Incidence, predictors and clinical outcome of post-operative cardiac tamponade in patients undergoing heart valve surgery

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Incidence, predictors and clinical outcome of post-operative cardiac tamponade in patients undergoing heart valve surgery

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ABSTRACT

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Background

The risk and clinical implication of cardiac tamponade following heart valve surgery are poorly defined in modern era, although cardiac tamponade is potential lethal complication.

Aims

We sought to investigate the incidence, predictors and clinical outcome of cardiac tamponade after valvular heart surgery.

Methods

A total of 556 patients (271 male, mean age 58.5 ± 12.9 years) who underwent heart valve surgery in a single tertiary center between January 2010 and March 2012 were identified. Transthoracic echocardiography was conducted to evaluate cardiac function and hemodynamic changes including the amount of pericardial effusion (PE) and presence of cardiac tamponade in every patient about a week after surgery and repeated regularly after surgery. Patients with suspected pericardial hemorrhage were excluded.

Results

Twenty-four (4.3%) of the 556 patients developed cardiac tamponade in the post-operative period. Among them, 16 (66.7%) and 8 (33.3%) patients underwent percutaneous catheter drainage or pericardial window formation, respectively. The median time of pericardial drainage after surgery was 17

(IQR 13-30) days. There was no difference in age and sex between the group with cardiac tamponade and without. Infective endocarditis as the etiology of valve disease, mechanical valve replacement of aortic or mitral valve and any amount of PE on the first post-operative echocardiography were related with development of cardiac tamponade (all $p<0.05$). After multivariate adjustment, development of cardiac tamponade was associated with any amount of PE on the echocardiography performed at 5-day after surgery (OR 15.8; $p<0.001$) and mechanical valve replacement with marginal statistical significance (OR 2.5; $p=0.051$). Mean hospital days in patients with cardiac tamponade was longer than those without (34.9 vs. 13.5, $p=0.031$). Re-hospitalization rate of patients after surgery was higher in the group of cardiac tamponade. After resolution of PE after pericardial drainage, there was no echocardiographic recurrence of PE more than small amount during a median of 1044 (IQR 446-1311) days after surgery.

Summary / Conclusion

Cardiac tamponade occurrence after heart valve surgery is uncommon, but it prolongs hospital stay. Presence of any amount of PE at the post-operative 5-day echocardiography and mechanical valve replacement have an independent predictive value for cardiac tamponade. It has benign clinical course without recurrence after timely intervention.

Key words: Cardiac tamponade, pericardial effusion, echocardiography, valve, surgery

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I. INTRODUCTION

Pericardial effusion (PE) after cardiac surgery is a commonly encountered finding after cardiac surgery even in patients without evidence of post-operative pericardial hemorrhage. It is known to be related to an inflammatory process of the pericardium so called post-pericardiotomy syndrome (PPS).[1] Though it mostly shows benign course without clinically serious complications, it is able to progress to potentially lethal cardiac tamponade which is associated with increased morbidity and mortality.[2–5] A few studied demonstrated that post-operative PE or consequent constrictive physiology were prevalent in patients undergoing coronary artery bypass grafting (CABG).[6,7] However, there would be considerable variations in incidence of PE and consequent clinical course according to the types of surgery, underlying diseases or the use of anticoagulants in patients undergoing heart valve surgery. Therefore, we sought

to define the incidence, predictors and clinical outcome of cardiac tamponade after heart valve surgery.

II. MATERIALS AND METHODS

1. Study population

The study consisted of a retrospective analysis of 556 consecutive adult patients from January 2010 through March 2012 at a single tertiary referral center. All patients underwent open heart surgery because of valvular heart disease. Medical records as well as serial echocardiographic data following valvular heart surgery were reviewed. Patients were classified as the etiologies of valve disease, underlying valve function, types of surgery (repair or replacement), and concomitant surgery (aorta surgery, CABG, or MAZE operation for atrial fibrillation). All the patients in this study underwent mandatory anticoagulation as the guideline recommended except for those with serious bleeding complications.[8] The length of stay in hospital was calculated in each patient from valvular operation to discharge. The patients were divided into two groups according to the occurrence of cardiac tamponade in the post-operative period. And then, the patients with cardiac tamponade were further classified into two subgroups based on the timing of its occurrence: early cardiac tamponade versus delayed cardiac tamponade.

