

Clinical Manifestation, Diagnosis, Management, and Treatment Outcome of Pericarditis in Patients with Systemic Lupus Erythematosus

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Objective: To investigate the clinical manifestations, diagnosis, etiology, management, and outcomes of patients with systemic lupus erythematosus (SLE) and pericarditis

Material and Method: The authors retrospectively reviewed the records of 81 patients who were diagnosed of SLE according to the American College of Rheumatology criteria and had 82 episodes of pericarditis between 2002 and 2010. The diagnosis of pericarditis was defined as the presence of pericardial effusion alone by echocardiography or having 2 out of 4 of the following criteria: retrosternal pain, pericardial friction rub, widespread ST-segment elevation, and new/worsening pericardial effusion.

Results: Most of them (92%) were female with the median disease duration (range) of 1 (0-312) month. Cardiac tamponade occurred in 16% (95% CI 8.72-25.58%). There was no statistically significant difference between patients who developed tamponade and those who did not. The causes of pericarditis included active SLE (93%), and suspected tuberculosis (TB) (5%), with 2% inconclusive. In patients with lupus pericarditis, 71% had other active organ involvement. Most lupus pericarditis patients (79%) had good response to steroid or NSAIDs. Diagnosis of TB pericarditis was made by clinical suspicion without microbiological or pathological evidence.

Conclusion: In an endemic area of TB, lupus pericarditis was still the most common cause of pericarditis in SLE. Most patients responded well to steroid.

Keywords: Systemic lupus erythematosus, Pericardial effusion, Pericarditis, Tuberculosis

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Pericarditis is one of the most characteristic manifestations and is included in the American College of Rheumatology (ACR) classification criteria for systemic lupus erythematosus (SLE). The incidence varied among studies between 12 and 48%⁽¹⁾. Most cases were asymptomatic. In autopsy patients, pericarditis was found between 25 and 62%⁽²⁾. Cardiac tamponade according to SLE itself was quite rare and found in only 1-2.5% in SLE patients and 4-13% among patients with all-causes pericarditis^(3,4). In one study, all cardiac tamponade episodes occurred in female gender and were associated with low complement (C) 4 level. The association with other

conditions such as age, duration of SLE, organ manifestation, anticardiolipin antibodies, lupus anticoagulant, steroid use, erythrocyte sedimentation rate (ESR), white blood cell (WBC) count, and C3 level was not found⁽⁵⁾. The common etiology was the active disease itself. Most of them responded well to non-steroidal anti-inflammatory drugs (NSAIDs) or steroid, and pericardiocentesis was usually not required for diagnosis or treatment^(6,7).

In Thailand, there was no study regarding the incidence and characteristics of pericarditis in SLE. Moreover, this is an endemic area of tuberculosis (TB); the incidence of TB pericarditis in SLE may be different from previous reports. Mortality rate of TB pericarditis was as high as 85% at 6 months with mean survival rate of 3.7 months⁽⁸⁾. Due to differences in management, treatment outcomes, and prognosis, the differential diagnosis of TB pericarditis should be considered in suspected patients. In such patients, pericardiocentesis should be done regardless of

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cardiac tamponade with the purpose of correct diagnosis and proper treatment.

In order to improve the quality of management and treatment outcomes, this study was designed to investigate the baseline characteristics, clinical features, diagnosis, etiologies, management, and outcomes of pericarditis in patients with SLE in Thailand.

Material and Method

The medical records of patients who were diagnosed of SLE and pericarditis between 2002 and 2010 at Siriraj Hospital, a university hospital in Thailand, were pulled and retrospectively reviewed if they had an ICD-10 code consistent with a diagnosis of SLE and pericarditis. Cases were included in this study if they were 18 years old or more and fulfilled at least 4 out of 11 of the ACR criteria⁽⁹⁾. Pericarditis was defined as the presence of pericardial effusion alone by echocardiography or having at least 2 out of 4 of the following criteria: retrosternal pain, pericardial friction rub, widespread ST-segment elevation, and new/worsening pericardial effusion⁽¹⁰⁾. Patients were excluded from this study if there were no data of pericarditis episodes in the hospital database.

