Constrictive Physiology After Coronary Artery Bypass Grafting: Its incidence and clinical outcome

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Constrictive Physiology After Coronary Artery Bypass Grafting: Its incidence and clinical outcome

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<ABSTRACT>

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BACKGROUND: Constrictive physiology (CP) is not an uncommon cause of heart failure after coronary artery bypass grafting (CABG) although it is frequently unrecognized. The objective of this study is to investigate the incidence and clinical course of CP after CABG. **METHODS:** From January 2004 to September 2006, 454 patients underwent isolated CABG with comprehensive pre- and post-operative transthoracic echocardiography (TTE). Their medical records, TTE data and post-operative multi-detector computed tomography (MDCT) findings were reviewed. Diagnosis of CP was based on post-operative TTE findings; abnormal ventricular septal motion, presence of a dilated inferior vena cava, typical respiratory variations in mitral and hepatic venous flow and preserved or exaggerated early diastolic mitral annular velocity. **RESULTS:** Seventy-eight (17%) of 454 patients showed CP after CABG. Their pre-operative baseline characteristics were not significantly different from those

of patients without CP. However, on post-operative TTE, left ventricular ejection fraction was significantly higher (59±12 vs. 55±13%; p = 0.030) and the frequency of regional wall motion abnormality was significantly lower (36% vs. 51%; p = 0.019) in patients with CP. In multivariate analysis, the independent risk factors for CP after CABG were post-operative pericardial effusion, low voltage QRS and no post-operative non-steroidal anti-inflammatory drugs use. During clinical follow-up, composite clinical event rate of patients with CP was not significantly higher compared to that of patients without CP (5% vs. 3%; p = 0.240). Among 61 patients (78%) who had follow-up TTE, 50 patients (82%) showed resolution of CP at mean 525±360 (18-1,296) days after CABG. The other 11 patients (18%) showed residual CP until mean 650±531 (10-1,316) days after CABG. Only 1 (9%) of 11 patients with residual CP re-admitted for heart failure due to CP but improved medically. None of patients with CP showed pericardial adhesion, fibrosis or calcification on post-operative MDCT. **CONCLUSIONS:** CP is commonly observed after CABG. The clinical course of CP after CABG is relatively benign and transient. It is resolved spontaneously or with conservative management in most patients and aggravation to permanent CP is rare. Therefore, for the management of CP after CABG, conservative follow-up without surgical intervention may be warranted.

Key words: constrictive physiology, coronary artery bypass grafting.

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I. INTRODUCTION

Classic constrictive pericarditis is a disease characterized by the encasement of the heart by a rigid non-pliable pericardium with thickening, adhesion, fibrosis, and often calcification. These cause impaired diastolic ventricular filling leading to heart failure, manifested as chronic insidious signs and symptoms of systemic venous congestion [1]. In the past 20 years, several reports have highlighted changes in the spectrum of constrictive pericarditis characterized chiefly by a declining incidence of tuberculous pericarditis and an increase in the frequency of cases resulting from cardiac surgery [2]. Although the prevalence of classic constrictive pericarditis after CABG is only 0.2-0.3% [3], cardiac surgery has emerged as an important cause of constrictive pericarditis, representing up to 11-37% of cases in some study [4-6]. Classic constrictive pericarditis is a well-recognized, but rare, complication of CABG these days. However, after comprehensive echocardiography has been widely available, it helped us to detect

CP after CABG more conveniently [7-12] and CP is not an uncommon cause of heart failure after CABG although it is frequently unrecognized. Few data are available regarding the incidence and clinical course of CP after CABG or its relation with classic constrictive pericarditis. Therefore, we investigated the incidence and clinical outcome of CP after CABG.

II. MATERIALS AND METHODS

1. Study population. We reviewed the Yonsei Cardiovascular Center cardiac surgery database to identify all patients who underwent isolated CABG from January 2004 to September 2006. A total of 573 patients underwent isolated CABG. Patients were excluded from the study if they had not undergone comprehensive pre- or post-operative TTE. Ultimately, a total of 454 patients were enrolled. Retrospective analysis of their medical records, TTE data and post-operative MDCT findings were done. The patients were divided into 2 groups according to post-operative TTE findings: CP group, No-CP group.

