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BRIEF REPORT

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Screening sites for detection of carbapenemase-producers– a retrospective cohort study

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Abstract

While screening the rectal site and urine may be appropriate for detection of carbapenemase-producing *Enterobacterales*, respiratory samples, throat and wound swabs may increase the sensitivity of screening protocols when aiming to detect colonization with carbapenemase-producing non-fermenting bacteria. Our results support the need for tailoring screening recommendations according to the bacterial species targeted.

Keywords Carbapenemases, Carbapenemase-producing bacteria, Carbapenemase-producing *Enterobacterales*, Non-fermenting bacteria, Screening

Introduction

Active surveillance by screening high-risk patients for carbapenemases-producing bacteria (CPB) is a crucial part of infection prevention and control measures (IPC) as recommended by international guidelines such as the ones published by the World Health Organization (https://iris.who.int/bitstream/handle/10665/259462/9789241550178-eng.pdf?sequence=1). Given the natural intestinal colonization with *Enterobacterales*, patients colonized with carbapenemase-producing *Enterobacterales* (CPE) can be identified by rectal swabs in a majority of the cases [1, 2]. In contrast, knowledge regarding the optimal screening sites for detection of carbapenemase-producing non-fermenting bacteria (CPNF), such as *Acinetobacter baumannii* complex or *Pseudomonas* species, is limited. Consequently, recommendations for active surveillance cultures remain inconclusive, leading to different screening practices within healthcare institutions [3]. Enhancing comprehension of body sites most likely colonized with CPE and CPNF may help tailoring species-specific screening strategies, facilitating early detection of patients at risk for CPB-colonization and possibly minimizing costs for screening of body sites unlikely to add further diagnostic value. To address this gap, we evaluated different body sites for the detection of CPB by comparing the proportions of positive sites between patients colonized with CPE and CPNF, to provide insights for the refinement of screening strategies.

Methods

This retrospective, single-center cohort study included consecutive patients with detection of CPB-colonization or infection between 01/2008 and 09/2023 in the in- and outpatient setting of the University Hospital Basel (UHB). The UHB is a tertiary academic care center in a

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low CPB-endemicity setting, admitting more than 40,000 patients annually.

Results of clinical and screening samples were assessed to determine sites most likely yielding growth of CPB. The following body sites are considered for CPB-screening at the UHB: rectum, groin, throat, wounds, urine and insertion sites of catheters or drainages. All patients with a known history of CPB-colonization as well as patients admitted directly from acute care facilities outside of Switzerland or prolonged contact to a foreign healthcare system within the prior twelve months are routinely screened for carriage of multi-drug resistant organisms (MDRO) on hospital admission. Furthermore, as part of our active surveillance infection prevention and control program, patients admitted to high-risk wards such as the hematologic ward or intensive care units (ICU), if the patient is intubated and has an expected length of ICU stay > 24 h, are screened for MDRO.

Patients were excluded if refusal of subsequent use of their data was documented.Data was retrospectively extracted from patients' medical charts. A "REDCap" database was used for collection of data. Definitions of the assessed variables are provided in the supplementary file.

Standard diagnostic approaches were applied for detection of CPB at the UHB laboratory for bacteriology and mycology. Testing for CPB changed within the study period, the following describes the most recent methods: Chrom ID.® Carba Smart agar (bioMérieux) was used for screening of CPB. Carbapenemase genes for *Enterobacterales* were tested using eazyplex Superbug CRE panel detecting OXA-48, OXA-181, KPC, NDM, VIM. *Pseudomonas* spp. were analyzed using GeneXpert Carba-R (Cepheid Switzerland, Thalwil, Switzerland) detecting OXA-48, KPC, NDM, VIM, IMP-1 and *Acinetobacter baumannii* complex using eazyplex SuperBug complete A test system including KPC, VIM, OXA-48, OXA-23, OXA-40, and OXA-58. [4, 5]

This study was approved by the local ethics committee (Ethikkomission Nordwest- und Zentralschweiz [EKNZ], Project-ID 2019–01548) and is a part of the clinicaltrials.gov-registered study "Epidemiology of Carbapenemase-producing Bacteria in a Swiss Tertiary Care Hospital" (ID NCT04098133). We adhered to the "Strengthening the Reporting of Observational Studies in Epidemiology" guidelines (https://www.equator-network.org/reporting-guidelines/strobe/).

