

Evaluation of Hemodynamic Parameters during Laparoscopic Cholecystectomy in Children with Sickle Cell Disease and Non-Sickle Cell Disease

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Abstract

Background: Aim of this study is to evaluate if there is any difference in terms of hemodynamic parameters during Laparoscopic Cholecystectomy (LC) in children with Sickle Cell Disease (SCD) and Non-Sickle Cell Disease (NSCD).

Methods: Twenty children with asymptomatic cholelithiasis, recruited for elective LC were allocated into two groups: group 1 (n: 10) children without SCD (control group), and group 2 (n: 10) children with SCD. All of the patients with SCD had preoperative hematological evaluation for the surgical procedure. Study parameters included: heart rate, mean blood pressure, Peak Inspiration Pressure (PIP), end-tidal carbon dioxide concentration (PETCO₂), expiratory tidal volume (Vt), O₂ saturation, arterial blood gases (pH, PaCO₂, PaO₂, HCO₃), hematocrit and intra-abdominal pressures. Parameters were recorded at five moments: before CO₂ insufflation, 15, 45, 60 minutes after insufflation in reverse Trendelenburg position and 5 minutes after CO₂ insufflation in the supine position.

Results: Regarding all parameters, before and after CO₂ insufflation and after CO₂ desufflation, there were insignificant changes in each group and between the two groups. Intra-abdominal pressure changes did not affect the hemodynamic parameters. No postoperative complication was observed and the post-operative hospital stay in two groups was similar.

Conclusion: Although sample size is not enough this study reveal that laparoscopic cholecystectomy does not have any adverse effects on hemodynamic parameters of children with sickle cell disease. Laparoscopic cholecystectomy may be feasible and as safe as in NSCD patients.

Introduction

Sickle cell disease in children is well known to have a high incidence of cholelithiasis. Laparoscopic Cholecystectomy (LC) is the treatment of choice in children with cholelithiasis. The safety and efficacy of LC in cholelithiasis with Sickle-Cell Disease (SCD) in adults is proved to be as safe as in patients with Non-Sickle-Cell Disease (NSCD) [1].

Minimally invasive surgery provides shorter hospital stay, less pain, early recovery and good cosmetic results. Due to severe operative complications, SCD patients require meticulously planned management procedures [2]. To the best of our knowledge, there is no prospective study evaluating hemodynamic parameters during laparoscopic cholecystectomy in children with cholelithiasis related to SCD.

Sickle cell disease is common in Mediterranean region of Turkey. Our aim is to evaluate the safety of LC in SCD with cholelithiasis in children comparing with the control group with NSCD children.

Materials and Methods

A prospective study is carried out at Çukurova University, Faculty of Medicine, Department of Pediatric Surgery, Adana, Turkey, after approving of our local research ethics committee and informed parental consent. Cukurova University Hospital is a reference hospital for the city of Adana and neighbor community living in the South part of Turkey. This corresponds to approximately 1.9 million inhabitants with a frequency of 0,5% -37% of sickle cell disease. Twenty two children with asymptomatic cholelithiasis had LC. We used the standard 4-port technique. 10 of them had SCD, 2 had β thalassemia and hereditary spherocytosis, 10 had no hematological disorder. Twenty children with asymptomatic cholelithiasis, recruited for elective LC, were allocated into two groups: group 1 (n: 10), children without SCD (control group), and group 2 (n:10) children with SCD. The SCD patients underwent partial exchange transfusions if the HbS level was more than 30% preoperatively. The ones with hemoglobin level less than 10gr/dl received blood transfusion.

Table 1: Main features of patients, mean, minimum and maximum data.

	Group 1 (NSCD)	Group 2 (SCD)
Age (year)	12±4.13 (3-16)	11.7±4.29 (2-16)
Sex (F/M)	5/5	4/6
Weight (kg)	38.8±14.66 (15-56)	39.9±14.9 (17-61)
Height (cm)	144.5±19.16 (112-171)	139.6±20.72 (96-165)
Operative time (min)	113±19.88 (90-155)	107±21.62 (70-130)
Hospital stay (day)	2.5±1.17 (1-5)	1.7±0.48 (1-2)