2. Echocardiographic diagnosis of constrictive physiology. Standard TTE 2-dimensional and Doppler measurements were obtained. Mitral and hepatic venous flow were obtained while simultaneously recording respiration. For the mitral flow, early transmitral flow velocity (E) and late transmitral flow velocity (A) were obtained during inspiration and expiration. Pulsed wave Doppler tissue imaging was performed at the septal side of mitral annulus. Early diastolic mitral annular velocity (E') and late diastolic mitral annular velocity (A') were also obtained. A diagnosis of CP after CABG was made on the basis of post-operative TTE findings; abnormal ventricular septal motion [7], presence of a dilated inferior vena cava [8], typical respiratory variations in

mitral and hepatic venous flow [9, 10], and preserved or exaggerated early diastolic mitral annular velocity [11, 12].

3. Statistical analysis. Continuous variables were expressed as mean±standard deviation and were compared using an unpaired Student *t*-test. Categorical variables were expressed as counts and percentages and the χ^2 or Fisher's exact test was used for comparison. Multivariate logistic regression analysis was performed to identify the independent risk factors for the development of CP after CABG. Clinical event rates of the 2 groups were calculated by the Kaplan-Meier method and compared using the Log-rank test. All statistical tests were two-tailed. Statistical analyses were performed using SPSS version 12.0 (SPSS Inc., Chicago, Illinois). A p value of <0.05 was considered to be statistically significant.

III. RESULTS

1. Pre-operative baseline characteristics. Seventy-eight (17%) of 454 patients who underwent isolated CABG showed CP on post-operative TTE. Pre-operative baseline characteristics of the CP group and No-CP group are shown in Table 1. There were no significant differences in demography, past history and pre-operative TTE findings between the 2 group except E/E', which was lower in CP group (11.4±4.1 in CP group vs. 12.8±5.6 in No-CP group; p = 0.010). Angina was the most common indication for CABG in both groups (74% in CP group vs. 72% in No-CP group; p = 0.703).

2. Operative characteristics. No significant differences were found in the frequency of cardiopulmonary bypass pump use and graft number between both groups. Post-operative fall in hemoglobin and the frequency of post-operative anticoagulation (mainly due to underlying or newly-developed atrial fibrillation) or transfusion were

also similar (Table 2).

Table 1. Pre-operative baseline characteristics

	CP (n=78)	no CP (n=376)	p value
Age(years)	62.2±8.3	62.7±8.2	0.570
Male	59(76%)	265(71%)	0.359
BSA(m^2)	1.73±0.14	1.72±0.16	0.600
Hypertension	44(56%)	242(64%)	0.186
Diabetes mellitus	29(37%)	154(41%)	0.536
Smoking	42(54%)	164(44%)	0.099
Dyslipidemia	36(46%)	144(38%)	0.197
CRF	8(10%)	41(11%)	0.867
Past history			
Tuberculosis	2(3%)	11(3%)	1.000
MI	9(12%)	54(14%)	0.512
UA	18(23%)	57(15%)	0.087
CHF	1(1%)	13(4%)	0.481
CVA	1(1%)	27(7%)	0.066
Cardiac surgery	2(3%)	10(3%)	1.000
CABG indication			
Angina	58(74%)	270(72%)	
MI	18(23%)	86(23%)	
CHF	2(3%)	20(5%)	0.703
pre-operative TTE			
LVESD(mm)	35±5	36±8	0.071
LVEDD(mm)	51±5	51±6	0.365
LAVI(mL/m^2)	24±8	26±9	0.079
EF(%)	59±12	56±15	0.118
RWMA	32(41%)	196(52%)	0.074
LVH	10(13%)	49(13)	0.960
Transmitral E velocity(m/s)	0.61±0.17	0.62±0.17	0.612
Transmitral A velocity(m/s)	0.86±0.19	0.75±0.18	0.457
E/A ratio	0.87±0.34	0.86±0.37	0.943
Deceleration time(msec)	210±47	210±46	0.987
Mitral annular E' velocity(cm/s)	5.7±1.7	5.3±1.9	0.166
Mitral annular A' velocity(cm/s)	8.6±1.9	8.2±2.1	0.225
E/E'	**11.4±4.1**	**12.8±5.6**	**0.010**
RVSP(mmHg)	25±6	26±7	0.445
Pericardial effusion	1(1%)	12(3%)	0.707