Statistical analyses

Counts and proportions were applied for categorical variables using Fisher's exact test, medians and interquartile ranges for continuous variables using Mann Whitney U test. Comparisons between the proportions of positive sampling sites as well as patient-related risk factors for carriage of CPE or CPNF were performed by univariate and multivariate logistic regression analyses. Odds ratio > 1 indicate higher odds for colonization with CPE, while OR < 1 indicate higher odds for colonization with CPNF. Missing information was considered as absent factor. Statistical significance was defined as p-values < 0.05. Statistical analyses were performed using STATA version 16.1 (StataCorp, College Station, TX).

Results

We identified 119 eligible patients colonized with a total of 158 CPB, accounting for 115 cases of CPE and 43 cases of CPNF. Co-colonization with both CPE and CPNF occurred in 11 patients. Baseline characteristics of the study cohort are summarized in supplementary Table 1. The median age was 65 years (IQR 51–73) and 38.7% were female (N = 46). 104 patients (87.4%) had been hospitalized in the 12 months prior to the index hospitalization and 66 patients (55.5%) had travelled outside of Switzerland. Of those, 60 patients (90.9%) were hospitalized abroad. Antibiotic therapies within three months prior to detection of CPB had been administered in 85 patients (71.4%). The median Charlson Comorbidity Index (CCI) was 2 (1–3). *Klebsiella pneumoniae* (N = 46, 29.1%) and *Escherichia coli* (N = 45, 28.5%) were the most frequently detected CPE, whereas the most frequent CPNF was *A. baumannii* (N = 32, 20.3%) (supplementary Table 2).

Table 1 summarizes the results of both screening and clinical samples based on each detected CPB species (N = 158). A higher percentage of positive rectal swabs (OR 4.62, 95%CI 1.93–11.07, $p < 0.001$) and urine screening samples (OR 3.90, 95%CI 1.06–14.32, $p = 0.040$) was associated with detection of CPE as compared to CPNF. Conversely, CPNF were more frequently found in respiratory samples (OR 0.11, 95%CI 0.04–0.29, $p < 0.001$), throat swabs (OR 0.23, 95%CI 0.07–0.79, $p = 0.020$) as well as in swabs of chronic or acute wounds (OR 0.07, 95%CI 0.01–0.80, $p = 0.032$ / OR 0.17, 95%CI 0.05–0.63, $p = 0.008$). Urinary catheterization at time of sampling was not associated with detection of either CPE or CPNF considering screening and clinical urine samples (Table 1). Analysis of positivity rates of screening sites in patients with detection of only CPE (N = 80) versus only CPNF (N = 28) was performed to account for the potential multiple inclusion of screening sites within the same patient (supplementary Table 3). Again, a higher percentage of positive rectal swabs (OR 9.0, 95%CI 2.60–31.18, $p = 0.001$) was associated with detection of CPE while urine screening tended to be associated with CPE detection (OR 5.0, 95%CI 0.97–25.38, $p = 0.052$). Respiratory samples (OR 0.46, 95%CI 0.01–0.16, $p < 0.001$), throat

