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CASE REPORT

Cerebellar involvement that occurred during treatment of *Legionella* pneumonia: A case report

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ABSTRACT

Legionnaires' disease can appear with different levels of severity. A case of a previously healthy lady with community-acquired pneumonia who progressed to severe acute respiratory distress syndrome and developed cerebellar dysfunction is reported. In patients presenting with neurological symptoms after an episode of pneumonia, *Legionella* infection should be considered. *J Microbiol Infect Dis 2013; 3(2): 83-85*

Key words: *Legionella*, cerebellar dysfunction, dysarthria, ataxia

Legionella pnömonisi tedavisi sırasında ortaya çıkan serebellar tutulum: Bir vaka sunumu

ÖZET

Lejyoner hastalığı farklı klinik tablolar ile karşımıza çıkabilmektedir. Bu vakada toplumdan kazanılmış *Lejyonella* pnömonisi tedavisi görmekte olan sağlıklı bir kadın hastanın, tedavisi sırasında ciddi akut solunum sıkıntısı sendromu ve serebellar fonksiyon bozukluğu gelişmesi bildirilmiştir. Pnömoni ile birlikte nörolojik semptomların görülmesi *Lejyonella* enfeksiyonunu akla getirmelidir.

Anahtar kelimeler: *Lejyonella*, sebellar tutulum, dizartri, ataksi

INTRODUCTION

Legionnaires' disease is one of the causes of atypical community-acquired pneumonia (CAP) which commonly presents as severe CAP requiring hospitalization and intensive care.[1,2] Legionnaires' disease is a systemic infection involving the lungs and rarely accompanied by extrapulmonary organ involvement, including the central nervous system.[1,3] The most common neurologic symptom is encephalopathy that presents broad spectrum of symptoms that range from confusion to coma.3 Other rare of central venous system (CNS) involvements include cerebellar dysfunction, encephalomyelitis, peripheral neuropathy, and myositis.[3]

We report herein a previously healthy woman who developed persistent cerebellar dysfunction that occurred during treatment of *Legionella* pneumonia.

CASE REPORT

A 40-year-old woman presented to the emergency department (ED) with five days of fatigue, abdominal pain, and high grade of fever. Upon admission she was febrile to 38°C, physical examination was normal. Only urinalysis revealed erythrocyte and 6-8 leukocyte per high power field (normal 0-3). Urinary tract infection was considered, and cefuroxime axetil was given orally.

On the following day, she presented to the ED again with fever, vomiting, urinary incontinence and confusion. She fell down to the ground with loss of consciousness at her home. On this second admission to the hospital, she was febrile to 39.7°C with a respiratory rate of 24 breaths/min. Her blood pressure was 130/85 mmHg, and heart rate was 110/min and arterial oxygen saturation was 95% on room air. Other findings of her physical examination were unremarkable, including those for the lungs, which were clear to auscultation. Neurological examination was normal except for mild neck stiffness. Her past and family history was not significant.

A chest radiograph showed that the extensive consolidation on upper lobe of the right lung (Figure 1). Abnormal laboratory data were as follows: White

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blood cell (WBC) count 19.200/mm3 with 86% of neutrophils, sodium 124 mmol/L, creatinine 1.4 mg/dL, lactate dehydrogenase (LDH) 465 U/L, C-reactive protein 46 mg/dL, and erythrocyte sedimentation rate of 103 mm/hr. Computerized tomography (CT) scan of the brain was normal. Lumbar puncture (LP) was performed and we observed cerebrospinal fluid (CSF) examination was normal. No organism observed on direct microscopy of CSF.

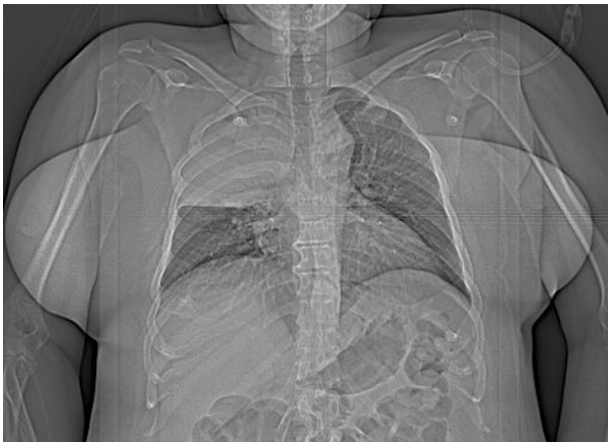

Figure 1. Chest radiograph of extensive consolidation present upper lobe of the right lung.

She was admitted to the Infectious Diseases Department with the diagnosis of CAP and administered a combination of ceftriaxone and azithromycin empirically. On the second day of admission, her clinical condition worsened and she showed respiratory distress (30 breaths/min). Arterial blood gas analysis revealed partial O2 pressure (PaO2) 89 mmHg. She was admitted to the intensive care unit (ICU) and placed on CPAP initially. Because of history of a trip and staying in several hotels in the last 2 weeks before beginning of the symptoms, and hyponatremia and confusion, we added rifampicin (600 mg/day) to support the azithromycin action against possible *Legionella* pneumonia.