CP:constrictive physiology, BSA:body surface area, CRF:chronic renal failure, MI:myocardial infarction, UA:unstable angina, CHF:congestive heart failure, CVA:cerebrovascular accident, CABG:coronary artery bypss grafting, TTE:transthoracic echocardiography, LVESD:left ventricular end systolic dimension, LVEDD:left ventricular end diastolic dimension, LAVI:left atrial volume index, EF:ejection fraction, RWMA:regional wall motion abnormality, LVH:left ventricular hypertrophy, RVSP:right ventricular systolic pressure.

Table 2. Operative characteristics

	CP (n=78)	No-CP (n=376)	p value
Off pump	55(70%)	300(80%)	
On pump	23(30%)	76(20%)	0.071
Graft number	3.2±0.8	3.3±0.9	0.273
Post-operative fall in hemoglobin*	3.5±1.6	3.3±1.7	0.355
Post-operative anticoagulation†	1(1%)	11(3%)	0.700
Post-operative transfusion‡	26(33%)	111(30%)	0.504

CP = constrictive physiology.

*The difference between the pre-operative and post-operative hemoglobin level.

†The number of patients who received warfarin or intravenous heparinization in post-operative period.

‡The number of patients who received more than three units of packed red blood cell in post-operative period.

3. Post-operative TTE and MDCT findings. Post-operative TTE was conducted at mean 14±27th day after CABG in CP group and at mean 13±36th day after CABG in No-CP group (p = 0.704). As shown in Table 3, Seventy-eight (17%) of 454 patients showed CP. Left ventricular ejection fraction (LVEF), E velocity, E/A ratio, E' velocity and the frequency of pericardial effusion (PE) were significantly higher in CP group. However, the frequency of regional wall motion abnormality (RWMA), A velocity and deceleration time were significantly lower in CP group. MDCT to evaluate the graft patency was conducted within post-operative 30th day (82% in CP group vs. 74% in No-CP group; p = 0.142). No significant difference was found in the graft patency rate between the 2 groups (89% in CP group vs. 87% in No-CP group; p = 0.717). Additional information about pericardium, mediastinum and pleura was obtained by MDCT. There were no significant differences in the frequency of pericardial thickening, PE, pericardial or mediastinal hematoma, mediastinal fluid collection and pleural effusion between the 2 groups. None of patients in both groups showed pericardial adhesion, fibrosis or calcification.

Table 3. Post-operative TTE and MDCT findings

	CP (n=78)	no CP (n=376)	p value
Post-operative TTE			
LVESD(mm)	34±6	35±7	0.456
LVEDD(mm)	49±5	49±6	0.722
LAVI(mL/m2)	25±8	25±8	0.964
EF(%)	**59±12**	**55±13**	**0.030**
RWMA	**28(36%)**	**190(51%)**	**0.019**
LVH	8(10%)	42(11%)	0.809
Transmitral E velocity(m/s)	**0.75±0.17**	**0.67±0.19**	**0.001**
Transmitral A velocity(m/s)	**0.62±0.13**	**0.69±0.17**	**0.000**
E/A ratio	**1.26±0.42**	**1.01±0.36**	**0.000**
Deceleration time(msec)	**177±34**	**195±41**	**0.000**
Mitral annular E' velocity(cm/s)	**6.7±1.8**	**6.0±2.0**	**0.012**
Mitral annular A' velocity(cm/s)	8.5±2.3	8.7±2.4	0.727
E/E'	12.4±5.7	12.2±5.0	0.810
RVSP(mmHg)	27±6	26±6	0.209
Pericardial effusion	**30(39%)**	**71(19%)**	**0.000**
Post-operative MDCT			
The number of patients*	64(82%)	279(74%)	0.142
Date of exam	post-operative 8±3th day	post-operative 8±4th day	0.620
Patent graft	57/64(89%)	243/279(87%)	0.717
Pericardial thickening	3/64(5%)	4/279 (1%)	0.124
Pericardial adhesion	0/64(0%)	0/279 (0%)	
Pericardial fibrosis	0/64 (0%)	0/279 (0%)	
Pericardial calcification	0/64 (0%)	0/279 (0%)	
Pericardial effusion	7/64 (11%)	19/279 (7%)	0.293
Pericardial or mediastinal hematoma	6/64 (9%)	20/279 (7%)	0.600
Mediastinal fluid collection	9/64 (14%)	34/279 (12%)	0.683
Pleural effusion	28/64 (44%)	123/279 (44%)	0.961

