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Possible causes of major pleural effusion in early period after cardiac surgery

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ABSTRACT

Objectives: This study aims to investigate possible causes of major pleural effusion in the early period after cardiac surgery.

Patients and methods: Between January 2012 and June 2012; 72 patients with major pleural effusion study group as confirmed by the chest X-ray two weeks after surgery were included. The control group consisted of 68 age- and sex-matched patients with minor or no effusion. Coronary artery bypass graft (CABG), valve replacement (VR), and CABG in combination with VR, the use of internal mammary artery, total perfusion time (TPT), and aortic cross-clamp time (ACCT) were compared between the two groups. The use of antiaggregants, anticoagulants, and diuretics was analyzed.

Results: The development of pleural effusion was found higher in CABG and CABG in combination with VR patients than only VR patients (p=0.007). Among CABG patients, the development rate of pleural effusion was higher in patients with a mammary artery than those with a non-mammary artery (p=0.043). In study group, TPT (p=0.007) and ACCT (p=0.042) were higher than those without pleural effusion. Logistic regression analysis showed that CABG was responsible for the development of major pleural effusion.

Conclusion: Based on our study results, CABG patients seems to be potential candidates for the development of major pleural effusion compared to VR patients possibly due to pleurotomy, atelectasis, impaired lymphatic drainage, and reduced sternal blood flow. Extended extracorporeal circulation time may also play a role in the development of pleural effusion through inflammatory responses.

Keywords: Cardiac surgery; chest-X-ray; pleural effusion.

It is estimated that about 66.000 cases of cardiac surgery are performed annually in Turkey.[1] After coronary artery bypass grafting (CABG), pleural effusion develops in 41 to 87% of patients, as confirmed by chest X-ray.[2-5]

Pleural effusions are usually minor and unilateral, asymptomatic, and resolve spontaneously or by conservative treatment.[6,7] Pleural effusion may lead to prolonged hospital stay and patient discomfort. It may also cause complications such as empyema and atelectasis. Despite of being an important issue, the reasons of pleural effusion which requires a therapeutic intervention after cardiac surgery still cannot be fully explained.

In this study, we aimed to investigate possible causes of major pleural effusion in the early period after cardiac surgery.

PATIENTS AND METHODS

Between January 2012 and July 2012, 74 patients (study group) who had major pleural effusion in the control chest X-ray two weeks after surgery were included. Major pleural effusion was defined as pleural effusion covering >25% of hemithorax in the control chest X-ray. The control group consisted of age- and sex-matched 68 patients with minor or no pleural effusion. The majority of the patients in control group were symptomatic and required intervention. Patients who were reoperated for bleeding revision, those who underwent complex cardiac surgery other than CABG and valve replacement (VR), and those who had an ejection fraction less than 35% were excluded. Six of 74 patients were excluded from the study group and, therefore, a total of 68 patients were included in the study.

Age, sex, diabetes mellitus (DM), hypertension (HT), chronic obstructive pulmonary disease (COPD), smoking history, ejection fraction (EF),

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Table 1
Pleural effusion levels of both groups

	Chest X-ray finding	Description
Control group	0	Effusion covering only costophrenic angle
	1	Effusion covering <25% of hemithorax
Study group	2	Effusion covering 25 to 50% of hemithorax
	3	Effusion covering 50 to 75% of hemithorax
	4	Effusion covering >75% of hemithorax

platelet count, urea and creatinine values, and whether or not emergency surgery were compared statistically between two groups. Also, CABG, VR and CABG with VR patients were compared in terms of development of pleural effusion. During CABG, patients with left internal mammary artery (LIMA), LIMA and right internal mammary artery (RIMA) being used and without mammary artery being used were also compared. The degree of hypothermia, total perfusion time (TPT) and aortic cross-clamp time (ACCT) were compared in terms of the impact on pleural effusion. The use of furosemide, aldactazide, furosemide with aldactazide and without use of diuretics during postoperative period was analyzed. Furthermore, patients who used only acetyl salicylic acid (ASA), ASA with clopidogrel, and ASA with warfarin postoperatively were studied. Logistic regression analysis was performed to analyze the operation, mammary artery use, TPT and ACCT.

Statistical analysis was performed using the NCSS 2007 for Windows statistical software program (Number Cruncher Statistical System, Kaysville, Utah, USA). Standard descriptive statistical calculations were made (mean and standard deviation) and unpaired t test was used to compare normally distributed data. The Mann-Whitney U test was performed to analyze abnormally distributed data. The chi square test was performed to evaluate the qualitative data. Significant univariate clinical variables were included in a multivariate logistic regression model which predicted the increased risk of effusion. A p value of <0.05 was considered statistically significant.

RESULTS

The mean age was 57.4±11 and M/F ratio was 59/13 in control group. In study group mean age was 60.1±11.3 and M/F ratio 48/20. The baseline characteristics of both groups in terms of pleural effusion levels are shown in Table 1.