The anesthesia induction was obtained with thiopental sodium (4-5 mg/kg), 4-5% sevoflurane and 50% nitrous oxide in oxygen using face mask. After the administration of vecuronium (0.1mg/kg iv) the trachea was intubated. Anesthesia was maintained with the inhalational anesthetic sevoflurane, 50 % nitrous oxide in oxygen. The patients were mechanically ventilated with a tidal volume of 10-12 ml/kg at a respiratory rate sufficient to maintain PETCO₂ of approximately 32-36 mmHg. All patients were given ringer lactate 5-10 ml/kg/hr to obtain proper Heart Rate (HR) and Mean Arterial Pressure (MAP) by anesthesiologist. Analgesia provided with tramadol 1-2 mg preemptively. Pulse-oximetry, arterial line, nasogastric tube and urinary bladder catheter were inserted. Pneumoperitoneum was established using a Veress needle at the umbilical site and intra-abdominal pressure was maintained at 12 mmHg. Study parameters included: heart rate per minute, mean blood pressure, Peak Inspiratory Pressure (PIP), Positive End Expiratory Pressure (PEEP), end-tidal carbon dioxide concentration (PETCO₂), expiratory tidal volume (Vt), O₂ saturation, arterial blood gases (pH, PaCO₂, PaO₂, HCO₃), hematocrit and intra-abdominal pressures. Intra-abdominal pressures were measured via nasogastric tube. Parameters were recorded at five moments: before CO₂ insufflation, 15, 45, 60 minutes after insufflation in the reverse Trendelenburg position and 5 minutes after CO₂ insufflation in the supine position.

Data were entered into a database and analyzed with SPSS 11.0 software. The Wilcoxon and Friedman tests were used to assess changes of measurements in time of 0, 15, 45, 60 minutes after inflation and 5 minutes after deflation in each groups. Differences between group 1 and 2 were compared using the Mann-Whitney U test. A p value less than 0.05 was taken to present statistical significance.

Results

There were no significant differences between the two study groups regarding age, sex, weight, height, operative time and hospital stay (Table1).

Table 2: Hemodynamic and intra-abdominal pressure changes, mean (SD).

	Heart Rate/min		Mean Arterial Pressure (mmHg)		Intra-abdominal Pressure cm/H ₂ O	
	NSCD (Group 1)	SCD (Group 2)	NSCD (Group 1)	SCD (Group 2)	NSCD (Group 1)	SCD (Group 2)
Before CO₂ insufflation	95.5 ±(21.38)	87.6±(16.87)	97.7±(9.54)	94.4±(13.92)	6.1±(1.26)	5.2±(3.82)
15 min after insufflation	95.1±(12.8)	94.2±(18.77)	93.2±(13.99)	91.6±(21.95)	13.6±(4.52)	10.6±(4.06)
45 min after insufflation	93.7±(18.01)	96.3±(23.45)	93.9±(11.5)	88.8±(15.03)	13.3±(4.62)	9.8±(3.99)
60 min after insufflation	99.4±(14.24)	91.8±(19.09)	95.10±(13.42)	91.5±(21.38)	12.7±(5.97)	9.7±(2.54)
5 min after deflation	98.8±(17.46)	92.37±(19.28)	93.88±(5.48)	90.01±(14.94)	4.1±(2.33)	2.7±(1.88)

Within in each group, after insufflation produced a significant increase and after deflation a significant decrease in intra-abdominal pressure but no heart rate and mean arterial pressure changes were detected throughout the procedure. Comparison between the two groups, heart rate, mean arterial pressure and intra-abdominal pressure revealed no significant differences at the moment of- before CO₂ insufflation, 15, 30, 45, 60 min and after deflation of CO₂ (Table 2).

Regarding PETCO₂, Vt and PEEP, there were no significant changes in each two groups themselves and between the two groups at all moments of the study. In each group, during 45 minutes after insufflation, there was not a significant increase in PETCO₂ (Table 3). There was not a significant correlation between PIP and IAP.

There was not any significant change in each groups and between the two groups regarding the arterial gases parameters including pH, PaCO₂, PaO₂, hematocrit and HCO₃ (pH, PaCO₂ and PaO₂ values summarized in table 4). No blood transfusion was needed in each group during the operation and post-operative period. None of the SCD patients had experienced a sickle cell crisis.

Discussion

Heart rate and mean arterial blood pressure commonly increase during laparoscopy due to CO₂ insufflation. An increase in mean arterial pressure, usually combined with an increase in systemic vascular resistance, has been a common finding in CO₂ pneumoperitoneum [3-6]. According to our results, within in each group, insufflation produced a significant increase and deflation a significant decrease In Intra-Abdominal Pressure (IAP) but no heart rate and mean arterial pressure changes were detected through the procedure. Anesthesiologist may interfere the arterial pressure and heart rate changes by giving proper fluid therapy during the operation. The mechanism behind the hemodynamic changes may be caused by CO₂ pneumoperitoneum and head-up positioning. Decreased preload, increased afterload, and the release of humoral factors all may contribute to the changes. The magnitude of Intra-Abdominal Pressure (IAP), the baseline hemodynamic function and the volume status of the patients, anesthetic techniques, and measures for decreasing peripheral pooling of extracellular fluid all are expected to influence hemodynamic results [7, 8]. The patients in our study, had no central venous and pulmonary artery catheter and these parameters were not studied. In an animal model, it was claimed that intraoperative normovolemia and fluid management seem to be the most important factors for minimizing the hemodynamic side effects of pneumoperitoneum [9]. In our study, the patients were hemodynamically stable due to appropriate fluid management.

