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ORIGINAL ARTICLE

A retrospective study of joint infections in patients with systemic lupus erythematosus

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Abstract The aim of this study was to analyze the clinical characteristics of systemic lupus erythematosus (SLE) patients with joint infections. We retrospectively reviewed the medical records of 11,734 SLE patients admitted to Peking Union Medical College Hospital (PUMCH) from January 1990 to December 2016. Twenty patients who developed joint infections were identified. Subjects without joint infections (designated as control patients) were selected from the pool of SLE patients using a 1:4 systematic sampling method. The median disease duration from SLE onset to joint infection was 23 months (range 4 to 156 months). The symptoms of patients with joint infections manifested as joint pain (all cases), swelling (14 cases), and fever (15 cases). All patients had oligoarthritis, and the knee was the joint most commonly affected joint. There were 7 patients in the *Salmonella* group and 5 in the *Staphylococcus aureus* group. One patient was infected with *Streptococcus*, and 7 patients were infected with *Mycobacterium*. SLE patients with and without joint infections demonstrated significant differences ($P < 0.05$) regarding the following symptoms: pre-existing arthritis (65.0 vs 33.8%), gastrointestinal involvement (5.0 vs 26.3%), cardiac damage (5.0 vs 31.3%), elevated C-reactive protein (CRP) (80.0 vs 22.5%), and elevated SLE Disease Activity Index (SLEDAI) score (≥5) (30.0 vs 77.5%). When an SLE patient presents with pre-existing arthritis and suddenly develops asymmetric oligo- or large-joint swelling and pain with elevated CRP levels and low disease activity, joint infections should be considered. Early treatment could protect the joint and improve functional outcomes.

Keywords Lupus erythematosus · Systemic · Joint infection

Abbreviations

SLE	systemic lupus erythematosus (SLE)
SLEDAI	systemic lupus erythematosus disease activity index
CTX	cyclophosphamide
LEF	leflunomide
MMF	mycophenolatemofetil
CRP	C-reactive protein
ESR	erythrocyte sedimentation rates
MRSA	methicillin-resistant *Staphylococcus aureus*
MSSA	methicillin-sensitive *Staphylococcus aureus*
INF	isoniazid
RFP	rifampicin
EMB	ethambutol
PZA	pyrazinamide

Introduction

Systemic lupus erythematosus (SLE) is a systemic autoimmune inflammatory disease with variable clinical manifestations. SLE mainly affects females. In the past decades, the survival rate of SLE patients has been greatly improved due to earlier diagnoses and administration of high-dose glucocorticoids and other immunosuppressive agents. With these intensive treatments for active SLE manifestations, the most

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frequent cause of death in patients with SLE is now infection [1, 2]. These infections can be viral, bacterial, fungal, or parasitic involving multiple organs [3]. The most common infection sites are the lung, blood stream, urinary tract, and gastrointestinal system. Joint infections in patients with SLE, which represent a destructive form of acute arthritis, have rarely been described.

The annual incidence of joint infection in the general population varies from 2 to 10 per 100,000 patients [4]. The incidence increases to 28–38 cases per 100,000 in patients with pre-existing inflammatory arthritis [5]. Despite the wide use of antibiotics, the mortality rate for in-hospital joint infections ranges from 7 to 15% [6–10] (most of these deaths are directly attributable to sepsis). Currently, there is still no single confirmatory test available for diagnosing joint infections. Joint infections are difficult to diagnose at an early stage, especially in SLE patients, more than half of whom present with arthritis [11, 12]. If infections are undetected, they can lead to rapid joint destruction. Therefore, identifying an infection and pathogen early is essential for successful treatment. In this study, we investigated the clinical characteristics and risk factors of joint infections in patients with SLE in China.

