Renal Cell Carcinoma encasing and invading inferior vena cavae: Anaesthetic concerns

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Citation

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Abstract

We report a unique case of renal cell carcinoma encasing the inferior vena cava (IVC). There was no distant metastasis, lymph node involvement, gross renal vein or IVC tumor invasion, which was misleading! The anesthetic management was challenging and the outcome fatal. The only forewarning signs of impending catastrophe were the presence of a small tumor thrombus in IVC detected on ultrasound abdomen and the CT scan report of IVC being encased by the renal cell carcinoma. Our failure to recognize the implications of IVC encasement resulted in a disastrous outcome. The surgical intervention, anesthetic management and an unexpected fatal outcome have been discussed.

IMPLICATION STATEMENT

Surgical management of renal cell carcinoma encasing and invading the inferior vena cava can result in massive intraoperative hemorrhage and death. Awareness of this complication and adequate anesthetic preparation for it have been discussed.

INTRODUCTION

Renal cell carcinoma (RCC) accounts for approximately 3% of adult malignancies and 90-95% of neoplasms arising from the kidney[1]. RCC is more common in men than in women (ratio 2:1), and the median age at diagnosis is approximately 60 years[2]. It is characterized by a lack of early warning signs, diverse clinical manifestations, resistance to radiation and chemotherapy[3]. The tumor extends into the vena cava in about 5% of patients, and aggressive surgical therapy is warranted if there is no evidence of metastasis[4]. Anesthetic considerations include massive blood loss in view of close proximity to major blood vessels, and optimisation of blood flow to the remaining kidney. Cardiopulmonary bypass and hypothermic circulatory arrest are the preferred modalities for intraoperative management of extensive caval tumor invasion.

CASE

A 43 year old, 90 kg male and ASA grade III presented to us with complaint of pain in right flank for one week, arising suspicion of a renal mass. An ultrasound abdomen revealed a right renal mass, possibly a RCC and a small thrombus 1.4x1.2 mm in IVC below the level of right renal hilum. Rest of IVC was compressed and no other thrombus was seen. IVC – right atrium junction was normal. A contrast enhanced CT scan abdomen and pelvis showed a large mass measuring 11.4x8.9x12.4 cm, arising from the upper pole of right kidney and reaching till inferior surface of right lobe of liver. Retroperitoneal or liver metastasis was not seen. His systemic examination revealed no abnormality. Haematological, biochemical investigations, coagulation profile, chest radiograph and electrocardiography were normal. An echocardiography confirmed the absence of any thrombi in the heart and a normal left ventricular function with ejection fraction 55-60%. His airway examination showed Mallampatti grade II with adequate neck movements. An exploratory laprotomy and right radical nephrectomy was planned.

During preanesthetic visit, a written informed consent was obtained and adequate blood and blood products were arranged. He was premedicated with tablet diazepam 10mg and tablet ranitidine 150 mg a night before and on the morning of surgery.

On the day of surgery, patient was shifted in the operating room. Electrocardiogram, pulse oximetry, non invasive blood pressure (NIBP) were attached. General anesthesia along with epidural analgesia (postoperative pain relief) was planned. Before anesthetic induction, intravenous lines (with two 16G wide bore cannulae after local infiltration) were secured. Also, under full aseptic precautions, an epidural

catheter was inserted in lumbar 3-4 interspace. Anesthesia was induced with fentanyl 2 mcg/kg, propofol 2mg/kg and tracheal intubation with 8.5mm ID ETT was facilitated with vecuronium 0.1mg/kg. In view of major blood loss and fluid shift, as the tumor was encasing and invading the IVC and adherent to surrounding structures, we had planned for invasive monitoring (central venous pressure and intra-arterial blood pressure). The left internal jugular vein and left radial artery were cannulated under strict asepsis. Patient was positioned supine and surgery started.

On surgical exploration, a huge right renal cell carcinoma encasing, compressing and obstructing the inferior vena cava was found. The right renal vein was hugely dilated. While attempting to resect the tumor mass from IVC, multiple rents were created in the IVC causing blood loss of about two litres over a period of 15 minutes. As a result, his blood pressure dropped to 80/40 mm Hg and CVP to 5 cm H2O but saturation remained above 98%. We started replacing the blood loss aggressively with warm crystalloids and colloids and maintained CVP near 10 cm H_2O, but the patient kept on bleeding. One day prior to surgery we were informed that four units of packed RBC, fresh frozen plasma and plasma each had been arranged for this patient. But, when requisition was sent, we were told by blood bank staff that only two units of packed RBC were available. About 20 minutes later, we got two units of PRBC and were administered. By then the blood loss was about 8 litres and blood pressure fell down to 40 mmHg and CVP to 2-3 cm H2O. Infusions of dopamine@ 5-10 mcg/kg/min, adrenaline @ 2-5 mcg/min and noradrenaline@ 2-5 mcg/min i/v were added. Since no more PRBCs of the same blood group of the patient were available, we transfused 11 units of O negative blood over a period of 1-2 hours alongwith 5 units of plasma and 4 units of FFPs, 1 litre of hetastarch and 7 litres of crystalloids. The surgery was completed, bleeders ligated and the abdomen packed with swabs to control bleeding. The total blood loss in the surgery was around 9 litres! Despite the massive blood transfusion, continuous inotropes and aggressive efforts, the heart rate at end of surgery was 125 bpm, invasive blood pressure at 46/23 mm Hg and NIBP, SpO2, CVP were unrecordable. It was difficult to shift the patient out of operating theatre with such unstable hemodynamics. Resuscitation with fluids, bolus doses of dopamine, adrenaline, noradrenaline and mephenteramine was continued for 50 minutes postoperatively, but the blood pressure did not improve. Meanwhile, we felt that the abdomen was distended and discussed it with the operating surgeon but he said that no intervention can be done at that moment, following which the decision was taken to shift him to surgical high dependency unit (HDU).