Data collection

The following data were collected: demographic data, clinical presentation, diagnostic criteria, laboratory investigation, etiologies, management, and outcomes. SLE status was classified into diagnosed SLE and first diagnosis of SLE. Disease activity was measured by Modified SLE Disease activity index (SLEDAI)-2K^(11,12). In pericarditis episodes, the clinical profiles were collected and defined whether patients had symptoms of chest pain, which is compatible with pericarditis. Cardiac tamponade was defined by any abnormal findings of pulsus paradoxus, arterial hypotension, or right ventricular diastolic collapse detected by echocardiographic study. The etiologies of pericarditis were grouped into lupus pericarditis, bacterial infection, TB pericarditis (definite/possible), malignancy, or inconclusive. The diagnosis of lupus pericarditis was made by excluding the other causes or having clinical response to specific treatment. Definite bacterial pericarditis was diagnosed by identification of organism in pericardial fluid using gram stain or culture. Definite TB pericarditis was diagnosed by isolation of *Mycobacterium tuberculosis* or demonstration of granulomatous inflammation on

histological examination of the pericardial fluid or tissue biopsy⁽¹³⁾. Suspected TB pericarditis was diagnosed using clinical response to anti-TB drug. Malignancy was defined by pericardial fluid cytology or tissue pathology.

Statistical analyses

Descriptive statistical analyses related to demographic data, clinical presentation, diagnostic criteria, laboratory investigation, etiologies, management, and outcomes were expressed as mean and standard deviation (SD) or median and range, as appropriate for continuous data and number and percentage for categorical data. To compare the characteristics of causes of pericarditis between patients who had and those who did not have cardiac tamponade, student t-test or Mann-Whitney U test were used as appropriate for continuous data and Chi-square test or Fisher's exact test were used as appropriate for categorical data. All analyses were performed using the SPSS software version 18. Statistical significance was defined as a *p*-value of less than 0.05. All reported *p*-value are two-sided.

Sample size was calculated based on the prevalence of lupus pericarditis (70%) and also increased by 20% for missing data. A total of 270 episodes of SLE patients with pericardial effusion were needed.

The present study was conducted in accordance with the ethical principles of the Declaration of Helsinki and adhered to the principles outlined in the Guideline for Good Clinical Practice International Conference on Harmonization (ICH) Tripartite Guideline (January 1997). The study protocol was approved by the Local Ethics Committee, the Siriraj Institutional Review Board.

Results

The present study included 81 patients with 82 episodes of pericarditis. Most of them (92%) were female with the mean age (\pm SD) of 29.3 \pm 11 years and short disease duration of 1 month (range 0-312). Half of them had pericarditis at the first presentation. Most patients (70%) had other active organs at the same time with the presence of pericarditis with the median modified SLEDAI-2K (range) of 12.5 (1-38). Renal and hematologic manifestations were commonly presented at 68% and 49%, respectively. Nineteen episodes (23%) were pleuropericarditis. Prior TB infection was uncommon (3.6%). Nearly half (47.6%) of episodes occurred while patients were receiving SLE

treatment. The treatments included corticosteroids (34%), disease modifying anti-rheumatic drugs or DMARDs (27%), and NSAIDs (2.4%). Only two patients had recurrent pericarditis.

Characteristics of pericarditis and pericardial effusion

Table 1 summarizes the clinical manifestations of pericarditis. The onset of pericarditis was mostly acute with the symptom duration of 14 days. Half of the patients had 'typical' symptomatic pericarditis. The asymptomatic patients presented with other active organs or other medical illnesses (e.g. infection, sepsis, pulmonary hypertension, etc.), and pericardial effusion was an incidental finding in echocardiographic studies. Cardiac tamponade was not uncommon and

occurred in 13 episodes (16%); however, only three had cardiovascular instability including hypotension or paradoxical pulse. There was no difference in clinical and laboratory findings between patients with and without cardiac tamponade (Table 2). Pericardiocentesis and pericardial biopsy were performed in only 17 (20.7%) and 10 episodes (12.2%), respectively. Pericardiocentesis was initiated in all episodes of cardiac tamponade except for one episode due to patient's rejection.

