Clinical Investigation

Anaesthesia for laparoscopic kidney transplantation: Influence of Trendelenburg position and CO_2 pneumoperitoneum on cardiovascular, respiratory and renal function

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ABSTRACT

Background: Laparoscopic donor nephrectomy is a routine practice since 1995. Until now, the recipient has always undergone open surgery for transplantation. In our institute, laparoscopic kidney transplantation (LKT) started in 2010. To facilitate this surgery, the patient must be in steep Trendelenburg position for a long duration. Hence, we decided to study the effect of CO2 pnuemoperitoneum and Trendelenburg position in chronic renal failure (CRF) patients undergoing LKT. **Methods:** A total of 20 adult CRF patients having mean age of 31.7±10.36 years and body mass index 19.65±3.41 kg/m^2 without significant coronary artery disease were selected for the procedure. Cardiovascular parameters heart rate (HR), mean arterial pressure (MAP), Central venous pressure (CVP) and respiratory parameters ($ETCO_2$, peak airway pressure) were noted at the time of induction, after induction, 15 min after creation of pnuemoperitoneum, 30 min after Trendelenburg position, 15 min after decompression of pnuemoperitonuem and after extubation. Arterial blood gas analysis was carried out after induction, 15 min after creation of pnuemoperitoneum, 30 min after Trendelenburg position and 15 min after clamp release. Total duration of surgery, anastomosis time, time for the establishment of urine output and total urine output were noted. Serum creatinine on the 1st and 7th post-operative day were recorded. **Results:** Significant increase in HR was observed after creation of CO_2 pneumoperitoneum and just before extubation. Significant increase in the MAP and CVP was noted after creation of pneumoperitoneum and after giving Trendelenburg position. No significant rise in the $ETCO_2$ and $PaCO_2$ was observed. Significant increase in the base deficit was observed after the clamp release, but none of the patients required correction. **Conclusion:** LKT performed in steep Trendelenburg position with CO_2 pneumoperitoneum significantly influenced cardiovascular and respiratory homeostasis; however, measured parameters remained within clinically acceptable range without affecting early function of the transplanted kidney.

Key words: CO_2 pneumoperitoneum, laparoscopic kidney transplantation, Trendelenburg position

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INTRODUCTION

Kidney transplantation is a preferred therapeutic option for patients with end stage renal disease compared to dialysis. The donor kidney is placed in the iliac fossa of the recipient by conventional open route and the technique has not changed since the first transplantation, which was performed in 1950. The removal of donor kidney by laparoscopic approach has become a routine procedure in transplant scenario due to the number of advantages and comparable outcome to open donor nephrectomy on graft function. Inspired by the success of laproscopic donor nephrectomy and feasibility of laparoscopic vascular reconstructive

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surgeries such as repair of abdominal aortic aneurysm and renal artery aneurysms described in the literature, we started performing laparoscopic kidney transplantation (LKT) from 2010 with permission of Internal Review Board of the Institution.

LKT is a complex surgery that requires previous experience in vascular and reconstructive laparoscopic surgery.[1] To facilitate this surgery, the patient must be placed in steep Trendelenburg position for potentially long duration with CO_2 pneumoperitoneum. This can produce adverse changes in the cardiovascular, respiratory and renal system of patients with chronic renal failure (CRF) due to associated co-morbid conditions such as hypertension, anaemia, fluid, electrolyte, and acid base imbalance. There are no reports in the literature on anaesthetic management of LKT; hence, we conducted a study with an aim to evaluate the safety of the procedure in terms of haemodynamic and respiratory parameters and early graft function.

METHODS

After approval by the Hospital Ethics Committee, 20 adult ASA physical status II to III patients scheduled for LKT were included in this prospective observational study. Out of these 20 patients 10 patients had chronic glomerular nephritis, 7 had hypertensive nephropathy and 3 had diabetic nephropathy. Patients with severe cardio-pulmonary dysfunction (patients with poor functional capacity, showing echocardiographic evidence of significant systolic/diastolic dysfunction/wall motion abnormalities, significant dysrhythmias) and morbid obesity were excluded from our study. All patients were thoroughly evaluated including echocardiography and coronary angiography when indicated and their written informed consent was obtained.