The laboratory findings including cardiac biomarkers, inflammatory markers,

and coagulation profiles were reviewed in every patient. The peak values of post-operative white blood cell count (WBC), creatine kinase (CK), CK-MB isoenzyme, C-reactive protein, activated partial thromboplastin time, and prothrombine time (PT), activated partial thromboplastin time (aPTT) were analyzed. This study was performed in accordance with the ethical guidelines of the 1975 Declaration of Helsinki.

2. Echocardiography and diagnosis of cardiac tamponade

All subjects underwent comprehensive transthoracic echocardiography using commercially available equipment before valvular heart surgery to assess cardiac function and severity of valvular dysfunction. Standard two-dimensional and Doppler measurements were performed per the recommendations of the American Society of Echocardiography guidelines.[9] The first follow-up echocardiography after heart valve surgery was performed on all patients at median 5 days after the surgery. After then, follow-up echocardiography was performed based on clinical decision. In general, if the patients were uneventful, the second follow-up echocardiography was performed at 6 months after the surgery, and then annually for the evaluation of post-operative cardiac remodeling and serial follow-up of the valve function.

PE was classified into three grades as follows: small, 10~14mm of loculated effusion or circumferential effusion <10mm; moderate, 15~19mm of loculated

effusion or 10~14mm of circumferential effusion; and large, any amount more than moderate effusion. This grade was determined during the diastolic cardiac phase.[10]

Cardiac tamponade was suspected in patients with otherwise unexplained tachycardia, hypotension or clinical signs including pulsus paradoxus, raised jugular venous pressure, and cardiomegaly. The diagnosis was supported by following findings on echocardiography; right or left atrial and ventricular collapse, inferior vena cava distension, and respiratory flow variation of mitral and tricuspid inflow velocities.[11] In terms of the timing of cardiac tamponade, early cardiac tamponade was defined if cardiac tamponade occurred earlier than 30 days after surgery. In contrast, delayed cardiac tamponade was defined if it occurred 30 days or later after surgery. To exclude pericardial hemorrhage related with bleeding complications immediately after the surgery, the patients who developed cardiac tamponade within 48 hours after surgery or continuous blood loss though pericardial tube were excluded in analysis.

3. Management of cardiac tamponade and clinical follow-ups

Urgent interventions were conducted for every patient with cardiac tamponade after the diagnosis. Selection of the pericardial drainage methods depends on the clinicians' decision. In general, surgical drainage was preferred in patients with loculated PE or PE with posterior-dominant location. Otherwise,

percutaneous catheter drainage under echocardiographic or fluoroscopic guidance was conducted.

Overall, the median follow-up duration was 1044 days. The median time of pericardial catheter drainage or surgical drainage following valvular surgery was evaluated. Readmission or emergency department visit was counted, only when requiring medical care from the department of cardiology or cardiovascular surgery after discharge. Recurrence of PE was defined when development of small or more amount of effusion was identified on echocardiography after drainage of PE during follow-up duration.

4. Statistical analysis

Results were expressed as mean ± SD or n (%), as appropriate. The means or percentages of baseline characteristics between patients with and without cardiac tamponade were compared using independent Student t-tests for continuous variables and Chi-square tests for categorical variables. Univariate and multivariate logistic regression analyses were performed for variables that were significantly different between groups with and without cardiac tamponade. All statistical analyses were performed with standard procedures (SPSS, version 21).

III. RESULTS

1. Patient characteristics

Baseline characteristics of the 556 study population (male 48.7%) are shown in Table 1. The mean age was 58.5 years. 41 patients (7.4%) had prior open heart surgery. 167 (30.0%), 333 (59.9%), and 44 (7.9%) patients had rheumatic valve disease, degenerative valve disease and infective endocarditis as the etiology of valvular dysfunction, respectively. Bicuspid aortic valve (BAV) was surgically identified in 86 patients (15.5%). 266 (47.9%) patents had their valve replaced with mechanical prosthesis. Concomitant aorta surgery, CABG and MAZE were performed in 58 (10.4%), 56 (10.1%) and 14 (2.5%) patients each. There was no difference in age, sex, a proportion of underlying valve function, or concomitant surgery between two groups based on occurrence of cardiac tamponade.