Table 1 Comparison of proportions of positive screening sites of CPE versus CPNF

Localization	CPE N = 115			CPNF N = 43			Univariable analysis	Multivariable correction for CCI Score[d]
	All N (%)	Positive N (%)	Negative N (%)	All N (%)	Positive N (%)	Negative N (%)	OR[e] (95%CI), *p*-value[f]	OR[e] (95%CI), p-value[f]
Screening samples (N = 453)								
Rectal swab	98 (85.2)	75 (76.5)	23 (23.5)	29 (67.4)	12 (41.4%)	17 (58.6)	4.62 (1.93–11.07), **0.001**	4.58 (1.82–11.53), **0.001**
Perianal swab	2 (1.7)	2 (100.)	0	0	0	0		
Urine	72 (62.6)	25 (34.7)	47 (65.3)	25 (58.1)	3 (12.0)	22 (88.0)	3.90 (1.06–14.32), **0.040**	4.68 (1.20–18.26), **0.026**
Urinary catheter[a]	20 (17.4)	7 (35.0)	13 (65.0)	9 (20.9)	0 (0.0)	9 (100)	–	
Groin swab	45 (39.1)	23 (51.1)	22 (48.9)	12 (27.9)	4 (33.3)	8 (66.7)	2.09 (0.55–7.95), 0.279	
Throat swab	43 (37.4)	11 (25.6)	32 (74.4)	15 (34.9)	9 (60.0)	6 (40.0)	0.23 (0.07–0.79), **0.020**	0.20 (0.05–0.76), **0.018**
Chronic wound swab[b]	11 (9.6)	4 (36.4)	7 (63.6)	9 (20.9)	8 (88.9)	1 (11.1)	0.07 (0.01–0.80), **0.032**	0.04 (0.00–0.72), **0.029**
Acute wound swab[c]	33 (28.7)	9 (27.3)	24 (72.7)	16 (37.2)	11 (68.8)	5 (31.3)	0.17 (0.05–0.63), **0.008**	0.16 (0.04–0.63), **0.009**
Vaginal swab	2 (1.7)	2 (100)	0	1 (2.3)	0	1 (100)	–	
Insertion site vascular catheter	22 (19.1)	0	22 (100)	12 (27.9)	1 (8.3)	11 (91.7)	–	
Insertion site drainage	3 (2.6)	3 (100)	0	3 (7.0)	0	3 (100)	–	
Clinical samples (N = 79)								
Abscess		4 (3.5)			0		–	
Ascites		1 (0.9)			0		–	
Blood culture		7 (6.1)			6 (14.0)		0.40 (0.13–1.27) 0.119	
Biopsy		10 (8.7)			4 (9.3)		0.93 (0.28–3.13), 0.905	
Deep swab		10 (8.7)			1 (2.3)		4.00 (0.50–32.20), 0.193	
Respiratory sample		7 (6.1)			16 (37.2)		0.11 (0.04–0.29), < **0.001**	0.11 (0.04–0.34), < **0.001**
Urine		12 (10.4)			1 (2.3)		4.89 (0.62–38.83), 0.133	
Urinary catheter[a]		2 (1.7)			1 (2.3)		0.74 (0.07–8.41), 0.811	

Significant *p*-values are indicated in bold and defined as a *p*-value < 0.05

CPE, carbapenemase-producing *Enterobacterales*; CPNF, carbapenemases-producing non-fermenting bacteria

[a] in place at time of sampling, transurethral or suprapubic

[b] Ulcers, decubiti

[c] e.g. traumatic wounds, surgical wounds

[d] CCI-Score OR 1.70 (95%CI 1.34–2.14), *p*-value < 0.001, see supplementary Table 3

[e] OR (Odds ratio) > 1 indicates higher odds for colonization with CPE, while OR < 1 indicates higher odds for colonization with CPNF

swabs (OR 0.09, 95%CI 0.02–0.49, $p = 0.005$) and swabs of acute wounds (OR 0.09, 95%CI 0.01–0.62, $p = 0.015$) remained associated with detection of CPNF.

Comparisons of demographics and treatment data between patients colonized with CPE versus those carrying CPNF are provided in Table 2. A history of colonization with CPB (OR 10.43, 95%CI 1.59–68.56, p = 0.015) and a higher CCI (OR 1.58, 95%CI 1.04–2.40, $p = 0.031$) was associated with the CPE-group after multivariate analysis, while urinary catheterization within the prior 30 days (OR 0.10, 95%CI 0.03–0.35, $p < 0.001$) was associated with the CPNF-group.

To minimize potential confounding of screening sites and clinical samples associated with either CPE or CPNF due to CCI, multivariate logistic regression analyses were applied to adjust for CCI. None of the assessed sampling sites lost statistical significance (i.e. *p*-value < 0.05) (Table 1).

Discussion

This retrospective cohort study, conducted in a low CPB endemicity setting, found rectal swabs and urine screening samples to be associated with detection of CPE, while respiratory samples including throat swabs and wound swabs were associated with detection of CPNF. Patients colonized with CPE had a higher CCI and were more often found to have a history of CPB-colonization within the previous 12 months compared to CPNF-colonized patients.

