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Pulmonary involvement in childhood-onset systemic lupus erythematosus: a report of five cases

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***Objective*. Systemic lupus erythematosus (SLE) is a chronic systemic disease, which can involve multiple organs such as kidney, skin and brain. Lung is another organ that can be affected. A number of pulmonary complications including pleuritis, pneumonitis, infectious pneumonia, pulmonary haemorrhage, pulmonary hypertension and pneumothorax have been reported in patients with SLE. Pulmonary involvement is relatively frequent in adult patients; it has infrequently been reported in children with SLE. However, pulmonary manifestations may be an initial and/or life-threatening complication of SLE in children. In this paper we aim to emphasize the pulmonary involvement in childhood-onset SLE via description of our patients.**

***Methods*. The patients, who were diagnosed with SLE at the Children's Hospital of Ankara University Medical School between 1993 and 2002, were retrospectively evaluated for evidence of pulmonary involvement. All patients fulfilled at least four of the classification criteria of the American Rheumatism Association. Using a standardized form, we obtained data regarding the age, sex and presenting complaints of the patients, previous therapies given, clinical and laboratory features, treatment and outcome. Informed consent was obtained from all patients.**

***Results*. During the 10-yr study period, 16 patients were diagnosed with childhood-onset SLE. Five of them (31%) had pulmonary involvement including acute lupus pneumonitis, invasive pulmonary aspergillosis, cytomegalovirus pneumonia and pulmonary haemorrhage (in two patients). These 5 patients with lupus lung disease are presented in more detail.**

KEY WORDS: Systemic lupus erythematosus, Lupus pneumonitis, Invasive aspergillosis, Pulmonary haemorrhage, CMV pneumonia.

Case 1

This patient presented with profound respiratory symptoms that began abruptly 5 days before admission. Her breathing sounds were diminished and fine crackles were noted bilaterally. Her body temperature was normal. Chest X-ray examination revealed bilateral lower lobe consolidation and obliteration of costodiaphragmatic sinuses (Fig. 1A). A thorax computed tomography (CT) scan confirmed the X-ray findings and revealed minimal pleural and pericardial effusion (Fig. 1B). The diagnosis of multi-lobar pneumonia was established. Despite triple antibiotics (vancomycin, ceftriaxone and clarithromycin) and oxygen therapy, the patient's respiratory distress progressed and mechanical ventilation was required at the fourth day of hospitalization. Blood and bronchoalveolar lavage (BAL) sample cultures were sterile. Serological tests for *Mycoplasma pneumoniae* and *Legionella pneumophila* were negative. A tuberculin skin test was negative. Although the criteria for systemic lupus erythematosus (SLE) were found to be incomplete (Table 1) corticosteroid treatment was initiated. After this therapeutic approach, pulmonary symptoms improved rapidly. The patient was weaned from mechanical ventilation on the 12th day of hospitalization. Antibiotics were given for 14 days. Pulmonary infiltration recovered completely, and the patient was discharged on the 20th day. The patient developed polyarthritis and glomerulonephritis on follow-up and then the diagnosis of SLE was established.

Case 2

This girl was admitted to the hospital with a history of anasarca oedema. The diagnosis of SLE was established (Table 1) and immunosuppressive therapy was initiated. On the 14th day of therapy, pulmonary symptoms appeared. Although fine crackles were noted on the right hemithorax, chest X-ray examination was within normal limits. Atypical pneumonia was considered, and clarithromycin therapy was initiated. However, her chest pain persisted and spontaneous pneumothorax was demonstrated by chest X-ray examination 2 days later (Fig. 2). Closed tube drainage was performed immediately, and the therapy was changed to vancomycin, meropenem and fluconazole. However, her clinical condition deteriorated and a bronchopleural fistula developed on 4th day of closed tube drainage. A thorax CT revealed pneumothorax, consolidation, atelectasis and minimal pleural effusion on the right. The patient was operated on and the bronchopleural fistula was repaired. A culture from the excised fistula was positive for *Staphylococcus aureus*. Histopathological examination of excised tissue revealed non-specific inflammatory changes. Despite treatment with vancomycin, meropenem and fluconazole, the patient's fever persisted. Repeated thorax CT revealed bibasilar consolidation, linear atelectasis and paravertebral abscesses on the right lung (Fig. 3). Thorax CT-guided abscess drainage was performed and *Aspergillus niger* was isolated from the obtained purulent abscess material. Fluconazole was discontinued and liposomal amphotericin B (L-AmB) 5 mg/kg/day was initiated.