In the HDU, patient was promptly connected to ventilatory support and inotropes were ensured to be running (dopamine@ 10mcg/kg/min, adrenaline/noradrenaline @ 10 mcg/min. Monitors were attached. However, pulse, blood pressure and SpO2 were not recordable. Only electrocardiogram trace was recordable which showed a heart rate of 120 beats per minute. 5 minutes later, patient developed sudden bradycardia with a heart rate 48bpm. Injection atropine 0.6 mg i/v was given but there was no response and the patient had a cardiac arrest. Cardiopulmonary resuscitation continued and patient was resuscitated as per standard ACLS resuscitation guidelines but he could not recover. He was finally declared dead after one hour and fifteen minutes of resuscitation.

DISCUSSION

Here we describe a unique case report of a 43 year old man with a massive right sided renal cell carcinoma compressing and encasing the inferior vena cava. Our failure to recognise the implications of IVC encasement resulted in a disastrous outcome. In a case of RCC with lymph node involvement, invasion of renal vein or IVC, spread to right atrium or history of pulmonary embolism, a thorough discussion of the clinical, radiological findings, extent of tumor involvement, operative procedure planned and the complications, should be done preoperatively for effective management. Our patient had a T3aNoMo stage.[3] Radical surgery for renal cell carcinoma extending to the vena cava is justified when the tumor thrombus does not extend beyond the level of the diaphragm in the cranial direction[5,6].

The successful management of radical nephrectomy with vena caval involvement using deep hypothermic circulatory arrest and cardiopulmonary bypass techniques have been described[5,6,7,8,9]. Cardiopulmonary bypass is required in these cases to prevent tumor embolisation; it may also be necessary in some with extension into the upper portion of hepatic vena cava or when venous return is significantly compromised[10]. Perhaps, an atrial caval shunt which bypasses the caval route could be employed as in surgical management of IVC injuries[11]. All these techniques can play a vital role in avoiding the massive blood loss and associated high perioperative mortality. As seen in our case, the attempt to free the tumor mass encasing the IVC can produce big rents in the vessel and result in massive bleeding. This if uncontrolled, may have fatal consequences. Hence, we

propose that the above mentioned techniques be the modality of choice in all cases of RCC with encasement of IVC too. The services of a specialised vascular surgeon along with a general surgeon in resecting the IVC from tumor mass should be imperative because of the risk of massive and uncontrolled bleeding in inexperienced hands. Good back up facilities in the form of adequate blood and blood products, rapid transfusion devices, cell saver and patient warming systems should be available. In our case, we fell short of blood products at the time of need. Normally, a blood loss of 800-1200 ml is expected in a radical nephrectomy, but in our case we had a loss of 9-10 litres of blood. We initially had only two units available!

Invasive monitoring in the form of arterial blood pressure, CVP and urine output should be established. Right heart catheterisation may be avoided in view of the danger of a part of tumor getting dislodged and embolised. Left internal/external jugular cannulation with catheter tip not going beyond the superior vena cava should be preferred[10]. CVP in these cases may not be accurately reflected because of impaired venous return. This impaired venous return also predisposes the patient to hypotension during induction of anesthesia. Hence, the patient needs to be adequately hydrated to optimise the blood flow to the remaining kidney and also prevent hypotension from IVC compression during positioning. Venous obstruction leads to dilation of epidural veins and hence caution should be exercised if epidural catheterisation is attempted. Pneumothorax may occur if chest is inadvertently entered during surgery which can be confirmed postoperatively by a chest radiograph.

Renal cell carcinoma is a unique and challenging tumor because of the frequent occurrence of paraneoplastic syndromes, including hypercalcemia, erythrocytosis, and nonmetastatic hepatic dysfunction (ie, Stauffer syndrome). Renal cell carcinoma develops in nearly 40% of patients with VHL disease[3,12] and is a major cause of death among these patients. At least 4 hereditary syndromes associated with renal cell carcinoma are recognized. Polyneuromyopathy, amyloidosis, anemia, fever, cachexia, weight loss, dermatomyositis, increased erythrocyte sedimentation rate, and hypertension also are associated with renal cell carcinoma. Again, the emphasis is on appropriate preoperative preparation and skillful intraoperative anesthetic management.

In conclusion, we reiterate that renal cell carcinoma encasing and invading the inferior vena cava may be associated with massive blood loss intraoperatively and that cardiopulmonary bypass, deep hypothermic circulatory arrest, vena caval shunts should be employed. A preoperative preparation, discussion of surgical plan with the surgeon and backup of good transfusion support and inotropes is the key to a successful anesthetic management.

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