EFFECT OF PNEUMOPERITONEUM ON THE LEVEL OF PLASMA POTASSIUM

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Abstract

The purpose of this study was to find out the impact of acute respiratory acidosis on serum potassium level during laparoscopy.

The study was performed on ninety patients who underwent laparoscopic surgery. Ventilation parameters were kept constant throughout the study. Samples for assessment of blood gases were collected in the following sequence: before pneumoperitoneum, a the 20^{th} minute of pneumoperitoneum, at the 60^{th} minute of pneumoperitoneum, and after extubation. The systolic, diastolic pressures and heart rate were recorded simultaneously.

Before pneumoperitoneum, heart rate, systolic and diastolic pressures were reduced according to baseline values. With the induction of pneumoperitoneum, both systolic and diastolic pressures returned to the baseline levels except the heart rate. There was a statistically significant increase in potassium level (Control: 3.49 $mEq.L^{-1}$, determined high level: 3.75 $mEq.L^{-1}$).

Electrolytes, especially potassium, should be monitored during

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laparoscopy utilizing CO_2 pneumoperitoneum and when necessary, ventilatory parameters should be adjusted.

Key Words: Laparoscopic surgery, pneumoperitoneum, hypercapnia, potassium.

Introduction

Recently, the frequency of and indications for laparoscopic surgery have increased. This has enabled surgery of longer duration to be performed on patients with various medical problems and the elderly[1]. Laparoscopic approach is performed by various ways such as intraabdominal, extraperitoneal etc. To carry out the laparoscopic procedure, a pressured gas, most commonly CO_2 is used. It is important to know the physiopathologic changes on cardiovascular and respiratory systems that are caused by an increase in intraabdominal pressure and by the absorbtion of CO_2 from an artificial CO_2 pneumoperitoneum, in order to take preventive measures[2].

The absorption of the insufflated CO_2 from the peritoneal cavity into the blood during the CO_2 pneumoperitoneum, and decrease in lung compliance and functional residual capacity due to increased intraperitoneal pressure, cause an increase in $PaCO_2$[3-5]. If the ventilation parameters are not adjusted, hypercapnia and secondary respiratory acidosis will occur[3]. Severity of respiratory acidosis is well correlated with the duration of pneumoperitoneum. It is asserted that the acidosis caused by absorption of CO_2 from peritoneal cavity directs the intracellular potassium out to plasma and this may result in hyperkalemia[6].

The purpose of our study is to find out the effects of respiratory acidosis on serum potassium levels during laparoscopy.

Material and Methods

This study was carried out in a general surgery operating room with the approval of the institutional ethics committee and the individual

written consent of the patients. 90 adults (ASA I-II) who underwent laparoscopic procedure were included in the study. 83 of the subjects underwent laparoscopic cholecystectomy while 7 subjects had laparoscopic hiatal hernia repair.

Subjects with acute or chronic lung, kidney, metabolic or endocrine diseases or who had been using drugs that could lead to acid-base and electrolyte disturbances were not included. Subjects were not given any solution containing potassium or drugs that could increase the plasma potassium level during the operation.

0.9% NaCl infusion was started intravenously in the operating room. Midazolam 0.015 $mg.kg^{-1}$ iv was given as premedication. After applying the Allen's test preoperatively a 20 G cannula was placed into radial artery under local anesthesia.

Anesthesia was induced with 2 $mg.kg^{-1}$ propofol and 2 $\mu g.kg^{-1}$ fentanyl. Following 0.5 $mg.kg^{-1}$ atracurium, the trachea was intubated with a cuffed tracheal tube. The cuff was inflated until there was no audible air leakage. During anesthesia, the mechanical ventilation was performed by a Drager Sulla 808 V ventilator to sustain the following parameters; FiO_2: 50%, tidal volume: 10 $ml.kg^{-1}$, respiratory frequency: 10 per minute. Ventilation parameters were unchanged throughout study unless $EtCO_2$ reached 60 mmHg. Anesthesia was maintanined with 0.5-1% Isoflurane. During operation 1 $\mu g.kg^{-1}$ fentanyl and 0.1 $mg.kg^{-1}$ atracurium were administered as required. The CO_2 pneumoperitoneum was achieved by 2 $L.min^{-1}$ flow with the laparoscopic insufflator machine. The intraabdominal pressure was maintained at 12 mmHg and all subjects were kept at the 15° head-up throughout the operation.

