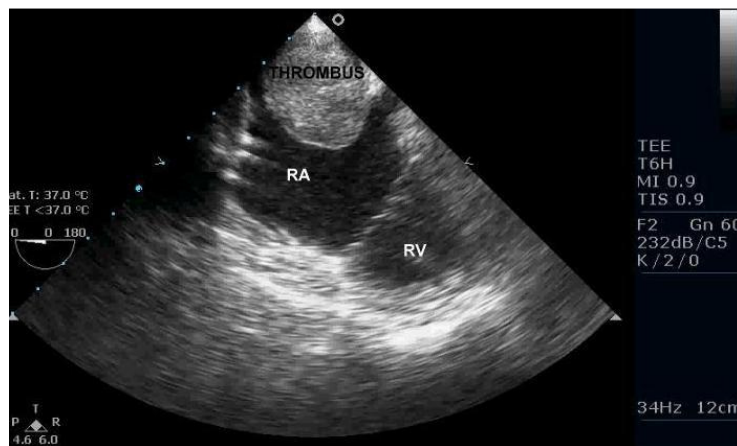


Fig 1: TEE visualisation of the intra atrial tumour thrombus

Anaesthesia was maintained with air, oxygen, isoflurane and supplemental doses of Vecuronium and Fentanyl. Monitoring to detect potential emboli was continued throughout the operation by TEE. Laparotomy was performed, and the left kidney, left renal vein and IVC were mobilized. Following Sternotomy and heparinisation (3 mg/kg); aortic, superior vena cava and infrarenal IVC cannulation were performed. Patient was put on full CPB and cooled to 20⁰ C. Methyl Prednisolone 1 gm and Thiopentone sodium 500 mg were added to the pump. Cardioplegia was started with an aortic root cannula. Radical nephrectomy was done. SVC and infra renal IVC were snared and atrial thrombus was removed via right atriotomy. An IVC incision was made and the IVC thrombus could be removed in piecemeal. The IVC was then flushed for removing any residual debris and repaired. The RA was closed. The patient was weaned off CPB in sinus rhythm and stable haemodynamics and Protamine was injected. TEE showed no residual atrial mass. Post surgery, the patient was transferred to the ICU. She was extubated after 5 hours and thereafter her postoperative course was uneventful.

Discussion: Intravascular tumour growth along the renal vein into the IVC and to the RA (Fig. 2) occurs in up to 15% and 1% of RCC patients respectively.³

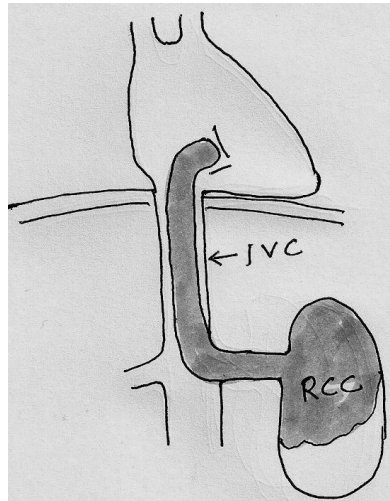


Fig 2: Schematic representation of pattern of RCC tumour thrombus extension.

The RCC caval thrombi can be classified as:⁴ (Fig. 3)

Group I: Venous thrombus in the renal vein not reaching IVC.

Group II: Infra-hepatic IVC thrombus.

Group III: Thrombi in retrohepatic or suprahepatic IVC not reaching the RA.

Group IV: RA thrombi.

