and 6.58 (95% CI, 6.37-6.79), respectively (Fig. 1). The Tru-D vegetative and 5-minute Moonbeam cycles were similarly effective (P > .99), and both were more effective than the 3-minute Moonbeam cycle (P <.001 and P < .001, respectively). MRSA samples receiving direct UV-C exposure had significantly greater log10 reductions (6.95; 95% CI, 6.89–7.01) than did indirect exposure (6.67; 95% CI, 6.46–6.87; P < .05) (Fig. 2). For C. difficile, the Tru-D sporicidal, the 5-, and 3-minute Moonbeam cycles resulted in average CFU log10 reductions of 1.78 (95% CI, 1.43-2.12), 0.57 (95% CI, 0.33-0.81) and 0.64 (95% CI, 0.42-0.86), respectively (Fig. 1). Tru-D was significantly more effective than either the 3- or 5-minute Moonbeam cycles (P < .00). C. difficile samples receiving direct UV-C exposure had higher dosage and significantly greater log10 reductions (1.34; 95% CI, 1.10-1.58) than did indirect exposure (0.58; 95% CI, 0.31–0.86; *P* < .01) (Fig. 2). Conclusions: Use of the Tru-D vegetative cycle and the Moonbeam 3- and 5-minute cycles resulted in similar reductions in MRSA; both resulted in

significantly greater reductions than the manufacturer's recommended 3-minute Moonbeam cycle. Therefore, healthcare facilities should carefully evaluate manufacturer-recommended run times in their specific clinical setting. For *C. difficile*, the Tru-D sporicidal cycle was significantly more effective than either of the Moonbeam cycles, likely due to higher irradiation levels. As such, direct UV-C exposure resulted in greater average reductions than indirect exposure.

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Presentation Type:

Poster Presentation

Emergence of Vancomycin Resistance after Treatment of Enterococcus: Risk Factors for Subsequent Pathogen Resistance

Variable	VSE-VSE (n=99)	VSE-VRE (n=76)	p value
Average Age Years (SD)	60.5 (17.4)	60.4 (16.5)	0.966
Male Gender (%)	45 (45.5)	32 (42.1)	0.7588
E.faecalis (%)	28 (28.2)	21 (27.6)	
Hospital Amission w/in 2y (%)	87 (89.9)	72 (94.7)	0.1848
ICU Admission w/in 2y (%)	35 (35.4)	41 (53.9)	0.0207
Prior Clostridium difficile infection (%)	11 (11.1)	20 (26.3)	0.0155
Prior H2 Blocker/PPI	75 (75.8)	56 (73.7)	0.8607
Prior Positive VRE Swab or Culture	28 (28.3)	36 (47.4)	0.0114
Time between Cultures in days (SD)	21.5 (21.7)	21.7 (22.2)	0.9694
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Initial VSE Culture Sites			
Abdominal Fluid (%)	13 (13.1)	10 (13.2)	
Abscess (%)	4 (4.0)	3 (4.0)	
Bile (%)	5 (5.1)	4 (5.3)	
Blood (%)	22 (22.2)	17 (22.4)	
Bone (%)	1 (1.0)	1 (1.3)	
Fluid NOS (%)	6 (6.1)	4 (5.3)	
Tissue (%)	10 (10.1)	8 (10.3)	
Urine (%)	38 (38.4)	29 (38.2)	
Antibiotics Prior to Initial VSE			
Amoxicillin-Clavulanic Acid (%)	14 (14.1)	13 (17.1)	0.6744
Ampicillin-Sulbactam (%)	15 (15.1)	5 (6.6)	0.095
Cefazolin (%)	25 (25.3)	3 (3.9)	0.0001
Cefepime (%)	23 (23.2)	32 (42.1)	0.0089
Ciprofloxacin (%)	29 (29.3)	29 (38.2)	0.2575
Daptomycin (%)	14 (14.1)	14 (18.4)	0.5337
Ertapenem (%)	13 (13.1)	8 (10.5)	0.6462
Levofloxacin (%)	28 (28.3)	25 (32.9)	0.5127
Linezolid (%)	10 (10.1)	12 (15.8)	0.3578
Piperacillin-Tazobactam (%)	37 (37.4)	38 (50.0)	0.1232
Trimethoprim-Sulfamethoxazole (%)	14 (14.1)	14 (18.4)	0.5337
Vancomycin (%)	46 (46.5)	53 (69.7)	0.0022
Antibiotics After initial VSE			
Cefepime (%)	15 (15.2)	20 (26.3)	0.0861
Ceftriaxone (%)	14 (14.1)	9 (11.8)	0.822
Cephalexin (%)	7 (7.1)	0 (0	0.0193
Ciprofloxacin (%)	14 (14.1)	8 (10.5)	0.5015
Ertapenem (%)	10 (10.1)	5 (6.6)	0.5873
Linezolid (%)	10 (10.1(7 (9.2)	1
Piperacillin-Tazobactam (%)	26 (26.3)	21 (27.6)	0.8647
Vancomycin (%)	32 (32.3)	41 (53.9)	0.0053

