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Rate of contamination of hospital privacy curtains on a burns and plastic surgery ward: a cross-sectional study

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SUMMARY

Background: Surfaces in the patient environment may play a role in microbial transmission if they become colonized by bacteria. Patient privacy curtains are one such surface that may pose a high risk for transmission because they are high-contact surfaces, are infrequently cleaned, and healthcare workers are less likely to wash their hands after contacting inanimate objects such as curtains.

Aim: To determine the amount and type of bacterial colonization of patient privacy curtains at a regional burns/plastic surgery unit.

Methods: Privacy curtain contamination on the burns/plastic surgery ward was determined for two separate occasions six months apart: 23 curtains on August 2015 and 26 curtains on January 2016. Dey—Engley neutralizing agar (DENA) replicate organism detection and counting (RODAC) contact plates were used daily to sample curtains near the edge hem where they are most frequently touched. Microbial contamination was reported as cfu/cm^2 and the presence of meticillin-resistant *Staphylococcus aureus* (MRSA) was determined. Swabs were also taken of any open wounds and from tracheostomy sites on the ward.

Findings: Curtain contamination in August 2015 was 0.7—4.7 cfu/cm^2 with 22% testing positive for MRSA, whereas contamination on January 2016 was 0.6—13.3 cfu/cm^2 with 31% of curtains testing positive for MRSA.

Conclusion: Curtains on the burns/plastic surgery ward become colonized with significant quantities of bacteria. Future studies will need to address the rate of colonization and the clinical impact of this colonization to better inform cleaning protocols.

Introduction

Hospital-acquired infections (HAIs) are associated with significant mortality, morbidity and resulting costs. Inanimate

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objects such as patient privacy curtains may be a potential source of transmission. As such, it is important to know the rate of bacterial contamination on privacy curtains.

It is estimated that at least 220,000 HAIs occur in Canada each year, causing more than 8000 deaths [1]. HAIs place significant stress on the healthcare system with 1—4 additional hospital days for a urinary tract infection, 7—8 days for a surgical site infection, 7—21 days for a bloodstream infection, and 7—30 days for pneumonia [2].

Efforts toward decreasing the rate of HAIs have often focused on promoting proper hand hygiene of healthcare workers before and after direct patient contact to decrease the chances of acting as a vector for pathogen transmission between patients [3]. However, environmental surfaces in hospitals are also potential sources of contamination. Bhalla *et al*. demonstrated that 53% of hands that were culture negative following hand hygiene became culture positive after coming into contact with environmental surfaces (this included vancomycin-resistant *Enterococcus* spp. (VRE) and meticillin-resistant *Staphylococcus aureus* (MRSA) [4]. Therefore, direct contact with a contaminated surface after performing hand hygiene may lead to unrecognized recontamination of caregiver hands.

Curtains are one possible environmental reservoir of microbes and have been implicated in HAI outbreaks. A 2002 study of a carbapenem-resistant *Acinetobacter baumannii* outbreak in an intensive care unit attributed curtains as the primary reservoir for the *A. baumannii* [5]. Another study identified curtains as the potential source of group A streptococcus cross-transmission in an ear—nose—throat ward [6].

Potential for cross-contamination to and from the curtains is increased in the presence of open wounds. Burns/plastic surgery units are especially at risk due to the number and complexity of patients with open wounds. This risk has been recognized by the Ontario Agency for Health Protection and Promotion who identified burn survivors as a highly vulnerable population and burns units as being at high risk for exposure from surfaces [7].

Due to the growing awareness of curtain contamination as a potential reservoir for microbes, we aimed to determine the amount and type of bacterial colonization of patient privacy curtains at a regional burns/plastic surgery unit. We hypothesized that the hospital privacy curtains were at risk of significant contamination both in the number of bacteria present, and in the presence of multi-drug resistant bacteria.

Methods

Sample collection

The study protocol was reviewed and approved by the local institutional ethics review board. Cultures were collected from patient privacy curtains on the burns/plastic surgery unit at a level 1 trauma referral centre in Winnipeg, Manitoba, Canada. Swabs were taken of any open wounds and from tracheostomy sites. To confirm the results, samples were collected on two separate occasions six months apart: 23 curtains were sampled on August 2015 and 26 were sampled on January 2016.

Bacterial cultures were obtained using Dey—Engley neutralizing agar (DENA) RODAC contact plates (Oxoid cat. no. RE111103). Each RODAC plate was handled by a gloved data collector, brought to room temperature before use, and two sites were sampled from each curtain. The sample sites were on the side of the inside surface of curtain facing the patient near the edge hem, with the first sample taken from above shoulder height and the second sample taken from below shoulder height. For each sample, the RODAC plate was pressed against the curtain for 1 min, while holding a RODAC plate lid covered with paper towel on the opposite side of the curtain to ensure a tight, flat contact between the RODAC plate and the curtain.

Bacterial isolation

Curtain contact plates were incubated at 37°C for 48 h and swab samples were streaked on lysogeny broth (LB) agar (Bacto™ LB agar, Lenox, BD, France) and incubated at 37°C for 48 h. After incubation, colony-forming units (cfu) were counted from each plate. As a representation of hospital-acquired bacteria, cultures were examined for the presence of MRSA. For the isolation of MRSA, bacterial colonies were streaked on to the mannitol salt agar (MSA) (with 6 μg/mL oxacillin; Oxoid, Canada) and incubated at 37°C for 48 h. Initially, phenotypic identification of MRSA strains was confirmed by growth on MSA—oxacillin agar. SA003 (CA-MRSA #40065) was used as a reference strain throughout the study. Due to the heavy growth on the first testing date, the decision was made to use MSA—oxacillin for screening for the second testing date.

Genotypic analysis

All strains isolated on MSA—oxacillin agar were further screened by nuclease (*nuc*) and *mecA* gene by colony polymerase chain reaction (PCR) method. Briefly, primers (Table I) for the amplification of *nuc* and *mecA* gene were used. The DNA template was prepared by resuspending bacterial colony in 50 μL of sterile water and incubated at 100°C for 10 min. One microlitre of crude extract was used as template in 10 μL of PCR reaction using 0.2 μM primers, 200 μM dNTPs, 1.25 U of *Taq polymerase* and 10× PCR buffer. PCR reaction steps were as follows: initial denaturation at 94°C for 2 min, denaturation at 94°C for 30 s, annealing temperature 51°C and 45°C for 30 s (for *nuc* and *mecA* gene, respectively), extension at 72°C for 1 min and final extension at 72°C for 5 min. Denaturation to extension steps was repeated 30 times. Eight microlitres of the amplified PCR mixture was loaded to 0.8% agarose gel containing ethidium bromide and then visualized in UV transilluminator. A 1 kb DNA ladder was used to compare amplified PCR product [8,9].

Table I
Primers used in this study to detect *nuc* and *mecA* genes

Gene	Primers	Product size (bp)
Nuclease (*nuc*)	FW 5′-GCGATTGAT GGTGATACGGTT-3′	279
	RV 5′-AGCCAAGCCTTGAC GAACTAAAGC-3′	
mecA	FW 5′-AACAGGTGAAT TATTAGCACTTGTAAG-3′	174
	RV 5′-ATTGCTGTTAAT ATTTTTTGAGTTGAA-3′	