The prevalence of infective endocarditis as a reason for heart valve surgery was significantly higher in patients with cardiac tamponade than those without (20.8% vs 7.3%, respectively; p= 0.017). In terms of types of surgery, more patients in the group of cardiac tamponade received mechanical valve replacement compared with the other group (66.7% vs. 46.1%, respectively; p=0.044)

Table 1. Characteristics of study population

	All (n = 556)	Cardiac Tamponade	
		No (n= 532)	Yes (n =24)
Demographic characteristics			
Age, years ± SD	58.5 ± 12.9	58.7 ± 12.9	54.3 ± 12.8
Male gender, n (%)	271 (48.7)	256 (48.1)	15 (62.5)
Body mass index, kg/m^2 ± SD	23.2 ± 3.1	23.2 ± 3.2	23.2 ± 2.4
Prior history of valve operation, n (%)	41 (7.4)	41 (7.7)	0 (0)
Etiology of valvular disease			
Rheumatic valve disease, n (%)	167 (30.0)	160 (30.1)	7 (29.2)
Degenerative valve disease, n (%)	225 (40.5)	219 (41.2)	6 (25.0)
Secondary valve disease, n (%)	13 (2.3)	13 (2.4)	0 (0.0)
Prosthetic valve failure, n (%)	21 (3.8)	21 (3.9)	0 (0.0)
Infective endocarditis, n(%)	44 (7.9)	39 (7.3)	5 (20.8)*
Bicuspid aortic valve, n(%)	86 (15.5)	80 (15.0)	6 (20.8)
Other valve disease, n (%)	130 (23.4)	119 (22.4)	7 (20.8)
Underlying valve function			
Mitral regurgitation, n (%)	133 (23.9)	129 (24.2)	4 (16.7)
Mitral stenosis, n (%)	167 (30)	160 (30.1)	7 (29.2)
Aortic regurgitation, n (%)	67 (12.1)	64 (12)	3 (12.5)
Aortic stenosis, n (%)	130 (23.4)	125 (23.5)	5 (20.8)
Tricuspid regurgitation, n (%)	3 (0.5)	3 (0.6)	0 (0.0)
Types of surgery			
Replacement with mechanical prosthesis	261 (46.9)	245 (46.1)	16 (66.7)*
Aortic valve, n (%)	157 (28.2)	146 (27.4)	11 (45.8)
Mitral valve, n (%)	146 (26.3)	137 (25.8)	9 (37.5)
Tricuspid valve, n (%)	7 (1.3)	7 (1.3)	0 (0.0)
Double valves, n (%)	49 (8.8)	45 (8.5)	4 (16.7)
Replacement with bioprosthesis	159 (28.6)	155 (29.1)	4 (16.7)
Aortic valve, n (%)	123 (22.1)	120 (22.6)	3 (12.5)

Mitral valve, n (%)	52 (9.4)	51 (9.6)	1 (4.2)
Double valves, n (%)	16 (2.9)	16 (3.0)	0 (0.0)
Repair			
Aortic valve, n (%)	3 (0.5)	3 (0.6)	0 (0.0)
Mitral valve, n (%)	146 (26.3)	141 (26.5)	5 (20.8)
Concomitant surgery			
Aorta surgery, n (%)	58 (10.4)	55 (10.3)	3 (12.5)
CABG, n (%)	56 (10.1)	55 (10.3)	1 (4.2)
MAZE operation, n (%)	14 (2.5)	12 (2.3)	2 (8.3)

* for p value < 0.05
CABG, coronary artery bypass grafting

2. Echocardiographic and laboratory characteristics

Thirty-three patients (6%) among total study population showed PE at TTE which performed at median 5 days following valvular surgery (Table 2). 24 (4.3%) patients developed cardiac tamponade on serial echocardiography. 30% among patients with small PE at 5-day TTE and 55% patients with large PE at 5-day TTE developed cardiac tamponade which needed urgent procedure (Fig 1). The median time of pericardiocentesis or surgical pericardial drainage following valvular surgery was 17 (IQR 13-30) days. Readmission was identified in 121 patients (22%).

