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Case report

Anesthesia Considerations for a Better Kidney Function in Patient with Radical Nephrectomy and Inferior Vena Cava Thrombectomy: A Case Report

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Abstract

Renal cell carcinoma (RCC) is the most common type of kidney cancer, originating from the epithelial cells of the renal tubules. Anesthesia management during surgical procedures to excise malignant tumors that have metastasized into the renal vein or inferior vena cava is exceedingly challenging due to the elevated risk of thrombus emboli displacement, potentially resulting in pulmonary obstructions or cardiac or neurological complications. The stable vital parameter should be also preventing development of acute kidney injury and in addition, the possible deterioration of kidney function on top of the already halved one with the nephrectomy.
We present a case of a 69-year-old male patient admitted to the University Clinic of Urology for scheduled radical nephrectomy due to RCC. An open nephrectomy was conducted under general anesthesia combined with epidural analgesia. During the operation, the patient had a significant hypotension, tachycardia, and a substantial reduction in end-tidal CO_2 levels. Bilateral mydriasis was also observed. Timely administration of anticoagulants and vasoactive drugs resulted in clinical stabilization. Thereafter, we experienced an additional episode of hemorrhage from the a. lienalis, requiring splenectomy as an appropriate therapeutic option. At the end of the surgery, the patient was extubated from mechanical ventilation within the subsequent 30 minutes. The postoperative period was steady, exhibiting optimal hemodynamic and clinical conditions. The patient was discharged seven days post-operation in a good health, with nearly the same kidney function as before the nephrectomy, and without neurological complications. This case highlights the importance of thorough anesthetic preparation, precise hemodynamic monitoring, and synchronized intraoperative treatment following radical nephrectomy with tumor thrombus extending into the inferior vena cava.

Keywords: Anesthesia management, radical nephrectomy, renal cell carcinoma, vena cava thrombus, kidney function

Introduction

Renal cell carcinoma (RCC) is the most prevalent kind of renal cancer, arising from the epithelial cells that are part of the kidney tubules [1]. Carcinoma cells may penetrate in the renal veins, leading to the development of a renal vein tumor thrombus, which may result in its infiltration into the inferior vena cava (IVC) and perhaps extend to the right atrium [2]. In around 4% to 19% of patients with RCCs, tumors infiltrate the IVC [2,3]. It is approximately twice as prevalent in men than in women and typically manifests in the seventh decade of adulthood. Cigarettes are the primary risk factor. Additional significant risk factors are overweight, high blood pressure, and acquired polycystic kidney disease [4]. Surgical resection remains the mainstay of treatment for localized as well as locally advanced disease [1]. Surgery may induce postoperative problems such as surgical access issues, hemorrhage, extensive blood transfusion, coagulopathy, lung embolism, and significant fluid changes [5]. Anesthetic handling for the surgical excision of malignant tumors complicated by thrombus metastasizing into the renal vein or inferior vena cava is highly challenging due to the elevated risk of tumor thrombus movement, potentially leading to embolism in the lungs or cardiovascular failure [5]. In the same line, one should consider also the residual kidney function after the nephrectomy. Certainly, the stable vital parameter should be also preventing deve-

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lopment of acute kidney injury and in addition, the possible deterioration of kidney function on top of the already halved one with the nephrectomy.
This case report analyzes our perioperative management and emphasizes the aspects that led to a satisfactory outcome.

