Early Period Effect of the Massive Pleural Effusion Drainage on Hemodynamic and Pulmonary Functions

Eurasian Clinical and Analytical Medicine Original Research

Effect of the massive pleural effusion drainage

Ömer Faruk Demir, Ömer Önal
Department of Thoracic Surgery, Faculty of Medicine, Erciyes University, Kayseri, Turkey

Abstract

Aim: The objective of our study was to put forth the acute changes in hemodynamic and pulmonary functions following massive pleural effusion via thoracoscopy.

Material and Methods: A total of 20 patients who underwent thoracoscopy due to massive pleural effusion between January 2012 and January 2013 were included in our study. Partial oxygen pressure, partial carbondioxide pressure, respiratory rate, pulse rate, systolic and diastolic blood pressures of the patients were measured one day before and one day after pleural effusions drainage. The central venous pressure, pulmonary capillaries wedge pressure and pulmonary artery pressure values of the patients were measured via Swan Ganz catheter before and after pleural effusion drainage.

Results: The average amount of pleural effusion drainage was 2205±734 cc. A statistically significant changes were observed in the respiratory rate, systolic blood pressure, partial oxygen pressure, pulmonary artery pressure, central venous pressure, and pulmonary capillaries wedge pressure with pleural effusion drainage ($p < 0.05$). No statistically significant changes were observed in diastolic blood pressure, partial carbondioxide pressure, and pulse rate ($p > 0.05$).

Discussion: Massive pleural effusion drainage is ineffective at the early stage on diastolic blood pressure, partial carbondioxide pressure, and pulse rate.

Keywords

Hemodynamic; Pleural Effusion; Pulmonary Function

DOI:10.4328/ECAM.118
Received : 17.10.2017
Accepted : 27.10.2017
Published Online : 01.09.2017
Printed Online : 01.09.2017
Eu Clin Anal Med 2017;5(3): 41-4

Corresponding Author: Name and address
GSM: +90 530 293 52 04 · **P..:** +90 352 207 66 66 · **F.:** +90 352 437 52 73 · **E-Mail:** ofdemir@erciyes.edu.tr

How to cite this article: Ömer Faruk Demir, Ömer Önal. Early Period Effect of the Massive Pleural Effusion Drainage on Hemodynamic and Pulmonary Functions. Eu Clin Anal Med 2017;5(3): 41-4.

Introduction

Malignant pleural effusion is a sign of poor prognosis in patients with malignity. Majority of these patients die within a year [1-2]. Massive effusion is defined as effusion that radiologically fills about 2/3 or more of the thoracic cavity and formation of it is frequently related with malignity [3-4]. Worsening of the quality of life is mostly related to cardiac and pulmonary functions. Drainage of massive pleural effusion has corrective effects on hemodynamic and pulmonary functions [5-6]. However, the number of studies carried out on this subject is very limited in the literature [7]. The objective of our study was to evaluate the changes in various hemodynamic and pulmonary parameters by measuring the pulse rate, arterial tension, pulmonary arterial pressure (PAP), pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP), respiratory rate, partial oxygen pressure (PaO2), partial carbondioxide pressure (PaCO2) in spontaneously breathing patients with massive pleural effusion due to malign reasons following the drainage of the effusion.

Material and Methods

A total of 20 patients who applied to our clinic between January 2012 and January 2013 with massive pleural effusion diagnosis were included in this study. Inclusion criteria of the patients are determined as 1) detection of massive pleural fluid, 2) verification of effusion in patients via thoracentesis, 3) thoracoscopy indication (pleural biopsy, evaluation of pleural space and simultaneous pleurodesis), 4) having primary malignity 5) no contraindication for Swan Ganz catheter, 6) volunteering to participate in the study.

Patients with pulmonary arterial tumor invasion, pulmonary embolism or cardiac disease were excluded. Posterior-anterior and lateral lung x-rays were used in the evaluation of massive pleural effusions. However, thoracic ultrasound and thorax tomography were also required in some patients to evaluate the mass or pulmonary artery invasion.