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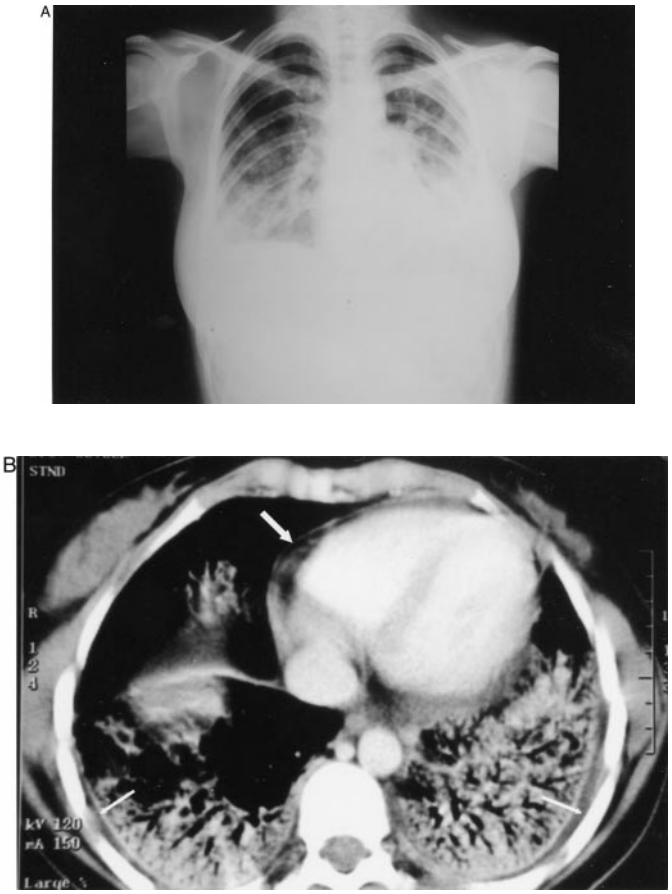


FIG. 1. A: Bibasilar infiltrates on chest X-ray. B: Simultaneous thorax CT shows bilateral lower lobe consolidation, and minimal pleural (thin arrows) and pericardial effusion (thick arrow).

The patient's fever resolved gradually, and L-AmB dosage was reduced to 3 mg/kg/day on the 30th day of the treatment and itraconazole 2 × 200 mg/day was added to the therapy. L-AmB was discontinued on the 50th day of therapy, and itraconazole was continued for another 2 months. The patient's chest examination and thorax CT were normal at the end of the therapy.

Case 3

This girl was admitted to the hospital with pulmonary symptoms 1 year after the diagnosis of SLE. On admission, fine crackles were noted at the bottom of both hemithoraxes. Chest X-ray showed bilateral interstitial infiltration. Cytomegalovirus (CMV) early antigen, anti-CMV IgM and IgG, and urinary CMV deoxyribonucleic acid (DNA) were positive. The diagnosis of active CMV infection and CMV pneumonia was established and ganciclovir therapy 10 mg/kg/day was initiated. The patient's respiratory symptoms and pulmonary infiltration were completely resolved by the first month of ganciclovir therapy. The dose of the drug was reduced to 5 mg/kg/day orally and continued for 4 months. CMV early antigen, anti-CMV IgM and urinary CMV DNA were found to be negative at the follow-up.

Case 4

This girl who had been followed with peritoneal dialysis for 3 yr was admitted to our hospital with a 3-day history of pulmonary symptoms. On admission, she had severe dyspnoea and pallor, but she had no fever. Fine crackles were noted on both hemithoraxes. Laboratory examinations included a haemoglobin level of 8.7 g/dl, leucocyte count of 10 100/mm^3 and platelet count of 117 000/mm^3. The erythrocyte sedimentation rate (ESR) was 50 mm/h, the C-reactive protein level was 0.68 mg/dl, the serum urea nitrogen level was 159 mg/dl and the serum creatinine level was 9.4 mg/dl. Chest X-ray examination showed bilateral perihilar patchy infiltrates. The diagnosis of pneumonia was considered and ceftriaxone therapy was initiated. However, the patient's dyspnoea persisted, her haemoglobin level decreased to 6.7 g/dl, pulmonary infiltration progressed and haemoptysis occurred. Coagulation tests were within normal limits and there were no haemolysis findings. Serological tests for *M. pneumoniae* and *L. pneumophila* were negative. Because of the unexplained abrupt decrease in haemoglobin value, haemoptysis and the concomitant increase in breathing difficulty the possibility of acute pulmonary haemorrhage was considered. A CT scan of the thorax revealed bilateral perihilar consolidation that supported the diagnosis of pulmonary haemorrhage. Haemosiderin-laden macrophages were demonstrated in BAL samples. BAL sample and blood cultures were sterile. The girl was treated with corticosteroids and erythrocyte transfusions. Her clinical condition improved gradually. After 22 days of hospitalization, she was discharged. The patient's chest X-ray and chest respiratory examination were normal at the control visit performed 1 month later.