TTE:transthoracic echocardiography, MDCT:multi-detector computed tomography, CP:constrictive physiology, LVESD:left ventricular end systolic dimension, LVEDD:left ventricular end diastolic dimension, LAVI:left atrial volume index, EF:ejection fraction, RWMA:regional wall motion abnormality, LVH:left ventricular hypertrophy, RVSP:right ventricular systolic pressure
*The number of patients who were evaluated by multi-detector computed tomography within post-operative 30th day.

4. Clinical characteristics in post-operative period. Various clinical characteristics in post-operative period are given in Table 4. White blood cell count was lower in CP group (8040±2805/mm^3 in CP group vs. 8673±2428/mm^3 in No-CP group; p = 0.042), but hemoglobin, albumin and creatine kinase MB isoenzyme levels were similar between both groups. The incidence of low voltage QRS on post-operative

electrocardiogram was significantly higher in CP group (17% in CP group vs. 7% in No-CP group; p = 0.010). The frequency of post-operative medications including beta blockers, angiotensin converting enzyme inhibitors, diuretics and digoxin was not significantly different in the 2 groups although non-steroidal anti-inflammatory drugs (NSAIDs) were prescribed less often in CP group (3% in CP group vs. 10% in No-CP group; p = 0.042). The most common clinical symptom in post-operative period was dyspnea (13% in CP group vs. 8% in No-CP group; p = 0.170). More patients in CP group developed chest pain, dyspnea, edema and fever after CABG. However, the differences between both groups were not statistically significant.

Table 4. Clinical characteristics in post-operative period

	CP (n=78)	No-CP (n=376)	p value
Laboratory results			
WBC(/mm^3)	**8040±2805**	**8673±2428**	**0.042**
Hemoglobin(g/dL)	9.4±1.1	9.5±1.3	0.433
Albumin(g/dL)	3.5±0.3	3.5±0.4	0.188
CKMB(ng/mL)	25.2±36.4	28.1±51.5	0.641
Electrocardiographic findings			
Atrial fibrillation	3(4%)	19(5%)	1.000
Low voltage QRS	**13(17%)**	**28(7%)**	**0.010**
Medications			
Beta blockers	59(76%)	263(70%)	0.314
ACE inhibitors	36(46%)	188(50%)	0.536
Diuretics	30(39%)	160(43%)	0.505
Digoxin	2(3%)	17(5%)	0.755
NSAIDs	**2(3%)**	**36(10%)**	**0.042**
Clinical symptoms			
Chest pain	3(4%)	4(1%)	0.102
Dyspnea	10(13%)	30(8%)	0.170
Edema	3(4%)	4(1%)	0.102
Fever	1(1%)	3(1%)	0.531

CP = constrictive physiology; WBC = white blood cell; CKMB = creatine kinase MB isoenzyme;
ACE = angiotensin converting enzyme; NSAIDs = non-steroidal anti-inflammatory drugs.

5. Independent risk factors for post-operative constrictive physiology. Multivariate logistic regression analysis was performed to identify the independent risk factors for CP after CABG. Various clinical, operative, TTE data and MDCT findings were entered into the model. Post-operative PE, low voltage QRS and no post-operative NSAIDs use were the independent risk factors for CP after CABG (Table 5).

