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Major Article

Nosocomial infections by diverse carbapenemase-producing *Aeromonas hydrophila* associated with combination of plumbing issues and heat waves

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Key Words:
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Background: Aquatic opportunistic pathogen *Aeromonas hydrophila*, known to persist in low-nutrient chlorinated waters, can cause life-threatening infections. Two intensive care units experienced a cluster of *Aeromonas* infections following outdoor temperature spikes coinciding with recurrent plumbing issues, with fatalities due to severe underlying comorbidities co-occurring with extensively-drug resistant (XDR) *Aeromonas*.

Methods: We investigated this cluster using whole genome sequencing to assess genetic relatedness of isolates and identify antimicrobial resistance determinants. Three *A. hydrophila* were isolated from patients staying in or adjacent to rooms with plumbing issues during or immediately after periods of elevated outdoor temperatures. Sinks and faucets were swabbed for culture.

Results: All *A. hydrophila* clinical isolates exhibited carbapenem resistance but were not genetically related. Diverse resistance determinants corresponding to extensively-drug resistant were found, including co-occurring *KPC-3* and *VIM-2*, *OXA-232*, and chromosomal *CphA*-like carbapenemase genes, contributing to major treatment challenges. All 3 patients were treated with multiple antibiotic regimens to overcome various carbapenemase classes and expired due to underlying comorbidities. Environmental culture yielded no *Aeromonas*.

Conclusions: While the investigation revealed no singular source of contamination, it supports a possible link between plumbing issues, elevated outdoor temperatures and incidence of nosocomial *Aeromonas* infections. The diversity of carbapenemase genes detected in these wastewater-derived *Aeromonas* warrants heightened infection prevention precautions during periods of plumbing problems especially with heat waves.

SUMMARY

A cluster of carbapenem-resistant Aeromonas infections occurred in intensive care units amidst elevated outdoor temperatures and plumbing problems. Sequencing revealed these isolates were unrelated and harbored diverse resistance genes. High temperature and wastewater leaks may contribute to Aeromonas nosocomial infections.

BACKGROUND

The aquatic opportunistic pathogen *Aeromonas* can cause severe infections among both immunocompetent and immunocompromised hosts.[1] Although typically associated with environmental aquatic exposures, *Aeromonas* has been implicated in nosocomial outbreaks.[2] While predominantly associated with gastroenteritis, *Aeromonas* can

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cause diseases ranging from milder infections like cellulitis to high-mortality illnesses such as pneumonia and bacteremia.[3] In addition to an arsenal of virulence factors such as adhesion and toxin production, *Aeromonas spp.* are known carriers of antibiotic resistance.[1,4] Numerous studies document extensive carriage of plasmids harboring resistance genes, in addition to chromosomally mediated β-lactam resistance in *A. hydrophila* and other *Aeromonas* species.[5,6]. Infections by resistant *Aeromonas* spp. have few treatment options, resulting in high-mortality, especially among immune-compromised patients.[2,7,8]

Studies of hospital effluent and surface waters illustrate the high burden of multidrug-resistant *Aeromonas* isolates sourced from hospitals capable of persisting in the environment.[9–15] With the ability to survive in chlorinated and nutrient-scare waters, water collection and distribution systems may be a continuous reservoir of resistant *Aeromonas*. Climate change compounds this risk. Warmer waters result in higher concentrations of *Aeromonas* in both environmental waters and distribution/collection systems, while residual chlorine inversely correlates to higher temperatures.[14,16–18] Thus, in the context of compromised water collection and distribution systems in clinical settings, extreme precautions should be taken to prevent outbreaks of multidrug-resistant *Aeromonas spp.*

In this study, we present 3 nosocomial infections with extensively drug resistant (XDR) *A. hydrophila* occurring in 2 intensive care units (ICU) in the late summer and early fall, contrasted with a case of community-acquired wild-type *A. veronii* infection. The *A. hydrophila* cases correspond to a period of extreme heat events overlapping with plumbing issues in these units. We aimed to determine the relatedness of this cluster of infections and characterize the resistance determinants along with the relationship between heat events and reported plumbing problems. Understanding the epidemiology of these infections will help inform future infection prevention practices in the face of a changing climate.

