CASE REPORT

Dilemma in management: a patient with active systemic lupus erythematosus presenting with pulmonary cavitary lesion

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Abstract

Pulmonary manifestations of systemic lupus erythematosus (SLE) include, but are not limited to, pneumonia, interstitial pneumonitis, atelectasis and pleural effusion. Cavitary lung lesions are rarely associated with SLE. We present herein the case of a female patient with SLE and lupus nephritis who presented to the hospital with respiratory failure, rash and arthralgias. She was found to have a cavitary lung lesion most concerning for infection. However, despite an extensive inpatient antibiotic course, her symptoms persisted. After a collaborative effort between the primary team, pulmonology, infectious disease and rheumatology, she was placed on systemic glucocorticoid therapy, which resolved not only her respiratory failure, but also her cavitary lung lesion on subsequent follow-up with imaging. The dilemma of management in such cases will be discussed in addition to a review of previously reported cases.

INTRODUCTION

Cavitary lung lesions are typically associated with bacterial or fungal infection [1] with viral disease occurring more often in immuno-compromised hosts [2]. Other less commonly reported causes include malignancy, pulmonary embolism complicated by necrosis, cryptogenic organizing pneumonia, Langerhans’ cell histiocytosis and rheumatologic disease [1]. We recount this case of cavitary lung disease associated with active systemic lupus erythematosus (SLE) in an attempt to increase provider awareness for early recognition and management of a potentially remedial complication of SLE.

CASE DESCRIPTION

A 36-year-old female with SLE and class II lupus nephritis presented to the emergency room with a 5-day history of severe right-sided pleuritic chest pain that was 10/10 in intensity. Associated symptoms included fever, dyspnea and non-productive cough. Because of inadequate prior access to healthcare, she was not on any medications for her SLE. Pertinent positive of review of systems included a 2-month history of polyarthralgia and facial and upper extremity rash. Vital signs revealed a temperature of 39.1°C, blood pressure 114/61 mmHg, heart rate 102 beats per minute, respiratory rate 24 and oxygen saturation 97% at room air. Physical examination was notable for a malar rash, a raised erythematous rash over bilateral upper extremities, and right upper chest rhonchi.

Laboratory studies revealed leukocyte count 3.7 × 10⁹/L (4.3 × 10⁹–11.3 × 10⁹/L), hemoglobin 10.8 g/dL (12.0–15.3 g/dL), platelet count 145 × 10⁹/L (147 × 10⁹–409 × 10⁹/L), creatinine 2.37 g/dL compared to her baseline creatinine 1.00 g/dL (0.550–1.02 g/dL), blood urea nitrogen 35 mg/dL (7–18 mg/dL), albumin 2.5 g/dL (3.4–5.0 g/dL).

Received: April 10, 2017. Revised: July 18, 2017. Accepted: August 12, 2017

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and total protein 8.5 g/dL (6.4–8.2 g/dL). The remaining electrolytes and liver enzymes were normal. Thyroid-stimulating hormone level was 1.91 mIU/L (0.358–3.740 mIU/L) but erythrocyte sedimentation rate and C-reactive protein were more than 120 mm/h (0–20 mm/h) and 30.6 mg/dL (0.00–0.30 mg/dL), respectively. Chest x-ray (CXR) revealed a right upper lobe opacity and pleural effusion (Fig. 1) that was confirmed on computed tomography with angiography of the chest as a new 5-cm cavitary lesion in the right hilum without evidence of pulmonary embolism (Fig. 2). Review of medical records indicated that the patient presented to her primary care provider 1 month prior to her admission with a 2-week history of non-productive cough, for which a CT chest was done but was without cavitary lesion (Fig. 3). Inpatient, she was started on vancomycin, piperacillin/tazobactam, levofloxacin and fluconazole. Infectious workup was negative, including blood and sputum cultures, and serum screening for cytomegalovirus, coccidioides, cryptococcus, histoplasma and aspergillus. Quantiferon-TB Gold result was indeterminate but subsequent acid-fast sputum culture was negative. Cytology of bronchoalveolar lavage and pleural fluid from thoracentesis were also negative. Despite negative infectious evaluation and antibiotics, pleuritic chest pain and intermittent fever persisted.