For pericardial fluid analysis in 17 episodes, the analyses were suggestive of exudative effusion. In addition, there was no microbiological evidence of bacterial or TB infection in all specimens. Pathologic studies, including fluid cytology and tissue biopsy, also

Table 1. Clinical characteristics of pericarditis (n = 82)

Parameters	Number (%) or median (min-max)
Duration of symptom (days)	14 (1-90)
Presence of pericardial effusion	78 (95)
Symptomatic pericarditis (n = 41, 50%)	
Chest pain	28 (68)
Dyspnea	24 (59)
Fever	31 (76)
Cough	20 (49)
Pericardial rub	8 (20)
Diagnosis of pericarditis	
At least 2 of 4 criteria	30 (36)
Pericardial effusion alone	52 (63)
Cardiac tamponade (n = 13, 16%)	
Hypotension	2 (15)
Pulsus paradoxus	2 (15)
Right ventricular collapse by echocardiography	13 (100)

Table 2. Comparisons of clinical and laboratory features between patients with and without cardiac tamponade

Clinical features	Tamponade (n = 13 episodes)	No tamponade (n = 69 episodes)	p-value
Age (year), mean (SD)	26.5 (7.6)	29.8 (12.0)	0.35
Female, n (%)	10 (76.9)	65 (94.2)	0.22
Duration from SLE diagnosis (month), median (min-max)	12 (0-204)	0 (0-312)	0.29
First presentation, n (%)	4 (30.8)	35 (50.7)	0.19
Other active organs, n (%)	9 (69.2)	47 (68.1)	1.00
Pleuritis, n (%)	4 (30.8)	15 (21.7)	0.49
Disease activity (modified SLEDAI-2K), median (min-max)	18 (3-38)	12 (1-34)	0.43
C3 (low), n (%)	3 (75.0)	28 (82.4)	1.00
C4 (low), n (%)	3 (75.0)	26 (83.9)	0.55

SLEDAI = systemic lupus erythematosus disease activity index; C3 = complement component 3; C4 = complement component 4

showed no evidence of granuloma or malignancy. Of these 17 episodes, 13 episodes were therefore considered as serositis associated with lupus, and another four were diagnosed of suspected TB. There was no significant difference in pericardial fluid analysis between patients with lupus pericarditis and those with suspected TB pericarditis. However, pericardial fluid analysis of patients with suspected TB pericarditis showed low sugar, high lactate dehydrogenase (LDH) level, and high pericardial/serum LDH ratio, while WBC count, protein in pericardial fluid, and pericardial/serum LDH ratio were comparable between two groups.

Causes of pericarditis and treatment outcomes

The leading cause of pericarditis in this study was active SLE (93%). Tuberculosis was suspected in only 5%, while 2% was inconclusive. Treatment outcomes are showed in Fig.1

Lupus pericarditis

In 76 episodes of lupus pericarditis, 62 (82%) were diagnosed using only clinical suspicion, while 14 (18%) were diagnosed using pericardial fluid analysis to exclude other causes. Seventy-one (71%) had other active organ involvement in the same period with lupus pericarditis. Most of lupus pericarditis (79%) had good response to treatment; 74 episodes were treated with steroid and the other two with NSAIDs.

TB pericarditis

TB pericarditis was suspected in four episodes. All of them were diagnosed by clinical suspicion without microbiological or pathological evidence. The diagnosis of the first two patients was based on echocardiographic finding of thick pericardium; the remaining two had pulmonary tuberculosis with minimal pericardial effusion. All of them responded well to anti-TB agents, but they also received high-dose steroid as conjunctive treatment for TB pericarditis.

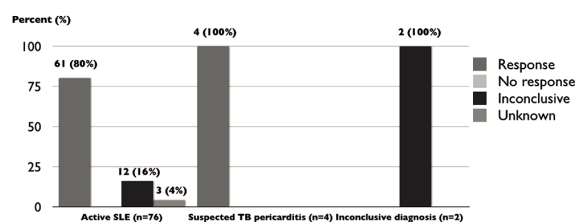


Fig. 1 Outcomes of patients with SLE and pericardial effusion (TB = tuberculosis).

Unknown or inconclusive causes

In these two cases, one had cardiac tamponade, but patient rejected treatment and died afterward. The other one was suspected of TB pericarditis first, but she had worsened clinical course after starting anti-TB agents. After stopping the treatment, she was improved.

Overall, 13 patients (16%) expired during the episodes of pericarditis. The causes of death included septicemia, pneumonia, and suspected diffuse alveolar hemorrhage. Sudden cardiac arrest occurred in one patient and the cause of death was suspected myocardial infarction; another one died from myocarditis and refractory heart failure.

Discussion

The present study described clinical presentation, diagnosis, etiologies, treatment, and outcomes of SLE patients with pericarditis in a university hospital in Thailand. Our results showed that pericarditis can be the first presentation in 50% of SLE, and 50% of all episodes were asymptomatic. The proportion of symptomatic pericarditis in the present study was higher than in a previous report, 50% vs. 25%⁽¹⁾. This difference may be explained by the population in this study. Identifying cases with ICD-10 searching system, all SLE patients included in this study were hospitalized and generally had more severe disease.