METHODS

Four isolates were collected from clinical specimens from 4 patients between August 2022 and October 2022. All isolates were identified as *Aeromonas spp.* using the Vitek MS Matrix-assisted laser desorption ionization–time of flight (MALDI-TOF) instrument (BioMerieux). Phenotypic antibiotic susceptibility testing was performed using in-house microbroth dilution in accordance with clinical and laboratory standards institute methodology. Carbapenemase and metallo-β-lactamase (MBL) activity was confirmed using the modified/ethylenediaminetetraacetic acid-modified carbapenem inactivation assay (mCIM/eCIM).

Environmental sampling of rooms under investigation was performed following plumbing repairs. Swabs from faucets and sinks from 10 rooms were directly plated on blood agar, MacConkey agar, and Aeromonas-selective Cefsulodin-Irgasan-Novobiocin (CIN) agar and incubated at room temperature. Plates were assessed for growth at 24, 48, and 72 hours, as well as assessment of CIN agar plates at 7 and 14 days. Isolates from CIN plates were streaked to purity and identified via MALDI-TOF.

Bacterial DNA was extracted using the Blood and Tissue extraction kit (Qiagen) and quantified using Qubit Fluorometric dsDNA Assay (Thermo Fisher). Libraries were prepared for whole genome sequencing using the Illumina DNA Library Prep kit and were sequenced using MiSeq (Illumina). De novo assembly, single-nucleotide polymorphism (SNP) calling, and tree generation were conducted using CLC Genomics Workbench (Qiagen). KmerFinder[19] was used for reference identification, and Geneious Prime (Biomatters, Inc) was used for reference-mapping, annotation, and sequencing and assembly QC. Resistance markers were identified using the comprehensive antibiotic resistance database (CARD) protein homology method,[20] with further resistance analyses performed with Abricate.[21] To investigate the mobility of these genes, Center for Genomic Epidemiology's PlasmidFinder tool [22] was used to detect plasmid replicon types along with Geneious Prime to explore gene locations.

Elevated temperature periods were determined to be peaks of temperature lasting at least 2 days with average temperature (± SD) exceeding 10-day average temperatures pre- and post-temperature peak. The peak overlapping with positive specimens from patients B and C were classified by the National Oceanic and Atmospheric Administration as an extreme heat wave, with peak outdoor temperature exceeding the historical average temperature for a period of 2 or more days.

RESULTS

Clinical histories

Patient A presented in mid-July with hematemesis in the setting of decompensated alcoholic cirrhosis. On hospital day (HD) 10, ceftriaxone was started for spontaneous bacterial peritonitis. On HD21, the patient was transferred to the ICU for vasopressor support and intubated for airway protection. A CT abdomen and pelvis showed pneumoperitoneum, and ertapenem was started for respiratory cultures growing extended spectrum beta-lactamase *Escherichia coli.* On HD33, meropenem and linezolid were started for escalating vasopressor requirements. Endotracheal respiratory cultures grew carbapenem-resistant *E. coli*, prompting the transition to tigecycline. On HD38, antimicrobials were changed to ceftazidime-avibactam, metronidazole and caspofungin due to a sudden increase in vasopressor requirements and leukocytosis. Respiratory and blood cultures grew XDR *A. hydrophila*, susceptible only to amikacin. Aztreonam and amikacin were added to ceftazidime-avibactam for disseminated *Aeromonas* infection. The patient had progressive mixed shock, and renal failure requiring hemodialysis. Repeat peritoneal fluid culture again grew XDR *A. hydrophila* despite targeted treatment with 3 agents. On HD50, the family transitioned the patient to comfort care, which expired shortly thereafter.