Patients and methods

Patients

We retrospectively reviewed the medical charts of SLE inpatients admitted to Peking Union Medical College Hospital (PUMCH) from January 1990 to December 2016 in the medical record system. Demographic data, clinical features, laboratory findings, treatments, and outcomes were recorded. Of the 11,734 SLE patients, 20 had joint infections. Subjects without joint infections (designated as control patients) were selected from the pool of SLE patients using a 1:4 systematic sampling method (selected during the same time period as the SLE patients). All patients fulfilled the 2009 American College of Rheumatology revised classification criteria for SLE. Active SLE was defined as an SLE Disease Activity Index (SLEDAI) score ≥ 5. A definitive diagnosis of a joint infection was based on clinical manifestations and one of the following criteria: a positive result for any microorganism in a tissue specimen, joint fluid, blood culture, Gram stain, or acid-fast stain; typical imaging suggestive of joint infections; or effectiveness of an empirical treatment. The institutional review board of PUMCH approved this study. The requirement for written informed consent was waived because this study was retrospective and only involved the review of records.

Statistical analyses

The software package SPSS 18.0 (IBM, Armonk, NY, USA) was used to perform statistical analyses. Means ± standard deviations (SDs) were used for descriptive analyses. A chi-square test and Fisher exact test were used to compare categorical data. An independent sample *t* test was used to compare quantitative data between groups. Statistical significance was set at $P < 0.05$.

Results

Patient demographics and clinical features

The overall prevalence of joint infections in the SLE patients was 0.2% (20 of 11,734). The male-to-female ratio was 1:9. The mean age at onset of a joint infection was 32.2 ± 10.8(range 17–51) years old. Two patients had undergone joint needle aspiration prior to the infection, and only 1 patient had experienced trauma before the joint infection. The duration from the onset of SLE to joint infection varied from 4 months to 13 years (median duration 23 months). The clinical features at presentation are shown in Table 1. Fourteen patients presented with a single joint infection, and 6 patients had infections in 2 joints. The knee joint was infected in 15 patients, followed by the hip (6 cases), the ankle (1 case), and the sacroiliac joint (1 case). Joint pain was the most frequently observed symptom (20 cases, 100%). Joint swelling was noted in 14 patients, except for 5 patients with infection of the hip infections or the sacroiliac joint (swelling of this joint is usually undetectable). Twelve patients experienced joint warmth, while 15 patients experienced fever.

All patients were treated with steroids at the time of SLE diagnosis. The median duration of glucocorticoid treatment was 11 months (range 1–156 months). Eighteen patients received a 1 mg/kg/day equivalent dose of prednisone, and 6 patients underwent methylprednisolone pulse therapy. Following the methylprednisolone pulse therapy, 3 patients were experiencing joint infections 3 months after therapy, and the other 3 patients were experiencing joint infections more than 3 years after therapy. When joint infections were diagnosed, seven patients were managed with a daily dose of prednisone ≥1 mg/kg/day. Four patients received ≥0.5 and <1 mg/kg/day prednisone. Nine patients received <0.5 mg/kg/day prednisone (≥15 mg in 3 cases, <15 mg in 6 cases). Seventeen patients received additional immunosuppressants, and 15 patients received a monotherapy: cyclophosphamide (CTX) (9 cases), leflunomide (2 cases), cyclosporine A (1 case), or mycophenolatemofetil (MMF) (3 cases). Two patients were treated with CTX and MMF (1 case) or CTX and