There is no difference between groups with and without cardiac tamponade in postoperative laboratory test including WBC count, CK-MB, CRP, aPTT and

PT level. Any effusion on the first postoperative echocardiography (OR 11.4; 95% CI 3.7-35.6) showed significant relationship with development of cardiac tamponade (p=0.001).

Table 2. Echocardiographic and laboratory characteristics

	All	Cardiac Tamponade	
	(n = 556)	No	Yes
		(n= 532)	(n =24)
Pericardial effusion			
No (< small), n (%)	523 (94.1)	509 (95.7)	14 (58.4)
Any amount (≥small), n (%)	33 (5.9)	23 (4.3)	10 (41.6)*
Small amount, n (%)	17 (3.1)	12 (2.3)	5 (20.8)*
Moderate amount, n (%)	7 (1.2)	7 (1.3)	0 (0.0)
Large amount, n (%)	9 (1.6)	4 (0.7)	5 (20.8)*
Timing of cardiac tamponade			
Early (<30 days), n (%)	17 (3.1)	0 (0)	17 (70.8)
Delayed (≥ 30days), n (%)	7 (1.3)	0 (0)	7 (29.2)
Postoperative laboratory characteristics			
WBC count, 10^9/mL	20.1 ± 7.3	20.2 ± 7.3	19.1 ± 7.2
CK-MB, IU/L	26.6 ± 22	26.2 ± 21.7	33.4 ± 26.9
CK, IU/L	621.5 ± 498.4	617.7 ± 499.6	692.9 ± 480.4
CRP, mg/L	90.8 ± 34	91.5 ± 33.9	78.5 ± 34.1
aPTT, sec	45.5 ± 15.7	45.5 ± 15.9	43.4 ± 13.6
PT, INR	1.83 ± 0.74	1.83 ± 0.75	1.71 ± 0.63

* for p value < 0.05

WBC, white blood cell; CK, creatine kinase; CRP, C-reactive protein; aPTT, activated partial thromboplastin time; PT, prothrombin time; INR, international normalized ratio

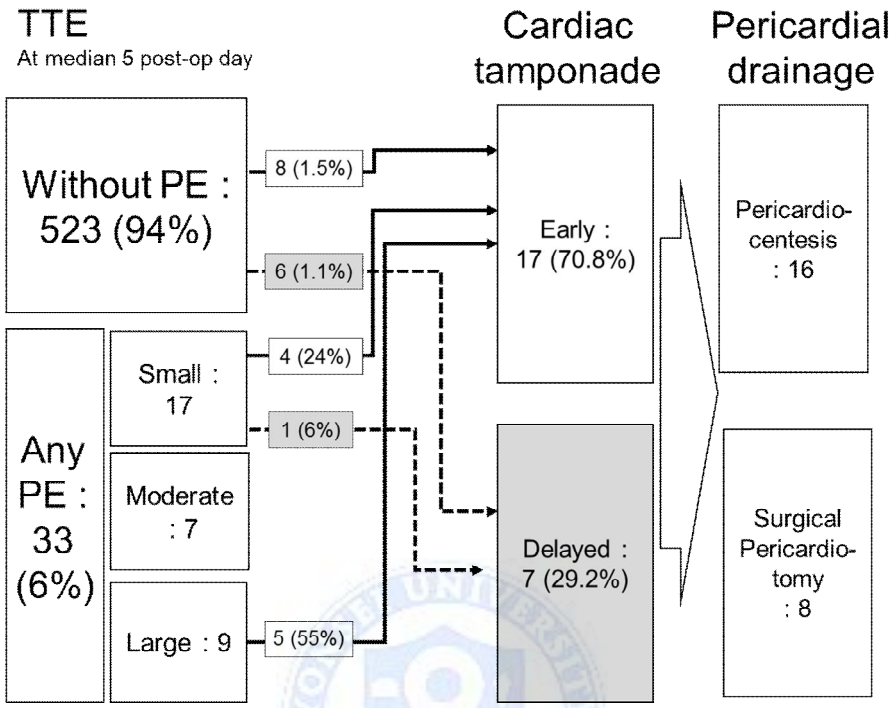

Figure 1. Incidence, grade and outcome of pericardial effusion.