There were 63 CABG patients, eight VR patients, and one CABG with VR patients in control group, while there were 52 CABG patients, five VR patients, and 11 CABG with VR patients in study group. The development of pleural effusion was significantly higher in CABG and CABG with VR patients compared to VR patients alone (p=0.007) (Table 2). During CABG, the number of patients who used LIMA, LIMA with RIMA and non-used both of them (only saphenous vein used) were 52, 2, and 18, respectively in the control group. These numbers were 60, 0, and 8, respectively in study group. The development rate of pleural effusion was significantly higher in LIMA and LIMA with RIMA used patients, than saphenous vein used patients alone (p=0.043) (Table 3).

In addition, TPT and ACCT in the control group were 85±27.5 min, and 47.8±23.6 min respectively,

Table 2
Comparison the groups in terms of operations

Operation	Control group		Study group		
	n	%	n	%	p
Coronary artery bypass graft	63	87.50	52	76.47	0.007†
Valve replacement	8	11.11	5	7.35	
Coronary artery bypass graft + valve replacement	1	1.39	11	16.18	

†: Statistically significant.

Table 3
Comparison of the groups in terms of grafts used

Greft	Control group		Study group		
	n	%	n	%	*p*
Only saphenous vein	18	25.00	8	11.76	0.043†
Left internal mammary artery (+)	52	72.22	60	88.24	
Left internal mammary artery + right internal mammary artery	2	2.78	0	0	

†: Statistically significant.

Table 4
Comparison of the groups in terms of peroperative variables

	Control group	Study group	
	Mean±SD	Mean±SD	*p*
Hypothermia °C	30.5±1.33	30.47±1.78	0.911
Total perfusion time (min)	84.97±27.45	101.53±42.76	0.007†
Aortic cross-clamp time (min)	47.76±23.61	57.91±34.26	0.042†

SD: Standard deviation; †: Statistically significant

while in the study group, these values were 101.5±42.8 min and 57.9±34.3 min, respectively. Also, TPT (p=0.007) and ACCT (p=0.042) were significantly lower in the control group, compared to the study group (Table 4). Although these parameters were found to cause the development of pleural effusion, there were no effects on the development of pleural effusion in the multivariate analysis in terms of TPT and ACCT.

There was no statistically significant difference between the two groups in terms of age, sex, DM, HT, COPD, smoking history, whether or not the emergency operation, EF, platelet count, urea and creatinine values (Table 5).

The mean hypothermia during surgery in the control group was 30.5±1.3 °C, while it was 30.5±1.8 °C in the study group. There was no statistically significant

Table 5
Comparison of the groups in terms of demographic characteristics

Demographic characteristics	Control group			Study group			
	n	%	Mean±SD	n	%	Mean±SD	*p*
Age			57.4±11.0			60.9±11.3	0.064
Gender							
Male	59			13			
Female	48			20			0.167
Body mass index	28.5	27.9					0.460
Diabetes mellitus	20	27.78		24	35.29		0.338
Hypertension	51	75.00		51	70.83		0.58
Chronic obstructive pulmonary disease	12	16.67		15	22.06		0.419
Smoking (pocket/year)			21.4±18.4			19.9±19.6	0.652
Ejection fraction (%)			53.7±9.4			52.6±9.7	0.516
Platelet count (n/mm^3)			265.9±113.4			267.2±79.9	0.938
Urea (mg/dL)			18.0±9.6			18.9±9.43	0.566
Creatinin (mg/dL)			1.1±0.8			1.0±0.3	0.399
Emergency	6	8.33		9	13.24		0.349

SD: Standard deviation.

Table 6
Comparison of the groups in terms of postoperative antiaggregant/anticoagulant use

Postoperative antiaggregant/anticoagulant use	Control group		Study group		
	n	%	n	%	*p*
Acetyl salicylic acid	51	70.83	40	58.82	0.189
Acetyl salicylic acid + warfarin	9	12.50	13	19.12	0.299
Acetyl salicylic acid + clopidogrel	12	16.67	15	22.06	0.401

difference in hypothermia between the two groups (Table 4).

When comparing the two groups in terms of using ASA, ASA with warfarin and ASA with clopidogrel which were prescribed during discharging, there was no statistically significant difference between the groups (Table 6). Also, there was no significant difference in the use of furosemide, aldactazide, and furosemide with aldactazide between the groups (Table 7). Also, CABG was found to be the main factor for the development of major pleural effusion (Table 8).