Both, *Acinetobacter baumannii* complex and *Pseudomonas* spp., are known for colonization of moist body sites such as the respiratory tract or wounds in addition

Table 2 Comparison of patients carrying only CPE versus patients carrying only CPNF

	CPE N = 80	CPNF N = 28	Univariable analysis		Multivariable analysis	
	N (%) or median (IQR)	N (%) or median (IQR)	OR[e] (95%CI)	*p*-value[f]	OR[e] (95%CI)	*p*-value[f]
Age (years)	68 (48–74)	65 (54–72)	0.99 (0.97–1.02)	0.441		
Female sex	34 (42.5)	11 (39.3)	1.14 (0.47–2.75)	0.767		
Localization before hospitalization			0.37 (0.23–0.60)	**< 0.001**	0.68 (0.36–1.30)	0.244
Home	61 (76.3)	9 (32.1)				
Nursing home	2 (2.5)	1 (3.6)				
Other acute care facility	17 (21.3)	18 (64.3)				
Discipline/ward			0.82 (0.54–1.23)	0.330		
Surgery	31 (38.8)	13 (46.4)				
Medicine	40 (50.0)	12 (42.9)				
Isolation ward	8 (10.0)	0				
Gynaecology	1 (1.3)	0				
Urology	0	3 (10.7)				
History of hospitalization[a]	69 (86.3)	24 (85.7)	1.05 (0.30–3.59)	0.944		
Stay in ICU	9 (11.3)	6 (21.4)	0.45 (0.14–1.43)	0.177		
History of colonization with CPB[a]	27 (33.8)	2 (7.1)	6.62 (1.46–30.01)	**0.014**	10.43 (1.59–68.56)	**0.015**
History of colonization with ESBL-PE[a]	28 (35.0)	4 (14.3)	3.23 (1.02–10.24)	**0.046**	2.65 (0.54–12.99)	0.230
Travel history[a]	35 (43.8)	20 (71.4)	0.31 (0.12–0.79)	**0.014**	0.68 (0.19–2.41)	0.550
Hospitalization abroad[a]	31 (38.8)	18 (90.0)	0.86 (0.14–5.18)	0.870		
CCI	2 (1–4)	1 (0–2)	1.84 (1.31–2.60)	**0.001**	1.58 (1.04–2.40)	**0.031**
Antibiotic therapy prior to detection of CPB[b]	53 (66.3)	24 (85.7)	0.33 (0.10–1.04)	0.058		
Immunosuppressing therapy[a]	20 (25.0)	3 (10.7)	2.78 (0.76–10.19)	0.124		
PPI[a]	43 (53.8)	12 (42.9)	1.55 (0.65–3.69)	0.323		
Chronic wounds	9 (11.3)	7 (25.0)	0.38 (0.13–1.14)	0.085		
Recent surgery[a]	36 (45.0)	15 (53.6)	0.71 (0.30–1.68)	0.435		
Urinary catheterization[c]	20 (25.0)	22 (78.6)	0.09 (0.03–0.26)	**< 0.001**	0.10 (0.03–0.35)	**< 0.001**
Vascular hardware[d]	7 (8.8)	5 (17.9)	0.44 (0.13–1.52)	0.196		
Outcome						
Infection due to CPB	17 (21.3)	8 (28.6)	0.67 (0.25–1.80)	0.431		
Length of hospital stay (days)	15 (9–32)	22 (9–41)	0.99 (0.97–1.00)	0.143		
Death	4 (5.1)	9 (32.1)	0.11 (0.03–0.41)	**0.001**		

Significant *p*-values are indicated in bold and defined as a *p*-value < 0.05

CPE, carbapenemase-producing *Enterobacterales*; CPNF, carbapenemases-producing non-fermenting bacteria; IQR, interquartile range; 95%CI, 95% confidence interval; ICU, intensive care unit; CCI, Charlson Comorbidity Index; PPI, proton pump inhibitor