Table 1 Characteristics of SLE patients with joint infections

No.	Site of joint infection	Pathogens	Resource	Method	Coexisting infection	Treatment
1	Left knee	MSSA	Synovial fluid	Joint aspiration	N	Ceftriaxone sodium
2	Right knee	Suspected Tuberculosis	Unidentified	N	N	INF + RFP + EMB + levofloxacin
3	Right knee	MRSA	Synovial fluid	Joint aspiration	Bacteremia	Vancomycin
4	Both knees	MRSA	Synovial fluid	Joint aspiration	Periarticular abscess	Linezolids + incision and abscess drainage
5	Left hip, left knee	*Salmonella typhi*	Synovial fluid	Arthroscopy, joint aspiration	N	Sulbactam and cefoperazone
6	Both knees	*Salmonella* D	Synovial fluid	Arthroscopy, joint aspiration	N	Ceftriaxone sodium, levofloxacin + synovectomy, joint drainage
7	Left hip	*Mycobacterium*	Synovial pathology	Arthroscopy	Pulmonary tuberculosis	INF + EMB + RFP + PZA + synovectomy, joint drainage
8	Left knee, left ankle	MSSA	Synovial fluid	Joint aspiration, arthroscopy	N	Ceftriaxone sodium, vancomycin + joint drainage
9	Right knee	*Salmonella* D group	Synovial fluid	Joint aspiration	Intestinal infection	Ceftriaxone sodium
10	Left hip	*Mycobacterium*	Synovial fluid	Arthroscopy	Periarticular abscess	INF, EMB, RFP, PZA + incision and abscess drainage
11	Right knee	*Mycobacterium*	Synovial pathology	Arthroscopy	N	INF, EMB, RFP, PZA
12	Both hips	MSSA	Synovial fluid	Joint aspiration	Periarticular abscess, pelvic abscess, bacteremia	Amoxicillin and clavulanate potassium, RFPs + incision and abscess drainage
13	Right knee	*Salmonella typhi*	synovial fluid	Joint aspiration	Periarticular abscess	Penicillin + incision and abscess drainage + joint drainage
14	Left knee	β-hemolytic streptococcus	Synovial fluid	Joint aspiration	Periarticular abscess	Ceftriaxone sodium +incision and abscess drainage
15	Right hip	*Salmonella typhi*	Synovial fluid	Arthroscopy	Bacteremia	Meropenem
16	Right knee	*Mycobacterium*	Synovial fluid	Joint aspiration	Periarticular abscess	INF, EMB, RFP, PZA + incision and abscess drainage
17	Sacroiliac joint	*Mycobacterium*	Synovial fluid	Joint aspiration	Bacteremia	INF, EMB, RFP, PZA
18	Right knee	*Salmonella typhi*	Synovial fluid	Joint aspiration	Bacteremia	Ciprofloxacin
19	Left knee	*Mycobacterium*	Synovial fluid	Joint aspiration	Popliteal cysts solid mass	INF, EMB, RFP, PZA
20	Right knee and hip	*Salmonella typhi*	synovial fluid	Joint aspiration	N	Ceftriaxone sodium + joint drainage

N negative, *INH* isoniazid, *RFP* rifampicin, *EMB* ethambutol, *PZA* pyrazinamide, *MRSA* methicillin-resistant *Staphylococcus aureus*, *MSSA* methicillin-sensitive *Staphylococcus aureus*

Tripterygiumwilfordii (1 case). It is worth mentioning that the treatment of all patients was adjusted after the infection was confirmed.

Pathogens

Seven patients had *Salmonella* infections (5 cases of *Salmonella typhi*, 2 cases of Salmonella D). Five patients had *Staphylococcus aureus* infections (2 cases of methicillin-resistant *S. aureus*, 3 cases of methicillin-sensitive *S. aureus*). One patient was infected with β-hemolytic *Streptococcus*. Seven patients had suspected *Mycobacterium* infections. The pathogen was confirmed in only 6 of these cases, and 1 patient in which *Mycobacterium* was not found (She had acute unilateral knee pain and swelling. MRI revealed cartilage destruction and purulent effusion. Purulent synovial fluid was aspirated from the right knee. The patient was treated with anti-tuberculosis drugs, and the symptoms improved.)

Six patients had simultaneous periarticular abscesses caused by the same pathogen, which was the most common coexisting infection. Bacteremia was found in 3 patients. Other organ infections occurred in 3 patients (pulmonary tuberculosis, intestinal infection, and vertebral infection). Osteomyelitis was noted in 3 patients.

Laboratory characteristics

Leukopenia was identified in three patients (15.0%), and lymphocytopenia was identified in 13 patients (65.0%, mean $0.9 \pm 0.5 \times 10^9$/L). A lymphocyte subset analysis was performed in seven patients. Depressed B cell function was observed in 7 patients (77.8%), and the CD4+ T cell count was decreased in all patients (100%) and was <200 cells/mm^3 in 6 patients (66.7%). Hypoalbuminemia was observed in eight patients (40.0%, mean 31.1 ± 5.4 g/L). Elevated erythrocyte sedimentation rates (ESR) were observed in all patients with joint infections. Elevated C-reactive protein (CRP) levels were observed in 16 patients. The CRP levels were more than 50 mg/L in 10 patients (50.0%).

