

นิพนธ์ต้นฉบับ

Cardiac Tamponade in Medical Patients : Five Years Analysis at Prapokklao Hospital.

Piyapong Permlarp M.D.*

Abstract

Objective : To analyse the medical patients with cardiac tamponade, in terms of etiologies, clinical manifestations, radiologic and echocardiographic findings.

Materials and methods : Retrospective descriptive study of the medical records with diagnoses of cardiac tamponade from department of internal medicine, Prapokklao Hospital from October 1999 through September 2004. Diagnosis of cardiac tamponade was based on consistent clinical manifestations and echocardiographic findings of pericardial effusion and right ventricular diastolic collapse.

Results and conclusion : There were 22 cases of cardiac tamponade. The age ranged from 21 to 78 years. The most common cause was idiopathic pericarditis (45.5 percent). The three most common clinical manifestations were tachypnea, dyspnea and tachycardia (100, 90.1 and 59.1 percent respectively). Chest roentgenograms revealed enlarged cardiac silhouette in all patients. Electrocardiograms revealed low QRS voltage, abnormalities of the T waves and abnormalities of the ST segments in 70, 60 and 25 percent respectively. This study provides information about cardiac tamponade in medical patients which differ from cardiac tamponade due to cardiac trauma.

Introduction

Cardiac tamponade is one of the serious cardiac disorders. Since immediate treatment of tamponade may be lifesaving, a high index of suspicion and prompt measures to establish early

diagnosis are required. Cardiac tamponade can result from a number of disease processes so the clinical manifestations may vary widely. Medical patients with cardiac tamponade clinically differ from those with acute tamponade due to cardiac

* Cardiovascular Unit, Department of Internal Medicine, Prapokklao Hospital, Chanthaburi Province.

trauma because the fluid accumulation in medical patients is often more gradual¹⁻⁹. This study aims to examine the medical patients with cardiac tamponade, in terms of etiologies, clinical manifestations, radiologic and electrocardiographic findings.

Materials and methods

The study population consisted of all patients with diagnoses of cardiac tamponade who were admitted to department of internal medicine, Prapokklao Hospital during a five – year period from October 1999 to September 2004. Cases selected for review were required to have consistent clinical manifestations and to have been confirmed by echocardiographic findings of pericardial effusion with right ventricular diastolic collapse.

A standard data abstraction form was developed for the medical records review. This form included information on demographic characteristics, etiologies, clinical manifestations, radiologic and echocardiographic findings. Pulsus paradoxus was defined as an inspiratory decrease in systolic blood pressure of 10 mm.Hg. or more. The diagnosis of tuberculous pericarditis was based on positive acid fast bacilli from pericardial fluid or pericardial biopsy specimen or microscopic findings of granulomatous pericarditis and response to antituberculous drugs therapy. Data were collected and analyzed descriptively.

Results

There were 26 medical patients with the final diagnoses of cardiac tamponade. Four patients

who did not meet the inclusion criteria were excluded.

Among 22 included patients, there were 13 men (59 percent) and 9 women (41 percent). The age ranged from 21 to 78 years.

The etiologies are listed in table 1. The most common cause was idiopathic pericarditis. Malignancies in 5 cases comprised CA lung, CA thyroid, CA colon, CA breast and metastatic squamous cell CA of unknown primary site. In 2 bacterial pericarditis, one had *S. aureus* and the other had *P. pseudomallei*.

Table 1 : Etiologies of Cardiac Tamponade.

Etiology	No of patients (percent)
Idiopathic pericarditis	10 (45.5)
Malignancy	5 (22.7)
Tuberculous pericarditis	5 (22.7)
Bacterial pericarditis	2 (9.1)

The most common clinical manifestations was respiratory rate ≥ 20 / min. Other clinical manifestations are shown in table 2

Table 2 : Clinical manifestations of cardiac tamponade.

