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BRIEF REPORTS

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Faucets as a reservoir of endemic *Pseudomonas aeruginosa* colonization/infections in intensive care units

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Abstract *Objective:* To evaluate the role of faucets as a reservoir for *Pseudomonas aeruginosa* colonization/infection of patients hospitalized in intensive care units (ICUs). *Design:* Prospective epidemiological investigation performed during a nonepidemic period of 1 year. The inner part of the ICU faucets were swabbed for *P. aeruginosa*. Data were recorded on all patients with at least one culture of a clinical specimens positive for *P. aeruginosa*. Pulsed-field gel electrophoresis was used to characterize the strains. *Setting:* Five ICUs of a university hospital which are supplied by two separate water distribution networks. *Patients:* During a 1-year period 132 cases were investigated. *Results:* In 42% of cases (56/132) there were isolates identical to those found in the faucets, with a total of nine different genotypes. Among the nine genotypes isolated from both patients and faucets one of them, the most prevalent, was isolated in the two networks and in 30 cases. The other eight genotypes were recovered almost exclusively from either one (three genotypes in 12 cases) or the other (five genotypes in 12 cases) network and from the patients in the corresponding ICUs. *Conclusions:* These results suggest that the water system of the ICUs was the primary reservoir of patient's colonization/infection with *P. aeruginosa* in a substantial proportion of patients, although the exact mode of acquisition could not be determined.

Keywords Hospital plumbing network · Intensive care units · *Pseudomonas aeruginosa* · Pulsed field-gel electrophoresis · Water contamination

Introduction

The main reservoir of *Pseudomonas aeruginosa* is humid environment. However, *P. aeruginosa* was also found to be part of the endogenous flora of 2.6–24% of hospitalized patients [1]. Sporadic or endemic infections have generally been considered as originating mainly from the endogenous flora. On the other hand, hospital epidemics linked to environmental sources have been observed on many occasions [2]. Drinking water has been associated with *P. aeruginosa* infection when used for hydrotherapy of burned patients or medical devices after disinfection. The importance of faucet aerators and sinks as a source of *P. aeruginosa* is debated [2], but rare well described outbreaks have been reported. Recently water was suspected to play a role as a reservoir for *P. aeruginosa* colonization/infectio of ICU patients [3]. The aim of the present study was to evaluate the importance of the water distribution system and faucet colonization as a reservoir of endemic *P. aeruginosa* colonization/infection of ICU patients using epidemiological and molecular typing techniques.

Materials and methods

The University Hospital of Lausanne is a 870-bed tertiary care hospital which was opened in 1982. The ICUs are located on the same floor but have two distinct water distribution networks: (a) surgical, burn, and pediatric ICUs (network I) and (b) medical and cardiology ICUs (network II). Patients were included in the study if they were hospitalized in one of the ICUs and had at least one culture of a clinical specimen positive for *P. aeruginosa* in 1998. All specimens were obtained because of a clinical indication. There was no routine bacteriological screening of the patients during the period of the study. Demographic and epidemiological data (unit and room of hospitalization, dates of admission and discharge) were retrieved from the hospital information system. The monthly number of patients with *P. aeruginosa* was recorded for the period of the study as well as for the previous year. During the period of the study (January–December 1998) there were 2,618 admissions accounting for 13,475 patient-days.

Swabs of all faucets found in patient's rooms were performed on four occasions (September 1997, February, June, and December 1998). The faucets were dismantled, and the hot-cold water mixing chamber was swabbed. Swabs were plated on blood and cetrimide agar plates and incubated at 35°C. During each screening water samples were obtained from 16 sites before swab sampling. The water was filtered, and the bacteria were resuspended in 10 ml sterile water. We then plated 100 µl of the resuspended solution on blood and cetrimide agar plates. Molecular typing was performed using the pulsed field-gel electrophoresis (PFGE) method as previously described [4]. In brief, plugs of genomic DNA were digested with *Spe*I, electrophoresis was performed with the use of the CHEF DR III system (Bio-Rad, Hercules, Calif., USA) at 6 V/cm at 12°C for 24 h with a linear pulsed time of 1–36 s.

A case was defined as a patient for whom a genotype of *P. aeruginosa* was found in a clinical specimen during its stay in an ICU. Thus two different genotypes found in one patient were considered as two cases. *P. aeruginosa* was considered as originating (directly or indirectly) from the water system when the isolate(s) from a patient was of the same PFGE genotype as one isolate recovered from the water distribution network where the patient was hospitalized. Cross-transmission was considered possible when indistinguishable isolates were found in patients hospitalized during overlapping periods in the same ICU, and a cluster was defined as two or more such cases.

Results

The monthly number of patients with at least one clinical specimen positive for *P. aeruginosa* over a 2-year period in the five ICUs is shown in Fig. 1. In total 139 patients had one or more clinical specimens positive for *P. aeruginosa*. The incidence densities did not differ between the five ICUs. The water system investigation showed that a total of 21 of 216 (9.7%) swabs from ICU faucets were positive for *P. aeruginosa* (Table 1). None of 64 water samples showed the presence of *P. aeruginosa*.

