An outbreak of *Legionella longbeachae* infection in an intensive care unit?

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**Summary:** During a nine-day period, five patients in a 14-bed intensive care unit (ICU) were shown to have seroconverted with a four-fold or greater rise in serum antibody titre to *Legionella longbeachae* serogroup 1. A further two patients were observed to have high titres consistent with previous exposure but earlier serum samples were not available for comparison. No patients had antibody responses to *Legionella pneumophila* serogroups 1 and 2. *L. longbeachae* was not cultured from respiratory secretions from patients or from the environment within the unit. *Legionella anisa* was recovered from one cooling tower on the ninth floor of the tower block. The ICU is located on the first floor of the same tower and receives external air from two vents, one on the eastern and the other on the western aspect. All patients with serological evidence of *L. longbeachae* infection were concomitantly infected with multiresistant *Staphylococcus aureus*, and were located in bays on the eastern side of the unit. A large pigeon nest was discovered within 1–2 m of the eastern vent. Following removal of the birds’ nest, no further cases were seen on routine screening of all patients within the unit over the next eight weeks. Alternatively, seroconversion may have been related to demolition of the adjacent nine-storey nurses home. This was begun one month before the first case was diagnosed and was completed four months later. The periodic northerly winds could have carried legionellae from the demolition site directly over the block housing the ICU and may have concentrated them near the eastern air vent. All patients had pneumonia, which was probably multifactorial in origin. There is some uncertainty whether the serological responses seen were an epiphenomenon or were truly indicative of infection with *L. longbeachae*.

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**Keywords:** *Legionella longbeachae*; intensive care unit; nosocomial; outbreak.

**Introduction**

There have been a number of reports of Legionnaires’ disease in patients in intensive care units (ICUs).

Many of these cases have been community-acquired but some have been nosocomial in origin.1–5 Most of these infections have been due to *Legionella pneumophila*. An outbreak of *L. pneumophila* infection in an ICU due to contamination of potable water used for administration through nasogastric tubes has been described.6 Similarly, nosocomial transmission in ICUs has been traced to contaminated ice machines.7,8

There has been one report of nine patients with adult respiratory distress syndrome in an ICU in
Tasmania, Australia who had serological evidence of recent infection with *Legionella longbeachae*. Environmental investigation of that outbreak did not reveal the source of infection. We report a similar series of patients in an ICU in Adelaide, South Australia in which there was also serological evidence of recent infection with that organism, and which appeared to be nosocomially acquired.

**Methods**

**Legionella serology**

Serum antibodies to *L. longbeachae* serogroup 1, *L. pneumophila* serogroup 1 and *L. pneumophila* serogroup 2 were measured using an indirect immunofluorescent assay as described by Wilkinson. In brief, heat-killed legionellae of the above species and serogroups were fixed to microscope slides then reacted with dilutions of patients’ sera, washed, then an anti-human globulin conjugated with fluorescein isothiocyanate was added. Bacteria were examined for fluorescence using an ultraviolet microscope. The performance characteristics of the *L. longbeachae* serogroup 1 test have been described elsewhere.

**Legionella culture**

Respiratory secretions were processed using the method described by Dournon. In brief, specimens were liquefied in dithiothreitol–phosphate buffer then centrifuged. Some deposit was cultured directly on BCYE (buffered charcoal yeast extract agar) and MWY (modified Wadowsky and Yee medium: charcoal yeast extract plus glycine, vancomycin and polymyxin B) media. The remainder of the deposit was heated at 50°C for 30 min then cultured as above. Cooling tower waters were processed using the methodology prescribed by the Australian/New Zealand Standard. Aliquots of water were cultured on MWY medium and BMPA (buffered charcoal yeast extract supplemented with cefamandole, polymyxin B and anisomycin), heated at 50°C for 30 min and cultured on BMPA. Processing of liquid specimens from the ICU was based upon the method described by Dennis. Specimens were cultured directly or in some cases after concentration by filtration on BCYE and MWY media. In all cases, plates were incubated at 35°C in 5% CO₂ and examined daily for 10 days; all media were supplied by MedVet (Adelaide, Australia). Any suspect colonies were referred to a reference laboratory for further characterization. Material from the birds’ nest and filters in the air intake was sent to a reference laboratory for examination.