All the patients were operated by a single surgeon having extensive experience in laparoscopic donor nephrectomy, urological reconstructive surgery and open kidney transplantation (OKT) surgery. With patients in the horizontal position with arms by the side of the patient, pneumoperitoneum was created by insufflation of carbon dioxide and four transabdominal ports were placed, one for the camera and the remainder for insertion of the surgical instruments. The intra-abdominal pressure (IAP) was monitored continuously and maintained at 12-15 mm Hg. The patients were then positioned in the Trendelenburg position with a 30-40° head-down tilt. After dissection of the external iliac vessels, donor kidney was placed into the peritoneal cavity through a 6-7 cm incision made in the skin crease in the iliac fossa. The wound was closed rapidly and pneumoperitoneum was re-established. Both arterial and venous anastomosis were performed in the end to side fashion and after testing for leak, graft was allowed to perfuse. Pnemoperitoneum pressure was reduced to 8-10 mm Hg for 10 min and ureteroneocystostomy was performed. The kidney was lifted and placed in the iliac fossa over the psoas muscle and previously opened peritoneum was closed in part to keep the kidney in extra-peritoneal position. The abdomen was deflated, ports were removed and wounds were closed after infiltration with bupivacaine 0.5% to a total volume of 20 mL.

After applying standard monitors such as ECG, NIBP, SaO_2, patients were pre-medicated with injection glycopyrrolate 4 μg/kg and fentanyl 5 μg/kg. Anaesthesia was induced with thiopentone sodium 5-7 mg/kg and endotracheal intubation was facilitated with atracurium 0.6 mg/kg. Anaesthesia was maintained with air, O_2, sevoflurane and atracurium repeated as needed for muscle relaxation. The lungs were mechanically ventilated with volume controlled mode (WATO 30 Anaesthesia Delivery Unit) with tidal volume (TV) 8-10 ml/kg, pressure limit of 35 cm H_2O and respiratory rate (RR) to keep $EtCO_2$ <40 mmHg. Nasogastic tube and nasopharyngeal temperature probe were inserted to monitor temperature. AV fistula was covered and all pressure points were adequately padded. Forced air warmers were used to maintain normothermia and warm NS was infused to maintain CVP. All patients received injection Albumin 100 ml (20%), and injection mannitol 0.5 g/kg before release of the clamps. At the completion of surgery, patients were reversed with injection glycopyrrolate 6-8 μg/kg and neostigmine 0.05 mg/kg and were extubated when awake following verbal commands and able to lift head for 5 s.

After induction of general anaesthesia, a radial arterial cannula was inserted for blood sampling and pressure monitoring. Double lumen central venous cannula was introduced percutaneously via the right internal jugular vein to measure CVP and infusion of fluid or vasoactive drugs. Heart rate (HR), SaO_2, mean arterial pressure (MAP), $EtCO_2$, CVP and peak airway pressure (PAP) were monitored continuously and recorded before induction (baseline), after induction, 15 min after creation of pneumoperitoneum, 30 min after Trendelenburg position, 15 min after decompression of pneumoperitoneum and after extubation. Tachycardia was defined as HR >90/min and hypertension as

MAP >110 mmHg, which was controlled with nitroglycerine (NTG). Arterial blood gas (ABG) analysis was carried out after induction, 15 min after creation of pneumoperitoneum, 30 min after Trendelenburg position and 15 min after clamp release. Total duration of surgery, anastomosis time and time for the establishment of urine output were noted. Urine output was measured before transferring the patient to ICU and S. Creatinine on 1st and 7th post-operative day were recorded.>

Statistical analysis

Statistical analysis was performed using the SPSS version 12. Data are expressed as mean ± SD (standard deviation) for continuous variables and number (%) for categorical variables. Continuous variables were compared using two sample dependent *t*-test. $P<0.05$ considered to be statistically significant.

RESULTS

Demographic data and surgical characteristics were as shown in Table 1. Statistically significant increase in the heart rate was observed after creation of pneumoperitoneum and just before extubation. A significant increase in the MAP and CVP was noted after creation of pneumoperitonium and after giving head down position [Figure 1].