Patient B had metastatic pancreatic cancer complicated by recurrent biliary obstruction and paracolic abscess status post drain placement with cultures positive for *E. coli* and bacteremia due to *Enterococcus faecalis.* She was discharged on amoxicillin and trimethoprim-sulfamethoxazole, then readmitted in late August due to a dislodged drain. The hospitalization was complicated by protracted hematochezia, renal failure requiring hemodialysis, fungemia, and mixed shock. She was started on caspofungin and empiric piperacillin-tazobactam. Imaging from HD22 revealed an increased size of the known intra-abdominal abscess. Fluid obtained at the time of drain exchange grew *A. hydrophila.* The isolate was resistant to piperacillin-tazobactam, and plans were to start a carbapenem. At that time, the patient decided to transition to comfort care and was discharged home with hospice on HD26. The patient expired 2 days later.

Patient C presented in early September with severe back pain due to a T6 fracture sustained after a fall. Past medical history was significant for decompensated cirrhosis due to nonalcoholic steatohepatitis, and the hospital course was complicated by a gastrointestinal bleed on HD5 requiring an upgrade to the ICU for vasopressor support. Endoscopic interventions did not reveal a source of the bleed. Her ICU course was complicated by ongoing hematemesis and melena, shock, renal failure requiring hemodialysis, and an aspiration event requiring intubation on HD10. She was started on empiric meropenem. Tracheal aspirate and bronchoalveolar lavage cultures from HD11 grew *A. hydrophila,* and the patient was transitioned to ceftazidime-avibactam on HD13. Despite appropriate antimicrobials,

Table 1
Overview of clinical, epidemiological, and genomic characteristics of Aeromonas infections in this study

Patient	Date isolated	Specimen	Unit prior to *Aeromonas* isolation	Outcome	Isolate MLST	Closely related reference genome
A	8/22/2022	Blood	ICU1; ICU2	Deceased	860	*A. hydrophila* ZYAH75 CP016990.1
B	9/12/2022	Perirectal drain	ICU1	Discharged to hospice	1083	*A. hydrophila* Ah2111 CP095280.1
C	9/18/2022	Respiratory, nasotracheal suction	ICU2	Deceased	1083	*A. hydrophila* Ah2111 CP095280.1
D	10/13/2022	Blood	Community	Resolved	Nearest 406,1971	*A. veronii* FDAARGOS_632 CP044060.1

MLST, multilocus sequence types.

the patient had escalating vasopressor requirements. The family transitioned the patient to comfort care, who expired on HD33.

Patient D was admitted in early October with polymicrobial bacteremia with *Aeromonas veronii* and *Lactobacillus species* in the setting of metastatic pancreatic cancer, thought to be due to gut translocation given extensive colonic wall inflammation on imaging. The isolation of *Lactobacillus* was attributed to the patient's use of *Lactobacillus*-containing probiotics. She was initially started on vancomycin and piperacillin-tazobactam pending susceptibilities and narrowed to cefepime. After receiving appropriate antibiotics, serial repeat blood cultures had no growth. However, due to several noninfectious complications throughout the hospitalization, the patient opted for discharge to a facility on hospice care on HD42.

A single *Aeromonas* isolate was submitted for sequencing from each patient. Isolates are henceforth referred to as isolates A-D, corresponding to sequenced isolates from patients A-D (Table 1).