Case presentation

A 69-year-old male patient was scheduled for a radical nephrectomy due to radiologically confirmed RCC with extension into the inferior vena cava. His medical history was significant for arterial hypertension and persistent left bundle branch block (LBBB). Long-term pharmacological treatment comprised a beta-adrenergic receptor antagonist, an angiotensin-converting enzyme (ACE) inhibitor, a lipid-lowering agent (statin), and an antiplatelet medication. Upon admission, the patient was hemodynamically stable, had preserved blood pressure and a mildly elevated heart rate, with an age related, slightly reduced kidney function, afebrile. Laboratory analyses revealed leukocytosis, with a white blood cell count modestly above the upper normal range. Abdominal ultrasound showed steatotically changed liver parenchyma. The right kidney was without obstructive changes, and the left kidney was with solid heteroechogeny, and a change that covers almost the entire cortex, requiring further investigations. The computed tomography has shown thickened left basal pleura. Right kidney was with postcontrast imbibition and elimination. A projection of the left splenic-renal region showed a solid hetero-dense change with dimensions of 120x110 mm, with zones of disintegration and calcifications, which most likely originated from the adrenal gland. The left kidney appeared smaller in size, but a clear differentiation could not be made whether the change described has been in close contact with it. Enlarged lymph nodes up to 1 cm have been seen para-aortically. Although radiological interpretation did not reveal any thrombotic material within the inferior vena cava, the attending surgeon retained a heightened suspicion of IVC involvement, which was later corroborated during the operation. The patient was given American Society of Anesthesiologists (ASA) physical status III. Based on the clinical and radiological findings, the patient underwent an open radical nephrectomy.
We evaluated the kidney function through laboratory analyses and glomerular filtration rate preoperatively. Our patient had blood urea nitrogen (BUN) and createnine in referent range preoperative with 3.6 mmol/L and 75 umol/L, respectively, with an eGFR of 89 mL/min/1.73 m^2.
Upon the patient's arrival in the operating room, one 18 G and one 16 G peripheral intravenous catheter were placed to secure venous access prior to anesthetic induction. Monitoring encompassed non-invasive blood pressure (NIBP), electrocardiography (ECG), pulse oximetry, and body temperature. His essential parameters were BP 200/100 mmHg, SpO_2 92%, and the ECG indicated sinus rhythm with a heart rate of 95 beats per minute. An epidural catheter was subsequently inserted at the Th10-Th11 level of the thoracic spine. A test dose of bupivacaine 0.5% was administered. Subsequently, the induction commenced. For induction of anesthesia, the subsequent drugs were delivered consecutively: 1 mg of midazolam, 100 mcg of fentanyl, 40 mg of lidocaine, 100 mg of propofol, and 50 mg of rocuronium bromide. Tracheal intubation was successfully performed utilizing an 8.0 endotracheal tube and a conventional laryngoscope. The tidal volume was sustained at 6-8 ml/kg, the respiratory rate was 12 breaths per minute, and PEEP was set at 5 in the pressure-controlled volume guarantee ventilation mode. $EtCO_2$ was sustained within the range of 35 to 45 mmHg with respiratory rate adjustment. To sustain anesthesia, 1%-2% sevoflurane was continuously administered via inhalation at a MAC of 0.6-1, propofol was infused continuously at a rate of 30-40 ml/h, and fentanyl was administered intravenously in an intermittent manner. Bupivacaine 0.25% was administered intermittently via the epidural catheter in a dose of 2mg hourly. An arterial blood line was inserted into the right brachial artery post-induction to measure invasive blood pressure (IBP). Central venous catheterization in the internal jugular vein was executed to enable volume management, delivery of vasoactive medications, and assessment of central venous pressure (CVP). During the initial two hours, the patient exhibited hemodynamic stability, with blood pressure ranging from 110/60 to 120/70 mmHg and a heart rate of 60 to 80 beats per minute and CVP was maintained at 10 mmHg.
During the operation, roughly two hours after its commencement, the patient suffered from a sudden and significant haemorrhage, resulting in a rapid decline in blood pressure to 60/40 mmHg, and a notable decrease in end-tidal CO_2 ($EtCO_2$) to 12 mmHg, an elevation in heart rate to 140 beats per minute, and an increase in CVP to 18 mmHg with a consequent bilateral mydriasis. The initial therapy comprised with an intravenous delivery of 5000 IU of heparin, bolus vasopressors (phenylephrine 500 mcg and adrenaline 100 mcg), succeeded by a continuous adrenaline infusion at a rate of 0.05 mcg/kg/hour. The mydriasis subsided within 30 seconds. Blood pressure progressively enhances, attaining 80/50 mmHg within 10 minutes and subsequently 90/60 mmHg. The EtCO2 reading incrementally rose to 25, subsequently to 28, and ultimately reached 31 mmHg. Hemorrhage was managed using mechanical means. One unit of packed red blood cells and one unit of fresh frozen plasma were delivered. Thirty minutes post-initial stabilization, rebleeding ensued, with the splenic artery identified as the source. An urgent splenectomy was performed.