Thoracentesis was applied to confirm pleural effusion following a clinical and radiological examination. The average values of the systolic-diastolic blood pressure, pulse rates, and respiratory rates those were measured at 4 hour intervals during the 24 hours before thoracoscopy were recorded as pre-operative values. The blood gas values of the patients were measured 24 hours before thoracoscopy. Swan Ganz catheter was applied to the patients under sedation in the operating room prior to thoracoscopy. The reason for this was that it allowed both Swan Ganz catheterization and drainage of pleural effusion under sedation without creating additional pain in patients. Central venous pressure, pulmonary arterial pressure and pulmonary capillary wedge pressures of the patients were measured. Afterwards, pleural fluid was drained via thoracoscopy. The drained pleural fluid amount was recorded. The central venous pressure, pulmonary arterial pressure, and pulmonary capillary wedge pressures of the patients, were re-measured via the Swan Ganz catheter and noted down after the patients were returned to the supine position. The systolic, diastolic blood pressures and pulse rates, respiratory rate were measured after a period of 24 hours with 4 hour intervals and after that their average values were recorded as the value after the process. Twenty-four hours after thoracoscopy, blood gas was evaluated again. None of our patients had re-expansion surveillance. Sudden lung opening was not observed similar to awake illness. As the effect of sedation diminished, patients' lung expansions occurred. This study was carried out with the approval of the Faculty of Medicine Ethical Council (No: 2012/589).

Statistical method

The analyses were carried out using the SPSS 20 software. Average, standard deviation, ratio, and frequency values were used for the descriptive statistics of the data. Kolmogorov Smirnov test was used to examine the data distribution. Independent sampling t-test was used for the analysis of quantitative data. Paired sample t-test was used for repeated measurements. Pearson analysis was used for correlation analysis.

Results

Sixty percent (n=12) of the patients were male, and 40% (n=8) were female. The ages of the patients were ranged between 39 and 82 (average 61.8±10.9). 65% of the patients (n=13) had lung cancer, 10 % (n= 2) had breast cancer, 10 % (n=2) had stomach cancer, 5 % (n=1) had liver cancer, 5 % (n=1) had colon cancer, 5 % (n=1) had pancreas cancer. Half of the pleural effusions had a right hemithorax placement (n=10), whereas the other half had left hemithorax placement (n=10). The average value of pleural fluid drainage was 2205±734 cc.

Hemodynamic and pulmonary parameters are summarized in Table 1. Preoperative and postoperative measurements were compared, and statistically significant difference was found in systolic blood pressure, PAP, CVP, PCWP, respiratory rate, PO2 ($p<0.001$). Despite that no statistically significant difference was detected between preoperative and postoperative values of diastolic blood pressure, pulse rate and PCO2 ($p>0.05$).

No correlation was determined between all the measured parameters and whether the pleural fluid was located in the right or left hemithorax ($p>0.05$).

Table 1. Hemodynamic and pulmonary parameters

	Preoperative	Postoperative	Change	P
Systolic pressure	118.2±18.6	108.0 ±2.0	-10.2	p<0.001
Diastolic pressure	79.1 ± 12.3	76.4 ± 25.1	-2.7	0.600
Pulse rate	103.0±11.7	99.7 ±11.0	-3.3	0.085
PAP	18.7±3.1	15.4±3	-3.3	p<0.001
CVP	12.7±2.5	9.1±2.6	-3.6	p<0.001
PCWP	13.7±3.1	9.7±2.5	-4.0	p<0.001
Respiratory rate	27.1±1.7	20.9± 1.8	-6.3± 2.5	p<0.001
PO2	61.8± 10.7	69.7± 10.7	7.2 ±4.3	p<0.001
PCO2	36.5 ±6.2	36.2± 6.8	-0.3± 2.5	0.666

Paired sample t test: Data are presented as mean ± standard deviation. Values in bold indicate values with significant difference.

Discussion

Improvement of hemodynamic and pulmonary parameters due to pleural fluid drainage is one of the goals of palliative treatment of patients who had terminal stage malignancy. Unfortunately, we were not able to detect a study about hemodynamic changes in patients with spontaneous respiration in the literature. Therefore, the comparison of this study was conducted with the studies including patients in mechanical ventilation support and with experimental studies.

Ahmed et al. [8] reported results of 22 patients who were subject to pleural fluid drainage via thoracentesis while under mechanical ventilatory support. They detected minimaly decreases in systolic-diastolic blood pressure, and it was not statistically significant. Also, no statistically significant change was determined in the average arterial pressures of patients monitored by ventilator at the intensive care unit who were subject to an average of 1.579 ± 684 cc pleural fluid drainage during the study carried out by Razazi et al. [9], whereas in our study, a statistically significant decrease was determined in the systolic blood pressure values, no statistically significant difference was observed in diastolic blood pressure. The blood pressure change may have been minimal due to both the lowering effect of positive pressure on the blood pressure applied by the ventilator into the thoracic cavity, as well as due to the sedative drugs given during the follow up period in intensive care units.