Imaging examinations of the involved joints were performed for all patients. The examinations revealed evidence of involved joint swelling, soft tissue thickening, and joint space narrowing (Fig. 1). Synovial fluid aspiration was performed for 16 patients. The fluid had a hazy, opaque, or bloody appearance and was characterized by an increased number of leukocytes. Seven patients underwent arthroscopy, and we observed narrowing of the joint space, joint cavities filled with necrotic tissue, synovial hyperplasia, and cartilage destruction. Caseating granulomas were visualized in patients infected with *Mycobacterium*. Infectious organisms were isolated from the synovial fluid cultures of 17 patients (obtained by synovial fluid aspiration or arthroscopy). Infectious organisms were isolated from two patients using synovial tissue pathology.

Comparison of clinical characteristics between SLE patients with and without joint infections

Table 2 shows a comparison of the SLE patients with and without joint infections. The proportion of patients with pre-existing arthritis was significantly higher among the patients with joints infections. However, these patients had less gastrointestinal involvement and cardiac damage. Additionally, the joint infection group had significantly lower SLEDAI scores than the control group. Patients with joint infections had higher incidence of elevated CRP levels (80.0 vs 22.5%, $P < 0.01$) than the control group.

Discussion

Joint infection is a serious condition that can lead to joint and cartilage destruction. Indeed, delayed or inadequate treatment of joint infection can lead to irreversible joint destruction, with subsequent disability. This study is the first serial investigation of joint infections in SLE patients in China. Distinguishing infection from underlying arthritis in SLE patients is a diagnostic challenge of great importance and urgency. Invasion of a pathogen into the synovial space generally occurs by 2 routes: hematogenous spread (most common) or direct invasion [13]. The risk factors for joint infections are penetrating trauma, therapeutic procedures, joint prostheses, periarticular abscesses, pre-existing joint disease, and aging [9, 14]. We observed 2 patients who had joint needle aspiration and 1 patient with trauma prior to joint infection, and no patients had a history of joint prosthesis. Due to their immune dysfunction, the risk factors for joint infections among SLE patients are higher than for normal people, and joint infection among SLE patients occurs at a younger age compared to the general public (32.2 vs 57.5 years old) [14]. Moreover, lymphopenia and depressed B and T cell function are observed in the majority of SLE joint infection cases. Humoral and cellular immunological dysfunction induced by SLE and treatment for SLE is assumed to be responsible for the development of infections, especially at uncommon sites [3, 15, 16]. Findings indicate that lymphocytes should be monitored to avoid excessive inhibition of immunity during treatment. Moreover, the occurrence of pre-existing arthritis was also higher among the SLE patients with joint infections. These symptoms may be related to SLE arthritis which can cause joint congestion and swelling. These symptoms further increase the challenge of differentiating an infection from SLE-associated arthritis at an early stage.

The classic presentation of a joint infection is warmth, swelling, pain, and restricted movement. These symptoms