Table 3: End-tidal CO₂ , Tidal volume and Positive End Expiratory Pressure (PEEP) changes in two groups, mean (SD).

	PETCO ₂ (mmHg)		Tidal volume		PEEP	
	NSCD (Group 1)	SCD (Group 2)	NSCD (Group 1)	SCD (Group 2)	NSCD (Group 1)	SCD (Group 2)
Before CO₂ insufflation	31.7±(4.9)	34.5±(3.5)	339.7±(96.34)	369.5±(118.6)	3.8±(1.22)	3.5±(0.97)
15 min after insufflation	30.3±(5.31)	34.5±(5.52)	395.9±(149.5)	387.9±(137.5)	4.3±(1.70)	3.6±(1.5)
45 min after insufflation	33.5±(3.02)	36.1±(3.31)	394±(152.06)	411.4±(159.8)	3.7±(1.56)	3.3±(1.94)
60 min after insufflation	34.7±(4.16)	35.5±(3.43)	385.3±(153.8)	396.2±(165.3)	4.1±(1.10)	3.2±(1.31)
5 min after deflation	34.8±(3.62)	34.75±(3.57)	398.5±(164.3)	432±(165.9)	4.1±(0.92)	3.3±(0.91)

Table 4: Arterial blood gases changes in the two groups, mean (SD).

	pH		PaCO ₂		PaO ₂	
	NSCD (Group 1)	SCD (Group 2)	NSCD (Group 1)	SCD (Group 2)	NSCD (Group 1)	SCD (Group 2)
Before CO₂ insufflation	7.42±(0.04)	7.42±(0.05)	30.75±(4.35)	34.5±(5.71)	251±(69.29)	240.±(57.4)
15 min after insufflation	7.42±(0.03)	7.39±(0.03)	33.05±(3.21)	36.18±(4.01)	228.2±(68.7)	237.3±(54.79)
45 min after insufflation	7.38±(0.03)	7.36±(0.05)	35.11±(5.5)	37.0±(4.5)	217±(69.1)	228.1±(42.77)
60 min after insufflation	7.35±(0.01)	7.34±(0.04)	38.2±(4.33)	37.87±(4.2)	215.2±(73.7)	216.42±(38.55)
5 min after deflation	7.36±(0.02)	7.36±(0.034)	38.85±(3.98)	38.08±(4.56)	219±(73.94)	237.12±(37.21)

Potential respiratory consequences of CO₂ pneumoperitoneum include increased intraabdominal pressure which may impair diaphragmatic motion, decreased functional residual capacity and pulmonary compliance, increased airway resistance and decreased tidal volume and minute ventilation [10,11]. In our study, changes in blood gases, PETCO₂, tidal volume (Vt), PEEP, peak inspiratory pressure and respiratory rate were not found to be significant in each groups themselves and between the two groups at all moments of the study. There was no significant increase in PETCO₂ during 45 minutes after insufflation in each group.

It was reported that CO₂ insufflations led to a significant increase in PETCO₂, reaching a maximum level just before abdominal deflation in the anti-Trendelenburg position. PaCO₂ also increased significantly 30 min after CO₂ insufflation, but there was a significant decrease in pH levels 30 min after CO₂ insufflation in the anti-Trendelenburg position [1]. In our study, there were no significant increases in PETCO₂ 45 minutes after insufflation in each group. Although we observed a correlation between PIP and IAP clinically, this correlation did not achieve statistical significance. Some authors suggested a significant correlation between insufflation pressure and PIP [12]. The increased IAP induces a cephalad shift leading to a compression of the intrathoracic organs and reduces functional residual capacity, thoracic compliance and increases the airway resistance, ventilation perfusion mismatch and intrapulmonary shunting [13]. In our study, Vt and respiratory rate were arranged for maintaining normocarbida and neutral pH. Probably for this reason, no patient had acidosis, hypoxia or hipercarbida during study period in our study [14].

According to Sfez, et al. a high end-tidal CO₂ in 37% of neonates and children under 4 months of age and hypoxemia in 0.5% when insufflations pressure was limited to 15 mmHg [15]. In our study, insufflation pressure was 12 mm Hg. Higher pressure can affect pulmonary mechanics strongly.

On the other hand, we observed no significant changes in both SpO₂ and PaO₂ through the study period in the two groups. These results are compatible with the literature [1,16].

Our major limitation is sample size. Actually sample size is not enough for statistical measurements. The sample size must be more than 250 patients for power >90% and this sample size for children may be large for one hospital. Thus multicenter studies are needed.

Children with cholelithiasis related to sickle cell disease may tolerate elective laparoscopic cholecystectomy. Laparoscopic cholecystectomy in children with SCD may be feasible and as safe as NSCD children.

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