Case 5

This girl with SLE who had been followed for 10 years was admitted to our hospital with a history of serious respiratory symptoms and pallor (Table 1). Laboratory examinations included a haemoglobin level of 6.7 g/dl, leucocyte count of 9000/mm^3 and platelet count of 112 000/mm^3. Sedimentation rate and C-reactive protein level were 40 mm/h and 0.88 mg/dl respectively. She had no fever and no findings consistent with haemolysis. Because of dyspnoea, haemoptysis, anaemia and pulmonary infiltration on chest X-ray examination, pulmonary haemorrhage was considered. The diagnosis of acute pulmonary haemorrhage was supported by the presence of haemosiderin-laden macrophages in sputum samples. After the treatment with corticosteroids and erythrocyte transfusion her clinical condition improved rapidly. Her chest X-ray returned to normal at the control visit performed 1 month later.

Discussion

It has been reported that pleuropulmonary manifestations are seen in approximately 50–70% of adult patients with SLE [1, 2]. Although pulmonary involvement in SLE has rarely been systematically investigated in children, pulmonary involvement is reported to be 5 to 67% [3–5]. Our retrospective survey for the last 10 yr shows that 5 of our 16 patients (31%) diagnosed with SLE developed pulmonary manifestations. Pulmonary hypertension, diffuse interstitial disease, pulmonary haemorrhage, pneumothorax, acute lupus pneumonitis and shrinking lungs have infrequently been reported in previous studies [3–6]. However, pleuropulmonary infections and subclinical lung disease are relatively common in childhood-onset SLE [4, 7].

Our first patient demonstrates that SLE patients might present with pulmonary involvement as the first manifestation. Similarly, Matthay *et al.* [8] reported that 6 of 12 SLE patients with pulmonary involvement presented with acute lupus pneumonitis at the time of diagnosis. Most of these patients had high fever, dyspnoea, tachypnoea, tachycardia and cyanosis. The most common auscultatory findings were bibasilar fine crackles. The predominant findings on chest X-ray examination were patchy areas of increased density, atelectasis, diaphragmatic elevation and

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TABLE 1. Clinical and laboratory features of 5 patients with lupus lung disease