A CT scan of her chest revealed multiple areas of parenchymal consolidation in the entire upper right pulmonary lobe with 21 mm pleural effusion (Figure 2). No organism was isolated on CSF, urine and blood cultures. On the fourth day, the patient's hypoxemia increased (arterial blood PaO2 50 mmHg). The patient was placed on mechanical ventilation and clarithromycin, ceftriaxone and rifampicin was stopped, intravenous levofloxacin 1000 mg per day was given. No organism was isolated on endotracheal aspirate culture. On the seventh day of admission, *Legionella* 1 antigen was detected in the urine, the diagnosis of *Legionella* pneumonia was confirmed. The hotels that she was stayed were investigated by the health authority and *Legionella* was isolated from water system by one of the hotels. She was continued receiving levofloxacin for 3 weeks. After the 11 days of hospitalization, her clinical findings gradually improved, and the patient was taken off the ventilator.

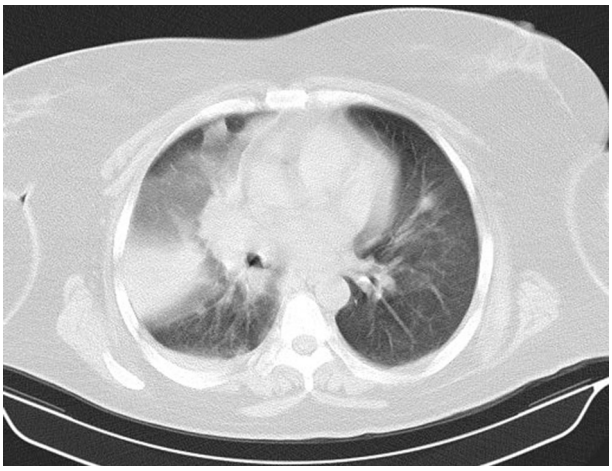

Figure 2. CT scan of parenchymal consolidation in the upper right pulmonary lobe.

On the 12th hospitalization day, she suffered from malaise and anxiety, and she developed hallucinations, slurred speech and difficulty with sitting and standing. Examination of her sensory and motor systems was normal. She had severe dysarthria, ataxia, and impaired finger-to-nose test. An electroencephalogram (EEG) and magnetic resonance imaging (MRI) scan of the brain were all within normal limits. No medical treatment, but only clinical observation was suggested by neurologist. The patient was discharged after 25 days of admission. After two months of discharge, her mild speech deficits and mild ataxia remained but she achieved unsupported walk. The chest X-ray showed that the pneumonia consolidation had completely resolved.

DISCUSSION

The signs and symptoms of Legionnaire's disease are non-specific and are similar to those of an atypical pneumonia, but may be more severe.[4] The most of the disease's symptoms are cough, high grade of fever, dyspnea, diarrhea, nausea, vomiting and abdominal pain due to multisystem involvement.[4,5]

In Legionnaire's disease neurological complications occur in 4-53% of cases.4 Common neurological symptoms include headache, somnolence, and varying degrees of encephalopathy.[5] Most of these involve encephalopathic signs like altera-

tions of mental status, confusion, disorientation, hallucinations and personality changes.[6] Less frequent presentations are brain stem and cerebellar dysfunctions, peripheral neuropathies, pyramidal tract dysfunction, cranial nerve palsies, memory loss, seizures and affective disorders. Cerebellar dysfunction is rare but well documented in Legionnaires' disease.[5,7-14] There are only case reports and no prospective studies of neurological manifestations in the literature due to limited number of cases. Shelburne et al.3 have reported 29 cases of cerebellar dysfunction in Legionnaires' disease and ataxia (72%) and dysarthria (79%) were reported as most frequent cerebellar symptoms. Plaschke et al.15 have also reported cerebellar dysfunction rate of 11.2% in 609 *legionella* infected patients with involvement of the CNS. The incidence of cerebellar dysfunction was reported in another study as 3.7% among the cases.[14]

In this case, the alteration of mental status and confusion were seen at the time of admission. Ataxia and dysarthria were seen as the late neurological manifestations. Shelburne et al.[3] reported that the ataxia and dysarthria have arisen a few days to one week after onset of the illness. In this case time from the onset of pulmonary symptoms to the onset of cerebellar symptoms was 12 days.

The pathogenesis of neurological dysfunction in Legionnaires' disease remains unclear.[5,6] Various theories have been proposed including direct invasion of brain parenchyma by the agent, the effect toxins to brain cells, or immune-mediated involvement of brain system or reversible ischemia.[5,16] The presence of *Legionella*e in the CNS has been showed in only rare instances.[3] The vast majority of analyses of CSF and post mortem brain examinations have been normal, suggesting that microorganism does not affect directly to the CNS.[3,6] Abnormalities on cranial CT or MRI scanning are rare, whereas the EEG often shows diffuse slowing consistent with toxic encephalopathy or it may be normal.[3,8] In this patient, performed LP, brain CT, MRI and EEG, all were normal. Although utility of CSF analysis is undetermined, it may helpful to rule out other infective causes. In the presence of neurological symptoms, the treatment modality such as antibiotics and supportive care does not have change. The prognosis of neurological deficits is not well studied. Cerebellar deficits may persist for a long time.

CONCLUSION

In conclusion, because of the limited number of cases, we believe that it is important to report of the Legionnaire's disease with neurological complications. Especially in countries which are difficult to reach specific diagnostic tests for the disease including Turkey, Legionnaires' disease should be strongly considered in patients having neurological symptoms accompanied by symptoms and signs of pneumonia especially. In such a setting, full evaluation has to be performed to exclude other etiologies. Failure to recognize and treat the infection may lead to poor outcomes.

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