Table 5. Independent risk factors for post-operative constrictive physiology

	Adjusted OR	95% CI	p value
Age	0.99	0.96 - 1.02	0.643
Hypertension	0.84	0.49 - 1.44	0.530
Cardiopulmonary bypass	1.56	0.85 - 2.86	0.148
Pre-operative LVEF	0.99	0.95 - 1.02	0.502
Pre-operative RWMA	1.15	0.42 - 3.12	0.643
Pre-operative pericardial effusion	0.31	0.04 - 2.66	0.286
Post-operative LVEF	1.02	0.98 - 1.06	0.336
Post-operative RWMA	1.75	0.60 - 1.02	0.303
Post-operative pericardial effusion	**2.57**	**1.46 - 4.53**	**0.001**
Pericardial thickening on MDCT	2.13	0.49 - 9.36	0.315
Post-operative WBC count	1.00	1.00 - 1.00	0.140
Post-operative low voltage QRS	**2.23**	**1.00 - 4.97**	**0.050**
Post-operative NSAIDs	**0.17**	**0.03 - 0.80**	**0.026**

OR = odds ratio; CI = confidence interval; LVEF = left ventricular ejection fraction; RWMA = regional wall motion abnormality; MDCT = multi-detector computed tomography; WBC = white blood cell; NSAIDs = non-steroidal anti-inflammatory drugs.

6. Clinical events after hospital discharge. The incidence of clinical events (congestive heart failure, constrictive pericarditis, pericardial effusion and death due to cardiovascular causes) in the 2 groups were investigated. In CP group, only the clinical events before the resolution of CP on follow-up TTE were included. No significant differences were found in the incidence of clinical events (separately and compositely) except constrictive pericarditis, which was higher in CP group (Table 6). Composite clinical events-free survival curves are presented in Figure 1.

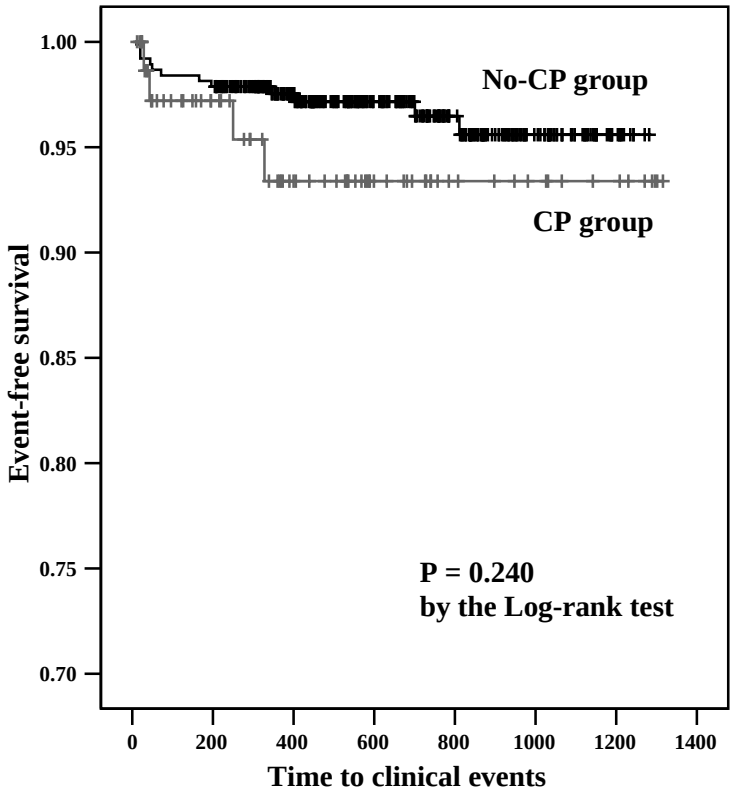
Table 6. Clinical follow-up after hospital discharge

	CP (n=78)	No-CP (n=376)	p value
F/U duration(days)	**515±381**	**623±292**	**0.020**
Congestive heart failure	2(3%)	8(2%)	0.630*
Constrictive pericarditis	**1(1%)**	**0(0%)**	**0.021***
pericardial effusion	1(1%)	1(0.3%)	0.166*
Death due to cardiovascular causes	0(0%)	3(1%)	0.460*
Composite clinical events	4(5%)	12(3%)	0.240*

CP = constrictive physiology; F/U = follow-up.

*By the Log-rank test.

Figure 1. Clinical events-free survival curves by the Kaplan-Meier method. CP = constrictive physiology.