Features	Case 1	Case 2	Case 3	Case 4	Case 5
Sex	F	F	F	F	F
Ethnicity	Turkish	Turkish	Turkish	Turkish	Turkish
Age at onset of SLE (yr)	14	16	13	12	13
Laboratory findings at onset of SLE					
Haemoglobin (g/dl)	13.3	10.1	12.0	9.0	7.0
Leucocytes (per mm^3)	10 600	2100	2700	3600	5800
Platelets (per mm^3)	285 000	111 000	171 000	282 000	108 000
ESR (mm/h)	15	114	44	90	43
ANA	(++)	(++)	(+)	(++++)	(+)
Anti-dsDNA (IU/ml)†	Normal	67	>100	>100	>100
C3 (mg/dl)†	75.8	36.8	53	18.7	38
C4 (mg/dl)†	14.4	4.0	8	11.5	11.6
Direct Coombs test	Negative	(++++)	Negative	(++++)	Negative
Urine examination	Normal	Gross haematuria and proteinuria	Proteinuria	Microscopic haematuria and proteinuria	Trace proteinuria and microscopic haematuria
Renal biopsy	Not performed	Diffuse proliferative glomerulonephritis	Diffuse mesangial proliferation	Membranous glomerulonephritis	Non-specific findings
Diagnostic criteria for SLE	Photosensitivity, serositis, positive test for ANA (arthritis and renal involvement later)	Renal involvement, haematological disorders, positive tests for ANA and anti-dsDNA	Photosensitivity, aphthous stomatitis, malar rash, arthritis, serositis, renal involvement, haematological disorders, positive tests for ANA and anti-dsDNA	Malar rash, arthritis, serositis, renal involvement, haematological disorders, positive tests for ANA and anti-dsDNA	Malar rash, renal involvement, haematological disorders, positive tests for ANA and anti-dsDNA
Treatment for SLE	P-MP followed by oral prednisone	P-MP followed by oral prednisone	P-MP followed by oral prednisone and P-CYC	P-MP followed by oral prednisone and P-CYC	P-MP followed by oral prednisone and P-CYC
Age at onset of pulmonary involvement (yr)	14	16	14	18	22
Respiratory symptoms and signs	Cough, dyspnoea, tachypnoea, cyanosis, diminished breath sounds and fine crackles	Fever, cough, chest pain, tachypnoea, fine crackles	Fever, cough, dyspnoea, tachypnoea, fine crackles	Cough, dyspnoea, tachypnoea, haemoptysis, pallor, fine crackles	Cough, dyspnoea, tachypnoea, haemoptysis, pallor, fine crackles
Radiographic findings	Infiltration	Infiltration, pneumothorax, lung abscesses	Infiltration	Infiltration	Infiltration
Pulmonary complication	Acute lupus pneumonitis	Invasive pulmonary aspergillosis	CMV pneumonia	Pulmonary haemorrhage	Pulmonary haemorrhage
Treatment for pulmonary complications	P-MP followed by prednisone	L-AmB and itraconazole	Ganciclovir	P-MP and erythrocyte transfusions	Prednisone and erythrocyte transfusions

†Normal ranges for anti-dsDNA, C3 and C4 are 0–7 IU/ml, 79–152 mg/dl and 16–38 mg/dl, respectively.
L-AmB, liposomal amphotericin B; P-MP, pulse methylprednisolone; P-CYC, pulse cyclophosphamide.

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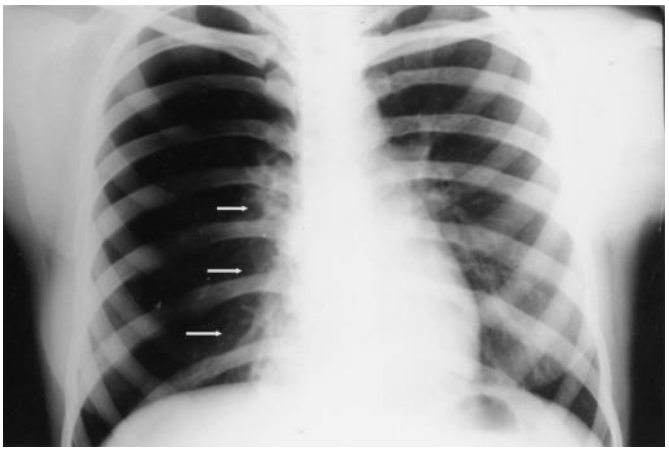


FIG. 2. Right-sided pneumothorax and lung atelectasis (arrows).

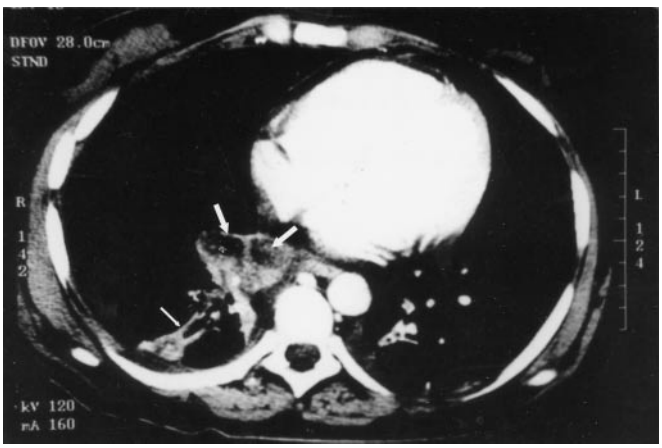


FIG. 3. Axial CT image demonstrating right-sided linear atelectasis (thin arrow) and paravertebral abscesses (thick arrows).