Arterial blood gas was obtained, and measurements revealed pH 7.31, pCO_2 35.7 mmHg, pO_2 131 mmHg, hemoglobin 9.5 g/dL, oxygen saturation 99%, and lactate 2.2 mmol/L. During the procedure, fluid resuscitation was sustained using crystalloids and colloids at a rate of 15 ml/kg/hour. Because of the hemodynamic instability and lack of urine production at the conclusion of the procedure, diuresis was pharmacologically induced with an intravenous furosemide at a dosage of 10 mg.
At the end of surgery, neuromuscular blockage was antagonized using neostigmine and atropine. Twenty minutes later, the patient was effectively weaned from mechanical ventilation and extubated. He was relocated to the post-anesthesia care unit, where he was monitored for one hour. Given that his vital signs and neurological condition were stable, he was transported to the urology department.
The postoperative laboratory findings from the first to the seventh postoperative day showed preserved renal function with Hgb from 85 to 100 g/L, BUN from 2.1 to 3 mmol/L and creatinine 71 -75 umol/L. Interestingly, his eGFR was around the same as previously 89 mL/min/1.73 m^2. Postoperative trends are presented in figure 1. The postoperative recovery was unremarkable, and the patient was discharged on the seventh day post-surgery.

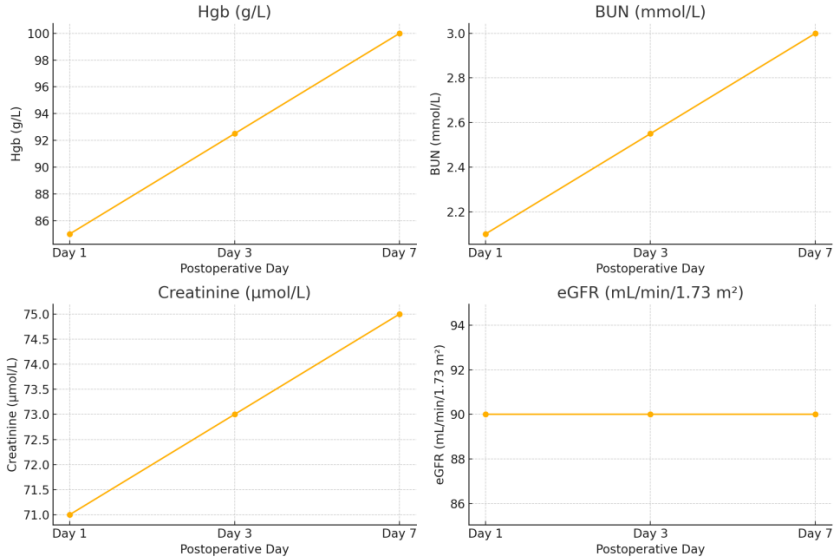

Fig. 1. Trend of Postoperative Hgb, BUN, Creatinine, and eGFR Levels Indicating Preserved Renal Function