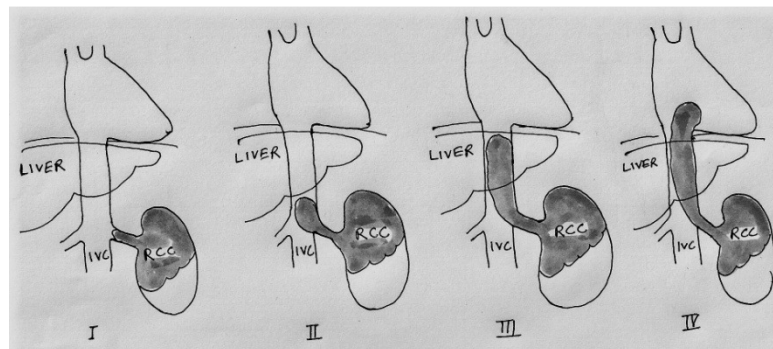


Fig 3: Levels of IVC tumour thrombi

RCC with cavo-atrial involvement by tumour thrombi may present unique challenges in anaesthetic management. The most notable among these are: anaemia secondary to decreased Erythropoietin, deranged kidney function with or without renal arterio-venous malformation, pathological fractures from bony metastasis, hypertension from secretion of Renin by the tumour cells, hypercalcemia, and paraneoplastic disease.⁵ An important paraneoplastic liver disorder (Stauffer syndrome) is associated with deranged liver function in patients with RCC.⁶ RCC is reported to develop in 24-45% of Von Hippel Lindau (VHL) Disease patients⁷, a rare genetic disorder characterized by visceral cysts, benign masses, and the potential for malignant transformation in



multiple organ systems. Polymyositis which can sometimes be associated with RCC may lead to atrioventricular conduction defects, tachyarrhythmias, dilated cardiomyopathy, pulmonary dysfunction due to weakness of thoracic muscles and interstitial lung disease.⁸ Some unusual presentations of RCC include pancreatitis, life-threatening polyneuropathy, gastrointestinal bleeding, hypercalcemia-induced coma and polymyalgia rheumatica. In addition, the pharmacodynamics of preoperative chemotherapeutic drugs if used should be taken into consideration.

CVP may not reflect the volume status of the patient because of impaired venous return. It also predisposes the patient to hypotension during induction of anaesthesia and positioning. Hence, the patient needs to be adequately hydrated beforehand. In patients with poor collateral development, IVC cross clamping can lead to central hypovolemic shock. Similarly, portal clamping leads to an accumulation of toxic metabolites in the gut and intestinal oedema necessitating portal decompression via inferior mesenteric vein cannulation. In case of a chronically obstructed IVC by the tumour thrombus, caval resection produces minimal postoperative morbidity because of the well developed collaterals.⁹ Otherwise graft reconstruction of IVC becomes a necessity.

In the presence of atrial thrombus, guide wires and central venous catheters should not be advanced too far and ideally should be placed under TEE guidance.¹⁰ Right heart catheterisation should be avoided in view of the danger of a part of tumour getting dislodged and embolised. The use of pulmonary artery catheter for hemodynamic monitoring can be withheld in favour of other safer modes of monitoring e.g. TEE, oesophageal doppler probes. Moreover, IVC obstruction leads to dilation of epidural veins and caution should be exercised if epidural catheterisation is attempted. Management of hypothermia, haemodilution, alpha-stat pH management, and normoglycemia are other important aspects of perioperative care.

Statistically, less than 0.4% of all renal cell carcinomas and less than 5% of those involving the IVC embolize during surgery.¹¹ Perioperative mortality rates have been reported between 2.7%–13% for IVC extension of RCC.¹² There was a high risk of pulmonary embolism leading to sudden death during surgical manipulation.¹³ Use of CPB enables careful and controlled dissection in essentially bloodless surgical fields and reduction in the risk of embolization. Techniques utilised ranges from CPB without circulatory arrest to deep hypothermia and circulatory arrest with or without cardioplegia.¹⁴ The veno-venous bypass (VVB) technique (shunt created between the femoral or saphenous vein to the axillary vein) has also been employed as an alternative when thrombus remains below the level of suprahepatic veins.¹⁵ The use of Heparin is another grey area. Anticoagulation in patients with accessory venous collaterals from IVC obstruction and extensive retroperitoneal dissection may be problematic especially if deep hypothermic cardiac arrest (DHCA) is used. There is a potential risk of bleeding and coagulopathy with CPB with or without DHCA, particularly when performed in conjunction with radical nephrectomy and lymph node dissection.

Conclusion: A good understanding of the anaesthetic implications brought about by the RCC is of paramount importance in its successful management. A thorough preoperative evaluation and good communication with the surgical team and blood bank is essential for smooth performance of the procedure. Major blood loss should be anticipated especially during nonbypass surgery and when



collaterals are well developed. Availability of rapid transfusion devices, cell saver, use of antifibrinolytic agents like epsilon aminocaproic acid and patient warming systems should be checked beforehand. The use of cell saver technique is controversial in malignancy patients because of the potential risk of systemic dissemination of tumour cells. So, we opt not to use it. Routine intraoperative TEE use, presence of the cardiac surgery team and CPB facility should be confirmed before trying to excise this peculiar tumour with cavo-atrial thrombus.

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