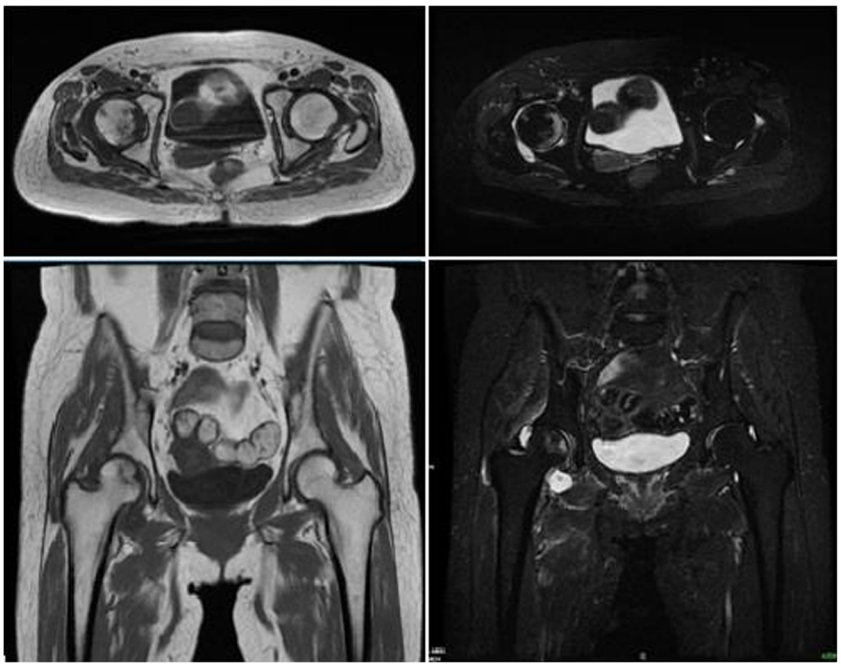

Fig. 1 MRI from an SLE patient with a right hip joint infection. The *right side* of the articular surface was not flat. The femoral head had long T1 and T2 signal intensities with fluid collection (long T1 and T2 signals)

are sometimes accompanied by fever and chill. However, a few patients may not present with classical symptoms. In our study, 25.0% of the SLE patients (5 cases) did not have fever when the joint infections occurred. In addition, disease activity was lower among the patients with joint infections. Of note, 18 patients (90.0%) received a 1 mg/kg/day equivalent dose of prednisone at the time of SLE diagnosis, and 17(85.0%) received additional immunosuppressants.

In this study, the joint infection group had significantly lower SLEDAI scores than the control group. Overall, infections often attributed to the use of corticosteroid and immunosuppressant medications [17]. Most infections occurring in SLE patients tend to be stabilized after progressive therapy. The risk of infection increases after the immune system is suppressed. However, most SLE-associated arthritis is relatively sensitive to glucocorticoids. These features may help us distinguish between SLE-associated arthritis and joint infections. All patients had oligo-arthritis in our series. The most frequently involved joints were large joints, such as hips and knees, which contrasts the multiple, symmetrical arthritis of

Table 2 Comparison of clinical characteristics of SLE patients with and without joint infections

Variable	SLE with joint infections (n = 20)	SLE without joint infections (n = 80)	U/χ^2	p value
Age (years)	32.2 ± 10.8	31.6 ± 12.8	0.968	0.328
Sex ratio(men/women)	2/18	13/67	0.490	0.484
Elevation of SLEDAI score(≥5)[#]	6(30.0)	62(77.5)	16.59	0.000
Oral/nasal ulcer	1(5.0)	15(18.8)	2.251	0.134
Rash	7(35.0)	35(43.8)	0.503	0.478
Photosensitization	3(15.0)	8(10.0)	0.409	0.523
Renal involvement	15(75.0)	45(56.3)	2.344	0.126
Previous joint syndrome[#]	13(65)	27(33.8)	6.51	0.011
Hematological system involvement	13(65.0)	38(47.5)	1.961	0.161
musculoskeletal involvement	9(0)	6(7.5)	1.596	0.207
Nervous system involvement	4(20.0)	20(25.0)	0.219	0.64
Cardiac damage[#]	1(5.0)	25(31.3)	5.73	0.017
Respiratory system involvement	3(15.0)	13(16.3)	0.019	0.892
Gastrointestinal involvement[#]	1(5.0)	21(26.3)	4.210	0.04

[#] $p < 0.05$

hand joints among SLE patients. Therefore, patients who present with a recent emergence or exacerbation of joint symptoms while taking medium-high steroid doses should be evaluated for a joint infection.

ESR and CRP are acute-phase reactants that become elevated during infections and many autoimmune states. According to our data, SLE patients had a higher incidence of CRP elevation ($P < 0.01$), and the CRP levels were greater 50 mg/L in 10 patients. Moreover, all of the patients with joint infections had elevated ESR, but there was no significant difference between the joint infection group and the control group. Many conditions can influence ESR levels. However, CRP levels increase more quickly compared to ESR. Elevated CRP levels are more common among patients with infections compared to SLE patients with active disease (and does not reflect SLE disease activity) [3]. Therefore, CRP levels can distinguish infections from lupus flares, especially when CRP levels are >50.0 mg/L [18]. In other standard laboratory settings, serum procalcitonin showed potential for diagnostic prediction of acute bacterial joint infections and was modestly correlated with fever, CRP, serum, and synovial fluid WBC [19].