Clinical manifestation	No of patients (percent)
Dyspnea	20 (90.1)
Chest pain	2 (9.1)
Respiratory rate ≥ 20 /min	22 (100)
Heart rate > 100 /min	13 (59.1)
Systolic blood pressure ≤ 100 /min	12 (54.5)
Pulse pressure ≤ 30 mmHg.	9 (40.9)
Pulsus paradoxus.	4 (18.2)
Diminished heart sounds.	7 (31.8)
Pericardial friction rub	2 (9.1)
Neck vein distension	8 (36.4)
Hepatomegaly	10 (45.5)
Leg edema	9 (40.9)

Table 3 : Electrocardiographic findings.

Finding	No of patients (percent)
- Sinus tachycardia	12 (60)
- Atrial fibrillation	1 (5)
- Low QRS voltage	14 (70)
- Non specific abnormalities of the T waves	12 (60)
- Non specific abnormalities of the ST segments	5 (25)

Chest roentgenograms were obtained in 19 of the 22 patients. All had an enlarged cardiac silhouette (cardiothoracic ratio greater than 50 percent). Pulmonary congestion was found in 4 patients.

Electrocardiograms were obtained in 20 of the 22 patients. Low QRS voltage was found in 14 patients (70 percent). None had electrical alternans. Other findings were shown in table 3.

Discussion

An increase in intrapericardial pressure secondary to fluid accumulation within the pericardial space results in cardiac tamponade. It may occur with almost any causes of pericarditis and may exist in either an acute or a chronic form. In this study, there were 4 causes of cardiac tamponade in medical patients including idiopathic, malignancy, tuberculous and bacterial pericarditis (45.5, 22.7, 22.7 and 9.1 percent respectively). This finding is not similar to other studies^{9,10} which had shown that malignancy was the most common cause. In part, a high frequency of idiopathic pericarditis in this study may be due to limited capability in laboratory investigation for viral study in this hospital.

Tuberculous pericarditis is frequently associated with pulmonary tuberculosis. In a large series of Strang JIG, et al,¹¹ 72 percent of 198 cases had evidence of active pulmonary tuberculosis. In this study, 2 of 5 patients (40 percent) with cardiac tamponade due to tuberculous pericarditis had active pulmonary tuberculosis.

The introduction of AIDS (acquired immunodeficiency syndrome) into the population has contributed to increase of new cases of tuberculosis. The prevalence of tuberculous pericarditis may be expected to follow the same pattern as that of pulmonary and extrapulmonary tuberculosis in general¹². In this study, 2 of 5 patients (40 percent) had HIV infection. None had pulmonary tuberculosis.

Pulsus paradoxus may be absent during cardiac tamponade in patients with some particular conditions, such as atrial septal defect, aortic regurgitation, severe aortic stenosis, uremia with left ventricular dysfunction and advanced tamponade with severe hypotension^{9,13-15}. However it may be due to lack of a careful measurement of blood pressure variation with respiration. In this study, only 4 of 22 patients (18.2 percent) had pulsus

paradoxus in contrast to 70–80 percent reported in other studies^{9,10}.

Tachypnea (respiratory rate greater than 20 /min) was the most common clinical manifestation. It was found in all patients. This finding is consistent with other studies^{9,10}. Tachycardia, despite a normal blood pressure, is also an important finding.

In a study of Gurerman BA, et al⁹ in 1981 in which 56 patients were studied, fifty – two patients (95 percent) had an enlarged cardiac silhouette by chest roentgenogram and two patients (3.6 percent) had pulmonary congestion. Each of them had myocardial failure. In this study chest roentgenogram revealed enlarged cardiac silhouette in all patients and pulmonary congestion was found in 4 patients. These findings suggest that although enlarged cardiac silhouette with clear lung fields suggest the possibility of pericardial disease rather than myocardial failure, evidence of enlarged cardiac silhouette with pulmonary congestion doesn't exclude the pericardial effusion with cardiac tamponade.