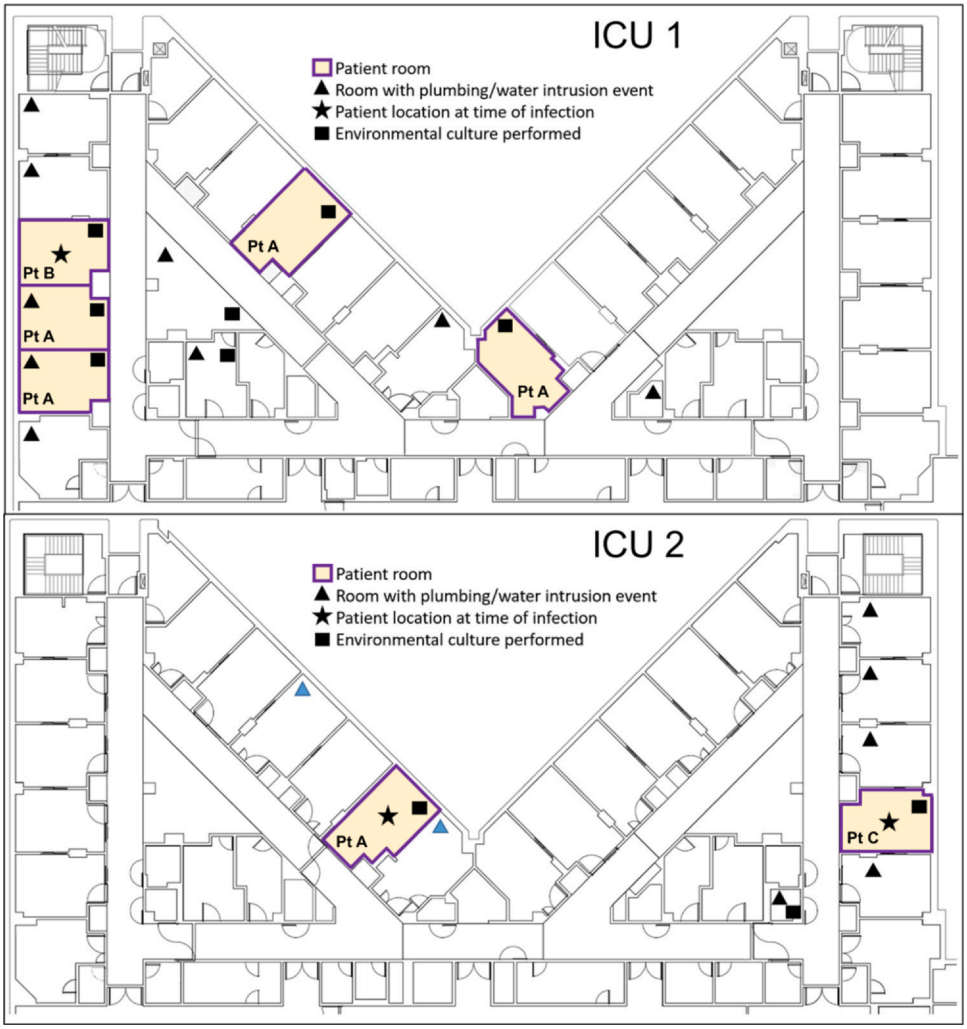


Fig. 1. Map of ICU1 and ICU2, with rooms of patients A-C indicated, including patient room during Aeromonas isolation. Rooms where significant plumbing events during the study period and rooms sampled during environmental sampling postresolution are also indicated.

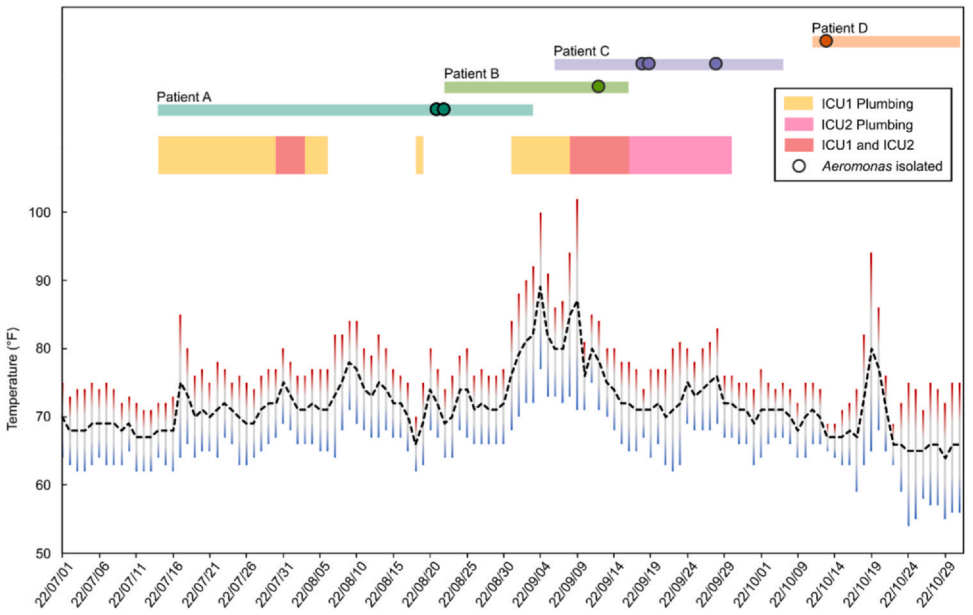


Fig. 2. Timeline of study period with minimum, maximum, and average temperature at the national weather station nearest to the institution. Periods of open service requests for plumbing issues in each ICU, as well as length of each patient stay are also indicated.