Relative to her known diagnosis of SLE, dsDNA-, Smith- and RNP-antibody were positive and ANA titer was 1:640 with speckled pattern. Serum complement C3 and C4 levels were decreased in addition to nephrotic-range proteinuria, implicating active SLE. Due to concern for progression of SLE-induced renal disease, a kidney biopsy was done and yielded a diagnosis of class IV lupus nephritis. In agreement with rheumatology, infectious disease and pulmonary care consultants, the patient was given intravenous methylprednisolone 500 mg daily for three days followed by 1 mg/kg/day. Of note, at the time when corticosteroids were started, the patient was solely on antibiotic therapy for 10 days without clinical or laboratory evidence of improvement. The patient quickly responded to corticosteroids with resolution of pleuritis, rash and polyarthralgia. She remained afebrile with substantial improvement of right upper lobe opacity and pleural effusion on follow-up CXR (Fig. 4). Antibiotics were de-escalated, and, at the time of discharge, the patient was switched to oral prednisone in addition to continuing hydroxychloroquine and mycophenolate mofetil. Repeat chest CT 16 weeks after her hospitalization revealed complete resolution of the lesion without evidence of recurrence or complications such as pneumothorax or pulmonary fibrosis (Fig. 5) also with normalized serum creatinine.

**DISCUSSION**

As previously mentioned, cavitary lung lesions are typically associated with bacterial, fungal and viral infections [1, 2]. While pulmonary manifestations of rheumatologic diseases are variable [3], with the exception of granulomatous with polyangiitis (GPA), cavitary lesions are not typical [1]. The frequency in GPA ranges from 16 to 50% on CT imaging [1]. Other less common autoimmune diseases that are associated with cavitary lung lesions include sarcoidosis and ankylosing spondylitis [1]. Pulmonary manifestations of SLE may include pneumonia, interstitial pneumonitis, atelectasis, pulmonary edema, pulmonary hypertension and diffuse alveolar hemorrhage [4].

As evidenced by a detailed review of the literature, the association between SLE and cavitary lung lesions is rare and its pathogenesis has yet to be established. The vast majority of the reported cases of SLE-related cavitary lung lesions were due to cytomegalovirus infection [2], bacterial/fungal infections,
vasculitis or as a result of necrotic infarction [4]. Torok et al. [5] reported a case of a pulmonary cavitary lesion in a patient who was newly diagnosed with SLE when he presented with the cavitary lung lesion. Lung biopsy was performed with pathology finding of venous thrombosis. The patient’s condition improved with 3 months of prednisone and low molecular weight heparin. In addition, the authors provided a comprehensive review of reported cases of pulmonary cavitary lesion associated with SLE [5]. Consistent with our review, majority of the cases were infectious in origin. Furthermore, none of the previously recorded cases provided information on follow-up condition of their patients (due to death in a few cases), the exact duration of the monitoring effort is not clear. As in our patient, a follow-up with repeat CT chest 4 months after her discharge from the hospital showed resolution of the cavitary lesion without evidence of recurrence or active infection. Further multidisciplinary collaboration between the patient’s primary care physician with rheumatology and pulmonology is essential to providing quality care to complex patient populations as well as to monitor any other pulmonary manifestations relating to rheumatologic conditions in general.

Our case underscores the importance of including SLE in the differential diagnosis of cavitary lung disease and we encourage further reports and research of the same.

ACKNOWLEDGMENTS

None.

CONFLICT OF INTEREST STATEMENT

All authors disclose no conflicts of interest with work involved in this article.

FUNDING

No funding was received for the work involved in this article.

ETHICAL APPROVAL

Not applicable as this article does not involve direct human subject research. No approval was required.

CONSENT

Patient’s consent was obtained for the publication of this case report.

GUARANTOR

All authors nominated Van T. La as a guarantor of the article.
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