Electrocardiography (ECG), heart rate, systolic and diastolic arterial pressures, oxygen saturation (SpO_2) and end tidal carbon dioxide ($EtCO_2$) values were recorded by the Millenia Monitor (Millenia, Orlando, FI, USA).

Arterial blood gas samples were taken as follows; before pneumoperitoneum (control [1. period]), 20 minutes after pneumoperitoneum (20 min of pneumoperitoneum [2. period]), 60

minutes after pneumoperitoneum (60 min of pneumoperitoneum [3. period]), and after extubation (extubation [4. period]). Systolic, diastolic pressures and the heart rate were recorded simultaneously and preoperative values of those parameters were also recorded. Ciba Corning 860 blood gas apparatus was used for the evaluation of arterial blood gases and electrolytes.

At the end of the operation, the neuromuscular block was reversed with 0.01 $mg.kg^{-1}$ atropine and 0.02 $mg.kg^{-1}$ neostigmine. After the subjects had recovered in the recovery room, they were sent to general surgery wards.

For statistical analysis of the collected data, ANOVA repeated measures analysis, along with posthoc Tukey-Kramer multiple comparisons tests, were used. Results were given as mean ± standard deviation. Values of $p < 0.05$ were accepted as statistically significant.

Statistical power analysis with an α error of 0.05 and a β of 80%, indicated 8 patients was required to detect a difference of 5 mmHg in $PaCO_2$ with a standard deviation of 4.9 mmHg (control value). 10 mmHg changes in $PaCO_2$ resulted in a potassium increase of 0.4 $mEq.L^{-1}$ [7]. Further differences in potassium would be due to metabolic effects. Power analysis for potassium with the same α and β indicated 32 patients was needed to detect a difference of 0.3 $mEq.L^{-1}$ in potassium and a standard deviation of 0.6 $mEq.L^{-1}$ (control value).

Results

The ninety subjects (65 women, 25 men) that were between the ages of 19-72 years (mean: 45.3 ± 12.3 years) and weighing of 53-120 kg (mean: 71.6 ± 12.03 kg) were included in our study. Eighty-three patients underwent laparoscopic cholecystectomy and the rest hiatal hernia repair.

The mean operating time was 83.9 ± 18.6 min. Changes in the hemodynamic parameters such as the heart rate and arterial pressures were always within the 20% limits of the control at all of the periods. Before pneumoperitoneum, it was observed that there had been a statistically significant decrease in systolic and diastolic arterial pressures

and heart rate when compared to baseline values. Systolic and diastolic arterial pressures increased to reach the values of preinduction with the creation of pneumoperitoneum. However, heart rate decreased meaningfully in all periods relative to the values at preinduction. No arrhythmia was observed in any of the subjects (Table 1).

Table 1

Hemodynamic values (mean ± SD)

Periods	SAP (mmHg)	DAP (mmHg)	HR (beat/min)
Preop	132.9 ± 18.1	81.4 ± 10.8	83.7 ± 13.03
Control	117.7 ± 15.8*	74.04 ± 12.6*	79.03 ± 12.5*
20 min of pneumoperitoneum	137.4 ± 19.9	86.01 ± 13.3	77.3 ± 12.01*
60 min of pneumoperitoneum	127.5 ± 18.05	79.7 ± 14.09	77.2 ± 13.03*
Extubation	131.7 ± 21.5	79.02 ± 13.8	76.7 ± 13.8*

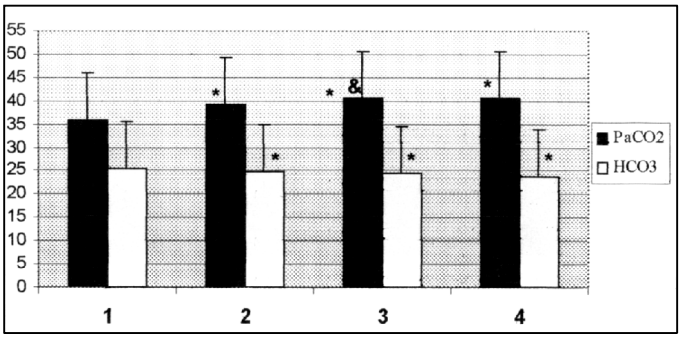
* $p < 0.05$ compared with preop value.