Insignificant rise in $ETCO_2$ is noted whereas statistically significant rise was noted in PAP after creation of pneumoperitoneum and after giving Trendelenberg position. Though, it remained <25 cm H_2O throughout the procedure, PAP returned to base line after decompression of pneumoperitonium [Figure 2]. Statistically, insignificant rise in gradient between $PaCO_2$ and $ETCO_2$ was observed throughout the surgery [Figure 3].

Insignificant increase in $PaCO_2$ is noted after pneumoperitonium. However, significant increase in base deficit is noted after clamp release [Table 2]. However, no patients required correction.

Table 1: Demographic data and surgery characteristics

Parameters	Mean±SD (min-max)
Age (years)	31.7±10.35
Gender (M:F)	19:1
BMI (kg/m²)	19.65±3.41
Anastomosis time (min)	47.85±6.30 (39-60)
Time of establishment of U/O after clamp release (min)	7.25±3.04 (1-15)
Total IV fluid (ml)	3981.5±811.4 (2400-5350)
Total U/O on table (ml)	918.5±453.1 (250-1700)
Duration of surgery (min)	224.5±32.4 (160-290)
S. Cr (mg/dL) at 1st/7th day	2.91±0.98/1.63±0.52

BMI – Body mass index; S. Cr – Serum creatinine

DISCUSSION

With the introduction of new surgical technique, anaesthesiologists must determine the need to alter their routine practice to ensure patient safety.