Salmonella and *S. aureus* are the predominant pathogens of joint infections [20–22], which is consistent with our results. Huang et al. [21] previously reported that SLE patients with *Salmonella* joint infections were relatively young. Changes in microorganisms involved in joint infections may be related to greater therapeutic aggressiveness (such as antibiotics and biological agents). *S. aureus* was the cause of 70% of joint infections before 2001 and only 35.8% after 2001 [20]. More unusual organisms, such as *Listeria monocytogenes* and *Mycobacterium tuberculosis* [23], caused some infections after 2001, especially in patients treated with TNF inhibitors. Because China has high tuberculosis rates, the prevalence of *Mycobacterium* in SLE patients in China (9.3%) is noteworthy [24]. Approximately 10% of extra-pulmonary tuberculosis cases are bone and joint tuberculosis [25]. In our study, we observed six confirmed cases of *Mycobacterium*, which is much higher than other reports on SLE joint infections (30.0 vs 0.0%) [18, 21]. Tuberculosis may be an occult infection and difficult to prove in some cases. However, eradicating the organism usually results in symptom relief.

Infections that cause joint inflammation must be diagnosed quickly. Because aspirated fluid is easy to check, especially the knee and wrist, joint aspiration is an urgent and vital step for assessing any acutely, hot, swollen joint when a joint infection is possible. For hip and shoulder infections, arthroscopy is often preferred because it allows for easier irrigation and provides better visualization of the joint to identify the pathogen.

Synovial fluid and tissue should be sent for culture, Gram stain, pathology, and polymerase chain reaction-restriction fragment length polymorphism analysis. In our study, pathogens were found by joint aspiration in 15 patients (75.0%), and coexisting infections were also higher among SLE patients with joint infections. Simultaneous examination for potential sites, such as blood culture, may help identify the pathogen. Additionally, if joint infection is suspected, empiric antibiotic therapy should be initiated immediately. The goal of treatment is to rapidly eradicate any infection and protect the joint. Thus, joint drainage or open surgical drainage is crucial for medical treatment, especially during osteomyelitis or hip infections. Early exercise may reduce the destruction of cartilage. The prognosis of joint infections depends mainly on the virulence of the pathogen and timing of effective treatments.

In summary, when an SLE patient with pre-existing arthritis suddenly develops asymmetric oligo- or large-joint involvement, especially when associated with elevated CRP levels and low disease activity, joint infections should be considered. A synovial fluid Gram stain, culture, blood culture, and arthroscopy should be promptly performed, and appropriate antibiotics (or empirical antibiotics) and joint drainage should be administered. Early diagnoses and timely treatments are critical for improving prognosis.

Compliance with ethical standards

Disclosures None.

References

1. Yee CS, Su L, Toescu V, Hickman R, Situnayake D, Bowman S et al (2015) Birmingham SLE cohort: outcomes of a large inception cohort followed for up to 21 years. Rheumatology (Oxford) 54: 836–843
2. Danza A, Ruiz-Irastorza G (2013) Infection risk in systemic lupus erythematosus patients: susceptibility factors and preventive strategies. Lupus 22:1286–1294
3. Zandman-Goddard G, Shoenfeld Y (2005) Infections and SLE. Autoimmunity 38:473–485
4. Clerc O, Prod'hom G, Greub G, Zanetti G, Senn L (2011) Adult native septic arthritis: a review of 10 years of experience and lessons for empirical antibiotic therapy. J Antimicrob Chemother 66: 1168–1173
5. Kherani RB, Shojania K (2007) Septic arthritis in patients with pre-existing inflammatory arthritis. CMAJ 176:1605–1608
6. Gupta MN, Sturrock RD, Field M (2001) A prospective 2-year study of 75 patients with adult-onset septic arthritis. Rheumatology (Oxford) 40:24–30
7. Swan A, Amer H, Dieppe P (2002) The value of synovial fluid assays in the diagnosis of joint disease: a literature survey. Ann Rheum Dis 61:493–498
8. Weston VC, Jones AC, Bradbury N, Fawthrop F, Doherty M (1999) Clinical features and outcome of septic arthritis in a single UK Health District 1982-1991. Ann Rheum Dis 58: 214–219
9. Gupta MN, Sturrock RD, Field M (2003) Prospective comparative study of patients with culture proven and high suspicion of adult onset septic arthritis. Ann Rheum Dis 62:327–331