The electrocardiographic abnormalities seen in cardiac tamponade include those of acute pericarditis and pericardial effusion per se. The development of electrical alternans is more specific indicator of cardiac tamponade. In series of Wall TC. et al¹⁰ low QRS voltage, abnormalities of the T waves, abnormalities of the ST segments and electrical alternans were found in 24, 12, 44 and 12 percent of 25 cases respectively. In this study, low QRS voltage, abnormalities of the T waves and abnormalities of the ST segments were found

in 70, 60 and 25 percent of 20 cases respectively. None had electrical alternans. All of these patients had no echocardiographic finding of swinging heart which had been reported as a cause of electrical alternans¹⁶.

In summary, data obtained by this study may provide clues for clinician to consider the possibility of cardiac tamponade in medical patients. This included clinical manifestations of unexplained dyspnea, unexplained tachypnea, unexplained tachycardia, unexplained narrow pulse pressure, pulsus paradoxus and unexplained signs of right – sided heart failure. Cardiac tamponade should also be considered in patients with low QRS voltage or abnormalities of the T waves or abnormalities of the ST segments in electrocardiogram and in patients with unexplained enlarged cardiac silhouette on chest roentgenogram. When a patient has one or more of the above findings, the possibility of tamponade is strengthened when certain predisposing conditions such as malignant neoplasm, tuberculosis and etc, are present. When the findings mentioned above are present and unexplained, echocardiogram should be performed and pericardial drainage must be carried out if there is cardiac tamponade.

References

1. Hoit BD. Disease of the pericardium. In : Fuster V, Alexander RW, O'Rourke RA. Editors. *Hurst's the Heart*. tenth edition. New York : Mc Graw-Hill, 2001.
2. Spodick DH. Pericardial diseases. In : Braunwald E, Zipes DP, Libby P. Editors. *Heart disease* 6 th edition. Philadelphia : W.B. Saunders company, 2001.

3. Cheitlin MD.: Cardiovascular trauma. Circulation 1982; 65:1529.
4. Henderson VJ, Smith RS, Fry WR, et al. Cardiac injuries : analysis of an unselected series of 251 cases. J Trauma 1994;36:341.
5. Mattox KL, Feliciano DV, Burch J, et al. Five thousand seven hundred sixty cardiovascular injuries in 4459 patients : Epidemiologic evolution 1958 to 1987. Ann Surg 1989;209:698.
6. Mattox KL, Estera AL, Wall JRMK. Traumatic heart disease. In : Bravwald E, Zipes DP, Libby P. Editors. Heart disease 6 th edition. Philaedlphia : W.B. saunders company, 2001.
7. Symbas PN. Traumatic heart disease. In : Fuster V, Alexande RW, O'rourke RA. Editors. Hurst's the Heart tenth edition New York : Mc Graw–Hill, 2001.
8. Brown J, Grover FL. Trauma to the heart. Chest Surg Clin North Am 1997;7:325.
9. Guberman BA, Fowler NO, Engel PJ, et al. Cardiac tamponade in medical patients. Circulation 1981;64:633.
10. Wall TC, Campbell PT, O'Conner CM, et al. Diagnosis and management (by subxiphoid pericardiotomy) of large pericardial effusions causing cardiac tamponade. Am J Cardiol 1992;69:1075–78.
11. Strang JIG, Kakaza HHS, Gibson DG, et al. Controlled clinical trial of complete open surgical drainage and of prednisolone in treatment of tuberculous pericardial effusion in Transkei. Lancet 1988;2:759–64.
12. Fowler NO, Tuberculosis Pericarditis. JAMA. 1991;266(1)99–103.
13. Winer HE, Kronzon I. Absence of paradoxical pulse in patients with cardiac tamponade and atrial septal defects. AM J Cardiol 1979; 44:378.
14. Lange RL, Boticelli JT, Tsagaris TJ, Walker JA, Gani M, Bustamante RA. Diagnostic signs in compressive cardiac disorders : constrictive pericarditis, pericardial effusion and tamponade. Circulation 1966;33:763.
15. Reddy PS, Curtiss EI, O' Toole JD, Shaver JA. Cardiac tamponade : hemodynamic observations in man. Circulation 1978;58:265.
16. Usher BM, Popp RL, Electrical alternans : mechanism in pericardial effusion. Am Heart J 1972; 83 (4):459–63.