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CASE REPORT

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Legionella pneumonia caused by *Legionella pneumophila* serogroup 2: second case report in Japan

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Abstract A 56-year-old man with a 3-day history of a chilly sensation and general fatigue presented to a hospital in his neighborhood. He was diagnosed as having pneumonia and immediately treated with intravenous ceftriaxone sodium, but his respiratory condition deteriorated and he developed symptoms of restlessness. Although *Legionella* urinary antigen detection tests were negative, his clinical course suggested *Legionella* pneumonia. After his treatment was changed to intravenous ciprofloxacin and oral clarithromycin, his general condition gradually improved. Later, *Legionella pneumophila* serogroup 2 was isolated from a bronchoalveolar lavage specimen. This was considered to be the causative organism. In our literature search, this was only the second case of *Legionella* pneumonia caused by *Legionella pneumophila* serogroup 2 in Japan.

Key words *Legionella* pneumonia · *Legionella pneumophila* serogroup 2 · Urinary antigen

Introduction

Among adults with community-acquired pneumonia, *Legionella* pneumonia has the potential to spread and be a fatal type of pneumonia similar to pneumococcal pneumonia.[1] *Legionella* pneumonia accounts for about 3% of community-acquired pneumonia, and most cases are caused by *Legionella pneumophila* serogroup (SG) 1.[1] *Legionella* urinary antigen detection tests are an effective tool for diagnosing *Legionella* pneumonia with high sensitivity and specificity. However, the sensitivity of *Legionella* infections, with the exception of *L. pneumophila* SG 1, is very low,[2,3] and so care must be taken in evaluating results. We present a case of *Legionella* pneumonia caused by *L. pneumophila* SG 2, for which a diagnosis was made by isolation of the bacteria, but in this case the *Legionella* urinary antigen detection tests were negative. This was only the second case of *Legionella* pneumonia caused by *L. pneumophila* SG 2 to be reported in Japan.

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Case report

A 56-year-old man, who presented to a general hospital in his neighborhood, had a 3-day history of a chilly sensation, nausea, and general fatigue, but no productive cough. He had a past history of tuberculous peritonitis, terminal ileitis, and cerebrovascular disease. He had smoked 80 pack-years, and his occupation was bus driver.

On admission, he was clearly conscious, and his vital signs were as follows: blood pressure 136/74 mmHg, pulse 102/min and regular, body temperature 39.8°, and oxygen saturation by pulse oxymetry (SpO_2) at 94% with 1 l/min oxygen therapy. A physical examination revealed no significant findings other than the abnormal vital signs. Neither crackle nor wheeze/rhonchi were heard in his chest, and no neurological deficit was recorded. Laboratory findings on admission showed a white blood cell count of 10700 cells/mm^3, C – reactive protein (CRP) of 31.02 mg/dl, and slight liver damage (Table 1). A chest roentgenogram on admission showed consolidation in the right middle lung field (Fig. 1). Expectorated sputum of satisfactory quality was not obtained.

The patient was treated with intravenous ceftriaxone sodium (CTRX) for 2 days after admission, but his condition deteriorated and symptoms of restlessness developed. On the third hospital day, his SpO_2 88% with 5 l/min oxygen therapy and inflammatory markers elevated further to a white blood cell count of 12100 cells/mm^3 with 89.8%

Table 1. Laboratory data

CBC		Blood chemistry		Serum	
WBC	10700/mm^3	TP	6.2 g/dl	CRP	31.02 mg/dl
RBC	436×10^4/mm^3	ALB	3.0 g/dl		
HGB	13.0 g/dl	GLU	131 mg/dl	Urinalysis	
MCV	89.7 fl	BUN	16 mg/dl	pH	6.0
MCH	29.8 pg	CRE	0.87 mg/dl	Protein	3+
MCHC	33.2%	Na	134 mEq/l	Sugar	–
HCT	39.1%	K	3.9 mEq/l	Ketone bodies	3+
PLT	22.7×10^4/mm^3	Cl	95 mEq/l	Occult blood	2+
		T.Bil	2.3 mg/dl	Urobilinogen	2+
		AST	58 IU/l	Bilirubin	2+
		ALT	60 IU/l		
		ALP	334 IU/l		
		LDH	501 IU/l		
		γ-GTP	208 IU/l		
		CPK	774 IU/l		
		AMY	120 IU/l		