DISCUSSION

Most of the effusions after CABG are minor, left-sided, and regress spontaneously. A small part of pleural effusions become permanent. Patients with pleural effusion had significantly longer ICU and hospital stays and experienced higher rates of complications than those without pleural effusion.[8]

Yıldırım et al.[9] showed that four of 62 patients (6.45%) with pleural effusion after cardiac surgery required toracotomy and decortication operation for pleural thickening. It suggests the importance of pleural effusion after cardiac surgery. In addition, Labidi et al.[8] demonstrated that peripheral arterial disease, atrial fibrillation, heart failure, and some anticoagulants may lead to symptomatic pleural effusion. The authors reported that older patients and high serum creatinine level patients had symptomatic pleural effusion. In our study, however, we found no significant difference in these preoperative parameters between the patients with major pleural effusion and

Table 7
Comparison of the groups in terms of diuretics

Diuretic	Control group		Study group		
	n	%	n	%	*p*
No diuretic	64	88.89	60	88.24	0.229
Furosemide	1	1.39	5	7.35	0.209
Aldactazide	7	9.72	2	2.94	0.241
Furosemide + aldactazide	0	0	1	1.47	0.981

Table 8
Logistic regression analysis of variables

Surgical data	Odds ratio	95% CI		*p*
		Lower	Upper	
Coronary artery bypass graft	0.03	0.002	0.39	0.027†
Valve replacement	0.21	0.017	2.66	0.229
Coronary artery bypass graft and valve replacement	0.15	0.00	0.97	0.008†
Left internal mammary artery usage	0	0	0.001	0.297
Left internal mammary artery + right internal mammary artery usage	0	0	0.001	0.998
Total perfusion time (min)	1.02	0.99	1.05	0.998
Aortic cross-clamp time (min)	0.99	0.95	1.02	0.116

CI: confidence interval.

those with minor or no pleural effusion. Besides, elective surgeries were significantly less associated with pleural effusions in Labidi's[8] study. However, we were unable to detect any difference in the development of pleural effusion between elective and urgent patients. The differences between these two studies can be explained by the fact that Labidi's study included only symptomatic or patients requiring an intervention, while our patients with minimal pleural effusion were included in the same group with non-effusion patients. Therefore, some variables of the patients with pleural effusion might have changed the results in favor of the group with effusion.

In another study, Light et al.[2] performed chest radiographs 28 days after surgery and showed a significantly higher rate of pleural effusions among patients undergoing either CABG surgery (63%) or combined CABG and valve surgery (62%) than those undergoing VR alone (45%). Labidi et al.[8] concluded that VR was more strongly associated with postoperative pleural effusions than CABG. However, we found that there were significantly higher rates of pleural effusion in CABG and CABG with VR patients than VR patients alone, similar to the Light's study findings (p=0.007). Unlike our study results, Light's study included only pure non-effusion patients in the control group. However, it did not change the results. In addition, pleural effusion values in CABG patients may be contributed by TPT length and pleural trauma. There were five VR patients in the study group. This may be caused by an inflammatory response triggered by cardiopulmonary bypass (CPB). Moreover, in CABG patients whose LIMA or LIMA with RIMA was used had a high pleural effusion rate than those whose only saphenous vein was used in our study. In addition, Hurlbut et al.[5] reported an incidence of left pleural effusion of 84% for on the sixth postoperative day following internal mammary artery (IMA) grafting compared with an incidence of 47% after saphenous vein grafting. Similarly, Yıldırım et al.[9] obtained a strong correlation between pleural effusion and IMA harvesting. There is a number of studies reporting similar results in the literature.[5,10] Christakis et al.[11] revealed that there was no difference in the development of pleural effusion between the use of mammary and saphenous vein groups. The causes of pleural effusion may be atelectasis, impaired lymphatic drainage, decreased sternal blood supply, and pleurotomy in mammary artery harvested patients.[6]

Furthermore, Payne et al.[12] found no differences related to TPT and ACCT between pleural effusion and without pleural effusion. Similarly, we found that TPT and ACCT had statistically no effect on the development of pleural effusion, based on the logistic regression analysis. Wynne and Botti[13] showed that use of CPB had clear negative consequences on postoperative pulmonary function. They compared CPB patients with other types of major surgery in terms of pulmonary function and were found more frequent lung injury and delayed pulmonary recovery in CPB patients. Extracorporeal bypass circuit time may also contribute to the increased complication by causing an inflammatory response. Therefore, the pulmonary dysfunction is thought to be due to effects of an acute systemic and pulmonary inflammatory response commonly referred to as "pump lung" [14] or "post-pump syndrome."[15]

Additionally, there was no difference in the use of several medications including ASA, ASA with clopidogrel and ASA with warfarin between patients with major pleural effusion and minor or no pleural effusion who were prescribed with variable medication as such. As a result, antiaggregants and anticoagulants were shown to have no effect on developing pleural effusion. On the other hand, pleural effusion was not found to be less than the patients prescribed with several diuretics such as furosemide, aldactazide, furosemide with aldactazide. Therefore, diuretic treatment which was prescribed to a discharged patient had no effect on the prevention of pleural effusion.

In conclusion, clinicians should be more alert to the development of pleural effusion in CABG patients than VR patients, as CABG and CABG with VR patients are more potential candidate for the development of major pleural effusion than VR patients alone. Major pleural effusion plays also an important role for mortality and morbidity, as they are caused by complications such as atelectasis and empyema in CABG patients. Prevention of some complications related to major pleural effusion after cardiac surgery should be considered in some variables such as mammary artery harvesting.

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