**Patients**

**Case 1**

A 52 year old woman was admitted on 11 September 2000 to the ICU after aerial retrieval from northern South Australia. She had been on doxycycline for one week because of a chest infection but then became acutely dyspnoeic. She had a past history of lung reduction surgery in 1997. By May 2000, she was severely compromised with a forced expiratory volume (FEV) of 0.64 L and could only walk 20 m. She was awaiting lung transplant and was on multiple drugs including 5–20 mg prednisolone. On arrival at The Queen Elizabeth Hospital (TQEH) she was intubated, ventilated and agitated. A chest X-ray showed that both lung fields were hyperinflated with emphysematous changes but there was no pulmonary opacification or pleural effusion. A diagnosis of an acute exacerbation of chronic obstructive airways disease was made and she was treated with sedatives, bronchodilators, methylprednisolone and antibiotics including timentin, gentamicin and erythromycin. Sputum examination at the time showed moderate numbers of polymorphs and occasional miscellaneous organisms but no pathogens were grown. She continued to be agitated and required a tracheotomy on 20 September 2000. Serology for respiratory pathogens was negative on 11 and 19 September 2000 so erythromycin treatment was stopped. She remained febrile and developed intestinal pseudo-obstruction On 28 September 2000, multi-resistant *Staphylococcus aureus* (MRSA) was isolated from the nose, tracheotomy site and sputum. At this point, she was receiving erythromycin for impaired gastrointestinal motility. The MRSA infection was treated with vancomycin. On 18 October 2000, a chest X-ray showed a small left upper lobe opacity. On 25 October 2000 she continued to be febrile so all antibiotics were stopped and cultures were repeated. A labelled white cell scan was suggestive of a focus in the left lung posteriorly. On 31 October 2000 she was transferred to a general ward then was almost immediately re-admitted to ICU after a respiratory arrest. By 6 November 2000, a lung aspirate showed numerous polymorphs and a few Gram-negative bacilli. Subsequently an
unidentifiable anaerobic Gram-negative bacillus was grown but *Legionella* spp. were not isolated. On 3 November 2000, repeat serological testing showed a four-fold rise in serum litres of antibodies against *L. longbeachae* serogroup 1 (Table I) with *L. pneumophila* serogroups 1 and 2 antibodies remaining negative. Therapy with erythromycin and other antibiotics was re-instituted. On 16 November she had been afebrile for six days and was discharged to a general ward. She continued to improve and on 18 December was transferred to a hospital in her home state.

Case 2
A 50 year old man with progressive syringomyelia with quadriplegia; sacral ulceration and a suprapubic catheter was admitted to another metropolitan hospital with myalgia, cough and shortness of breath. On 16 October 2000 he was transferred to TQEH ICU because of respiratory failure. A chest X-ray showed right lower lobe collapse and consolidation. He was treated with masked continuous positive airways pressure, cefotaxime and erythromycin. Subsequently he developed a urinary tract infection with MRSA and this organism was also found in his respiratory secretions; this infection was treated with vancomycin. He showed seroconversion to both influenza A virus and *L. longbeachae* serogroup 1 infection, with at least an eight-fold rise in titre to the latter organism (Table I). The influenza antibody titres were <1:20 on 19 October 2000 and 1:1280 on 5 November 2000. Despite treatment, he continued to deteriorate and died 24 November 2000. Post-mortem examination disclosed cytomegalovirus infection in the lungs both by histology and by antigen detection. *Legionella* spp. were not grown from lung tissue.

