

Letter to the editor

**Legionnaires' disease as an occupational risk related to decontamination work after the Fukushima nuclear disaster: A case report**

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**Key words:** Antibiotics, Decontamination, Legionnaires' disease, Urinary antigen test

Dear Editor:

Sawano et al<sup>1)</sup> have reported a 53-year-old male with Legionnaires' disease (LD) during the engagement into the decontamination work in Fukushima. Laboratory examinations showed decreased potassium concentration and significantly elevated serum creatine kinase without renal failure on admission day. LD is a well-known bacterial cause of rhabdomyolysis among numerous infectious agents. Legionellosis-induced rhabdomyolysis is thought to be due to endotoxins or exotoxins and direct bacterial invasion<sup>2)</sup>. In striking contrast to Pontiac fever, which is an influenza-like illness characterized by fever, chills, headache and myalgia, LD is a severe pneumonia type which is often accompanied by altered mental status and unresponsiveness to conventional antibiotics such as  $\beta$ -lactam antibiotics and ampicillin-sulbactam<sup>3)</sup>. Despite the atypical pneumonia, because the white-blood-cell count was over 10,000, complications with other types of bacterial pneumonia should have also been considered in this case. Given the positive result of the urinary antigen test (UAT), the antibiotic regimen was changed to levoflox-

acin. However, multiple organ failure led to the subject's death 5 days after admission to the hospital<sup>1)</sup>. While it is challenging to suspect the possibility of LD and make an early diagnosis, because legionellosis can be effectively treated with antibiotics such as macrolides, quinolones, or tetracyclines, we medical doctors should always keep LD in mind when making a differential diagnosis, and rapidly perform both the UAT and polymerase chain reaction (PCR) even if occupational exposure has not been clearly demonstrated.

Decontamination work includes the removal of topsoil in mountainous areas and cleaning roads and the roofs of residential buildings with high-pressure water, which frequently exposes workers to water and soil. Furthermore, the poor living conditions of the decontamination workers may increase the risk of developing legionellosis in this occupational setting. *Legionella pneumophila* thrive in water systems maintained at warm temperatures between 26.7 and 48.9 °C<sup>2,4)</sup>. Mechanistically, the biofilm-derived *L. pneumophila* evades the innate immune response in alveolar macrophages<sup>2)</sup>. Thus, the appropriate cleaning and maintenance of potential environmental reservoirs is essential for preventing LD outbreaks.

However, there are two major points which I think should be further discussed. Firstly, at present only 3 cases of LD have been reported among the large number of decontamination workers in Fukushima Prefecture. This is not at all typical of pandemic legionellosis, because approximately 30,000 to 40,000 workers are involved in the decontamination as of this writing, most of whom have lived in shared spaces<sup>1)</sup>. The intracellular pathogen *L. pneumophila* spreads in water and ventilation systems, in which aerosolization is likely to occur<sup>3)</sup>. Because the number of cases remains quite low despite these conditions, it seems likely that the infection of these patients occurred in a non-occupational environment, and therefore other routes of infection should be considered in this patient<sup>1)</sup>. For example, the travel history of the 3 LD cases among the decontamination workers should be investigated by thoroughly interviewing their family.

Secondly, the authors state that they cannot directly prove the existence of *Legionella* bacteria in this case report<sup>1)</sup>. Isolation of the organisms from water systems and expectorated sputum and characterization by species and serogroup would provide more accurate evidence for or against a causal association between occupational exposure and legionellosis development. Intriguingly, not all *Legionella* species show the same pathogenicity. Among the 58 species currently identified, only about 20 have been associated with human disease, and *L. pneumophila* serogroup 1 is responsible for over 80% of total LD

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cases<sup>2,4</sup>). Importantly, the sensitivity and specificity of the UAT is 87% and 94.7%, respectively, despite the fact that UAT can only detect serogroup 1<sup>5</sup>). Unfortunately, the methods other than PCR and UAT to identify the pathogen including culture on buffered charcoal yeast extract (BCYE) with L-cysteine and indirect fluorescent antibody method take too long time for us to administer the proper antibiotics during the treatable period.

*Conflicts of interest:* None declared.

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