It is expected that the discharge of the pleural effusion that will cause a decrease in intrathoracic pressure will decrease the pulse rate by removing the pressure on the heart. In a relevant study in the literature carried out on 22 piglets, a massive saline infusion was administered to imitate pleural effusion, and a correlation was determined between the saline infusions administered intrathoracically and the change in pulse rate ($p < 0.05$) [10]. Whereas in a study carried out by Ahmed et al. [8] pulse rate was measured as 109 ± 19 before pleural fluid discharge and as 107 ± 14.8 after fluid discharge for which the difference was not statistically significant. A statistically significant change was not determined in the pulse rate in the study carried out by Razazi et al. [9]. In our study, the preoperative pulse rate was measured as 103.0 ± 11.7, and the postop pulse rate was measured as 99.7 ± 11. However, this difference was not determined to be statistically significant ($p>0.05$). Despite that, a statistically significant difference was determined in pulse rate during the experimental study carried out by Wemmelund et al. [10]. We are of the opinion that the administration of the artificial pleural effusion generated in such experimental studies to the subjects might lead to an acute and strong response in the hemodynamics and respiratory system of the experimental subjects.

Massive pleural effusion yields findings similar to that of cardiac tamponade and the drainage of pleural effusion causes regression in these findings [11-14]. Ahmed et al. [8] reported that pulmonary capillary wedge pressure ($p <0.01$) and central venous pressure ($p<0.02$) were decreased statistically significantly following pleural fluid discharge. However, despite the decreases they detected in pulmonary artery pressure, they also put forth that these differences were not statistically significant. It was determined during the measurements made as part of the experimental study carried out by Wemmelund et al. [10] that there were statistically significant changes in central venous pressure and pulmonary artery pressure values. Statistically significant decrease was determined in our study for pulmonary artery pressure, pulmonary capillary wedge pressure and central venous pressure.

As can be seen, there was a significant change in pulmonary artery pressure in experimental studies, but this was not shown in patients with mechanical ventilation support.

When case reports are excluded, the studies about the effect of pleural effusion on hemodynamic parameters were identified in two groups.

These studies mostly consist of either patients followed by mechanical ventilation or experimental animal studies [8-10]. Our study, as far as we know, is the first study to evaluate hemodynamic changes after massive pleural effusion drainage in spontaneously breathing patients. Another point of interest the experimental hemodynamic response in experimental studies was higher than in patients with ventilatory support [10]. Drainage of massive pleural effusion cause more powerful response in hemodynamic parameters (especially PAP and systolic blood flow) in spontaneously breathing patients, than patients in mechanical ventilatory support, even if not emphasized in experimental studies.

Ahmet et al. [8] reported that although the respiratory rates of the patients were decreased due to drainage of pleural effusion, it was not statistically significant. Additionally, no significant difference was found in partial oxygen pressures and carbon dioxide pressures.

Whereas, in the study conducted by Wang et al. [14], a significant increase in the PO2 value was observed before the treatment of the patients, but no significant decrease in the PCO2 values could be shown.

Some studies in the literature have even noted hypoxemia in the early period (first 2 hours) after pleural drainage [15]. However, when review and meta-analyses of the subject are reviewed, it is stated that pleural effusion drainage may give rise to the improvement in PO2 and respiratory rate values in the late period (after 24h), but it does not decrease PCO2 levels markedly [7,16,17,18].

In our study, the changes in PO2 and respiratory rate were statistically significant in accordance with the literature, but the decrease in PCO2 was not statistically significant. In our opinion, the effect of pleural effusion drainage on pulmonary functions is similar both in spontaneously breathing patients and in patients with mechanical ventilation. Wemmelund et al. [10] carried out an experimental study in which they generated right pleural effusion in half of the subjects and left pleural effusion in the remaining half. However, no statistically significant difference was determined about hemodynamic values, and also no statistically significant difference was determined between the right and left pleural effusions during hemodynamic evaluations in our study ($p > 0.05$).

Our study was limited to a postoperative 24 hour period. The reason for this was the concern for returning the patients with a limited life expectancy to their daily lives as soon as possible. Also, since CVP, PCWB and PAP pressure were measured via Swan Ganz catheter and the same pressure monitor, the measurements were made during the early postoperative period to attain standardization.

In this study, we preferred an objective evaluation of the hemodynamic parameters that cause the symptoms instead of a subjective evaluation of these symptoms since the general status of our patients was poor.