Case 3
A 65 year old man was admitted to the ICU with respiratory failure on 19 October 2000 following aerial retrieval from rural New South Wales (NSW). He had been on holiday travelling through NSW, the Northern Territory and South Australia. He had a long-standing history of chronic obstructive airways disease with an exacerbation beginning five days earlier. A chest X-ray showed left lower lobe consolidation and collapse. He was treated with cefotaxime and erythromycin. He then developed MRSA septicaemia which was treated with vancomycin. He showed seroconversion to both influenza A virus and *L. longbeachae* serogroup 1 infection, with at least an eight-fold rise in titre to the latter organism (Table I). The influenza antibody titres were <1:20 on 19 October 2000 and 1:1280 on 5 November 2000. Despite treatment, he continued to deteriorate and died 24 November 2000. Post-mortem examination disclosed cytomegalovirus infection in the lungs both by histology and by antigen detection. *Legionella* spp. were not grown from lung tissue.

Case 4
A 56 year old man who had been quadriplegic for 29 years and had bilateral leg amputations had multiple admissions to ICU over many months for respiratory failure and for fever, the cause of which was usually unclear. On 3 November 2000 he had a single high titre of antibodies to *L. longbeachae* serogroup 1, which was consistent with previous exposure. Only one serum sample, which was unfortunately recent, was available for retrospective assessment. He was treated with erythromycin but still developed recurrent fevers.

Case 5
A 78 year old man had an anterior resection for carcinoma of the colon on 30 October 2000 and was then admitted to the ICU with probable aspiration pneumonia. He was intubated for 11 days. Routine screening for MRSA showed that he was a nasal carrier of MRSA. Empirical antibiotic therapy included erythromycin. On 13 November 2000 he was discharged to the ward. At this point his *L. longbeachae* serology returned (Table I). He had a single high titre to serogroup 1. No previous sera were available for testing.

Case 6
A 55 year old aboriginal woman with a past history of rheumatoid arthritis, chronic obstructive airways disease and corticosteroid treatment was admitted to

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Significantly rising titres were found in five patients.
TQEH ICU on 22 August 2000 from another metropolitan hospital with pseudomonas pneumonia requiring ventilation. She had a difficult course including MRSA septicemia, intestinal pseudo-obstruction and was difficult to wean off the ventilator. On 11 November 2000 she was shown to have at least a four-fold rise in titre of antibodies against \textit{L. longbeachae} serogroup 1 (Table I) with \textit{L. pneumophila} antibodies remaining negative. Macrolide therapy was included in the regimen of antibiotic treatment. She was eventually discharged to the ward after 100 days in intensive care.

**Case 7**

A 65 year old man who had been in a nursing home because of a severe stroke was admitted to TQEH on 12 October 2000 with bilateral pneumonia. On 28 October 2000 he was transferred to ICU for ventilation. MRSA and \textit{Pseudomonas} spp. were recovered from his respiratory secretions. On 12 November 2000 he was found to have at least a four-fold rise in antibody titre to \textit{L. longbeachae} serogroup 1 (Table I) with \textit{L. pneumophila} antibodies remaining negative. He died on 19 December 2000.

In this nine-day period, on the basis of serological testing, there had been five presumed definite cases with at least a four-fold rise in titre and one probable case with a single high titre. Consequently respiratory secretions from these patients were cultured for \textit{Legionella}, and all other patients in the ICU were tested for \textit{L. longbeachae} antibodies. Inanimate specimens in the ICU and waters from the air cooling tower were cultured for \textit{Legionella}.

For environmental investigation of the ICU, specimens were taken for \textit{Legionella} culture as follows: tap water from 14 bays, ventilator condensate from 11 bays, chlorhexidine solution from 11 bays, nebulizer fluid from 10 bays, mouthwash from three bays, ice water from two bays, the ice water machine, and hot and cold water from a sluice. \textit{Legionella} spp. were not grown from any specimen.

In the seven serologically suspect patients, \textit{Legionella} spp. were not grown from any respiratory specimens. Weekly samples were then taken for the next six to eight weeks from all patients in ICU for serology for antibodies against \textit{Legionella} spp., and respiratory secretions were cultured for \textit{Legionella} spp.

