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Aeromonas hydrophila and Aspiration Pneumonia: A Diverse Presentation

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Although there are ever increasing reports of extraintestinal human infections caused by Aeromonads, in both immunocompromised and immunocompetent patients, respiratory tract infections remain uncommon. We describe a case of aspiration pneumonia in an immunocompetent patient with multiple sclerosis, caused by a community acquired, multidrug resistant strain of *Aeromonas hydrophila* sensitive only to meropenem. The case highlights the clinical significance of *Aeromonas hydrophila* as a respiratory pathogen, as well as the community origin of multidrug resistance and the utility of newer carbapenems in such cases.

Key Words: *Aeromonas hydrophila*, pneumonia, multidrug resistant

INTRODUCTION

Microorganisms of the genus *Aeromonas* (family Vibrionaceae) are gram negative, oxidase positive, non-sporulating facultatively anaerobic rods that ferment carbohydrates. *Aeromonas* species have been associated with gastroenteritis in both adults and children.[1,2] However, they have also been occasionally associated with various extraintestinal infections in both the immunocompromised and healthy hosts.[3] We describe a case of bilateral pneumonia due to *Aeromonas hydrophila*.

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CASE REPORT

A 13-year-old female was admitted in March 2001 with respiratory failure and quadriparesis. She was a known case of multiple sclerosis, having recurrent episodes of cervical myelitis and optic neuritis (first episode on 1st March 2000). She was admitted to the Surgical Intensive Care Unit (ICU) for ventilatory support. On examination she was drowsy; pupils were dilated with decreased reaction to light, nystagmus in both eyes, tone increased, power 2/5, DTR (deep tendon reflexes) increased, planter bilateral extensor, facial nerve palsy along with bilateral lower lobe consolidation of the lung.

The initial laboratory evaluation revealed a peripheral leukocyte count of 5,700/mm^3, with 84% granulocyte and hematocrit of 23%. Chest X-ray revealed bilateral lower lobe opacities with prominent perihilar markings. There was no evidence of pleural effusion. Treatment with methylprednisolone, IV Ig, and acyclovir injection, along with other supportive measures, was started. Cultures of blood, CSF and urine were sterile, but that of bronchoalveolar lavage (BAL) showed growth of *Aeromonas hydrophila* (10^4 CFU/ml) as identified by biochemical tests.[4,5] An API 20 E (BioMerieux, Marcy L'Etoile, France) automated system was used for confirmation. Swabs were also taken simultaneously from ventilator tubings and humidifier for culture and antibiotic sensitivity, with sterile findings.

Nowadays most Aeromonads (>95%) can be accurately identified biochemically to the genomospecies level with conventional, albeit unusual, phenotypic tests and dichotomous schemes to identify the major *Aeromonas* species involved

in human infections; several species of which have been proposed.[6]

The *in-vitro* antibiotic sensitivity showed the organism to be resistant to ampicillin, ampicillin-sulbactum, ceftriaxone, cefotaxime, ciprofloxacin, ofloxacin, amikacin, gentamycin, cefazolin, piperacillin, tetracycline and trimethoprim-sulphamethoxazole. It was then tested against a panel of second line antibiotics consisting of cefpirome, cefaperazone-sulbactum, piperacillin-tazobactum, meropenem, netilmicin and gatifloxacin and was found to be sensitive to only meropenem and moderately sensitive to cefpirome. Antibiotic therapy was started on the third hospital day with intravenous meropenem at 1 gm every 8 hours. The area of consolidation gradually decreased both clinically and radiographically. The patient became afebrile and remained so up to the 22nd day. Repeated culture of BAL was done on every 4th day. The second culture showed growth of 10^2 CFU/ml of *A. hydrophila* followed by 8 colonies on the 4th culture of BAL, which later became sterile from the 12th day onwards. No such episode of pneumonia by *A. hydrophila* was noticed in any other patients in the ICU who were on a ventilator.