The presenting symptoms in the present study are quite different from previous study. The common clinical presentations in ours were fever (76%), chest pain (68%), and dyspnea (59%), while Man et al found that dyspnea was the most common symptom accounting for 61% and precordial pain was found in 39%⁽⁷⁾.

Interestingly, prevalence of cardiac tamponade in SLE patients with pericarditis found in the present study was quite high with 13 episodes (16%), compared to only 4-13% in previous studies^(3,4). However, recent study by Rosenbaum et al showed even higher incidence of tamponade (21.9%)⁽⁵⁾. This diversity could mainly occur from the variation in the method used to detect this condition. In the present study with the lowest incidence of 4%, cardiac tamponade was defined as echocardiographic finding combined with abnormal physical examination: pulsus paradoxus or hypotension. Similar to our study, Rosenbaum et al defined this condition using echocardiographic finding alone without clinical findings⁽⁵⁾.

The prevalence of tamponade at the first presentation of SLE with pericarditis was also high in the current study (31%) and Rosenbaum et al's study

(27%)⁽⁵⁾. In addition, similar to our study, the latter found no relationship between cardiac tamponade and clinical or laboratory characteristics, except for C4 level. They found that patients with cardiac tamponade had lower level of C4, compared to those without tamponade, while this finding cannot be demonstrated in our study due to a small number of patients who had data on complement level⁽⁵⁾.

The main etiology of pericarditis in SLE patients in this study remains lupus pericarditis as in previously reported. As Thailand is an endemic area of tuberculosis, TB pericarditis should be highly suspected in immunocompromised hosts including patients with SLE who are usually treated with steroid or immunosuppressive agents or in patients who had history of previous TB infection. However, in the present study, TB pericarditis was suspected in only four episodes (5%) and lupus pericarditis was final diagnosis in one of them. One of them had previous history of tuberculosis. The diagnosis of TB was made by clinical and laboratory findings without microbiology or pathology proven in all three patients; they all received and responded well to high-dose steroid in addition to anti-TB drugs. Hence, the diagnosis of TB pericarditis in these patients cannot be actually established.

It has been shown that adenosine deaminase (ADA) cut-off level of 40 U/L is useful for the diagnosis of TB pericarditis with sensitivity and specificity of 88% and 83%, respectively^(13,14). Pericardial fluid for ADA was analyzed only in six episodes in our study ranging from 11 to 56.8 U/L; two episodes with suspicious TB pericarditis had ADA value of 18.1 and 41 U/L. Because of no definite diagnosis of TB pericarditis in our report and the small number of fluid ADA samples, the authors cannot demonstrate the relationship between ADA and diagnosis of TB pericarditis.

Overall treatment response rate was 79%. In 41 episodes of symptomatic pericarditis, 35 (85%) episodes responded well to treatment. One patient suspected to be TB pericarditis did not respond to treatment, the other five expired from other causes. In asymptomatic episodes, 30 (73%) episodes had good response. In eight episodes, patients died from other causes and the rest were lost to follow-up and could not be contacted. Excluding those who expired from other causes, lupus pericarditis generally did respond well to treatment.

Due to the nature of retrospective study, there was much missing data, such as acute phase

reactant C3, C4, antiphospholipid antibodies, and pericardial fluid ADA. Thus, we cannot demonstrate the relationship between these factors and lupus pericarditis. Another limitation in the study was the sample size. The population in the study was less than the sample size estimation, so the significant differences of clinical characteristics between patients with tamponade and without tamponade or pericardial fluid analysis between lupus pericarditis and suspected TB pericarditis might not clearly be demonstrated. Up until now, there was no data regarding pericardial ADA in SLE patients with pericarditis. Further studies concerning the clinical application of ADA for the diagnosis of TB pericarditis in patients with SLE are encouraged for improving the standard of care.

In conclusion, lupus pericarditis is still the most common cause of pericarditis in SLE. Cardiac tamponade is not uncommon but usually does not lead to cardiovascular compromise. This condition responds well to corticosteroid or NSAIDs. Although TB is an uncommon causative agent in SLE patients with pericardial effusion, even in an endemic area, definite diagnosis should be instituted, especially in patients who had prior TB infection or clinical suspicion.

What is already known on this topic?