On 6 November 2000, routine collections were taken from all hospital cooling towers for \textit{Legionella} spp. culture. Cooling tower No. 4 on the ninth floor of the main block was found to be positive for \textit{Legionella} spp. (not \textit{L. pneumophila}) in a concentration of 200 cfu/mL. This organism was subsequently identified as \textit{L. anisa}. The tower was cleaned and hyperchlorinated on 13 November 2000. A specimen collected on 14 November 2000 grew 10 cfu/mL \textit{Legionella} species (not \textit{L. pneumophila}). This tower was again cleaned and hyperchlorinated with repeat testing being negative for \textit{Legionella} species.

![Figure 1](image-url)  
*Figure 1* Time line showing the appearance of antibodies against \textit{L. longbeachae} in the seven patients. Each horizontal bar indicates the time of admission to, and length of stay in, intensive care. Within each bar, the grey line indicates the period in which the patient is known to be seronegative while the time from seroconversion is shown in black. The grey shaded area denotes the period of demolition of the adjacent Nurses Home.
Physical layout

The Queen Elizabeth Hospital is currently undergoing a rebuilding programme, the basic layout of the site is shown in Figure 2. The main block is a nine-storey tower with three wings. The ICU was relocated to the first floor of the C wing in June 1999. The cooling towers for the main block are located on the ninth floor of the C wing. The air supply for the ICU does not come from the ninth floor but from two external vents, one on each side at first floor level (above the ground floor). This would imply that infected air would have drifted down on each side of the building and enter through those vents if the cooling towers were the source of infection.

The geographical location of the patients within the ICU was then investigated. The unit has 14 beds: seven beds are on the western aspect and seven beds on the eastern aspect (Figure 3). All of the seven patients described above were colonized/infected with MRSA and were nursed together on the eastern aspect of the unit as an infection control measure. This led to an investigation of the air supply to the ICU. There are two fan-driven air inlets on the outside of the building at the same level—on the western and one on the eastern aspect. Lint filters are in place for each ventilation system and air is cooled by heat exchange with sealed cold water lines.

Hypothesis 1 and intervention

Inspection of the western air inlet indicated no obvious problem. However, on the eastern side, the vent was located just under a ledge on the second floor. On that ledge was a pigeon nest [Figure 4(a) and (b)]. Much of the nest material had drifted down past the vent onto the ledge of the first floor. Samples of the bird’s nest, droppings and filter material were sent to an environmental laboratory for *Legionella* spp. isolation but no legionellae were detected by culture. While the results of those investigations were being awaited, the bird’s nest was removed on 17 November 2000, and the air filters were changed on the same day. Two weeks later, a pigeon cull was undertaken.

Following removal of the bird’s nest, no further cases of seroconversion to *L. longbeachae* serogroup 1 were seen on routine screening of all ICU patients over the next eight weeks nor were legionellae cultured in respiratory secretions.
Hypothesis 2

In mid-March 2001, five cooling towers on the main block and the maternity block were heavily contaminated with non-
 pneumophila Legionella spp. In retrospect we realized that contamination of the cooling towers could be related to demolition of the nurses home which also was nine-storeys high (Figure 2). External demolition was begun on 4 October 2000 with removal of the roof from the ninth floor of the nurses home which is adjacent to and on a similar level with the cooling towers on the ninth floor of C wing of the main block. The time line of the demolition is shown in Figure 1. The first case was diagnosed on 3 November 2000. By 17 November 2000, demolition had progressed down to first floor. By 1 March 2001, the final earthworks were nearing completion.

If contamination of the ICU ventilation system did result from the demolition activities, it was more difficult to explain why all of the patients appeared to have been infected via the eastern air system. The prevailing winds at that time of the year are generally from the west but there are periods of northerly winds. These would pass directly over the nurses home to the C wing of the main block. Eddies of contaminated air may have accumulated between the B and C wings on the eastern aspect and entered the ICU via the eastern vent (Figure 2).

There was no further significant building activity over the next 12 months and no further cases have been detected in the ICU.