CBC, complete blood count; HGB, hemoglobin; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; HCT, hematocrit; PLT, platelet; TP, total protein; ALB, albumin; BUN, blood urea nitrogen; CRE, creatinine; T.Bil, total bilirubin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; LDH, lactate dehydrogenase; γ-GTP, γ-glutamyl transpeptidase; CPK, creatine phosphokinase; AMY, amylase; CRP, C-reactive protein

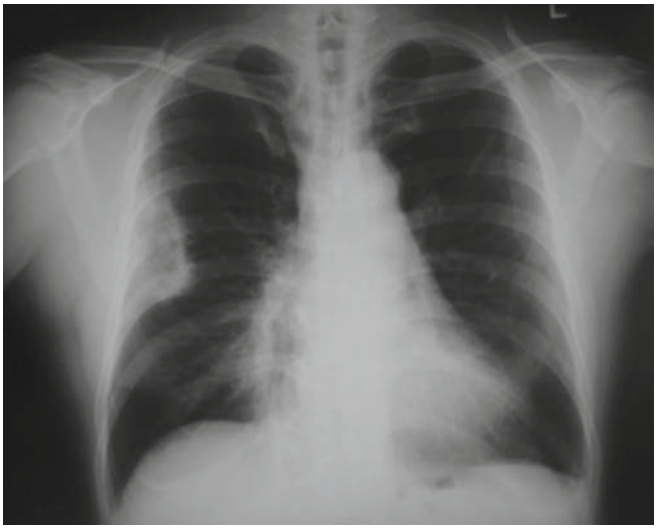

Fig. 1. A chest roentgenogram on admission showing consolidation in the right middle lung field

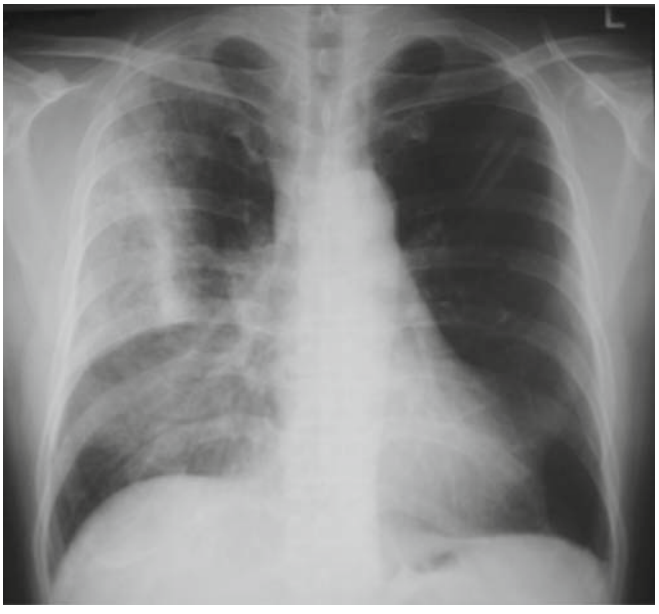

Fig. 2. A chest roentgenogram on the third hospital day, showing extended consolidation and a ground-glass shadow in the right lung fields

neutrophils, CRP of 51.30 mg/dl, and lactate dehydrogenase (LDH) of 501 IU/L. His chest radiographic findings showed that the consolidation extended with a ground-glass shadow in the right lung field (Fig. 2), and a chest computed tomography (CT) scan showed consolidation and a ground-glass shadow with pleural effusion over the right lobes (Fig. 3). He was suspected of having *Legionella* pneumonia because of the high fever, extraordinary elevation of inflammatory markers, slight liver damage, no effect of third-generation cephalosporin, and his history of using public baths for bathing. Although *Legionella* urinary antigen detection tests of his urine were negative, his treatment was changed to intravenous ciprofloxacin (CPFX) (600 mg/day) and oral clarithromycin (CAM) (400 mg/day) after bronchoalveolar lavage (BAL) on the third hospital day. In addition, to address the severe pneumonia he was given intravenous hydrocortisone (200 mg/day). Within 3 days, the patient was afebrile and his general condition was gradually improving (Fig. 4). Consolidation had extended over the right lung fields for a time, but the shadow was getting better. Therefore, we judged the treatment to be effective. Since his liver damage was getting worse after the administration of CPFX and CAM, a switch was made to intravenous pazufloxacin mesilate (PZFX) (1000 mg/day) on the 8th hospital day. It was then switched to oral gatifloxacin (GFLX) on the 15th hospital day to correspond with his discharge from hospital. The fibrotic change in the upper right lung field was still