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Opportunist pulmonary infection with *Legionella bozemanii*

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ABSTRACT Three cases of pulmonary *Legionella bozemanii* infection in immunocompromised patients are described. The diagnosis was made by culture in each case and would not otherwise have been made, and it is recommended that a culture specific for *Legionella* species should be included in the investigation of patients with suspected opportunist pulmonary infections.

Legionella pneumophila is a well recognised cause of both community acquired and nosocomial pneumonia. Other *Legionella* species—for example, *L micdadei* (the Pittsburgh agent)[1]—have been associated with human infection. Pulmonary infection in immunosuppressed patients due to *Legionella bozemanii* is little known to clinicians. The diagnosis of *L pneumophila* infection is usually made serologically, on the basis of a rise in specific antibody titre, though a direct fluorescent antibody test has recently been applied successfully to bronchial secretions.[2] There is heterogeneity between *Legionella* species, however, so that these tests as currently used are specific for *L pneumophila* and would not detect infection with *L bozemanii*. It is not universal practice to culture for *Legionella* species, which are fastidious organisms. *L bozemanii* and other *Legionella* species may therefore be an underrecognised cause of pulmonary infection in immunocompromised patients.

Case reports

CASE 1

A 44 year old woman who had undergone orthotopic cardiac transplantation six months earlier was admitted in congestive cardiac failure. She was receiving cyclosporin A, azathioprine, and prednisolone. Examination and investigation suggested the presence of pericardial constriction and restriction. She initially responded well to diuretics and dopamine but seven days after admission became febrile with a reaccumulation of her pleural effusion. Blood cultures and serological tests for adenovirus, parainfluenza viruses, influenza A and B viruses, cytomegalovirus, *L pneumophila, Mycoplasma pneumoniae,* chlamydia, and *Coxiella burnetti* gave negative results. Specimens of pleural fluid and pericardial fluid were obtained for culture and at that point the patient underwent emergency retransplantation.

Postoperatively she developed severe pyrexia (up to 40°C) and a profuse growth of *L bozemanii* was then obtained from the preoperative pleural and peritoneal aspirates. She was treated with erythromycin and rifampicin but developed disseminated intravascular coagulation and died from a cerebral haemorrhage.

CASE 2

A 60 year old woman with a six month history of biopsy proved Wegener's granulomatosis affecting the nasal and respiratory systems presented with a 10 day history of dry cough, pleuritic chest pain, breathlessness, and fever. She was receiving cyclophosphamide and prednisolone. A chest radiograph showed increased right basal shadowing and cavitating consolidation in the apical segment of the right lower lobe. Blood cultures and a serological screen for atypical pneumonia, as in case 1, gave negative results. Bronchoscopy and bronchoalveolar lavage were performed. No pathogens were seen in stained smear preparations, including a silver stain for *Pneumocystis carinii*. The result of a direct immunofluorescent antibody test on the bronchial washings for common viruses was negative. Culture of the bronchial washings, however, produced a growth of *L bozemanii* after three days' incubation. The patient responded rapidly to treatment with erythromycin and rifampicin and recovered.

CASE 3

A 48 year old man underwent orthotopic cardiac transplantation for ischaemic heart disease. He received immunosuppression with cyclosporin A, azathioprine, and prednisolone. Rejection, indicated by endomyocardial biopsy specimens 10 and 20 days after operation, was managed by additional methylprednisolone and antithymocyte globulin for five days. On the 33rd postoperative day he became febrile and a right sided pleural effusion was seen on the chest radiograph. Blood cultures and a serological screen for atypical pneumonia gave negative results. The pleural fluid was aspirated and contained numerous polymorphonuclear leucocytes, but no organisms were seen in stained smear preparations. The initial cultures of the pleural fluid were negative, so bronchoscopy and bronchoalveolar lavage were performed. The washings yielded a moderate growth of *Haemophilus influenzae* and he was treated with ampicillin. A direct immunofluorescent antibody screen for common

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viruses and a silver stain for *P carinii* on the bronchial washings gave negative results. The patient, however, felt increasingly unwell with abdominal pain and developed an acute symmetrical peripheral polyarthropathy. After three days' incubation a heavy growth of *L bozemanii* was isolated from the pleural aspirate and the treatment was changed to ciprofloxacin. His symptoms improved dramatically, with resolution of the pleural effusion. *L bozemanii* was subsequently cultured from the bronchial washings.

Discussion

We have described three cases of pulmonary infection in immunocompromised patients due to *L bozemanii*. This organism, originally termed WIGA, was first isolated from guinea pigs inoculated with material from the lungs of a diver who died of extensive bronchopneumonia.[3] Eleven culture positive cases of pulmonary infection with *L bozemanii* have been reported, of which nine occurred in immunocompromised patients. Five of these cases were nosocomial in origin, believed to be the result of contamination of a hospital water supply.[4] All but one[5] of the reported culture positive cases occurred in North America.

The cases we report occurred in two wards within the same block during eight months. There is evidence that *L bozemanii*, like *L pneumophila*, is associated with water.[4,5] We subsequently isolated the organism from water outlets within the building and have introduced appropriate control measures. This series of cases may therefore also represent a nosocomial outbreak and further environmental studies are in progress.

Several clinical features were noted in relation to infection with *L bozemanii*. Two patients developed hyponatraemia and one abdominal pain. Two patients had a pleural effusion (from which *L bozemanii* was cultured) and in one there was radiographic evidence of cavitation. Although of interest, these features are non-specific and of no value in predicting the causative organism.[6] Two of the patients, however, rapidly recovered from their infection with appropriate chemotherapy, thus emphasising the importance of correctly identifying the responsible organism.

We were able to identify *L bozemanii* as the pathogen in these cases only because our policy has been to culture all bronchoalveolar lavage and pleural aspirate specimens from immunocompromised patients for both *Mycoplasma* and *Legionella* species. The diagnoses would not have been made if we had relied on serological or direct fluorescent antibody testing. This raises the possibility that *L bozemanii* may have been responsible for opportunist pulmonary infection where no pathogen has been isolated or identified in other centres. We therefore emphasise the importance of including specific cultures appropriate for *Legionella* species[7] in patients undergoing investigation for suspected opportunist pneumonia.

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