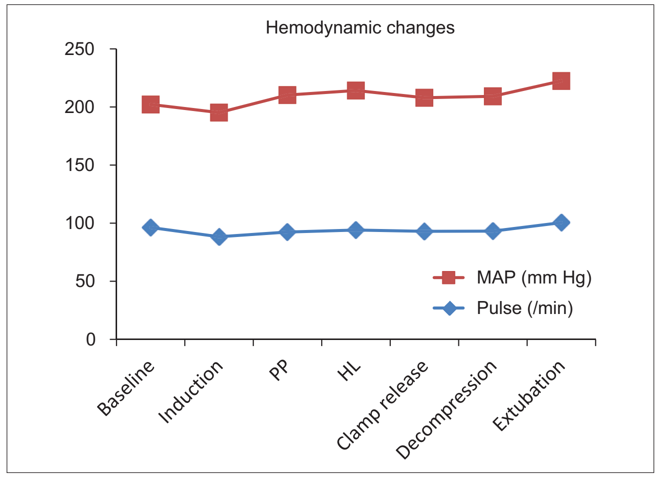

Figure 1: Heart rate and mean arterial pressure changes

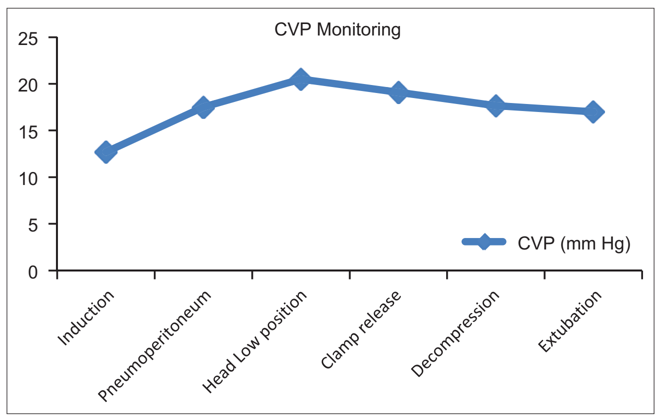

Figure 2: Central venous pressure monitoring

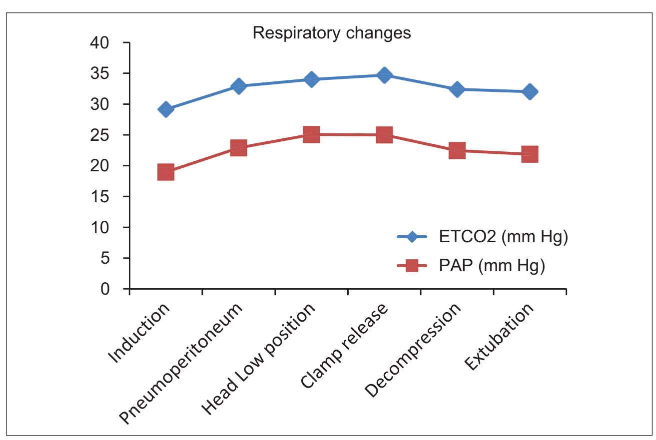

Figure 3: Respiratory parameters

Table 2: Arterial blood gas analysis

Parameters	Induction	Pneumoperitoneum	Clamp release	Extubation
PH	7.45±0.06	7.39±0.08	7.32±0.07	7.31±0.08
PaO_2	218.4±101.8	220.2±71.5	221.4±76.1	228.1±88.6
$PaCo_2$	34.1±5.17	40.7±7.11	41.8±6.38	40.8±6.88
BE	0.70±4.36	−1.19±3.76	−4.85±2.98	−5.63±2.36
HCO_3	24.4±4.22	23.6±3.3	21.02±2.34	20.2±1.71
SaO_2	98.9±1.68	99.2±1.18	99.13±1.11	99.08±1.35

Laparoscopy surgery is associated with significant haemodynamic and respiratory changes even in normal patients. Additionally changes in body position, especially, the head-down or Trendelenburg position can further exacerbate these changes. Apart from these, effect of pneumoperitoneum on kidney allograft function was also a major concern.

In conventional OKT performed in the supine position, apart from standard monitoring for major surgical procedures, we routinely monitor CVP to guide fluid therapy. We do not place arterial catheter in these patients; however, we decided to place it in LKT for IBP monitoring and ABG analysis as Trendelenburg position and pneumoperitoneum are known to induce changes in BP and acid base balance. We did not place pulmonary arterial catheter for haemodynamic measurements as patients selected were without significant coronary artery disease with good ventricular function and their pre-operative BP was well controlled.

Haemodynamic changes associated with Trendelenburg position and pneumoperitoneum are well described in the literature in patients undergoing laparoscopy radical prostatectomy.[2-4] There is an increase in MAP, central venous and pulmonary artery pressure, whereas the effect on HR is variable. Kalmar *et al*.[2] observed that during the institution of the steep Trendelenburg position, both MAP and CVP increased significantly; however, increase was greater in MAP than CVP suggesting increased cardiac output (CO), systemic vascular resistance, or both as a cause. O'Malley and Cunningham[5] demonstrated that these changes are caused by an increased IAP compressing the aorta and increasing the afterload, possibly further enhanced by humoral factors. We also observed a significant increase in the MAP, which required pharmacological control with NTG in 65% of patients. Increase in HR was significant after pneumoperitoneum possibly due to CO_2 absorption and at the time of extubation due to lighter plane of anaesthesia whereas it remained stable throughout surgery.

Although, we have not measured CO, clinical studies using invasive haemodynamic monitoring have reported decreased, stable or increased CO after Trendelenburg position and pneumoperitoneum. Meininger *et al*.[6] and Hofer *et al*.[7] have reported significant, but clinically not relevant decrease in CO with significantly increased intra-thoracic blood volume (ITBV) attributing it to decrease in cardiac contractility. Haas *et al*.[4] observed 20% increase in CO due to increased right as well as left ventricular filling whereas Falabella *et al*.[3] have shown a significant increase in stroke volume with concomitant slight increase in CO by transoesophageal Doppler measurements. Meininger *et al*.[8] demonstrated that CO was not affected when an insufflation pressure was limited to 12 mmHg over a period of 4 h reflecting stable cardiac contractility. In view of conflicting results on CO, it may be necessary to measure CO in cardiac compromised patients.

Measurement of CVP is traditionally used to assess the adequacy of intravascular fluid replacement during open renal transplantation in patients with good cardiac function; however, the effects of pneumoperitoneum and Trendelenburg position on CVP make it unreliable as a measure of right ventricular filling pressure. This is because the increase in IAP is transmitted to thorax via the diaphragm.[4,6] We observed 46% increase in CVP after pneumoperitoneum and 72% increase after head down position and tried to maintain it at a higher level. Studies measuring CVP, cardiac index and ITBV have observed that an increase in CVP results from increased intrathoracic pressure related to the pneumoperitoneum and head tilt, rather than from changes in the ITBV. What is really needed is simultaneous measurement of intra-thoracic pressure, which can be obtained by oesophageal pressure and to deduct this from the measured central venous pressure.

