Acute Heart Failure after Relief of Massive Pericardial Effusion

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Severe left ventricular dysfunction after relief of massive pericardial effusion has been rarely reported. Interventricular volume mismatch, acute distention of the cardiac chambers and interplay of autonomic nerve system are believed to be the possible causes for ventricular dysfunction. Presenting two patients who had marked decrease in global ventricular systolic function after relief of pericardial tamponade by subxyphoid pericardial window, we recommend gradual removal of pericardial fluid under hemodynamic monitoring, especially in patient with postcardiotomy tamponade.

(Key words: 1. Heart failure 2. Pericardial effusion 3. Pericardiomy)

CASE REPORT

Case 1

A 34-year-old female was hospitalized because of her increasing shortness of breath. Two months previously, she had undergone mitral valve replacement and tricuspid valve annuloplasty due to severe mitral stenosis and tricuspid insufficiency. She had been taking digoxin, warfarin, and diuretics. On admission, her electrocardiogram showed sinus tachycardia and transthoracic echocardiogram (TTE) showed a large amount of effusion in lateral and posterior space of pericardium, but both ventricular systolic functions were relatively normal and the prosthetic mitral valve function was also normal. The pressure gradient across the prosthetic mitral valve was within the expected limits. Her physical examination was noticeable for jugular venous distension, hypotension (blood pressure of 80/40 mmHg), tachycardia (heart rate of 120 beats/min). Because of her clinical sign of cardiac tamponade, the patient underwent pericardial window creation and about 650 mL of serous fluid was removed. Immediately after evacuation, the central venous pressure (CVP) was slightly decreased from 23 to 18 mmHg. Repeat echocardiogram showed normal valve motion and minimal pericardial effusion, but severe global RV and LV hypokinesia. Because of the presence of hypotension, the patient received maximal inotropic support and put on the intra-aortic balloon pump. However, the patient was succumbed to her illness one day later.
Case 2
A 46-year-old female patient with a history of mitral valve replacement with tricuspid annuloplasty eight months ago presented with progressively worsening dyspnea over the previous 2 months. Echocardiogram revealed a large amount of effusion in posterolateral space of pericardium (Fig. 1). Because of lack of typical clinical sign of tamponade, she was first treated conservatively with high dose diuretics. But during the following days, the patient’s condition was not improved and she became more tachypneic. Her physical examination was noticeable for jugular venous distension, hypotension, and tachycardia. Repeated TTE revealed a still remain large amount of pericardial effusion. The ejection fraction (EF) of LV was 53% and there were no regional wall motion abnormalities. An urgent pericardial window was performed and 700 mL of serosanguineous fluid was removed and preoperative high CVP (22 mmHg) was decreased to 12 mmHg. But over the following 10 hours, the CVP was increased back to 22 mmHg and the patient again complained about shortness of breath. TTE showed only minimal pericardial effusion with a severe global LV hypokinesia and EF fraction was 25%. Dopamin, Dobutamin and ACE inhibitor were initiated. Serial echocardiograms taken during a period of 2 weeks documented substantial improvement in left ventricular systolic function. Three weeks later she was discharged with the EF returning to the preoperative state and normally functioning prosthetic mitral valve.

DISCUSSION
Severe left ventricular dysfunction after relief of massive pericardial effusion has been rarely reported[1-3] and includes cases of pulmonary edema, and adult respiratory distress syndrome[4-7]. Both of our cases were in postcardiac surgery status and the pericardial effusion mainly collected from the posterior and lateral spaces. In the first case, the patient did not respond to inotropics and died of biventricular failure. In second case, preoperative status was similar with first case but postoperative course was more benign. In our patients, left ventricular systolic function appeared to be normal and no wall motion abnormalities were detected before pericardial window creation. After emergency pericardial window procedure, marked ventricular dysfunction developed. We do not understand the exact causative factors of severe cardiac failure after relief of pericardial effusion. The adaptive cardiac mechanisms to the increased intrapericardial pressure of effusion were well known[4]. The external compression of the right heart by large volume of pericardial effusion leads to a decrease in stroke volume and cardiac output. Compensatory mechanisms include tachycardia, an increase in the RV preload through expansion of the intravascular volume, and the results from sympathetic activation including stimulation by high catecholamine levels. However, experimental and cli-
nical studies have shown that cardiac tamponade does not appear to impair left ventricular systolic function. The possible mechanism of LV dysfunction after relief of massive pericardial effusion might be related to failure of these adaptive mechanisms. Several explanations have been suggested. One is the release of pericardial constraint, which could lead to a disproportional increase in the RV end-diastolic volume compared with LV end-diastolic volume[5]. This interventricular volume mismatch in the presence of vasoconstriction due to high catecholamine levels could lead to an increase in LV end-diastolic pressure and transient LV dysfunction for the previously diseased heart. When acute distention of the cardiac chambers secondary to increased venous return at high filling pressures is combined with a negative pressure in the pericardial cavity immediately after large-volume pericardiocentesis, it may be another cause of cardiac failure. In our patients, RV was hypokinetic and CVP returned to a high following the transient decrease during evacuation of effusion; this supports that these mechanisms might be responsible for the cardiac failure. We cannot rule out other proposed mechanisms that might have lead to cardiac failure. Myocardial stunning due to mismatch of oxygen supply for the acutely increased wall stress in myocardium might have resulted[1]. Tampone had resulted in a disproportionately greater decrease of the coronary blood flow, but any occult dysfunction was masked by the reduction in chamber sizes and associated tachycardia. The autonomic nerve system is also likely to have influenced the cardiac function before and after pericardiocentesis[2]. The removal of the stimulus for an increased sympathetic outflow when relieving the tamponade might have lead to left ventricular dysfunction.

The patients had suffered from long-standing myocardial dysfunction before the cardiac surgery, and they also suffered from postcardiotomy pericardial effusion. This suggests that the patients might have occult ventricular dysfunction, although the left ventricular systolic function appeared to be normal and no wall motion abnormalities were detected before pericardial window creation. The diseased heart would be more prone to maladapt to the changes in cardiac load. Hypokinetic RV and the absence of a decrease of CVP during evacuation might be predicting signs for cardiac failure. Although the incidence of severe LV dysfunction is rare, it is important to be aware of the possibility of LV dysfunction after rapid relief of massive pericardial effusion. We recommend gradual removal of pericardial effusion under hemodynamic monitoring, especially in patient with postcardiotomy tamponade.

REFERENCES

국문 초록

대량의 심낭삼출액을 제거한 뒤 발생할 수 있는 심한 좌심실 부전에 관한 보고는 많지 않다. 심실 간의 용적 차이, 심장의 감각스런 확장 그리고 교감신경계의 작용 등이 심실기능 부전의 원인으로 생각될 수 있다. 감상동기화 심낭장을 통해 심장 압전을 완화한 뒤 심실 기능 부전이 발생한 두 증례를 보고하면서, 특히 심장수술 후 심장 압전이 있는 환자의 경우 혈류학적을 잘 관찰하면서 심낭삼출액을 서서히 제거할 것을 제안한다.

중점 단어: 1. 심부전
2. 심낭삼출액
3. 심낭장