PE, pericardial effusion.

3. Factors associated with cardiac tamponade

Infective endocarditis as the etiology of valve disease (OR 3.3; 95% CI 1.2-9.4) and mechanical valve replacement of aortic or mitral valve (OR 2.4; 95% CI 1.0-5.7) were related with development of cardiac tamponade (all $p<0.05$). Any effusion on the first post-operative echocardiography (OR 11.4; 95% CI 3.7-35.6) showed significant relationship with development of cardiac tamponade ($p=0.001$). After multivariate adjustment, development of cardiac

tamponade was associated with any amount of PE on the first post-operative echocardiography about 5-day after surgery (OR 15.8; $p<0.001$) and mechanical valve replacement with marginal statistical significance (OR 2.5; $p=0.051$, Table 3).

Table 3. Factors associated with post-operative cardiac tamponade

Variable	Univariate	Multivariate		
	p value	OR	95% CI	*p*
Infective endocarditis	0.017	2.1	0.637-6.680	0.227
Any mechanical valve replacement	0.044	2.5	0.997-6.404	0.051
Any pericardial effusion at 5-day TTE	0.001	15.8	6.105-40.889	<0.001

TTE, transthoracic echocardiography; OR, odds ratio; CI, confidence interval

4. Factors associated with delayed cardiac tamponade

When patients with cardiac tamponade were divided into two groups according to timing of occurrence, early cardiac tamponade was developed in 17 patients (71%) and delayed cardiac tamponade was in 7 patients (29%). Further analysis revealed BAV and concomitant MAZE operation were demonstrated to have relationship with delayed cardiac tamponade (all $p<0.05$, Table 4). Any PE at the first post-operative echocardiography or mechanical valve replacement did not have relationship with delayed cardiac tamponade.

Table 4. Factors associated with delayed cardiac tamponade

Cardiac tamponade (total n=24)	Variables	Odd ratio	*p*
Delayed cardiac tamponade (≥ 30 days) n=7 (29%)	Bicuspid aortic valve	4.2	0.040
	Concomitant MAZE	6.9	0.046

5. Clinical outcome of the patients with post-operative cardiac tamponade

Sixteen (66.7%) and eight (33.3%) patients underwent percutaneous catheter drainage or pericardial window formation, respectively. The median time of pericardial catheter drainage or surgical drainage following valvular surgery was 17 (IQR 13-30) days.

Mean hospital days in patients with cardiac tamponade was longer than those without (43.6 vs 19.5, P=0.031). Readmission was identified in 121 patients (21.8%) due to cardiovascular cause. Echocardiography following procedural intervention revealed resolution of PE in all patients. There was no echocardiographic evidence of small or more amount PE a median of 1044 (IQR 446-1311) days after surgery.

IV. DISCUSSION

Overall, cardiac tamponade occurred in 24 patients (4.3%) in this study. Development of cardiac tamponade was related with infective endocarditis,

valve replacement using mechanical valve, and any amount of PE at median 5 days following surgery. In multivariate analysis, cardiac tamponade was independently associated with early PE (p<0.001) and surgical replacement with mechanical valve with marginal statistical significance (p=0.051). Subgroup analysis revealed that delayed cardiac tamponade, which developed later than 30 days after surgery, was prone to develop in patients with BAV or concomitant MAZE procedure.

The incidence of this study is higher than that in previous study, which reported the only 0.8~0.9% patients experienced cardiac tamponade after open heart surgery.[2,10] This study analyzed only patients undergoing valvular surgery, which were associated with more risk of cardiac tamponade than those undergoing CABG only.[2,3,10] 29% among all cardiac tamponades occurred after 30 days following surgery during median 1044 follow-up days. Surgical types confined to valvular surgery and long-term follow up duration might partially explain higher incidence of cardiac tamponade in this study.