7. Follow-up of constrictive physiology. A total of 61 patients (78%) in CP group underwent follow-up TTE. Among the patients who had follow-up TTE, 50 patients (82%) showed resolution of CP at mean 525±360 (18-1,296) days after CABG. The other 11 patients (18%) showed residual CP until the last follow-up TTE which was

conducted at mean 650±531 (10-1,316) days after CABG. Of them, 6 patients (55%) never developed clinical symptoms and 4 patients (36%) developed chest pain, dyspnea or edema that has resolved during clinical follow-up. Only 1 of 11 patients (9%) who showed residual CP re-admitted for heart failure due to constrictive pericarditis, but improved medically. At last follow-up, total 9 of 78 patients (12%) with CP had persistently shown mild chest pain or dyspnea. However, it was well-tolerated without further necessity for re-hospitalization (Figure 2).

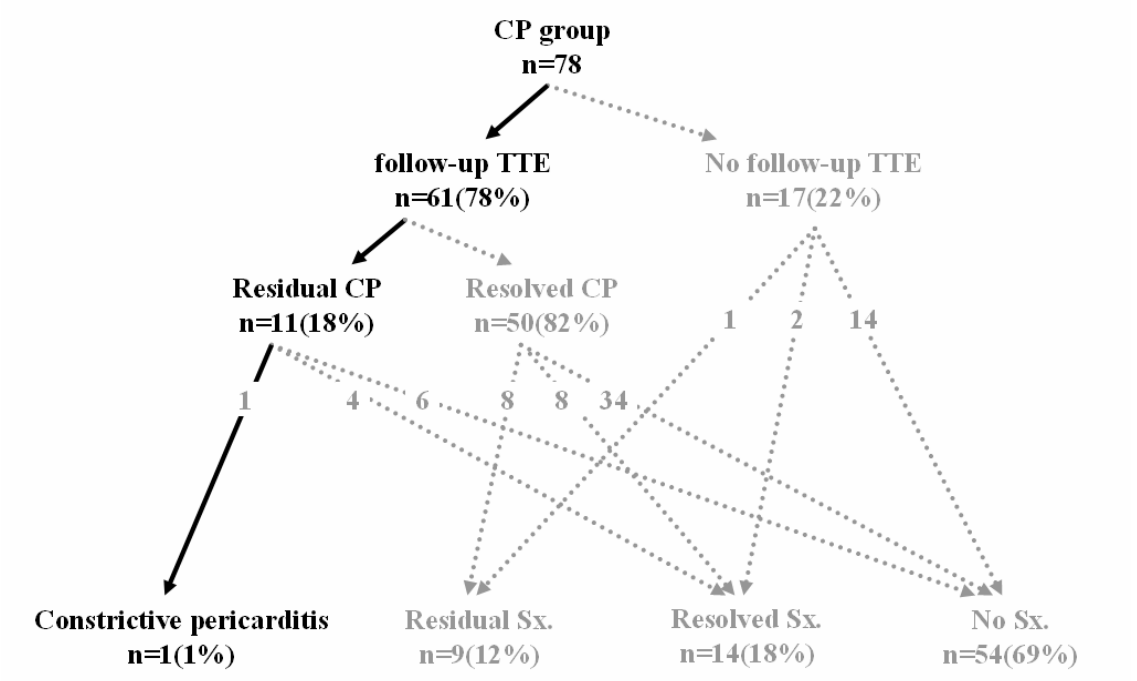


Figure 2. Follow-up of constrictive physiology. CP = constrictive physiology; TTE = transthoracic echocardiography; Sx. = clinical symptoms.

IV. DISCUSSION

Constrictive pericarditis, as a complication after cardiac surgery, was first reported in 1972 [13] and the prevalence of constrictive pericarditis after CABG is known to be 0.2-0.3% [3]. Traditionally, diagnosis of constrictive pericarditis was made by invasive cardiac catheterization or surgically confirmed pericardium with thickening, adhesion,

fibrosis or calcification. However, better insight into the mechanism of CP has allowed more reliable and convenient diagnostic criteria using comprehensive echocardiography with 2D, Doppler and tissue Doppler imaging [7-12]. Therefore, CP is not an uncommon condition after CABG despite preserved left ventricular systolic function although it is frequently unrecognized. To our best knowledge, no data are available regarding the incidence and clinical course of CP after CABG or its relation with classic constrictive pericarditis. Therefore, we investigated the incidence and clinical outcome of CP after CABG.