10. Li SF, Henderson J, Dickman E, Darzynkiewicz R (2004) Laboratory tests in adults with monoarticular arthritis: can they rule out a septic joint? Acad Emerg Med 11:276–280
11. Cervera R, Khamashta MA, Hughes GR (2009) The Euro-lupus project: epidemiology of systemic lupus erythematosus in Europe. Lupus 18:869–874
12. Li M, Zhang W, Leng X, Li Z, Ye Z, Li C et al (2013) Chinese SLE treatment and research group (CSTAR) registry: *I. Major* clinical characteristics of Chinese patients with systemic lupus erythematosus. Lupus 22:1192–1199
13. Ho G Jr (2001) Bacterial arthritis. Curr Opin Rheumatol 13:310–314
14. Margaretten ME, Kohlwes J, Moore D, Bent S (2007) Does this adult patient have septic arthritis? JAMA 297:1478–1488
15. Ruiz-Irastorza G, Olivares N, Ruiz-Arruza I, Martinez-Berriotxoa A, Egurbide MV, Aguirre C (2009) Predictors of major infections in systemic lupus erythematosus. Arthritis Res Ther 11:R109
16. Xu Y, Xu D, Zhang T, Leng XM, Zhang FC, Zeng XF (2012) The prevalence and clinical characteristics of systemic lupus erythematosus with infectious brain lesions in China. Scand J Rheumatol 41:466–471
17. Fei Y, Shi X, Gan F, Li X, Zhang W, Li M et al (2014) Death causes and pathogens analysis of systemic lupus erythematosus during the past 26 years. Clin Rheumatol 33:57–63
18. Kim HA, Jeon JY, An JM, Koh BR, Suh CH (2012) C-reactive protein is a more sensitive and specific marker for diagnosing bacterial infections in systemic lupus erythematosus compared to S100A8/A9 and procalcitonin. J Rheumatol 39:728–734
19. Paosong S, Narongroeknawin P, Pakchotanon R, Asavatanabodee P, Chaiamnuay S (2015) Serum procalcitonin as a diagnostic aid in patients with acute bacterial septic arthritis. Int J Rheum Dis 18:352–359
20. Favero M, Schiavon F, Riato L, Carraro V, Punzi L (2008) Septic arthritis: a 12 years retrospective study in a rheumatological university clinic. Reumatismo 60:260–267
21. Huang JL, Hung JJ, Wu KC, Lee WI, Chan CK, Ou LS (2006) Septic arthritis in patients with systemic lupus erythematosus: salmonella and nonsalmonella infections compared. Semin Arthritis Rheum 36:61–67
22. Madruga Dias J, Costa MM, Pereira da Silva JA, Viana de Queiroz M (2014) Septic arthritis: patients with or without isolated infectious agents have similar characteristics. Infection 42:385–391
23. Lee H, Hwang D, Jeon M, Lee E, Kim T, Yu SN et al (2016) Clinical features and treatment outcomes of septic arthritis due to Mycobacterium massiliense associated with intra-articular injection: a case report. BMC Res Notes 9:443
24. Zhang L, Wang DX, Ma L (2008) A clinical study of tuberculosis infection in systemic lupus erythematosus. Zhonghua nei ke za zhi 47:808–810
25. Chen ST, Zhao LP, Dong WJ, Gu YT, Li YX, Dong LL et al (2015) The clinical features and bacteriological characterizations of bone and joint tuberculosis in China. Sci Rep 5:11084