It seems evident that smaller PE presages overt cardiac tamponade after open heart surgery as demonstrated in multiple previous studies.[3,10] Though the exact pathophysiology generating post-cardiotomy PE or tamponade is still elusive, some mechanisms were suggested in previous studies. Peri-operative anticoagulation had been considered an important risk for cardiac tamponade by promoting pericardial bleeding.[2,10] However, Ofori-Krakye et al reported that

cardiac tamponade occurred in patients without prior anticoagulation in the study analyzing 1,290 patients.[12] Two other small studies did not indicate a detrimental impact of anticoagulation therapy either.[5,13] In this study, the post-operative levels of anticoagulation including both aPTT and INR between groups with and without cardiac tamponade were not different. Exclusion of immediate post-operative cardiac tamponade which occurred within 2 days after surgery, which usually associated with surgical bleeding may explain why anticoagulation was not associated with occurrence of cardiac tamponade.

Development of post-operative PE was also regarded as one of the major components and complications of PPS, which is characterized by lasting fever, pleuritic symptoms, pericardial or pleural effusions after cardiac surgery. Imazio et al reported PE was found in about 90% of patients with PPS.[1] Immunopathic etiology appears to be most acceptable mechanism of development of PPS based on various studies.[14,15] If post-operative PE shares pathophysiology with PPS, it is possible that increased both local and systemic inflammation after surgery may play a role in development of PE and tamponade.

Mechanical valve can promote both systemic and local inflammation, represented by serum inflammatory markers such as IL-6 [16] and metallosis of the adjacent tissue.[17] Infective endocarditis is undoubtedly pro-inflammatory status.[18] Concomitant MAZE procedure may enhance local inflammation

caused by surgical incision.[19] The pathophysiology of degenerative changes in BAV also includes inflammatory activation, evident from both excised tissues and venous blood samples in patients with BAV.[20,21] Therefore, infective endocarditis, BAV and concomitant MAZE procedure may raise pericardial and systemic inflammation, which could mediate relation between cardiac surgery and development or worsening of PE.

Especially late cardiac tamponade may develop silently without evidence clinical signs. Since it can be easily missed, and without early diagnosis and treatment, can be life threatening, early decompression is required as soon as its presence confirmed.[22,23] We treated patients with cardiac tamponade by either percutaneous pericardiocentesis or surgery in loculated posterior effusions. No in-hospital death occurred due to cardiac tamponade.

Post-operative cardiac tamponade prolonged hospital stay and was related with high rate of cardiovascular readmission. Post-operative constriction is associated with PE,[6,7] which can be associated with high readmission rate in patients with PE. Generally, however, cardiac tamponade has benign clinical course without recurrence after timely intervention.

To date, there is no obvious way to prevent cardiac tamponade after open heart surgery. Erdil's group suggested that posterior cardiotomy during valve replacement operation might reduce the risk of last cardiac tamponade, but they failed to show statistical significance in their study.[24] Imazio et al demonstrated

that prophylactic colchicine could prevent incident PE and worsening of PE as well as PPS.[25] It might be particularly helpful to use prophylactic colchicine for preventing cardiac tamponade in high-risk patients, such as those with pre-operative infective endocarditis or mechanical prosthesis.

Several limitations exist in this study. Because of retrospective property, there is no strict rule for regular echocardiographic follow-up. It is possible that some cases with PE were missed. The treatments for the PE less than the amount causing cardiac tamponade were different in each patient ranging from observation to stop of anticoagulation or adding anti-inflammatory medication. Serum levels of anticoagulation were not continuously measured after surgery, which might cause insufficient evidence of anticoagulation in study population. Only CRP was measured to represent systemic inflammatory reaction, which could be suboptimal to demonstrate systemic inflammation.

V. CONCLUSION

Cardiac tamponade is more common in patients with any PE echocardiography at 5 days after valvular heart surgery or mechanical valve replacement. Although post-operative cardiac tamponade prolongs hospital stay and requires more readmission compared with those without, it has benign clinical course without recurrence after timely intervention. This result might be helpful to identify high-risk patients for developing cardiac tamponade who

require prophylactic treatment. Also, we expect this study to help to investigate the pathophysiology of post-cardiotomy PE. Further large prospective study is required to validate the risk among patients or benefit of potential prevention.