DISCUSSION

It has been found that *A. hydrophila* has the best survival and replication attitude in low polluted environments, more commonly in clean water, which is mainly used for household purposes.[7] Since no other patients in the ICU suffered from a similar attack of *A. hydrophila* in more or less the same period, it was deduced that this infection did not originate in the hospital. It can be hypothesized that this infection might have been acquired through aspiration of drinking water at home when the patient was drowsy. No *Aeromonas* species was isolated from cultures of hospital water samples, ventilator tubings or humidifier.

In spite of increasing reports of extraintestinal human infections caused by *Aeromonas* spp., respiratory tract infection (RTI) by the pathogen is an uncommon clinical entity. Sputum isolates of *Aeromonas* were almost invariably judged to represent transient colonization, since oral secretions often harbored *Aeromonas* for short periods due to ingestion of contaminated potable water during warmer months. This viewpoint has radically changed now. Patients with *Aeromonas* RTI basically fall into two categories. One group is of immunocompetent individuals who develop symptoms after contact with an aquatic environment[8,9] and the other group comprises patients with an underlying disease for whom the infection appears to arise from haematogenous dissemination of aeromonads from the gastrointestinal tract to the respiratory tree.[10-12] A couple of cases of lung abscess have been described with no underlying systemic or pulmonary disease. The first isolate was cultured from BAL and the patient was treated with ampicillin and cefotaxime, while the latter was grown from two sputum samples and one bronchial wash and treated with gentamicin, cephalothin and cefuroxime. Both these patients were cured.[11,13] Three cases of pneumonia have also been described, one with no underlying disease and of the other two, one was diabetic and both were alcohol abusers. In all these cases, *A. hydrophila* was grown from blood apart from respiratory specimens and despite antibiotic treatment, all patients died.[9,12,14] Among these, one was a case of near drowning associated *Aeromonas* pneumonia.[14] A case of pulmonary edema and ARDS with septicemia was reported in which *A. hydrophila* was isolated from endotracheal aspirate and blood. This patient also died despite administration of tobramycin.[8] Other respiratory tract infections reported so far include parapharyngeal soft tissue infection, epiglottitis and empyema. The former two patients survived, while the latter died.[10,15,16] To identify the source of the infection in our case, water from the patient's home was cultured, but the organism could not be isolated, perhaps due to its dilution to a level below detection. However, the water supply authority of the city was notified.

Another important feature that should be emphasized was the multidrug resistant nature of the pathogen. This prompted us to believe that it could be a hospital isolate, but all the other evidence was against this conclusion. *Aeromonas* isolate of community origin might

have acquired inducible resistance from the drug pressure after indiscriminate use of antibiotics in the hospital. Thus, the microbiologist and physician must be vigilant with the awareness that such an inherently multidrug resistant microorganism can come from the community as well.

An earlier study showed that *A. veronii* is the most susceptible species to the antibacterial agents, while *A. hydrophila* was the most resistant, among blood isolates.[17] They found quinolones to be the most active antibiotic against this pathogen. This isolate was however multidrug resistant. Certain species are inherently more pathogenic (e.g. *A. hydrophila* and *A. veronii*) than others and are associated with higher fatality rates among immunocompromised individuals.[18] *Aeromonas* spp. harbouring a conjugative plasmid that confers multiple antibiotic resistance has been identified.[19] It produces as many as three β-lactamases, including a Bush group 2d penicilllinase, a group 1 cephalosporinase and a metallocarbapenamase.[20] Despite the presence of carbapenemases, minimum inhibitory concentrations to imipenem typically remain low. Considering the above fact, meropenem was tested *in-vitro* against this pathogen and was found to be sensitive. On administration to the patient it showed excellent clinical and microbiological response.

The overall mortality rate attributed to this pathogen is approximately 50%, which may be due to several factors. Poor prognostic indicators are pneumonia, hemoptysis, an acute, rapidly progressing infection and concomitant sepsis.[8,9,12,14] Fortunately our patient had only one of these factors and was cured with the help of appropriate antibiotic treatment.

Thus Aeromonads from respiratory samples should not be discarded as a contaminant and their clinical significance should always be determined by clinical impression. Clinical microbiological laboratories should exert an extra effort in identifying them to the species level and newer drugs like meropenem should be placed in the first line antibiotic sensitivity panel for such rare isolates.

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