Discussion

All of the patients in this series had fever, radiological features consistent with pneumonia and were intubated and ventilated for a variable period. Ventilator-associated pneumonia is often multifactorial in origin, and the causative microbial factors in individual patients are frequently difficult to identify.15,16 In many patients, this condition merges with adult respiratory distress syndrome characterized by oedematous pulmonary infiltrates on chest radiography and hypoxaemia despite oxygen therapy together with exclusion of raised pulmonary capillary pressure.17

Serology for respiratory pathogens including L. pneumophila and L. longbeachae is performed routinely on all patients in our ICU who are admitted from the community with pneumonia for which the cause is not clear and who require ventilation. One patient had serological evidence for recent infection with influenza A virus. In addition, that patient had at least an eight-fold rise in titre against L. longbeachae serogroup 1 antigens. Another four patients had a four-fold or more rise in titre against this organism, and two had single high titres regarded as suggestive of infection with this organism in the past. A four-fold or greater rise in serum antibody titre to an organism has traditionally been considered indicative of a recent infection.18 Five patients met this criterion. In fact, the rises in titres observed may well have been impaired to some degree by the compromised state of health including in some patients the administration of corticosteroids for chronic obstructive lung disease.

Given the serological findings, several questions arose. First, was the serodiagnosis truly specific for L. longbeachae serogroup 1 infection? Second, if the patients were infected with L. longbeachae, what was
the source of infection? Finally, if the source of infection could be identified, could transmission be interrupted? Unfortunately, despite intensive investigation, we cannot provide unequivocal answers to these questions.

Although *L. longbeachae* serogroup 1 was first described from Long Beach, California, USA, it appears to be an infrequent cause of pneumonia in the United States of America. Conversely, it is relatively common in South Australia, and has accounted for over 20% of patients with Legionnaires' disease admitted to this hospital over a 14 year period. The organism is widespread in the environment in South Australia. It was first isolated from potting soil following a statewide outbreak of Legionnaires' disease in South Australia in 1988 and 1989. Subsequent studies demonstrated the *L. longbeachae* serogroup 1 in 73% of potting soils in Australia made by 13 manufacturers but not in any of 19 potting soils made in Greece, Switzerland and the United Kingdom. Thus the finding of positive serology in these patients was not altogether surprising. The sero-diagnostic assay employed is known to be sensitive in patients with confirmed *L. longbeachae* serogroup 1 infection. Less is known, however, about its specificity. Serological cross-reactivity has been described between *L. longbeachae* and *Chlamydia psittaci*. All the patients in these series, however, had negative serology for chlamydial infection.

When the first patient was diagnosed on 3 November 2000, it seemed most likely that she had acquired *L. longbeachae* infection prior to admission even though there was no obvious source of exposure and that seroconversion was delayed because of her corticosteroid therapy. This became much less likely when two further cases of seroconversion were found two days later, then untenable when another two cases of seroconversion were confirmed in the following week. If this were the case, then the infections must have been nosocomial in origin with the infecting organism being either *L. longbeachae*, some other species of *Legionella* that cross-reacted in the assay, or another non-*Legionella* organism, presumably Gram-negative in nature, that also cross-reacted in the assay. If the serological response indicated a cross-reacting infection, then it was unclear whether or not such an infection was clinically relevant.

Even though the patients had negative serology for *L. pneumophila*, the air cooling towers on the top of the building were immediately investigated. *L. anisa* was isolated from one of these towers then subsequently removed by hyperchlorination. This organism has been associated with Pontiac fever and rarely causes pneumonic Legionnaires' disease. Further, there is some cross-reactivity between *L. anisa* and *L. longbeachae* serogroup 1 in the assay that we used. Although it seems unlikely, *L. anisa* may have caused either infection or colonization in these intensive care patients.

*Legionella* spp. were not identified either in patient specimens or in the immediate environment of the ICU. Routine treatment of all patients with community-acquired pneumonia in our ICU includes therapy with erythromycin, and this may have accounted for the failure to isolate the organism from these patients. Extensive testing of moist specimens within the unit failed to find any evidence of *Legionella*.