REFERENCES

1. Imazio, M. The post-pericardiotomy syndrome: *Curr. Opin. Pulm. Med.* **18,** 366–374 (2012).
2. Kuvin, J. T., Harati, N. A., Pandian, N. G., Bojar, R. M. & Khabbaz, K. R. Postoperative cardiac tamponade in the modern surgical era. *Ann. Thorac. Surg.* **74,** 1148–1153 (2002).
3. Pepi, M. *et al.* Pericardial effusion after cardiac surgery: incidence, site, size, and haemodynamic consequences. *Br. Heart J.* **72,** 327–331 (1994).
4. Weitzman, L. B. *et al.* The incidence and natural history of pericardial effusion after cardiac surgery--an echocardiographic study. *Circulation* **69,** 506–511 (1984).
5. Ikäheimo, M. J. *et al.* Pericardial effusion after cardiac surgery: incidence, relation to the type of surgery, antithrombotic therapy, and early coronary bypass graft patency. *Am. Heart J.* **116,** 97–102 (1988).
6. Matsuyama, K. *et al.* Clinical Characteristics of Patients With Constrictive Pericarditis After Coronary Bypass Surgery. *Jpn. Circ. J.* **65,** 480–482 (2001).
7. Im, E. *et al.* The Incidence and Clinical Outcome of Constrictive Physiology After Coronary Artery Bypass Graft Surgery. *J. Am. Coll. Cardiol.* **61,** 2110–2112 (2013).
8. Joint Task Force on the Management of Valvular Heart Disease of the

European Society of Cardiology (ESC) *et al.* Guidelines on the management of valvular heart disease (version 2012). *Eur. Heart J.* **33,** 2451–2496 (2012).

9. Zoghbi, W. A. *et al.* Recommendations for evaluation of prosthetic valves with echocardiography and doppler ultrasound: a report From the American Society of Echocardiography's Guidelines and Standards Committee and the Task Force on Prosthetic Valves, developed in conjunction with the American College of Cardiology Cardiovascular Imaging Committee, Cardiac Imaging Committee of the American Heart Association, the European Association of Echocardiography, a registered branch of the European Society of Cardiology, the Japanese Society of Echocardiography and the Canadian Society of Echocardiography, endorsed by the American College of Cardiology Foundation, American Heart Association, European Association of Echocardiography, a registered branch of the European Society of Cardiology, the Japanese Society of Echocardiography, and Canadian Society of Echocardiography. *J. Am. Soc. Echocardiogr. Off. Publ. Am. Soc. Echocardiogr.* **22,** 975–1014; quiz 1082–1084 (2009).

10. Meurin, P., Weber, H., Renaud, N., Larrazet, F. & al, et. Evolution of the Postoperative Pericardial Effusion After Day 15*: The Problem of the Late Tamponade. *Chest* **125,** 2182–7 (2004).

11. Authors/Task Force Members *et al.* 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the

Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur. Heart J.* **36,** 2921–2964 (2015).

12. Ofori-Krakye, S. K. *et al.* Late cardiac tamponade after open heart surgery: incidence, role of anticoagulants in its pathogenesis and its relationship to the postpericardiotomy syndrome. *Circulation* **63,** 1323–1328 (1981).

13. Stevenson, L. W., Child, J. S., Laks, H. & Kern, L. Incidence and significance of early pericardial effusions after cardiac surgery. *Am. J. Cardiol.* **54,** 848–851 (1984).

14. Khan, A. H. The postcardiac injury syndromes. *Clin. Cardiol.* **15,** 67–72 (1992).

15. Gaudino, M., Anselmi, A., Pavone, N. & Massetti, M. Constrictive Pericarditis After Cardiac Surgery. *Ann. Thorac. Surg.* **95,** 731–736 (2013).

16. Liebe, V. *et al.* Coagulation activation is associated with interleukin-6 plasma levels in patients with mechanical prosthetic heart valves. *Vivo Athens Greece* **20,** 427–430 (2006).