1. Incidence and risk factors of constrictive physiology. The results of our study shows that CP after CABG is relatively commonly observed (in 17% of patients after CABG) compared to classic constrictive pericarditis which is a well-recognized, but rare, complication occurring in 0.2-0.3% of patients after CABG. Independent risk factors for CP after CABG are post-operative PE, low voltage QRS and no post-operative NSAIDs use. Matsuyama K et al. suggested that post-operative PE with warfarin administration and normal LVEF is a significant risk factor for constrictive pericarditis after CABG [14]. The pathophysiology was explained by a theory that there is continuous violent friction between the pericardium and epicardium under normal LVEF and the blood in PE due to warfarin, which may eventually result in pericardial injury, inflammation and pericarditis. Similarly, in our study, post-operative PE and resultant low voltage QRS are the independent risk factors for CP after CABG. Also, post-operative LVEF is significantly higher in CP group (59±12% in CP group vs. 55±13% in No-CP group; p = 0.030), although it is not significant in multivariate analysis. Only 5 patients (6%) in CP group showed LVEF below 40%. Furthermore, the

frequency of post-operative RWMA is significantly lower in CP group (36% in CP group vs. 51% in No-CP group; p = 0.019). These findings may explain the role of continuous violent friction between the pericardium and epicardium in the pathogenesis of post-operative CP. In addition, our study shows that no post-operative NSAIDs use is another independent risk factor for CP after CABG. NSAIDs (high dose aspirin in most cases) are usually prescribed empirically by house staffs under suspicion of post-pericardiotomy syndrome when patients complain of heating sensation, malaise with suspicious diffuse ST segment elevation on electrocardiogram. It is possible that pericardial inflammation which had potential to aggravate to CP may have been suppressed by empirical post-operative NSAIDs in some patients. Post-pericardiotomy syndrome and CP may be thought of as having similar pathophysiology which is different in the extent; systemic vs. pericardial inflammation.

2. Clinical events during constrictive physiology. One patient re-admitted for post-operative constrictive pericarditis. The patient showed mild PE and normal LVEF without RWMA on post-operative TTE. He also showed pericardial thickening on post-operative MDCT. The patient took post-operative NSAIDs for a few days due to mild fever and malaise and was successfully discharged. About one month later, the patient re-admitted for post-operative constrictive pericarditis and improved medically with oral corticosteroids. Although only a single case has occurred, our study shows that the incidence of classic constrictive pericarditis is higher in patients with CP compared to patients without CP. However, composite clinical event rate (congestive heart failure, constrictive pericarditis, pericardial effusion and death due to cardiovascular causes) was similar in both groups. Therefore, CP after CABG is not an indicator of poor

prognosis.

3. Fate of constrictive physiology. In our study, most patients (82%) with CP showed resolution of CP on follow-up TTE which was done at mean 525±360 (18-1,296) days after CABG. The other patients (18%) showed residual CP until the last follow-up TTE which was conducted at mean 650±531 (10-1,316) days after CABG. Only 1 (9%) of them re-admitted for heart failure due to constrictive pericarditis, but improved medically. Until relative recently, the development of CP was presumed to be irreversible and pericardiectomy was treatment of choice. But, in recent years there have been reports describing a transient form of CP that resolves without surgical intervention. The development of CP and subsequent resolution with conservative treatment was first described by Sagrista-Sauleda et al. in 1987 in a group of 16 patients with idiopathic pericarditis [15]. The time to resolution of CP ranged from 7 days to 58 months (mean 9.4 month). Interestingly, one recent study identified that the most common cause of transient CP is cardiac surgery [16]. The pathophysiology of transient post-operative CP is unknown, although several possibilities can be suggested. Cardiac surgery is traumatic, with intra-operative irritation of the pericardium by physical manipulation of the heart and from the saline solution [14, 17-19]. Trauma to the pericardium in the course of cardiac surgery and continuous violent friction (previously mentioned) may possibly result in pericardial inflammation, edema and transiently thickened in-elastic pericardium. These seem to be major events. In our study, no patient with transient CP showed pericardial adhesion, fibrosis or calcification on post-operative MDCT which means irreversible organic change and high potential to aggravate to permanent CP. Only 3 patients in CP group showed pericardial thickening. However,

focal inflammation and fibrin deposit can be observed microscopically even in patients with normal pericardial thickness [20]. Our study suggests that patients with post-operative CP can be managed conservatively if the patients are clinically stable and do not show pericardial adhesion, fibrosis or calcification on post-operative MDCT.