Molecular typing (PFGE) was performed on 71 isolates recovered from the 25 positive swabs of the faucets and on 144 isolates recovered from the clinical specimens of 129 patients. For 10 patients *P. aeruginosa* isolates were not available or not typable. PFGE typing distributed the isolates into 59 different genotypes: 47 were recovered only from patients, 9 from patients and faucets, and 3 from faucets only. Multiple isolates from the same environmental sample and showing similar colony morphology all showed identical PFGE patterns.

Among the 129 patients for whom isolates could be typed three harbored two different genotypes; thus the number of cases was 132. The cases were divided into three groups according to epidemiological and molecular data (Table 1). Group A includes cases who harbored a genotype identical to one of those recovered from the faucets. Group B includes cases with a genotype that was found in at least one other patient but was not recovered from the faucets. Group C includes cases harboring a unique genotype. The mean duration (in days) of ICU stay before the isolation of the first *P. aeruginosa* strain was similar between the three groups: A:11.2±12.0, B: 10.2± 12.3, and C: 9.5±5.9. The majority (64%) of clinical samples were from the lower respiratory tract, followed by wounds (12%), endotracheal secretions (10%), and others sites (14%; e.g., blood, catheters). There was no significant difference in the frequency distribution of samples between the three groups of cases A, B, and C.

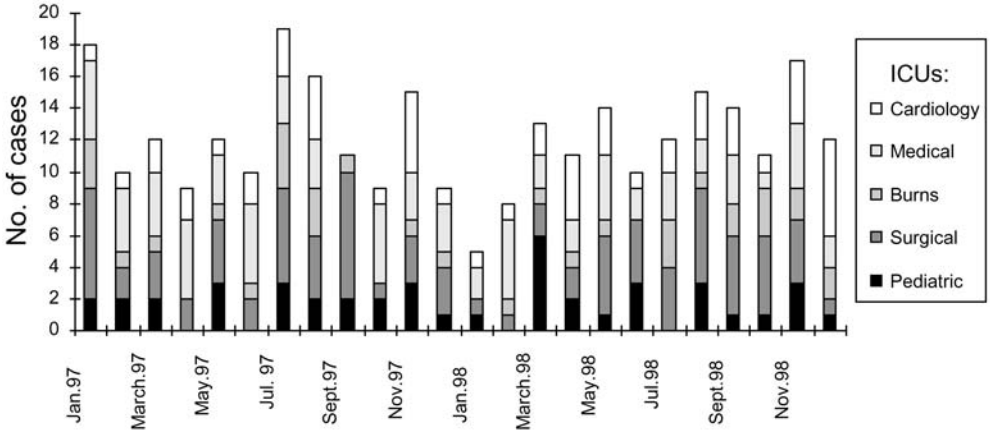

Fig. 1 Number of patients per month hospitalized in the different ICUs in whom *P. aeruginosa* was recovered from clinical specimen(s)

Table 1 *P. aeruginosa* recovered from patients and/or faucets in the ICUs between January and December 1998. Group A includes cases who harbored a genotype identical to one of those recovered from the faucets. Group B includes cases with a genotype that was found in at least one other patient, but was not recovered from the faucets. Group C includes cases harboring a unique genotype. The number of cases observed in clusters (cases of the same ICU with overlapping period of stay and with the same *P. aeruginosa* genotype) are also indicated for groups A and B

Water network	ICUs	Group A		Group B		Group C	Tap colonization[a] (%)
		No. of cases	No. of cases in clusters	No. of cases	No. of cases in clusters	No. of cases	
I	Pediatric	6	2, 2	5	–	8	1/63 (1.6%)
I	Surgical	10	–	12	2, 2	14	6/62 (9.7%)
I	Burns	9	2, 4	3	–	5	3/31 (9.7%)
II	Medical	21	4, 2, 2, 2, 2	5	3	4	5/28 (17.8%)
II	Cardiology	10	3	14	6, 2, 2	6	6/32 (18.8%)
	Total	56	25	39	17	37	21/216 (9.7%)

[a] No. positive/no. investigated

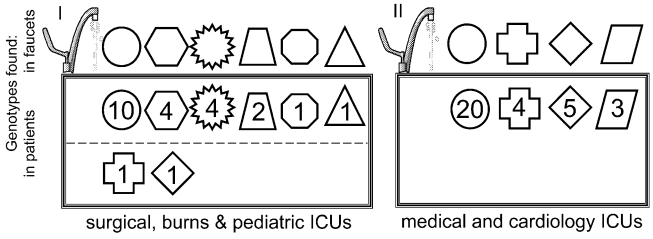

Fig. 2 Geographical and cases distributions of the nine *P. aeruginosa* genotypes isolated from both the faucets and the patients hospitalized in the different ICUs. Each genotype is represented by a different shape; the number of cases harboring each genotype are indicated in the shape

Group A includes 56 cases (42%) and nine genotypes (Fig. 2). The most prevalent genotype was isolated in the two water distribution networks and in 30 cases. The other eight genotypes were recovered only from either one network or the other. In ICUs of network I five genotypes were found in the faucets and in 12 patients hospitalized in these ICUs, and two cases harbored genotypes found in faucets of network II. In ICUs of network II three genotypes were found in faucets and in 12 patients hospitalized in these ICUs (Fig. 2). Six genotypes were recovered in the faucets before being isolated from 46 cases, and three genotypes were first recovered from ten patients cases before being recovered from faucets.