17. den Otter, G. Total prosthetic replacement of atrioventricular valves in the dog. 3. local tissue reactions in the heart caused by tricuspid valve prostheses. *Thorax* **27,** 105–110 (1972).

18. Watkin, R. W. *et al.* Pro-inflammatory cytokines IL6, TNF-alpha,

IL1beta, procalcitonin, lipopolysaccharide binding protein and C-reactive protein in infective endocarditis. *J. Infect.* **55,** 220–225 (2007).

19. Maesen, B., Nijs, J., Maessen, J., Allessie, M. & Schotten, U. Post-operative atrial fibrillation: a maze of mechanisms. *Eur. Eur. Pacing Arrhythm. Card. Electrophysiol. J. Work. Groups Card. Pacing Arrhythm. Card. Cell. Electrophysiol. Eur. Soc. Cardiol.* **14,** 159–174 (2012).

20. Forte, A. *et al.* Early cell changes and TGF pathway alterations in the aortopathy associated with bicuspid aortic valve stenosis. *Clin. Sci. Lond. Engl. 1979* **124,** 97–108 (2013).

21. Ali, O. A. *et al.* Interactions between inflammatory activation and endothelial dysfunction selectively modulate valve disease progression in patients with bicuspid aortic valve. *Heart Br. Card. Soc.* **100,** 800–805 (2014).

22. Borkon, A. M. *et al.* Diagnosis and management of postoperative pericardial effusions and late cardiac tamponade following open-heart surgery. *Ann. Thorac. Surg.* **31,** 512–519 (1981).

23. Sahni, J., Ivert, T., Herzfeld, I. & Brodin, L. A. Late cardiac tamponade after open-heart surgery. *Scand. J. Thorac. Cardiovasc. Surg.* **25,** 63–68 (1991).

24. Erdil, N. *et al.* Effect of Posterior Pericardiotomy on Early and Late Pericardial Effusion After Valve Replacement. *J. Card. Surg.* **20,** 257–260

(2005).

25. Imazio, M. *et al.* COlchicine for the Prevention of the Post-pericardiotomy Syndrome (COPPS): a multicentre, randomized, double-blind, placebo-controlled trial. *Eur. Heart J.* **31,** 2749–2754 (2010).

ABSTRACT (IN KOREAN)

개흉 판막 수술을 받은 환자에서 술 후 심낭압전의 발생률, 예측인자 및 임상예후

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목적

현대에 개흉 수술 이후의 심낭압전의 위험성 및 임상 예후에 대해서는 알려진 바가 적다. 따라서, 본 연구에서는 개흉 판막 수술 이후 심낭 압전의 발생률과 예측인자 및 임상 경과에 대해 확인하고자 한다.

환자 및 방법

본원에서 2010년 1월부터 2012년 3월까지 개흉 판막 수술을 받은 556 명의 환자를 분석 대상으로 하였다. 경흉부 초음파를 통하여 수술 1주일 이후의 심낭삼출액의 여부 및 양과 혈역학적 관계에 대해 확인하였고, 1번 이상의 재검을 하였다. 수술 직후의 출혈에 의한 심낭 삼출액이 의심되는 경우는 분석에서 제외하였다.

결과

대상 환자 군 중 4.3%에서 수술 후 심낭압전이 발생하였다. 수술 이후 심낭압전에 대한 시술 또는 수술까지의 중앙값은 17일이었으며 양 군사의 나이와 성별의 유의미한 차이는 없었다. 수술 후 5일째 시행한 경흉부 초음파에서의 심낭 삼출액의 유무

및 기계판막치환술이 유일한 독립적 위험인자로 밝혀졌다. 심낭압전으로 시술 또는 수술을 받은 환자들은 중앙값 1044일의 추적관찰기간 중에 추가적인 심낭삼출액의 발생은 보이지 않았다.

결론

개흉 수술 약 1주 후의 심낭삼출액 유무가 개흉 판막 수술 이후 심낭압전 발생의 독립적 위험인자였으며, 심낭압전은 발생은 재원일수를 연장시키기는 하나 장기적인 예후는 비교적 양호하였다.

핵심되는 말 : 심낭 압전, 수술 이후 심낭삼출액, 경흉부 심장 초음파