Both the steep Trendelenburg position and pneumoperitoneum influence the respiratory system increasing arterial CO_2 and PAP during the mechanical ventilation; hence, capnography and monitoring of airway pressures are essential to alert the Anaesthesiologist of these problems. As expected, PAP increased significantly with an institution of the pneumoperitoneum and the Trendelenburg position, which may be related to the cranial displacement of the diaphragm due to the patient's position, increased IAP and decreased lung compliance.[2] There was no further increase in PAP during the course of the operation, which returned to a level slightly above baseline values after reinstitution of the supine position. Our strategy to increase MV by increasing RR instead of TV may be responsible for steady state of PAP during surgery.

$EtCO_2$ measurement is an acceptable alternative to $PaCO_2$ in many clinical circumstances in patients without cardio-respiratory disease; however, the utility of $EtCO_2$ monitoring in the assessment and management of patients with a combined steep Trendelenburg position and CO_2 peritoneum has remained largely undefined. Normal gradient between $EtCO_2$ and $PaCO_2$ is 5-10 mmHg, which increases during laparoscopic surgery in Trendelenburg position due to continuous insufflation of CO_2, V/P mismatch and reduced functional residual capacity leading to potential atelectasis.[9] Kalmar *et al*.[2] observed that there is a stronger underestimation of the true carboxaemia at higher levels of $EtCO_2$ in patients undergoing robotic prostatectomy in steep Trendelenburg position, Klopfenstein *et al*.[10] have observed inconsistent correlation between $PaCO_2$ and $EtCO_2$ during laparoscopic colon surgery mainly due to inter- and intra-individual variability. In our study, we observed mild rise in gradient between $EtCO_2$ and $PaCO_2$ after creation of pneumoperitoneum, which persisted throughout the procedure. Generally, $PaCO_2$ returns to normal range within 1 h of desufflation, but after prolonged laparoscopic surgery, it may take several hours to achieve a steady state of CO_2 as considerable amounts of CO_2 is stored in extravascular compartments of the body that are slowly redistributed and metabolized or exhaled. One of our patients in whom surgery lasted 6 h required post-operative mechanical ventilation for 4 h for hypercarbia.

Controversy exists concerning the nature of acid-base alteration in laparoscopic surgery. Some authors believe that acidosis is of respiratory type due to CO_2 absorption, others claim it predominantly to be of metabolic type produced by tissue hypoperfusion whereas one report showed it to be of mixed type.[11] We performed ABG analysis as renal failure patients may have pre-existing metabolic acidosis and hypoperfusion of lower extremities in steep Trendelenburg position as well as release of anaerobic metabolites from ischaemic graft and leg on reperfusion can produce metabolic acidosis. We observed mild metabolic acidosis on release of clamp; however, it did not require any treatment.

The most important concern related to laparoscopic approach was the deleterious effect of CO_2 pneumoperitoneum on blood flow and functional integrity of the transplanted kidney. Experimental and clinical studies in animals[12] and human[13] indicate that effective renal plasma flow, glomerular filtration rate and urine output decreases during pneumoperitoneum, the magnitude depends on the degree and duration of pneumoperitoneum and level of hydration. This decrease is not due to haemodynamic alterations but due to renal ischemia resulting from extravascular compression on renal vessels as well as release of ADH, renin and aldosterone. In our study, we did not encounter adverse effects of pneumoperitoneum on renal allograft as time to the establishment of U/O, U/O on the table and S. Creatinine on 1st and 7th post-operative day were acceptable. Whether this was due to proper hydration, shorter duration of pneumoperitoneum with less IAP (8-10 mmHg) after reperfusion or denervated transplanted kidney is protected from neuroendocrine response remains to be further investigated.

Overall LKT performed in steep Trendelenburg position with CO_2 pneumoperitoneum significantly influenced cardiovascular and respiratory homeostasis; however, measured parameters remained within clinically acceptable range without affecting early function of the transplanted kidney.

CONCLUSION

LKT is a well-tolerated procedure in CRF patients without significant comorbidities; however, it is more demanding for anaesthesiologists compared to OKT. In depth knowledge of deleterious effects of pneumoperitoneum and Trendelenburg position on cardiovascular and respiratory system along with appropriate monitoring is essential for safe management. Renal function does not seem to be adversely affected by pneumoperitoneum.

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