Discussion

Regardless of advancements in radiation, chemotherapy, and immunotherapy, surgical excision is still the mainstay approach for RCC, including tumor thrombus [6]. Performing a radical nephrectomy involving the incision of the inferior vena cava to excise invasive tumors or thrombus has a 1-month death rate of 1-10% and a serious complications risk of 18-47% [7]. The perioperative assessment and anesthetic management during radical nephrectomy with inferior vena cava thrombectomy can provide significant challenges. Consequently, it is essential for the team of specialists to recognize the hemodynamic alterations that transpire during caval manipulation. The primary elements of intraoperative monitoring encompass fluid management, the preservation of steady hemodynamic parameters, and the prompt management of potential embolism [8]. We did not use transthoracic echocardiography (TTE), but we vigilantly followed invasive blood pressure, clinical signs, and $EtCO_2$.
$EtCO_2$ encompasses all facets of respiration and circulation, consequently offering a comprehensive evaluation of pulmonary and cardiovascular dynamics. Under stable respiratory conditions, $EtCO_2$ serves as a metric for assessing the cardiac output and pulmonary blood flow.
In general anesthesia patients, it could indicate perioperative poor output of the heart, organ inadequate perfusion i.e. kidneys, and consequent postoperative organ failure [9]. Perioperative pulmonary embolism (PE) affects nearly six percent of patients with IVC tumor thrombus, with death rates ranging from around 70%. PE often occurs during renal and inferior vena cava dissection and clamps [10]. In our case, considering all the details, PE was one of the possible causes of hemodynamic and respiratory abnormalities. Next, in both the Trendelenburg and kidney positions, the surgical field is elevated above the heart, establishing a negative pressure gradient that exists between the area of surgery and the heart. Air embolism may arise if the venous plexus is compromised when the patient is in a head-down posture during radical prostatectomy or cystoprostatectomy, or if the vena cava is breached during the radical nephrectomy. A large air embolism may result in a reduction of end-tidal carbon dioxide or an elevation of end-tidal nitrogen. Physical manifes-

tations indicative of air embolism encompass abrupt hypotension, hypoxemia, arrhythmia, and the detection of a mill-wheel murmur [11]. The decrease in $EtCO_2$ could be due to the loss of lung perfusion, and dilated pupils were most probably secondary to the cerebral hypoxia.

Furthermore, left radical nephrectomy was noted as the second most prevalent reason for inadvertent spleen removal in surgical abdominal procedures. Intraoperative documentation commonly indicates that nonspecific "spleno-renal adhesions" result in avulsion of the splenic capsule, predominantly at the lower pole of the spleen. Another frequently cited cause of splenic injury is the trauma from a hand retractor, and occasionally, unintentional laceration of the splenic capsule [12]. The reduction in the renal mass usually results in a diminished renal function. The excision of fifty percent of the renal mass with a radical nephrectomy promptly diminishes renal function to approximately half of its pre-nephrectomy level. Nevertheless, within a brief period, the surviving contralateral kidney starts to compensate for the diminished renal function [13], certainly, in cases without ensued acute kidney injury. In our case despite the intraoperative haemodynamic instability and the hypoperfusion in a short period of time, the patient had a satisfactory outcome with a preserved renal function. Following the patient one year after the operation he is with BUN and creatinine in normal ranges, good protein status, a satisfactory dieresis and eGFR in the normal age adjusted range.

Notwithstanding the intricacies of the intraoperative process, the patient was adeptly handled, achieving effective bleeding control and a successful completion of the treatment, which included splenectomy. We assert that the intraoperative application of transesophageal echocardiography (TEE) would have been beneficial for prompt diagnosis and real-time hemodynamic evaluation. Nonetheless, meticulous analysis of clinical indicators, invasive blood pressure monitoring, and $EtCO_2$ changes facilitated swift decision-making and a positive conclusion.

Conclusion

A nephrectomy including thrombus in the inferior vena cava necessitates meticulous planning and specialized anesthetic management. Effective management of hemorrhage and vascular manipulation during radical nephrectomy necessitates the implementation of accurate hemodynamic monitoring thus preserving also the kidney function. A multidisciplinary team approach is essential with anesthesia and surgery teams coordinated as to provide best patient outcomes.

Conflict of interest statement. None declared.

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