Epidemiologic investigation

Patients A-C stayed in multiple rooms during their ICU admissions (Fig 1). All three stayed in rooms with or directly adjacent to reported sites of plumbing issues and open service tickets for plumbing repairs. Reported plumbing concerns and maintenance requests included dripping water, sink leaks, sewage odors, toilet clogs, clogged sinks, and floor and ceiling leaks. Patient A received procedures in both ICUs (ICU1 and ICU2) and switched rooms 5 times during the course of his hospitalization. Patients B and C stayed in ICU1 and ICU2, respectively, and did not switch rooms.

All isolates from this study were collected during a three-month period between July and October (Table 1 and Fig 2). *Aeromonas* isolates from patients A-C were collected during or immediately following (≤5 days) periods of elevated outdoor temperature, with average temperatures exceeding 80 °F (27 °C) (Fig 2). Patient D was admitted to either ICU and no plumbing concerns were reported during their stay.

Environmental sampling of impacted rooms in both ICUs occurred in mid-Autumn after the resolution of plumbing concerns (Fig 1). Sinks and faucets were swabbed from ten rooms under outbreak investigation. No *Aeromonas* species were isolated. Only a few known environmental bacteria in low quantities were recovered, including *Stenotrophomonas maltophilia* and *Pseudomonas* species, which were considered nonsignificant.

Microbiological investigation

Of the clinical isolates, isolates A-C were confirmed to be *A. hydrophila* via MALDI-TOF, while the last isolate, not associated with nosocomial infection, was identified as *A. veronii.* Antimicrobial susceptibility testing on all isolates revealed extensive drug resistance among all 3 *A. hydrophila* strains, while broad susceptibility was observed in the *A. veronii* isolate (Table 2). Isolates A-C exhibited resistance to ertapenem, third generation cephalosporins and trimethoprim or sulfamethoxazole. Isolates A and B also were resistant to meropenem and imipenem, as well as cefepime and piperacillin or tazobactam. Notably, mCIM and eCIM tests were positive for all 4 *Aeromonas* isolates, indicating carbapenemase production.

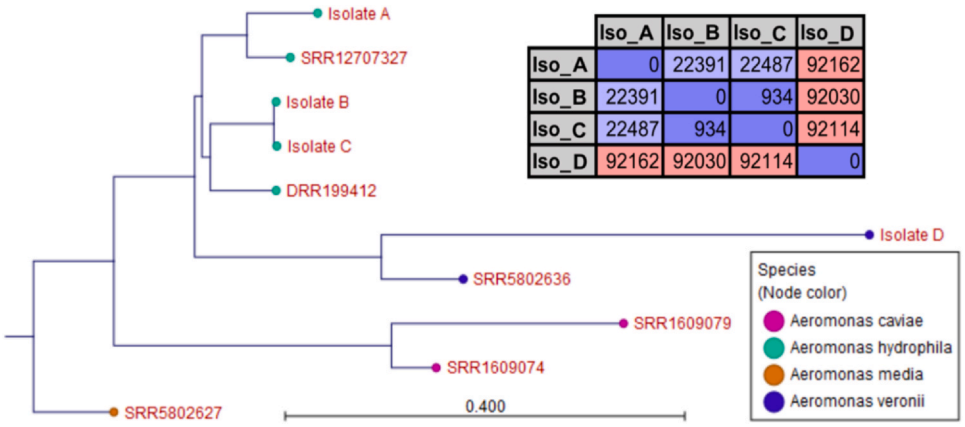
Table 2
Drug susceptibility of Aeromonas isolates