If infection was indeed occurring, the aerosol route was most likely. The proximity of a pigeon nest and droppings to the air inlet valve was noted. Pigeons have been a recurring problem at this hospital and have been culled from time to time. Pigeon nests have been noted in the location on previous occasions, but this may well have been the first time since a new ICU was built and moved to this area in 1999. The likelihood of this being the causative factor was perceived to be greater when it was realized that all the infections were in patients on the side of the unit that received air from this inlet whereas no cases occurred in patients on the other side of the unit which received air from an inlet on the western side of the building. All of these patients had been placed on the same side of the unit as an infection control measure as they were all co-incidentally infected with MRSA. Positive *L. longbeachae* serology is not related to cross-reactivity with this MRSA and no previous patients have shown such changes. Unfortunately, we were unable to confirm the hypothesis that the pigeon nest was the source of infection as legionellae could not be identified in samples of nests. Isolation of legionellae from this material is difficult as it is grossly contaminated with a multitude of other organisms. Furthermore, the outbreak resolved once the nests were removed and the pigeons culled.

When this episode was reviewed several months later, it was realized that it had occurred concurrently with the demolition of the old nurses home. We are now aware that the only other reported outbreak of *L. longbeachae* infection in an ICU occurred during building works in the immediate environs of
the unit in the Royal Hobart Hospital (P. Bell, personal communication). A possible relationship between an outbreak of Legionnaires’ disease due to \(L.\) \textit{pneumophila} and removal of demolition materials has been reported from Spain.\(^{26}\) An outbreak of Legionnaires’ disease due to \(L.\) \textit{michaeli} at a building site was described in China.\(^{27}\) Widespread contamination of potable water with \(L.\) \textit{pneumophila} during a period of major construction was found in the United States. In our case, \(L.\) \textit{amisa} was isolated on one occasion from a cooling tower. This occurred at the time when demolition was beginning. Demolition commenced at the top of the building, which was at a similar level to the affected cooling tower on the main block of the building and cross-contamination would be relatively easy. Demolition progressed rapidly and with in two to three weeks, demolition was down to one floor above ground level. Contamination of cooling towers on the ninth floor of the adjacent block was then less likely to be infective. The finding of \(L.\) \textit{longbeachae} in the cooling towers may simply indicate that these towers were sentinels of air-borne contamination. Alternatively, it is possible that they provide an amplification mechanism for dissemination of infection.

If contamination of the cooling tower with one or more \(L.\) \textit{Legionella} species was indeed the cause of the serological conversions to \(L.\) \textit{longbeachae} serogroup 1 that were seen, it must be presumed that infected aerosols drifted down from the top of the building and entered the air vents for the ICU on the first floor. If this is the case, then it is necessary to explain why all the cases appeared to be associated only with the eastern air vent. The prevailing winds may have produced eddies that concentrated contaminated air in the corner of two adjacent wings in which the air vent was placed.

This outbreak was short-lived, lasting only a month or so. During the five years prior to the outbreak, \(L.\) \textit{Legionella} spp. serology was performed on 169 patients and all were negative for \(L.\) \textit{longbeachae} infection. Despite intensive investigation for the two months following the outbreak and then routine serological observations for the next year, no further cases have been identified. Clearly an event occurred that was limited in time and space. The sero-conversions seen may simply be an epiphenomenon pointing towards an unknown event, presumably infection with a cross-reacting organism. Alternatively, the patients may truly have been exposed to \(L.\) \textit{longbeachae}. We incline to the view that there was probably \(L.\) \textit{longbeachae} infection, and in view of its circumscribed distribution within the ICU and rapid disappearance after removal of the birds’ nest, it was most likely associated with pigeon infestation. This report may alert others to the possibility of a similar association.

References

5. el-Abiary M, Sarmiento X, Torres A \textit{et al.} Prognostic factors of severe \(L.\) \textit{Legionella} pneumonia requiring admission to ICU. \textit{Am J Respir Crit Care Med} 1997; \textbf{156}: 1467–1472.
13. Australian/New Zealand Standard\textsuperscript{8}. Waters—examination for legionellae including \(L.\) \textit{Legionella}