[a] within the prior 12 months

[b] within the prior 3 months

[c] within the prior 30 days

[d] in place ≥ 7 days

[e] OR (Odds ratio) > 1 indicates higher odds for colonization with CPE, while OR < 1 indicates higher odds for colonization with CPNF

to the gastrointestinal tract [6]. Our results indicate that rectal swabs alone may prove insufficient for detection of CPNF and adding respiratory samples or wound swabs might enhance sensitivity of testing. These findings are in line with a study by Bopp et al. [2], revealing the highest percentage of positive sites for multi-drug-resistant non-fermenting gram-negative bacteria in respiratory and wound samples, while positivity rates for rectal samples or urine were low (45.5% and 15.4% respectively). Other studies identified screening of buccal mucosa or skin [7, 8] to provide the highest yield for detection of carbapenem-resistant *A. baumannii* (CRAB), with no significant additional value of rectal screening samples. Both buccal mucosa and skin have not been considered for screening at the UHB so far, potentially leading to missed cases of CRAB carriers. Furthermore, *Pseudomonas* spp. were found in 19.1% of oral cavities of Polish adolescents, all of which were categorized as multi-drug-resistant (MDR), likely facilitating spread to and colonization of the respiratory tract [9]. Considering these findings, our results

support the inclusion of wound swabs and respiratory tract sampling in CPNF screening protocols.

The observed differences concerning patient-related factors between patients colonized with CPE compared to CPNF observed in our cohort indicate two different patient profiles. The association of a history of colonization with CPB within the previous 12 months with the CPE-group, along with a higher CCI, are pointing to a chronically ill patient group, characterized by frequent hospitalizations and potential exposure to antibiotic selection pressure. Colonization or infection with CPB has previously been described as a risk factor for current CPE carriage [10]. These results are not surprising considering time to intestinal clearance of CPE might take months to years [11].

In contrast, the CPNF group was associated with a higher rate of urinary catheterization within the previous 30 days and in-hospital mortality rates were higher compared to CPE, a finding consistent with previous research by Kassem et al. [12]. Given these results, urinary catheterization is less likely an independent risk factor for CPNF colonization in itself, but may rather serve as an indicative parameter pointing towards an acutely and severely ill patient population. These findings may be helpful in assessing the necessary screening strategy based on the patients' individual risk profile.

As this is a retrospective, single-center study, conducted in a country of low CPB-prevalence, its results may not be generalizable to other settings. Furthermore, due to the study design, screening for CPB was not performed systematically and patients with detection of CPB from clinical samples did not necessarily receive a screening of further sites, hence testing was heterogeneous. While the case numbers of CPNF are low and may therefore be underpowered to adequately identify optimal screening sites, the results nonetheless provide valuable insights into a topic that remains insufficiently studied. Furthermore, this study does not specifically analyze potential differences in the positivity rates of screening sites between *A. baumannii* and *P. aeruginosa*, particularly due to the low case numbers. In clinical practice however, a tailored screening approach targeting each specific pathogen could prove challenging to implement.

While this study is not the first to point to differences between optimal screening sites for CPE and CPNF, it does provide a direct comparison between colonized sites, thereby further supporting the findings of previous studies and strengthening the evidence base for future screening recommendations.

In conclusion, while screening the rectal site and urine may be appropriate for detection of CPE, respiratory samples, throat and wound swabs may increase the sensitivity of screening protocols when aiming to detect CPNF colonization. Our results support the need for tailoring screening recommendations according to the bacterial species targeted.

List of abbreviations

CPB	Carbapenemase-producing bacteria
CPE	Carbapenemase-producing *Enterobacterales*
CPNF	Carbapenemase-producing non-fermenting bacteria
CRAB	Carbapenem-resistant *Acinetobacter baumannii*
CCI	Charlson comorbidity index
EKNZ	Ethikkommission Nordwest- und Zentralschweiz
IPC	Infection prevention and control measures
ICU	Intensive care unit
IQR	Interquartile range
MDRO	Multi-drug resistant organisms
OR	Odds ratio
UHB	University Hospital Basel
95%CI	95% Confidence interval

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13756-024-01513-2.

Additional file1

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Author contributions
IV performed data acquisition, data analyses, interpretation of results and wrote the manuscript. SR, PU, MB, GC critically revised the manuscript. PMK described microbiological analyses und critically revised the manuscript. STS conceived and supervised the study, analysed data, interpreted results and critically revised the manuscript. All authors read and approved the final manuscript.

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Data availability
The datasets used and analysed during this study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by the local ethics committee (Ethikkomission Nordwest- und Zentralschweiz (EKNZ), Project-ID 2019–01548).

Competing interests
All authors declare, that they do not have competing interests. S. Tschudin-Sutter serves as editor of the journal Antimicrobial Resistance and Infection Control.

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