Preop: preoperative value, control: before pneumoperitoneum, 20 min of pneumoperitoneum: 20 minutes after pneumopertoneum, 60 min of pneumoperitoneum: 60 minutes after pneumoperitoneum Extubation: after extubation, SAP: Systolic arterial pressure, DAP: Diastolic arterial pressure, HR: Heart rate.

Throughout the operation $PaCO_2$ showed a tendency to increase while the pH tended to decrease when compared to the control values (Graphic 1, 2).

Graphic 1

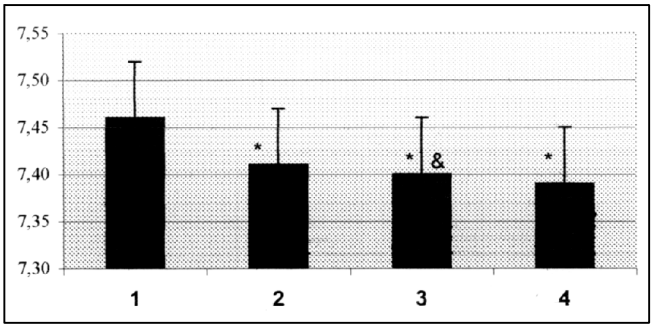
$PaCO_2$ (mmHg) and HCO_3 values (mean ± SD)



* $p < 0.05$ compared with 1 period & $p < 0.05$ 3 compared with 2 period.

Periods: 1: before pneumoperitoneum, 2: 20 minutes after pneumoperitoneum, 3: 60 minutes after pneumoperitoneum, 4: After extubation.

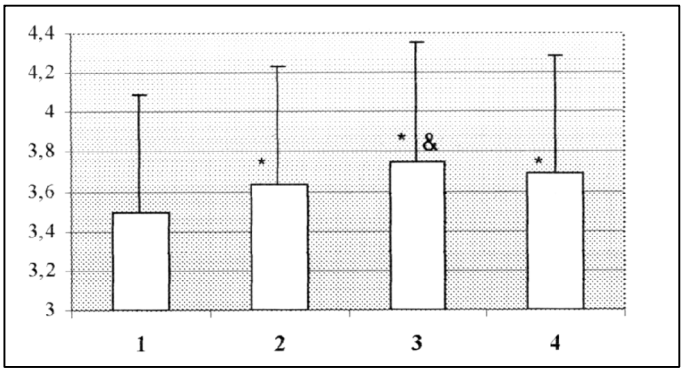
Graphic 2
pH values (mean ± SD)



* p < 0.05 compared with 1 period & p < 0.05 3 compared with 2 period.
Periods: 1: before pneumoperitoneum, 2: 20 minutes after pneumoperitoneum, 3: 60 minutes after pneumoperitoneum, 4: After extubation.

A statistically significant increase in serum potassium levels occurred at the 20th and 60th minutes but was not clinically important. Potassium level was determined as 3.49 ± 0.6, 3.63 ± 3.75, 3.75 ± 0.6, and 3.69 ± 0.6 mEq.L^{-1} respectively (Graphic 3).

Graphic 3
Potassium values (mEq.L^{-1}) (mean ± SD)



* p < 0.05 compared with 1 period & p < 0.05 3 compared with 2 period.
Periods: 1: before pneumoperitoneum, 2: 20 minutes after pneumoperitoneum, 3: 60 minutes after pneumoperitoneum, 4: After extubation.

Discussion

The essential feature of laparoscopic surgery is to create a pneumoperitoneum with pressured gas. CO_2 is preferred because of its highly soluble, non-flammable properties and it also reduces the frequency and severity of the emboli that can occur[1]. Highly pressured insufflation of CO_2 to produce pneumoperitoneum can lead to unfavorable conditions during anesthesia by causing changes in respiratory, hemodynamic and acid-base balances[8]. Hypercapnia may be contributed by absorption of CO_2 from the intraperitoneal cavity during pneumoperitoneum and depression of the functional residual capacity and lung compliance due to the increase in the intraabdominal pressure[3,4,9]. The elevation of the $PaCO_2$ level that is seen when the CO_2 gas is used for the pneumoperitoneum is not seen when N_2O or other gases are used and this shows that the principle mechanism of this elevation depends on the direct diffusion of CO_2 from peritoneal cavity to blood[10-12]. If the respiratory parameters are constant throughout the operation, development of hypercarbia and respiratory acidosis is inevitable with fixed minute volume[4].