4. Study limitations. This study has several important limitations including its retrospective design. The patients were not followed at uniform intervals. Follow-up echocardiogram and office visits were performed at the discretion of the treating physician. We do not know the exact time when CP has been resolved. Mean follow-up duration was shorter than 2 years and it is possible for some patients with residual CP to develop constrictive pericarditis in the future. Finally, the diagnosis of CP can be elusive, even after extensive investigation by the experienced sonographers and echocardiologists.

V. CONCLUSION

Our study demonstrates that CP after CABG is commonly observed. Post-operative PE, low voltage QRS and no post-operative NSAIDs use are the independent risk factors for CP after CABG. The clinical course of CP after CABG is relatively benign and transient. Most patients with CP are free of clinical symptoms and their incidence of clinical events is not higher compared to that of patients without CP. It is resolved spontaneously or with conservative management in most patients and aggravation to permanent CP is rare. Therefore, for the management of CP after CABG, conservative follow-up without surgical intervention may be warranted.

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< ABSTRACT(IN KOREAN)>

관상동맥 우회술 이후의 교착성 생리

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임의

관상동맥 우회술 후 교착성 생리는 수술 후 심부전의 드물지 않은 원인이지만 임상에서 흔히 간과되고 있고, 아직까지 이러한 환자들의 정확한 빈도나 임상경과에 대한 연구는 없는 실정이다. 따라서 본 연구에서는 관상동맥 우회술 후 발생할 수 있는 교착성 생리의 빈도와 임상경과에 대해 조사하였다. 2004년 1월부터 2006년 9월까지 연세 심장혈관센터에서 단독 관상동맥 우회술과 수술 전, 후의 종합적인 심초음파 검사를 받은 454명을 대상으로 하였으며 그들의 의무기록, 심초음파 결과 등을 분석하였다. 관상동맥 우회술 후 발생하는 교착성 생리의 진단은 수술 후 경흉부 심초음파 소견을 바탕으로 하였다. 454명 중, 총 78명 (17%)이 관상동맥 우회술 후 심초음파에서 교착성 생리를 보였다. 수술 후 교착성 생리를 보인 환자들의 수술 전 기본적 특성은 교착성 생리를 보이지 않는 환자들과 큰 차이가 없었다. 하지만, 수술 후 심초음파에서, 교착성 생리를 보인 환자들의 좌심실구출율은 교착성 생리를 보이지 않은 환자들보다 더 컸으며 (59%±12 대 55±13%, p = 0.030), 국소벽운동장애의 빈도는 더 낮은 것으로 나타났다 (36% vs. 51%, p = 0.019). 다변량 분석결과, 수술 후 교착성 생리의 독립적 위험 인자는 수술 후 심낭 삼출, 저전압 QRS와 수술 후 비스테로이드성

소염제를 사용하지 않는 경우였다. 퇴원 후 유의한 임상 사건의 발생빈도는, 양쪽 환자들간에 유의한 차이가 없었다. 추적 심초음파 검사를 받은 61명 (78%)의 환자 중, 50명 (82%)이 수술 후 평균 525±360 (18-1,296)일 후 시행한 추적 심초음파에서 교착성 생리가 소실되었고, 추적 심초음파에서 지속적으로 교착성 생리를 보인 환자 11명 중 단 1명 (9%)만이 교착성 심낭염으로 재입원하였지만 보전적 치료로 호전되었다. 결론적으로, 관상동맥 우회술 후 교착성 생리는 비교적 흔하게 생길 수 있지만, 임상경과는 양호하고 일시적인 경우가 많다. 대부분의 환자에서 자연적으로 또는 보존적 치료로 소실되며, 영구적인 교착성 심낭염으로 진행하는 경우는 드물다. 따라서 관상동맥 우회술 후 교착성 생리는 수술적 치료 없이 보존적 경과 관찰이 가능할 것이다.

핵심되는 말 : 교착성 생리, 관상동맥 우회술.