	Isolate A	Isolate B	Isolate C	Isolate D
Piperacillin/Tazobactam	>128	>128	32	≤8
Cefazolin	R	R	R	R
Ceftriaxone	>64	>64	64	≤1
Ceftazidime	>32	>32	>32	≤0.5
Ceftolozane/Tazobactam	32	32	2	≤0.5
Cefepime	>32	32	4	≤0.5
Meropenem	>16	>16	≤0.25	≤0.25
Imipenem	>8	>8	≤1	≤1
Ertapenem	>4	>4	4	≤0.25
Amoxicillin/Clavulanate	>32	>32	32	32
Gentamicin	>16	≤1	>16	≤1
Tobramycin	16	2	>16	≤1
Amikacin	≤4	≤4	≤4	≤4
Ceftazidime/Avibactam	≤2	4	≤2	≤2
Ciprofloxacin	>4	>4	>4	≤0.25
Levofloxacin	4	>8	2	≤0.5
Trimethoprim/ Sulfamethoxazole	>4/80	>4/80	>4/80	≤1/20

Genomic characterization

Closely related reference isolates and multilocus sequence types were identified for all isolates using KmerFinder (Table 1). The closest reference for the Isolate A was *A. hydrophila* ZYAH75 (NCBI CP169901.1) isolated from a wound specimen in Wuhan, China. Isolates B and C were most closely related with *A. hydrophila* Ah2111 (NCBI CP095280.1), isolated from an ascites specimen from Hangzhou, China. Isolate D was closest in homology to *A. veronii* FDAARGOS_632 (NCBI CP044060.1), a clinical isolate from Kentucky, USA.

Using *A. hydrophila* reference genome NCBI CP095280.1, variant detection was performed for all isolates, as well as 6 reference genomes representing *A. hydrophila, A. caviae, A. media,* and *A. veronii*. A maximum likelihood SNP tree and SNP matrix (Fig 3)



	Iso_A	Iso_B	Iso_C	Iso_D
Iso_A	0	22391	22487	92162
Iso_B	22391	0	934	92030
Iso_C	22487	934	0	92114
Iso_D	92162	92030	92114	0

Fig. 3. Single-nucleotide polymorphism (SNP) phylogram of isolates and Aeromonas reference genomes and corresponding matrix of SNPs for all clinical isolates.

indicated that none of these isolates were genetically related. Isolates B and C had a difference of 934 SNPs; Isolate A differed from isolates B and C by roughly 22,000 SNPs; isolate D, being a different species, differed by over 92,000 SNPs.

The number of antimicrobial resistance genes detected using the comprehensive antimicrobial resistance database ranged from 4 in isolate D to 19 in isolate C (Table 3). Chromosomal inducible Class B MBL genes were detected in all 4 isolates, including *cphA* in isolates A and D, and *imiH* in isolates B and C. All 3 *A. hydrophila* isolates (A, B, and C) contained AmpC-like Class C β-lactamase gene *CepS*. All 4 isolates possessed at least one Class D *OXA*-type β-lactamase gene (OXA-726 in A-C and OXA-912 in D), with isolates B and C containing additional ones (OXA-232 in B and OXA-1 in C). Nonintrinsic carbapenemase genes were detected in 2 isolates: *VIM-2* and *KPC-3* in isolate A and *OXA-232* in isolate B.

Plasmid-associated markers were detected on all *A. hydrophila* isolates (Table 3). An IncP1 plasmid containing a *VIM-2* gene was identified in Isolate A. Isolate B harbored a ColKP3 plasmid that was determined to contain *OXA-232*. This plasmid aligned to previously characterized *Klebsiella pneumoniae* ColKP3 plasmids with *OXA-232* isolated in China and the US (100% coverage, 99% identity.) Plasmid replicon type Col440I was identified in Isolate C, although no resistance genes were identified adjacent to that marker.

DISCUSSION

Patients A, B, and C began to experience symptoms consistent with nosocomial infectious processes after hospitalization for initial causes, with their first *Aeromonas* isolate collected between 12 and 38 days postadmission, strongly indicating nosocomial *Aeromonas* infections. A review of institutional *Aeromonas* infections of the prior year revealed no other hospital-onset cases prior to this cluster.