Among the remaining 76 cases 37 had a strain of a unique genotype (group C, Table 1), and 39 harbored an isolate indistinguishable from at least one other patient (group B, Table 1). Among the 39 cases of group B 17 (43%) occurred in clusters (6, 3, 2, 2, 2, 2 cases, respectively). Similarly, in the 56 cases of group A, 25 (45%) were involved in ten clusters (of 4, 4, 3, 2, 2, 2, 2, 2, 2, 2 cases, respectively), suggesting that patient-to-patient transmissions also occurred (Table 1).

Discussion

The results of the present study show several interesting features. Nearly one-half (42%) of cases colonized/infected with *P. aeruginosa* harbored strains which were genotypically identical to those recovered from the inner part of the faucets of the ICUs. Moreover, when genotypes were recovered only from the faucets of one of the two water distribution systems, the cases harboring theses genotypes were observed almost exclusively in the ICUs supplied by this system. This strongly suggest that the faucets were the ultimate reservoir for a substantial proportion of cases colonized/infected with *P. aeruginosa*. On the other hand, clusters of cases were observed both with genotypes recovered (group A) and not recovered (group B) from the water systems, suggesting that cross-transmission also accounts for part of the cases with genotypes identical to those of the faucets.

In several recent studies using molecular typing in a nonepidemic ICU setting, the major reservoir of *P. aeruginosa* was reported to be the endogenous flora of the patient. In one of the first studies that investigated this topic a German team prospectively searched for *P. aeruginosa* in patients, staff members, and the environment during a 4-month period in a surgical ICU [5, 6]. They found a low number of patients colonized with *P. aeruginosa* (18/153, 12%) and identified only two possible transmissions from patient-to-patient. Berthelot et al. [7] investigated the respective contribution of endogenous and exogenous sources of *P. aeruginosa* in mechanically ventilated patients. The presence of *P. aeruginosa* was prospectively assessed in the patients and in the environment. They concluded that for 80% of the cases (21/26) the origin of lungs colonization was endogenous. In a Dutch ICU Bonten et al. [8] prospectively investigated the patient's colonization and infection with *P. aeruginosa* during a period of endemicity. They concluded that the respiratory tract colonization was of exogenous origin in only 8% of the cases. In a similar study Speijer et al. [9] observed a large number of genotypes

and a small number of possible transmissions (5 among 49 patients with *P. aeruginosa*), indicating that most *P. aeruginosa* strains probably derived from the patients themselves.

On the other hand, other studies have shown that transmission from patient to patient or from environment to patient may play an important role. Cross-colonization was highlighted in a study of Bergmans et al. [10]. They prospectively investigated the colonization/infection of 100 patients in two ICUs during a period of endemicity. Nosocomial acquisition was suspected in 16 of 23 patients with *P. aeruginosa*. Other studies showed that drinking water was associated with *P. aeruginosa* infection when it was used for hydrotherapy of burned patients [11, 12] or for rinsing medical devices after disinfections [13]. Faucets and sinks were also suspected to be a nonnegligible source of *P. aeruginosa* when highly contaminated [3, 14].

Some of the apparently conflicting finding in the above studies may be due to methodological issues. In our study it was striking that only approximately 10% of the faucets were found to be positive, sometimes intermittently (data not shown), despite thorough swabbing of the inner part, and that all of the numerous water samples were negative for *P. aeruginosa*. Thus reservoirs such as faucets may be undetected in the absence of extensive and repeated sampling with an appropriate swabbing method.

The present report demonstrates that the water distribution system may be an important reservoir of nosocomial *P. aeruginosa* even when the contamination is low and despite the fact that the water meet the criteria for drinking water. Pathogen-free water should be used for mouth rinse and external care of high-risk patients. The eradication of *P. aeruginosa* in a hospital plumbing network might prove difficult, if at all possible [15]. In certain situations the use of filters connected at each faucet (end-line filtration) might help to better control *P. aeruginosa* and other water-borne bacteria. The hands of health care workers are probably responsible for most transmission of pathogens in ICU patients, including *P. aeruginosa* [14, 16]. Given the difficulty in controlling the water reservoir of *P. aeruginosa*, compliance to standard precautions and hand disinfection remain the cornerstone of prevention. Surveillance should be implemented, including molecular typing to assess the diversity of the strains and thus to measure the magnitude of potential exogenous infections.

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