Main etiology of pericarditis in SLE patients is lupus pericarditis.

Cardiac tamponade is uncommon in lupus pericarditis.

What this study adds?

Even in endemic area of tuberculosis, lupus pericarditis is the most common cause of pericardial effusion in SLE.

Cardiac tamponade is not uncommon; however, cardiovascular instability is infrequent.

Incidence of tamponade at the first presentation of lupus pericarditis is high.

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Potential conflicts of interest

None.

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ลักษณะทางคลินิก การวินิจฉัยโรค การดูแลรักษาและผลการรักษาของภาวะเยื่อหุ้มหัวใจอักเสบในผู้ป่วยลูปัส

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วัตถุประสงค์: เพื่อศึกษาลักษณะทางคลินิก การวินิจฉัยโรค สาเหตุ การดูแลรักษา และผลการรักษาของภาวะเยื่อหุ้มหัวใจอักเสบในผู้ป่วยลูปัส

วัสดุและวิธีการ: การศึกษาทำโดยการทบทวนประวัติย้อนหลังในผู้ป่วยที่ได้รับการวินิจฉัยโรคลูปัส (SLE) จำนวน 81 ราย และมีภาวะเยื่อหุ้มหัวใจอักเสบ 82 ครั้ง ตามเกณฑ์การวินิจฉัยโรคของสมาคมโรคข้อและรูมาติสซั่มประเทศ สหรัฐอเมริกา ในโรงพยาบาลศิริราชระหว่าง พ.ศ. 2545 ถึง พ.ศ. 2553 ส่วนเกณฑ์การวินิจฉัยภาวะเยื่อหุ้มหัวใจอักเสบนั้น ต้องมีการตรวจพบน้ำในเยื่อหุ้มหัวใจจากการตรวจคลื่นเสียงสะท้อนหัวใจ หรือ มีเกณฑ์อย่างน้อย 2 ข้อ จาก 4 ข้อ ได้แก่ อาการเจ็บหน้าอก (*retrosternal pain*), *pericardial rub*, ลักษณะคลื่นไฟฟ้าหัวใจที่มีการยกตัวของส่วน ST segment และตรวจพบน้ำในเยื่อหุ้มใจใหม่หรือเพิ่มขึ้น

ผลการศึกษา: ผู้ป่วยส่วนใหญ่ (ร้อยละ 92) เป็นผู้หญิง ช่วงระยะเวลาที่เป็นโรคลูปัสพบตั้งแต่ 0 ถึง 312 เดือน ค่ามัธยฐานอยู่ที่ 1 เดือน ภาวะหัวใจถูกบีบรัด (*cardiac tamponade*) นั้นพบร้อยละ 16 โดยพบว่าผู้ป่วยที่มีและไม่มีภาวะนี้ไม่มีลักษณะทางคลินิกที่แตกต่างกัน สาเหตุของภาวะเยื่อหุ้มหัวใจอักเสบได้แก่ เยื่อหุ้มหัวใจอักเสบจากโรคลูปัสพบร้อยละ 93 คาดว่าเกิดจากการติดเชื้อวัณโรคร้อยละ 5 และไม่ทราบสาเหตุชัดเจนร้อยละ 2 ในผู้ป่วยกลุ่มที่เกิดจากโรคลูปัสพบว่าผู้ป่วยร้อยละ 71 มีอาการของโรคลูปัสที่ร้ายแรงอื่นร่วมด้วย และพบว่าผู้ป่วยส่วนมาก (ร้อยละ 79) ตอบสนองดีต่อการรักษาด้วยยาคอร์ติโคสเตียรอยด์ และยาต้านอักเสบที่ไม่ใช่สเตียรอยด์ การวินิจฉัยภาวะเยื่อหุ้มหัวใจอักเสบจากเชื้อวัณโรคนั้นอาศัยลักษณะอาการทางคลินิก โดยไม่พบหลักฐานทางจุลชีววิทยา หรือ หลักฐานทางพยาธิวิทยา

สรุป: ในบริเวณที่โรควัณโรคพบบ่อย ภาวะเยื่อหุ้มหัวใจอักเสบจากโรคลูปัสก็ยังคงเป็นสาเหตุที่พบบ่อยที่สุดในผู้ป่วยลูปัสที่มีภาวะเยื่อหุ้มหัวใจอักเสบ ผู้ป่วยส่วนมากนั้นตอบสนองดีต่อการรักษาด้วยยาคอร์ติโคสเตียรอยด์