A retrospective study of *Aeromonas* bacteremia indicated that hospital-acquired *Aeromonas* infections were primarily caused by *A. caviae*, but mortality was higher for patients with *A. hydrophila* and *A. veronii* bacteremia.[7] *Aeromonas* was found to be a main causative

Table 3
Antimicrobial resistance genes detected, grouped by antibiotic class. β-lactam resistance genes are grouped by β-lactamase Ambler Class

	Isolate A *A. hydrophila*	Isolate B *A. hydrophila*	Isolate C *A. hydrophila*	Isolate D *A. veronii*
Aminoglycoside	*aadA2* *aph(3′)-Ia* *aac(6′)-IIa*	*aadA2* *aph(3′)-Ia*	*aadA2* *aph(3′)-Ia* *aac(3)-IId*	
β-lactam				
Ambler Class A	*KPC-3* *TEM-1*	*SHV-5*	*SHV-5* *TEM-1*	
Ambler Class B	*cphA2* *VIM-2*	*imiH*	*imiH*	*cphA3*
Ambler Class C	*CepS*	*CepS*	*CepS*	
Ambler Class D	*OXA-726*	*OXA-726* *OXA-232*	*OXA-726* *OXA-1*	*OXA-912*
Diaminopyrimidine	*dfrA12*	*dfrA12*	*dfrA12*	
Disinfectant	*qacEdelta1* *qacJ*	*qacEdelta1* *qacJ*	*qacEdelta1* *qacJ*	
Fluoroquinolone	*rsmA* *adeF*	*rsmA* *adeF* *QnrS2*	*rsmA* *adeF*	*rsmA* *adeF*
Macrolide	*mphA*	*mphA*	*mphA*	
Peptide	*arnA*	*mcr-7.1*	*mcr-7.1*	
Phenicol			*catB3*	
Rifamycin			*arr-3*	
Sulfonamide	*sul1*	*sul1*	*sul1*	
Tetracycline	*tet(D)*			
Plasmid Markers	IncP1	ColKP3	Col440I Col440II	

NOTE. Carbapenemase genes are bolded.

agent of bacteremia in patients with intra-abdominal infections or malignancies,[7] which is consistent with *A. hydrophila* infections in this study. Other studies have reported *A. hydrophila* to be the most common cause of nosocomial *Aeromonas* infections overall.[23] *Aeromonas* bacteremia case fatality rates have previously been reported to range from 27.5% to 46%.[2]

Patient C developed right upper and middle lobe pneumonia and pulmonary edema requiring intubation, and *Aeromonas* was cultured from a nasotracheal suction specimen. Respiratory *Aeromonas* infections are considered rare, though *Aeromonas* pneumonia has been observed particularly after near-drowning events.[24] Given the patient's absence of environmental aquatic exposures, other potential sources of infection include aspiration and contaminated water.[4] However, in numerous cases of respiratory *Aeromonas* infection in immunocompromised patients, the source of infection was not determined. *Aeromonas* pneumonia has an estimated mortality rate of over 50% even with aggressive treatment.[25]

Seasonal periodicity of *Aeromonas* infections has previously been described, with cases typically peaking in late summer and early autumn,[14,16] as was observed with this cluster. Environmental *Aeromonas* prevalence has similarly been demonstrated, as concentrations of *Aeromonas* in environmental waters peak during these warmer months in the Northern hemisphere.[26] The 3 cases described in this report were isolated during this time period. Moreover, these isolates corresponded to extreme heat events, which could result in *Aeromonas* overgrowth in the water. Other studies have demonstrated that even fecal carriage of *Aeromonas* displays temperature-dependent periodicity,[27] likely due to elevated concentrations of *Aeromonas* in the water supply.[4] The potential association between temperature surges and *Aeromonas* isolation in our report suggests a more direct relationship between discrete elevated outdoor temperature events and nosocomial acquisition of these infections.