chronic interstitial infiltrates. All patients received corticosteroids and more acutely ill patients received azathioprine. Our first patient had only three criteria for SLE diagnosis including photosensitivity, serositis, and an abnormal titer of antinuclear antibodies (ANA) at the initial evaluation [9]. Because the pneumonia did not resolve with antibiotics, and the aetiological agent was not demonstrated, a diagnosis of SLE was suspected. The patient's pulmonary findings were resolved with corticosteroids and two other SLE criteria appeared during follow-up. We think that corticosteroid therapy was a lifesaving approach in this patient. We suggest that a high degree of suspicion for the diagnosis of SLE is required in patients with severe pulmonary involvement, even when they do not fulfil the American College of Rheumatology criteria. This approach might enable the initiation of the life-saving corticosteroid therapy as in our patient.

Because infections are the leading cause of death in patients with SLE, all pulmonary infiltrates in SLE should be considered infectious in origin until proven otherwise. This requires a thorough and aggressive approach using appropriate cultures and, if needed, fibreoptic bronchoscopy, transbronchial biopsy and open lung biopsy. The infectious pulmonary infiltrations are caused by viruses, bacteria, fungi and protozoa [1, 10–12]. To the best of our knowledge, approximately 33 SLE patients with invasive aspergillosis have been reported in the English-language literature [13–30]. Most of these patients died and the diagnosis was established during postmortem examination. The similarity between the clinical findings of SLE and invasive aspergillosis was reported as the cause for delayed diagnosis [13]. Our second patient is one of the rare SLE patients with invasive aspergillosis who was diagnosed during the pneumonia episode and was treated successfully. Neutropaenia and immunosuppressive therapy are reported as the leading causes predisposing to invasive aspergillosis in SLE as in our patient [13]. Although the optimal duration of antifungal therapy in invasive pulmonary aspergillosis is controversial, treatment with high-dose amphotericin B for 4–6 weeks or until resolution of neutropaenia or remission of the underlying disease is recommended [31]. Our patient received L-AmB for approximately 7 weeks and itraconazole for 2 months. Our experience underlined that invasive pulmonary aspergillosis must be taken into consideration as a cause of pneumonia in SLE patients. All of the few reported patients with CMV infection had pneumonia and two of these patients died [32–37]. Our patient with CMV pneumonia was successfully treated with ganciclovir. Although it is infrequently reported, CMV infection should not be overlooked in SLE patients with pneumonia.

Pulmonary haemorrhage secondary to SLE is a relatively uncommon and frequently lethal complication. Mortality approaches up to 80% despite the use of high-dose corticosteroids and other immunosuppressive agents [1, 10]. Common clinical features include cough, dyspnoea, fine crackles, severe hypoxaemia and sudden drops in haematocrit levels. Because not all patients demonstrate haemoptysis, a high degree of suspicion is necessary for prompt recognition. Our two patients with pulmonary haemorrhage had cough, haemoptysis and anaemia. Both patients were successfully treated with high-dose corticosteroids and supportive therapy.

Invasive procedures to obtain tissue samples for microbiological and histopathological studies can provide valuable information in lupus lung disease. Bronchoscopy, bronchoalveolar lavage, transbronchial biopsy and open lung biopsy have been previously used [1, 2, 4, 6, 10, 13]. We successfully performed a CT-guided transthoracic needle aspiration in the patient with invasive pulmonary aspergillosis. An open lung biopsy might have been valuable in our first case. Acute alveolar damage with interstitial oedema, hyaline membranes and immune complex deposition in pulmonary tissue has been demonstrated in patients with acute lupus pneumonitis [1, 10]. However, this intervention could not be performed because of the poor clinical condition of the patient. CMV infection was easily detected by non-invasive procedures in our patient 3, and lung biopsy was unnecessary. The diagnosis of pulmonary haemorrhage was clinically obvious in our two patients, and the presence of haemosiderin-laden macrophages supported the diagnosis. We think that lung biopsy was unnecessary and even dangerous in these two patients. There are no well-defined recommendations for lung biopsy indications in SLE patients with respiratory symptoms. We suggest that invasive procedures should be performed to explore the causes of unexplained and unresolved infiltrations. In particular, opportunistic infections such as tuberculosis, aspergillosis and nocardiosis may only be demonstrated by tissue cultures or histopathological examinations [13, 38, 39].

In conclusion, the lung is a major organ involved in childhood SLE, and early diagnosis and appropriate treatment may improve the prognosis.

The authors have declared no conflicts of interest.

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