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Background: Vancomycin-resistant Enterococcus (VRE) is a leading cause of nosocomial infections that carries an increased risk of mortality when compared to vancomycin-sensitive Enterococcus (VSE). Data on the frequency of conversion between VSE and VRE in patients are scarce. Among patients presenting with VSE infections, little is known about the subsequent risk of conversion to VRE in the initial treatment period. Methods: A descriptive analysis of VSE to VRE conversion and a retrospective case-control study were performed examining cases of VSE that had subsequent cultures positive for VRE within 90 days within a quaternary healthcare system. Cases were obtained from June 2013 through December 2018. Controls were patients who had VSE culture followed by another VSE culture and were matched by organism (E. faecalis or E. faecium), time between cultures, and initial culture site. Age, gender, healthcare, antibiotic, Clostridiodes difficile, proton pump inhibitor (PPI) exposure, and H2 blocker exposures, and prior VRE infection or colonization were abstracted from the electronic medical record. A univariate analysis with the Fisher exact test was performed with significance considered for P < .05. Results: In total, 8,913 cases of *E. faecalis* and 2,322 cases of E. faecium were included in the study. Of 8,913 cases of E. faecalis, 51 of 8,503 (0.6%) cultured VRE after VSE, and 47 of 403 (11.7%) cultured VSE after initial VRE. Of E. faecium, 51 of 783 (6.5%) cultured VRE after VSE, and 76 of 1,532 (5.0%) cultured VSE after initial VRE. In total, 76 cases were matched with 99 controls. Patients converting from VSE to VRE were more likely to have prior admission to an intensive care unit (P = .0207), prior positive swab or culture for VRE (P = .0114), previous C. difficile infection (P =.0155), prior vancomycin (P = .0022) and cefepime (P =.0089) exposure. Patients receiving vancomycin after initial VSE culture were more likely to have subsequent cultures positive for VRE (P = .0053). There was no difference in age (P =.966) or male sex (P = .7588). Conclusions: Conversion from VSE to VRE is common, and *E. faecium* is more likely to become resistant than E. faecalis. Reversion to a vancomycin-sensitive phenotype is also common, and E. faecalis is more likely to show subsequent sensitivity than E. faecium. Previous admission to an intensive care unit, prior colonization or infection with VRE, prior C. difficile infection, and exposure to vancomycin and cefepime are risk factors for emergence of VRE after treatment for vancomycin-sensitive Enterococcus.

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Poster Presentation

Enhanced Bundled Interventions to Reduce Surgical Site Infections for Patients with Congenital Cardiac Disease

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Background: Surgical site infections (SSIs) among cardiothoracic (CT) patients are associated with high rates of morbidity and mortality. Data are limited regarding SSI incidence among pediatric patients undergoing primary reparative procedures for congenital cardiac disease. Published evidence on targeted interventions to prevent pediatric CT-surgery SSI is lacking. We aimed to establish standard metrics for measuring CT-surgery SSI incidence and to implement bundled interventions for SSI prevention. Methods: A dedicated CT-surgery SSI prevention workgroup was established, consisting of hospital leadership, CT surgeons, cardiac critical care unit staff, anesthesia, perfusion, environmental services, instrument sterile processing, risk management, infection prevention and antibiotic stewardship. We created a standard definition for CT-surgery SSI and calculated retrospective SSI rates over a 24-month period (2017-2019). The outcome measured was incidence of CT-surgery SSI per 100 primary cardiac procedures with delayed (\geq 3 days after primary surgery) or non-delayed chest closure. The difference in proportion of SSI was reported separately for delayed closure and non-delayed closure; statistical significance was tested using a Fisher's Exact test. We identified many potential improvement opportunities, including gaps in SSI surveillance, poor compliance with daily bathing, inconsistent perioperative antimicrobial prophylaxis, lack of controlled environment for bedside chest closures, and lapses in environmental cleaning. These issues informed the enhanced SSI prevention bundle, which included education on sterility with the operating room (OR) staff. Protocols for care of cardiac patients with delayed chest closures focused on universal daily and preoperative chlorhexidine baths. In addition, the bundle incorporated stringent environmental cleaning interventions including scheduled decluttering of patient rooms and clinical spaces, terminal cleaning of patient rooms prior to returning from the OR, and use of adjunctive ultraviolet light for the daily cleaning of operating rooms and patient rooms at discharge. Results: Surveillance definition of microbiological growth from a clinical sample obtained within 30 days of primary cardiac procedure sufficiently captured all CT-surgery SSIs. Of 551 CT-surgery procedures prior to intervention, 91 (17%) had delayed final operative closures. Prior to the intervention, 16 SSIs were identified from July 2017 - May 2019 for a rate of 2.90 per /100 procedures, and was higher among patients with delayed chest closure 6.59 per /100 procedures (6 SSIs/91 procedures) versus those with primary chest closure 2.17 per /100 procedures (10 SSIs/460 procedures; P = 0.034). Gram-positive organisms, including coagulase coagulase-negative Staphylococci, were most frequently identified as the causative organisms for SSIs. Compliance with bundled intervention, rolled out over a 2-month period, was associated with an immediate decrease in the number of SSIs for primary and delayed chest closures 6SSIs /185 procedures in the initial quarters (August - December 2019) of the postintervention period. However, this decrease was not reflected in the overall rate (3.24 per /100 procedures) due to fewer procedures performed. Data collection to measure sustainability is ongoing. Conclusions: Bundled interventions targeting skin antisepsis and environmental cleaning may be associated with a decrease in SSIs among pediatric CT-surgery patients.