The ability of *Aeromonas* to survive in chlorinated waters in low-nutrient conditions has been well-established.[13] Burke et al demonstrated both the seasonal fluctuations in *Aeromonas* concentrations in raw water and underground water as well as the presence of *Aeromonas* spp. in treated and chlorinated water in distribution systems.[14] Warmer temperatures were shown to correlate with lower free chlorine levels in the distribution system as well as *Aeromonas* gastroenteritis. Compounding this, the Los Angeles Department of Water and Power also reported changes in the municipal water supply during this period, resulting in a brief period of hypochlorination in our institution. Despite secondary disinfection at our institution, the plumbing disruptions in ICU1 and ICU2 introduced leakages and periods of water stagnation paired with elevated temperature, likely reducing residual disinfectant in the distribution system and allowing *Aeromonas* to proliferate. Moreover, the high resistance observed in isolates A, B, and C may correspond with the reported plumbing issues potentially involving toilet leaks resulting in wastewater contamination. Wastewater isolates of *Aeromonas* are more often drug-resistant,[9] and numerous studies report carbapenemase-producing *Aeromonas* isolated from wastewater and hospital effluent.[10,28–30]

Climate change will result in increasingly frequent and extended periods of elevated temperature, which may have direct impacts on temperature fluctuations in water supplies and distribution systems. Opportunistic pathogens colonize water distribution systems and form biofilms more readily during periods of elevated temperature.[31] Especially in the setting of water stagnation, elevated temperatures may also contribute to the release of opportunistic pathogens into bulk water.[17] Water and wastewater conveyance may also be impacted by climate change, resulting increased blockages and breakages.[18,32] Under these conditions, contamination between distribution and collection, especially in the event of leaks and stagnation, may result in more frequent incidents in which opportunistic pathogens entering otherwise assumed "safe" water sources. In clinical settings with large populations of immunocompromised patients, extreme care must be taken during periods of elevated outside temperature especially in the context of plumbing issues to prevent infections due to *Aeromonas* and other hydrophilic opportunistic pathogens.

The wild-type *Aeromonas* species are largely susceptible to cefepime, quinolones, and tetracyclines.[4] Chromosomal MBL carbapenemases are intrinsic in *A. hydrophila*, *A. jandaei*, and *A. veronii*.[33] Isolates A, B, and C in this study were XDR and mCIM and eCIM positive. This phenotype has previously been reported locally in intra-abdominal infections in solid organ transplant patients.[6] In isolate A, 3 carbapenemase genes and 3 additional β-lactamase genes were detected, including a combination of Class A bla*KPC-3* and Class B bla*VIM-2*, previously unreported in *Aeromonas*. These 2 genes, in addition to *cphA*, *cepS*, bla*TEM-1*, and bla*OXA-726*, confer resistance to all beta-lactams. The co-occurrence of *KPC* and *VIM* has been described in case reports in *Pseudomonas aeruginosa*,[34] *Klebsiella pneumoniae*,[35] *E. coli*,[36] and *Enterobacter cloacae*,[37] with the earliest reports from 2010. This combination of resistance also supports possible wastewater origins of this isolate, as extensive gene exchange present in wastewater can result in novel resistance genotypes. Isolate B harbored the first reported case of *OXA-232* in *A. hydrophila*. Historically reported in Enterobacterales, *OXA-232* was identified in clinical isolates implicated in an outbreak of carbapenem-resistant *K. pneumoniae* associated with a contaminated endoscopy.[38] To our knowledge, this is the first study reporting such a rare genotype of OXA-232 producing *A. hydrophila* in the clinical setting.

There are several limitations of this study. First, the culture of environmental samples did not yield any *A. hydrophila*, thus there was no direct evidence linking the plumbing leaks to these nosocomial infections. Second, the small case number (n = 3) also limited the significance of this study. Other causes/sources of the spread of these XDR *A. hydrophila* remain possible.

CONCLUSIONS

In the presence of plumbing leaks and heat waves, nosocomial infections due to diverse carbapenemase-producing *Aeromonas* were observed in our institution. In a changing climate, warmer ambient temperatures may facilitate additional growth of *Aeromonas*, which can be XDR and lead to treatment challenges. Extreme caution must be taken during these periods to protect vulnerable patients from exposure, especially due to breaches in water distribution and collection systems.

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