El-Minavi et al[13] found a meaningful increase at $PaCO_2$ levels in contrast to the value of pH when pneumoperitoneum was made by CO_2. The lack of finding any significant changes in HCO_3 level shows that this condition is a respiratory event secondary to CO_2 Mango et al[10] declared that there was a significant elevation in $PaCO_2$ and a depression in pH level secondary to intraperitoneal CO_2 insufflation in the patients to whom volume-controlled ventilation was applied during laparoscopic cholecystectomy. In the study of Iwasaka et al, which was performed on patients who underwent laparoscopic cholecystectomy, it was shown that during insufflation, $PaCO_2$ and $EtCO_2$ increase, and secondary to this effect, the pH decreases, but there is no change in the concentration of HCO_3[3].

In our study, similar to the other studies mentioned above, at the first 20^{th} minute of the insufflation there was a significant increase in $PaCO_2$ and a significant decrease in the pH level relative to the control period. At

this period a significant decrease in HCO_3 level was observed. At the 60th minute of insufflation there was a significant increase in $PaCO_2$ when compared with that of control and the 20th minute values. There was a significant decrease in the level of pH in comparison with that of the 20th minute and the control period. There was a significant difference at HCO_3 levels relative to that of 20th minute. In our study acidosis that has been observed during the first 20 minutes was a respiratory acidosis due to an increase of $PaCO_2$ level. The decrease in HCO_3 level could be either due to compensation of the respiratory acidosis or, as some researchers have mentioned, could be a sign of metabolic acidosis because of the decrease in peripheral perfusion[14].

It is claimed that hyperkalemia can occur secondary to the movement of intracellular potassium into the serum resulting in acidosis caused by CO_2 absorption from the peritoneal cavity[6]. Extracellular acidosis or acidemia causes hyperkalemia by the movement of potassium from intracellular media outwards as a result of the cellular tampon of H^+[15,16]. On the other hand, abdominal wall ischemia that is secondary to the increase in intraabdominal pressure and local intracellular acidosis that is created by peritoneal CO_2 insufflation, can cause the increase in serum potassium level[6,17].

Pearson et al[6] observed the increase in serum potassium concentration with the ECG changes reflecting hyperkalemia in 2 to 3 hours after CO_2 insufflation in pigs. Hassan et al[18] demonstrated that there was a relation between the serum potassium level and $PaCO_2$. Parallel to the increase in $PaCO_2$, there was an elevation of the serum potassium level that was clinically irrelevant and did not cause ECG changes. They set forth an association between the serum potassium level and $PaCO_2$ with even minimal changes of hypercapnia. Edwards et al[7] found that there was an increase of 0.4 meq/L in the serum potassium level for each 10-mmHg increase in $PaCO_2$ and there was a relation between the serum potassium level and $PaCO_2$. Perner et al[17] claimed that in young patients and patients with normal renal functions to whom CO_2 pneumoperitoneum was applied for a short time, there was no hyperkalemia. They observed that especially with old patients there was a

clinically and statistically insignificant increase in the potassium level in the case of pneumoperitoneum which took longer time.

In our study, similar to results reached by the aforementioned authors, there was a statistically significant increase in potassium level associated with hypercapnia in the period after pneumoperitoneum. The increase in potassium level was not relevant clinically and there was no ECG changes or arrhythmia in any patient.

Intracellular potassium moves into the plasma as a result of acidosis associated with hypercapnia. Development of hyperkalemia or the reaching of the dangerous level has been protected and avoided by increasing elimination of urinary potassium with the effects of aldosterone mediators. On the other hand in patients taking aldosterone inhibitory treatment with poor renal functions, potassium level can reach dangerous levels in the state of prolonged pneumoperitoneum[17,18].

In our study, the average time of pneumoperitoneum was 88 mintes and measurements were done at the 20^{th} and 60^{th} minutes. There was a significant increase in the serum potassium level, although intraabdominal pressure was kept stable at 12 mmHg. This increase was not clinically important because our subjects were healthy.

During prolonged pneumoperitoneum with high pressure, the serum potassium level can increase more evidently. Potassium level can reach dangerous levels in patients taking potassium sparing diuretics and digitalis with poor renal functions. During CO_2 pneumoperitoneum, changes in potassium level associated with the increase of $PaCO_2$ should be observed carefully and ventilation parameters should be adjusted. Positional changes and intraabdominal pressure should be limited.

We conclude that for patients using potassium sparing diuretics and digitalis with limited renal and cardiac reserve, more extended monitoring including blood gases and potassium level measurement should be applied.

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