Conclusion

As a result, drainage of massive pleural effusion improves hemodynamics and pulmonary functions by decreasing systolic pressure, PAP, CVP, PCWP, respiratory rates, and by increasing PO2 levels.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Funding: None

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

References

1. Egan AM, McPhillips D, Sarkar S, Breen DP. Malignant pleural effusion. Q J Med. 2013; 107.3:179–84.
2. Jimenez D, Diaz G, Gil D, Cicero A, Pérez-Rodríguez E, Sueiro A, et al. Etiology and prognostic significance of massive pleural effusions. Respiratory Medicine. 2005; 99.9 :1183-7.
3. Porcel JM, Vives M. Etiology, and pleural fluid characteristics of massive and massive Effusions. Chest. 2003;124.3:978–83.
4. Sakuraba M, Masuda K, Hebisawa A, Sagara Y, Komatsu H. Pleural Effusion Adenosine Deaminase (ADA) Level and Occult Tuberculous Pleurisy. Ann Thorac Cardiovasc Surg. 2009; 15: 294–6.
5. Chidambaram S, Sangareddi V, Ganesan G, Dhandapani VE, Ravi MS, Meenakshi K, et al. An echocardiographic assessment of cardiovascular hemodynamics in patients with massive pleural effusion. Indian Heart Journal. 2013;65.6: 666-70.
6. Hermansen JF, Juhl-Olsen P, Frederiksen CA, Christiansen LK, Hørlyck A, Solth E. Drainage of Massive Pleural Effusions Increases Left Ventricular Preload. Journal of Cardiothoracic and Vascular Anesthesia. 2014; 28.4:885-9.
7. Thomas R, Jenkins S, Eastwood PR, Lee YCG, Singh B. Physiology of breathlessness associated with pleural effusions. Current Opinion in Pulmonary Medicine Issue. 2015; 21.4: 338–45.
8. Ahmed SH, Ouzounian SP, DiRusso S, Sullivan T, Savino J, Del Guercio L. Hemodynamic and pulmonary changes after drainage of significant pleural effusions in critically ill, mechanically ventilated surgical patients. Journal of Trauma and Acute Care Surgery. 2004; 57:1184 –8.
9. Razazi K, Thille AW, Carteaux G, Beji O, Brun-Buisson C, Brochard L, et al. Effects of pleural effusion drainage on oxygenation, respiratory mechanics, and hemodynamics in mechanically ventilated patients. Annals of the American Thoracic Society. 2014; 11.7: 1018-24.
10. Wemmelund KB, Lie RH, Juhl-Olsen P, Frederiksen CA, Hermansen JF, Sloth E. Pleural effusion decreases left ventricular pre-load and causes haemodynamic compromise: an experimental porcine study. Acta Anaesthesiologica Scandinavica. 2012; 56.7: 833-9.

11. Traylor JJ, Chan K, Wong I, Roxas JN, Chandraratna PA. Massive pleural effusions producing signs of cardiac tamponade resolved by thoracentesis. Am J Cardiol. 2002; 89: 106–8.
12. Kaplan LM, Epstein SK, Schwartz SL, Cao QL, Pandian NG. Clinical, echocardiographic, and hemodynamic evidence of cardiac tamponade caused by massive pleural effusions. American journal of respiratory and critical care medicine. 1995; 151:904–8.
13. Venkatesh G, Tomlinson CW, O'Sullivan T, McKelvie RS. Right ventricular diastolic collapse without hemodynamic compromise in a patient with massive, bilateral pleural effusions. Journal of the American Society of Echocardiography. 1995; 8: 551-3.
14. Wang JS, Tseng CH. Changes in pulmonary mechanics and gas exchange after thoracentesis on patients with inversion of a hemidiaphragm secondary to massive pleural effusion. Chest. 1995; 107.6: 1610-4.
15. Brandstetter RD, Cohen RP. Hypoxemia after thoracentesis: a predictable and treatable condition. JAMA. 1979; 242.10: 1060-1.
16. Goligher EC, Leis JA, Fowler RA, Pinto R, Adhikari NK, Ferguson ND. Utility and safety of draining pleural effusions in mechanically ventilated patients: a systematic review and meta-analysis. Critical Care. 2011; 15.1: R46.
17. Doelken P, Abreu R, Sahn SA, Mayo PH. Effect of thoracentesis on respiratory mechanics and gas exchange in the patient receiving mechanical ventilation. Chest. 2006; 130:1354–61.
18. Karetzky MS, Kothari GA, Fourre JA, Khan AU. Effect of thoracentesis on arterial oxygen tension. Respiration 1978; 36:96–103.